TEN SURGICAL CONDITIONS

BY

NKINDA MARVIN MBELENGE, MD (CLUJ-NAPOCA)

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This is to certify that the surgical conditions presented in this book were managed by Dr. Nkinda M. Mbelenge during his postgraduate training in general surgery under my direct or delegated supervision.

.....
Dr. M.R. Aziz
SUPERVISOR

.....
Dr. R.E. Kisanga,
HEAD, DEPARTMENT OF SURGERY

ABSTRACT

This work is fulfilment of part of the requirements for the award of the degree of Master of Medicine in General Surgery.

It consists of ten surgical conditions divided in 3 sections: five conditions of general surgery, 2 conditions of paediatric surgery, and 3 conditions of orthopaedics and trauma.

The conditions presented are those commonly met or are of practical significance. All are entirely of the author's choice. In every condition one case is presented in detail, the rest of the patients are analysed in a tabular form followed by discussion and review of the literature. A conclusion is finally made.

Nkinda M. Mbelenge

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SOLITARY THYROID NODULE

SUMMARY

Thirteen patients with solitary thyroid nodules were seen at the Muhimbili Medical Centre between November 1989 and October 1991. Ten were females and 3 were males and the F/M ratio was 3.3:1. The age range was between 23 and 60 years with on average of 32:2 years. Six patients were from goitre endemic areas. The commonest presenting symptom was a painless anterior neck swelling. Eleven patients had a duration of between 7 to 48 months at the time of presentation to hospital. All the patients had thyroid nodules whose history and physical characteristics were consistent with those of benign lesions.

The diagnosis was confirmed by histology of the surgical specimens. Four patients with clinically solitary nodule proved to have multinodular goitre at exploration. The rest of them were adenomas. The indications for surgery were mainly cosmetic in young individuals and in 2 patients in whom fine needle aspiration cytology could not rule out malignancy. The only complication encountered was transient vocal cord paralysis and slight bleeding from the throat in the same patient due to trauma from the endotracheal tube during extubation. The average post operative hospital stay was 7 days.

INTRODUCTION

Wade[1] has defined the solitary thyroid nodule as being a goitre which, on clinical examination, appears to be a single nodule in an otherwise normal gland. Solitary thyroid nodules are mentioned to be a common problem in clinical practice in Western literature[2,3]. The vast majority of these nodules are benign. However concern that such a nodule may harbour malignancy demands prompt and accurate diagnosis. It is also clear that when properly treated, differentiated

thyroid carcinomas are associated with an excellent prognosis. The objective of investigating patients presenting with thyroid nodules is to define the small number of malignancies requiring surgery. The clinical evaluation of a patient with a thyroid nodule is a common problem confronting the clinician wherever he may be. But if selecting patients who require surgery is a problem to the clinician in the developed world, it is even more so to the practicing clinician in a developing country like Tanzania where the diagnostic facilities are far from ideal.

CASE PRESENTATION

S.L. was a 23 year old female who was referred to the Muhimbili Medical Centre from a regional hospital with a nodular swelling in the front of the neck of 6 years duration. The neck swelling was painless and had gradually increased in size as to cause concern to the patient. The neck swelling was associated with voice hoarseness for 3 years prior to admission. There was no history of fever and her menses had been normal. There was no history of weight loss, palpitations, anxiety, heat intolerance or diarrhoea. There was no history of breathing or swallowing difficuties. She was a single nullipara who lived with parents and had no previous history of admission to hospital. She was a nonsmoker and consumed no alcoholic drinks. She was the third born in a family of 3 girls. None of her close relatives had similar complaints of an anterior neck swelling.

Physical examination revealed a young female in apparent good health. She was not wasted. She was neither pale nor jaundiced. There was a mild hoarseness of the voice as she spoke. She was not dyspnoeic. The radial pulse was 88/min and the blood pressure was 130/70 mm Hg. She was not anxious and had no other features of thyrotoxicosis. The mucous membranes were normal and she had no peripheral lymphadenopathy or oedema. Local examination revealed a

solitary swelling in the anterior aspect of the neck. The swelling was visible from the front of the neck with the neck in the normal position but was not visible from the back. The swelling was more to the right rather than central. Its size was about 2cm. in the largest diameter. It was globular in shape, cystic and nontender. It moved up with swallowing but did not move up with tongue protrusion. The swelling was not pulsatile and there was no bruit over it. The overlying skin was normal. No neck vein engorgement was observed. The cervical and lymphnodes were not palpable. The trachea was central and the chest movements were symetrical with normal chest expansion. The respiratory and cardiovascular systems were normal. Abdominal examination was normal. The liver and the spleen were not palpable. The vaginal and rectal examinations were normal. The muscle power and tone were normal. The tendon reflexes were normal.

A provisional diagnosis of solitary thyroid nodule was made. The following investigations were requested

Full blood picture:

Haemoglobin 11.8 gm/dl

White cell count - total 5,950/ul

Differential - Neutrophils = 65%, Eosinophils = 3%
Basophils = 2%, lymphocytes = 25%
Monocytes = 5%

Erythrocyte sedimentation rate (Westergreen Method) - 10 mm/first hour, Red blood cell film - normochromic, normocytic, Platelets were reported as adequate, the Electrocardiograph = normal sinus rythm, Chest Xray and Xray of the thoracic inlet were reported as normal.

T_3, T_4 and TSH assays were not performed due to lack of reagents. An indirect laryngoscopy was done and both vocal cords were normal with good mobility. A fine needle aspiration biopsy was performed and was reported as showing features suggestive of colloid goitre. an ultrasound of the thyroid swelling could not be done. It was then explained to

the patient about the nature of her disease; that it was a small benign thyroid swelling that needed no surgical treatment at that time. When she still insisted to be operated on, an informed consent was obtained by explaining to her the nature of the operation and some of the possible complications like voice changes that may occur due to recurrent laryngeal nerve injury.

At operation, under general anaesthesia and transoral endotracheal intubation the patient was placed in a supine position with the neck extended by placing a small sandbag between the shoulders and lowering the head piece of the operating table. In this way the neck swelling was pushed forwards and the skin over it was almost taut. Overextension of the neck was avoided by ensuring that the head rested on a head ring. The neck was cleaned with antiseptics and then draped with sterile towels. A skin mark was made by pressing a length of thread onto the skin of the anterior aspect of the neck. A crease anterior neck incision was made 4 cm above the sternoclavicular joints. Skin flaps which included the skin, subacutaneous tissue and platysma were created. The upper flap was extended to the superior notch of the thyroid cartilage while the lower flap was extended to the suprasternal notch. The flaps were held apart by a self retaining retractor. The deep fascia along the anterior border of the sternomastoid muscles was incised and the muscle was fully mobilised to expose the lateral borders of the pretracheal muscles on the right. The anterior jugular veins were ligated and divided. A mildline incision through the deep fascia was made from the superior thyroid notch to the suprasternal notch. While retracting the sternomastoid muscles, the pretracheal muscles (sternohyoid and sternothyroid) were retracted laterally so that the single nodule of the right thyroid lobe was exposed. The nodule was found to be well encapsulated. The nodule was adequately mobilized, displaced medially and enucleated. The specimen

was sent for histology.

Haemostasis was achieved by ligating the vessels as well as by electrocoagulation. Closure of the wound was done when no bleeding was observed. A small rubber drain was placed within the deep cervical space and brought out through the skin laterally. The deep fascia in the midline was repaired with nylon number 2/0. The wound was then inspected to ensure that it was dry. The skin was closed using subcuticular nylon stitch number 4/0. Postoperatively the patient was put on intravenous drip dextrose-saline 2.5 litres/24 hours and pethidine intramuscular injection 100mg 6 hourly for the first 24 hours. The nursing staff were instructed to record routine observations like pulse blood pressure and to report immediately any bleeding, difficulty in breathing or the occurrence of any swelling in the neck. The rubber drain was removed 24 hours after. The subcuticular stitch was pulled out 4 days after the operation. A postoperative indirect laryngoscopy was done to assess the vocal cord movements and were found normal. The patient was discharged home on the fifth postoperative day. She was seen again at the follow-up clinic 6 weeks after. Her condition was good and had no complaints. A serum calcium estimation was found to be normal. The histological specimen showed features consistent with colloid goitre.

RESULTS

Table 1: Age and Sex Distribution

Age in years	Number of patients		Total
	Female	Male	
20 - 25	2	1	3
26 - 30	2	2	4
31 - 35	2	-	2
37 - 40	1	-	1
41 - 45	0	-	-
47 - 50	1	-	1
Total	10	3	13

The age range was between 23 and 60 years. The peak incidence was 20 - 35 years. The F:M ratio = 3.3:1

Table 2 Region of Residence

Region of residence	Number of patients
Dar es Salaam	7
Morogoro	2
Tanga	2
Kilimanjaro	2

Six patients came from endemic mountaineous areas of Morogoro, Tanga and Kilimanjaro. The study was conducted in Dar es Salaam.

Table 3: Presenting symptoms

Symptoms	No of patients
Painless ant neck swelling	13
Pain	1
Difficulty in breathing	1
Loss of weight	1
Irregular menses	1
Hoarseness of voice (undocumented by vocal cord examination)	1

The commonest presenting symptom was a painless anterior neck swelling.

Table 4: Duration of symptoms at time of presentation

Duration in months	No of patients	Percentage
0 - 12	4	30.8
13 - 24	3	23.1
25 - 36	2	15.4
37 - 48	2	15.4
49 - 60	-	-
61 - 72	2	15.4

More than 60% of the patents had a duration of between 6 and 48 months at the time of presentation to hospital.

Table 5: Physical features looked for in the thyroid nodules

Case No	Size (cm)	Consistency			Surface outline		Mobility		Tenderness	Palpable nodes
		Soft	Firm	Hard	Smooth	Irregular	Mobile	Fixed		
1	2	+	-	-	+	-	+	-	-	-
2	3	-	+	-	+	-	+	-	-	-
3	2.5	-	+	-	+	-	+	-	-	-
4	4	-	+	-	+	-	+	-	-	-
5	2.5	+	-	-	+	-	+	-	-	-
6	3	-	+	-	+	-	+	-	-	-
7	3	-	+	-	+	-	+	-	-	-
8	2	-	+	±*	+	-	+	-	-	-
9	3	+	-	-	+	-	+	-	-	-
10	2	-	+	-	+	-	+	-	-	-
11	7	-	+	-	+	-	+	-	-	-
12	6	-	+	-	+	-	-	-	-	-
13	7	-	+	-	+	-	+	-	-	-

±* Firm to hard
All the patients had nodules with physical characteristics consistent with benign lesion

Table 6: Results of Fine Needle Aspiration Cytology

Total No. of patients in study	No of patients who underwent FNA Cytology	No of patients in whom malignancy was R/O	No of patients in whom FNAC was indeterminate
13	8	6	2

From the 8 patients who underwent Fine Needle biopsy, malignancy was ruled out in 6 while in 2 patients the results were indeterminate.

Table 7: Fine Needle Aspiration Cytology results versus histology results (Actual reports (n = 8)

Case No	Results of Fine Needle Aspiration Cytology	Histology of surgical specimen
1.	Colloid goitre	struma nodosa et micro follicullaris
2.	Cystic degeneration in a Colloid goitre	Colloid adenoma
3.	Haemarrgic smear with benign clusters. To R/O Malignancy, Biopsy recommended	Microfollicular adenoma
4.	Normal thyroid follicles	Thyroid adenoma
5.	Cellular smear with large Cells. To R/O Malignancy. Biopsy recommended.	Multinodular goitre
11.	Mostly blood smear with few thyroid follicular cells, atypia, inflamatory cells.	Struma colloides nodosa, macro et microfollicularis
12.	Many RBC's Serosity smear. No other cells. Cytic change?	Diffuse hyperplasia of thyroid gland.
13.	Cluster of follicular cells. No atypia Haemorrhage. No malignancy	Adenomatous goitre

Table 8: Histological results of the surgical specimen (n = 15)

Histological results	No of patients
Colloid adenoma	2
Micro follicular adenoma	4
Difuse hyperplasia	1
Adenomatous goitre	2
Nodular goitre	4
Total	13

Table 9: Surgical procedure performed

Surgical procedures	No of patients
Enucleation of thyroid cyst	1
Unilateral lobectomy	9
Unilateral partical lobectomy	1
Unilateral lobectomy and isthmusectomy	2

Most patients underwent Unilateral lobectomy.

DISCUSSION

EPIDEMIOLOGY

It is estimated that approximately 4-7% of the population in the USA have thyroid nodules that are clinically palpable[4]. Other figures obtained from autopsy studies on unselected patients who had been hospitalized indicate that almost half the adult autopsy population had one or more thyroid nodules[5]. Most of these nodules were not palpable, prior to autopsy because they were in the interior or posterior part of the gland[5]. Studies have also shown that in fact up to 50% of apparently solitary thyroid nodules prove to be dominant nodules in multinodular goitres at surgical

exploration[3]. Multinodularity increases with age even in those patients who present with one palpable nodule[5]. The risk of malignancy in multinodular goitre is low[6]. Despite the high incidence of solitary thyroid nodules, the risk of malignancy of these nodules in life is minimal since only 2-5% of these have been found to be cancer histologically[5]. The rest being benign lesions[3,7] in the form of simple cysts, colloid nodules or benign adenomas.

Solitary thyroid nodules can occur at any age but occur most commonly between the ages of 30 and 50 years[8]. In the middle aged less than 10% (2-5%) of the solitary thyroid nodules are malignant[3,8]. In the young (below 14 years) and the old this figure exceeds 50%[8].

In the present series of 13 patients the age range was 23 to 60 years and the peak incidence was between 20 and 35 years (see table 1).

In general thyroid disease is more common in females than in males. This is also true in benign thyroid nodules and malignancy. Solitary thyroid nodules are less common in males but when they occur, particularly in young males, the chances of malignancy is greater[6]. The female: male ratio of thyroid nodules in the UK is estimated to be 4:1[8]. Multinodularily is also common in females[8]. In the present series the female : male ratio was 3.3:1. This may be too small a series to acquire firm conclusions. Figures from Tanzania are scarce.

AETIOLOGY AND PATHOLOGY

Six patients in this study came from endemic goitrous mountaneous areas of Tanzania (see table 2 and figure I).

Apart from this finding, no other aetiological or predisposing factor could be elicited from the history of any of the patients.

Although thyroid neoplasms are said to be a common clinical occurrence their aetiology is still indeterminate[9]. However, in view of their varied manifestations, it is possible and

indeed likely that no single aetiological factor is responsible. Several predisposing factors have been incriminated:

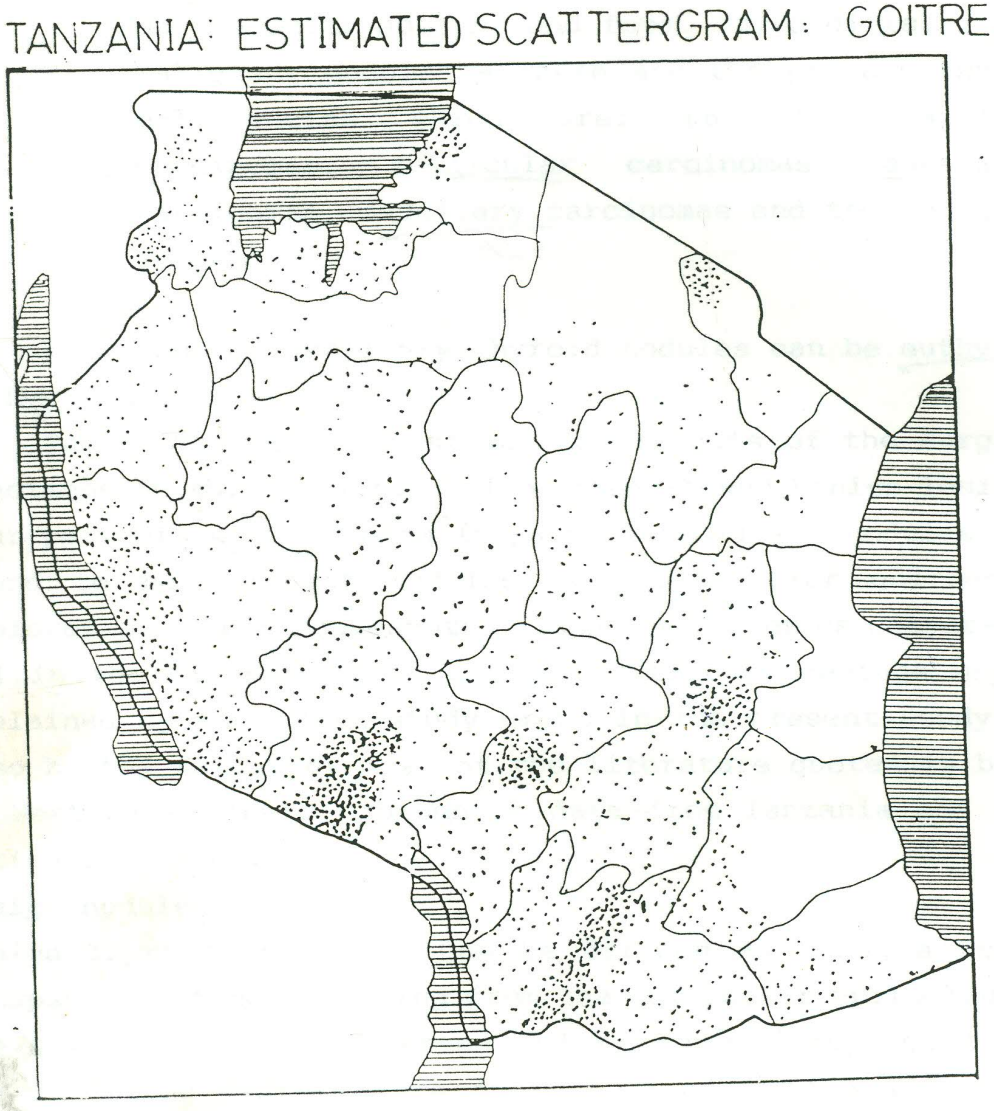
Geographical factors and goitre: It has been noted that in certain geographycal area where endemic goitre is common, benign thyroid nodules are also common[10]. Belfiore et al[11] analysing the thyroid cancer morbidity in two adjacent areas of Sicily, differing in iodine intake observed that follicular and anaplastic carcinomas were found to be 3 times more frequent in the iodine dificient area than in the control area.

Age and sex: Thyroid nodules are likely to be found in females rather than in males[6]. A thyroid nodule is more likely to be a cancer in a man than in a woman[10] and in a young patient than in an old one. Female preponderance was also noted in this small study group. In contrast to the above though the majority of our patients were relatively young individuals, none of them had malignant thyroid lesions. This finding comes in support of the fact that malignant solitary nodules are rare[2,3]. Multinodularity is also more common in females than in males. It was found in 4 patients (31%) in this series on surgical exploration.

Low or high dose irradiation: to the head and neck in infancy and childhood has been associated with increased incidence of thyroid malignancy[7,10]. Among all the incriminated aetiological factors this one is probably the least disputed[7]. Fjalling et al[12] found out that the median latency for carcinoma in his series was 40 years, suggesting that the increased risk of thyroid carcinoma after irradiation remains almost for the rest of the patients life. One would recommend to make a lifelong follow up of all the patients undergoing radiation for diagnostic or for therapeutic purposes.

Radioisotopes: have also been implicated in the causation of thyroid neoplasms[7].

Genetic predispositions: Thyroid cancer has been described in families as well as in identical twins[10]. The familial multiple endocrine neoplasm is a well documented clinical entity[6]. Others factors implicated include autoimmune thyroiditis, dyshormonogenesis and primary hyperthyroidism[13].



From Iodine deficiency disorders - A review by Kavishe FP, Maletrileme T.N. TMJ Vol.2 (1), 5-9, 1985

The following are the commonest causes of solitary thyroid nodules and according to their biological behaviour thyroid nodules can be divided into benign and malignant nodules[1,2,3,8,10]:

1. Benign nodules - These include benign, adenomas, cysts, colloid nodules, dominant nodules within a multinodular goitre, and focal thyroiditis.
2. Malignant nodules - These are the primary thyroid malignancies which are: (6) the papillary carcinomas, follicular carcinomas, anaplastic carcinomas, medullary carcinomas and the malignant lymphomas.

Functionally, solitary thyroid nodules can be euthyroid or hyperthyroid.

Table 8 shows the histological results of the surgical specimens in this series. All of them showed benign lesions. Four patients with clinically palpable solitary nodules were found to be dominant nodules in multinodular goitres at exploration. This represented 31% of the cases as compared to 50% in the literature[2,3,10]. The smaller percentage may be explained by the small study group in the present study and also by the fact that most of the literature quoted is based on Western studies. Authentic data from Tanzania and East Africa is scarce.

Benign nodules

Benign Thyroid Adenoma: Can be defined as being a benign encapsulated tumour arising from the follicular epithelium of the thyroid gland[10]. Warren and Meissner[11] suggested that the following features are indicative of a true adenoma: a solitary nodule, good encapsulation, uniform structure, a different microscopic pattern from the adjacent gland and compression of the surrounding thyroid by expansile growth of tumour. A benign thyroid adenoma is a typically solitary spheroidal or ovoid nodule encapsulated; and measuring up to

several centimetres. In the present series adenomas measured between 2 and 7cm. clinically. (see table 5). They may be merely a cosmetic embarrassment but may sometimes grow to considerable size to produce pressure effects. They may also undergo sudden enlargement from a haemorrhage, may show calcification, cystic changes and fibrosis. Only about 30 to 50% of seemingly solitary nodules prove to be adenomas of one type or another[4]. In this study 9 patients had adenomas of one type or the other representing 69% of the cases.

The histological classification of thyroid adenomas continues to be unsettled[4]. All adenomas contain follicles of varying sizes and in variable proportions and hence all might well be called follicular adenomas. However 3 major histological types are described[4,12] (a) Papillary, (b) follicular and (c) Hurthle cell adenoma (rare). The follicular adenomas can be subdivided according to the size of the follicles into: macrofollicular (colloid), microfollicular (or fetal) and embryonal varieties.

Papillary adenomas are today considered as papillary carcinomas because they have been found to invade locally or spread to regional nodes with time[4].

Thyroid adenomas can be associated with hyperthyroidism. When they do, such nodules function independent of pituitary control. The clinical entity produced by such an autonomously functioning nodule or toxic adenoma is called Plummer's disease[2]. In a study from Sheffield in the UK.[2] only 35 out of a series of 630 patients with hyperthyroidism had a solitary toxic adenoma representing only 5.6% of all operation for toxic thyroid disease. A solitary Toxic Adenoma is rarely malignant[12].

Simple Cyst: True cysts are quite rare[2]. Usually they are a result of cystic degeneration within a colloid nodule or a follicular adenoma. They can have variable sizes. Sometimes they may suddenly increase in size due to haemorrhage within it producing serious tracheal compression and even require

emergency surgery. Blood stained fluid within it is an indication of previous haemorrhage. Occasionally its wall can undergo calcification.. Needle aspiration and cytology is indicated for these lesions because of their rare occurrence and also because both benign and malignant lesions can have cystic components[2]. Sometimes the cyst wall can harbour malignant changes that can easily be missed by fine needle aspiration. This makes histological analysis of the cyst wall mandatory using ultrasound guideance(13). Solitary cysts of less than 4cm diametre are almost always benign. Those above 4cm are suspect. There was one thyroid cyts in the present study group. The histology turned out to be a colloid goitre which had undergone cystic degeneration.

Dominant Nodule Within a Multinodular Gland:

Up to 50% of clinically solitary nodules prove to be multinodular goitres at operative exploration[5] or on radionuclide scanning. This is especially true in obese or muscular patients since these features can make clinical examination difficult[3]. Multinodular goitres derive from simple goitre and may appear in both sporadic and endemic goitres, usually as late complications of these and usually in the elderly. The basis for their transformation into the nodular pattern is not yet known[8] but they are considered as the end result of patchy and disorganised thyroid metabolism where areas of hyperplasia progress to hyperplastic nodules[8]. Some of them undergo thyroid involution, haemorrhage, cystic degeneration necrosis fibrosis and calcification. These may also be nontoxic or toxic (toxic multinodular goitre) which differs from Grave's disease in that here there is no ophthalmopathy, no dermopathy and the hypermetabolism is usually less severe[8]. Rarely are these associated with hypothyroidism or myxoedema[8]. Malignant changes can occur in about 1-5% of untreated multinodular goitres[8,14]. Malignant change should be suspected if there is rapid enlargement of one of the nodules, adjacent lymph node enlargement or a

recurrent laryngeal nerve palsy.

Focal Thyroiditis: is a rare cause of thyroid nodule and is usually caused by bacterial infection but may be of any other type except viral[2]. The most frequent bacteria are staphillococcus aureus, streptococci, salmonella usually secondary to a primary focus elsewhere. It is associated with symptoms and signs of acute inflamation. Almost always the condition is transient and self-limited or is controllable with appropriate therapy.

Hashmoto's Thyroiditis is an autoimmune disease in which patients become sensitised to their own thyroid tissue. They form antithyroid antibodies. High serum titres of antimicrosomal and antithyroglobulin antibodies are found and are used for diagnostic purposes.

Carcinomas:

Carcinomas: The incidence of thyroid carcinoma in a solitary thyroid nodule varies from country to country[3]. However it is generally agreed that thyroid carcinomas occur in at least 2-5% of all solitary nodules in the middle aged(2,3,8). This figure exceeds 50% in the young below 14 years and in the old[6,14]. A thyroid nodule is more likely to be a cancer in a man than in a woman[14].

Primary thyroid malignancy can be classified as follows[15]. This classification has a prognostic value:-

1. **Papillary carcinoma**
   (a) Occult. The tumour is <1.5cm. in size and is not palpable
   (b) Intrathyroid. This is a larger tumour but is limited to the substance of the gland.
   (c) Extrathyroid - This tumour is locally invasive and has breached the capsule of the gland and has invaded local structures such as the larynx, trachea and oesophagus. Spread to the lymph nodes does not categorize a papillary tumour as extrathyroid. This depends on the degree of local

invasion. These tumours classically spread via lymphatic nodes but are unusual in that this type of spread does not always worsen the prognosis.

2. **Follicular Carcinoma:**
   (a) Minimally angioinvasive
   (b) Angioinvasive tumours which microscopically show tumour within the thyroid blood vessels. These tumours classically spread via the blood stream and are associated with secondaries in the bone and lung.
3. **Anaplastic Carcinoma:** This tumour occurs in the elderly and grows rapidly into all neighbouring structures including the trachea. It rarely presents as thyroid nodule.
4. **Medullary Carcinoma:** This tumour arises from the thyroid C cells and can form part of the multiple endocrine neoplasia group of disorders (MEN IIa and IIb).
5. **Malignant lymphoma:** These are rare. They have a variable prognosis depending on the degree of extrathyroid spread. They have the advantage of being the only thyroid tumour that can be radiosensitive.

CLINICAL DIAGNOSIS

When confronted by a patient with a clinically palpable solitary thyroid nodule two major issues need to be considered: Is the nodule symptomatic? Is it benign or malignant? Sometimes even sophisticated laboratory and Xray tests cannot differentiate with certainty between a benign and a malignant nodule of the thyroid[3]. Any patient who presents with a palpable thyroid nodule must therefore have a careful history and a complete physical examination.

The history: should be carefully taken and should always stress on the patients age, sex, place of domicile and family history. These factors may have a predictive value for thyroid malignancy. Children below the age 14 and the old are

more likely to have malignant nodules than the middle aged. Males are more likely to harbour malignant nodules than females (see pathology above). However there were three males in this study still none had a malignant nodule. Residence in an iodine deficient area may predispose one both for benign and for malignant thyroid nodules. (see aetiology above). Family history of thyroid tumour or goitre may predispose one to malignant thyroid nodules. This is especially relevant in the medullary carcinomas. The duration of the thyroid swelling or the rate of enlargement of the swelling are important. Recent enlargement of a thyroid swelling especially if it is accompanied by pain or discomfort may indicate either haemorrhage into the nodule or a malignant change. When haemorrhage occurs in a nodule the pain is usually of sudden onset and subsides within a few weeks. Sometimes this is accompanied by diminution in the size of the swelling. On the other hand, in malignancy, the pain persists and the swelling gradually increases in size. Vague discomfort in a thyroid swelling of recent origin particularly if it is accompanied by sensation that the swelling is becoming bigger or firmer is very suggestive of malignancy. Recent onset of the nodule or rapid and painless growth also increases the suspicion of malignancy. Local symptoms of pain and discomfort difficulty of breathing or swallowing (dysphagia) and voice changes (hoarseness) must be looked for. Systemic symptoms like weight loss, symptoms of hyperthyroidism must be looked for. History of exposure to radiation to the head and neck is important.

In the present series only 2 cases (15.4%) had a history of less than 6 months on presentation to hospital. In 9 cases (69.3%) symptoms had been present for between 6 months and 5 years and in 2 cases (15.4%) for over 5 years before they presented to hospital (see table). Four of the patients had a history of less than 12 months duration and yet none had a malignant nodule. Comparable figures of malignant solitary

thyroid nodules from the Royal Marsden Hospital series[10] show that 42.2% (108 cases) had a history of less than 6 months on presentation to hospital, in 26.6% (68 cases) symptoms had been present for between 6 months and 5 years and in 22.6% (58 cases) for over 5 years before presenting to hospital. This means that the duration of symptoms is just a helpful indicator but not a diagnostic factor. Similarly the sizes of thyroid nodule in this series measured between 2 and 7cm. Neither the 2cm nor the 7cm nodules were malignant. Six of the patients in this series came from the goitrous areas of Kilimanjaro, Morogoro and Tanga. None of the patients in the series had any history of known exposure to radiation in the past.

Careful physical examination is essential by the usual systematic procedure of inspection, palpation and auscultation.

The nodule should be inspected to determine its origin, size, shape, symmetry and movement. Restriction of movement on swallowing suggests abnormal fixation of the thyroid to the surrounding structures by neoplastic infiltration or inflamation[10].

The thyroid is then palpated to determine whether there is a solitary thyroid nodule or if it is a multinodular gland and whether or not there are palpable lymph nodes.

A solitary thyroid nodule is comparably more likely to be malignant than a multinodular goitre[4]. The nodule itself is examined for size, symmetry, consistency, outline, fixity to surrounding structures and tenderness. The rest of the gland is examined for enlargement and for other smaller nodules. The presence or absence of a pyramidal lobe is noted wherever possible. Rarely, one lobe, usually the left may be congenitally absent giving rise to compensatory hypertrophy of the opposite lobe which may be considered a pathological abnormality[10]. The position of the trachea is noted. It may be deviated to one side or the other. The cervical area and

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the neck are assessed for lymphadenopathy from the angle of the jaw to the clavicle, including the posterior triangle nodes. These may be involved by metastases from a malignant thyroid nodule even though their absence does not exclude this possibility.

Palpation of the thyroid is subject to observer error and thyroid ultrasound may detect a nodule when palpation is recorded as normal[13]. A thyroid mass is palpable when it is more than 1.5cm large[15]. For practical purposes a lump in the thyroid is significant if it can be palpated. The most important physical sign is the presence of a clinically palpable solitary nodule.

Table 5 shows the features assessed in the description of the thyroid nodules in this series. The sizes of the nodules ranged from 2 to 7cm. in the largest diameter. The consistency of the nodules was recorded as soft in 5 patients (38.5%) and as firm in 8 patients (61.5). None of them were hard in consistency.

The surface outline was smooth in all the patients. All the patients had mobile nodules, none were fixed. Fixation to the strap muscles, trachea, or larynx can sometimes be detected and is a sign of infiltration by a malignant lesion. the actual size of the nodule is quite unimportant as far as the malignant character of the nodule is concerned. Usually a small 2cm. nodule is often more likely to be malignant than a large swelling. The surface of a thyroid carcinoma may be smooth or nodular. Nodularity by itself is not a diagnostic feature, but it is suggestive[10]. In this series the high proportion of cases in which the surface was smooth and the absence of palpable regional nodes are important findings suggestive of the benign nature of these swellings in this series. Firm, hard, irregular or fixed lesions are more likely to be malignant though some palpillary lesions, are cystic and follicular tumours may be soft[6]. Extreme hardness in a thyroid swelling may be due to calcification and may be

confirmed by soft tissue radiology of the neck. Minor degrees of calcification can be seen in some tumours (particularly in papillary carcinomas). This can usually be easily destinguished from the grosser forms of calcification seen in the nodules or adenomas.

Vocal cord paralysis is due to unilateral laryngeal nerve involvement can sometimes be detected but is uncommon. In this series a 23 years old female with a 6 years history of a thyroid swelling presented with a 3 years history of hoarseness of the voice for which no basis could be established at laryngoscopy. Horner's syndrome may also occasionally be detected. None of the patients presented with signs of hyperthyroidism.

A careful general examination of the patient is made with particular reference to systemic effects of the thyroid nodule or distant metastases like in the abdomen, chest, bone etc. At the conclusion of the examination an indirect laryngoscopy is recommended in every case. It was not possible to carry out indirect laryngoscopy in every case in this study.

On the basis of the aforegoing discussion, clinically, a solitary thyroid nodule can present in the following manner[10]: (a) A solitary nodule in an otherwise normal gland (b). A solitary nodule with local spread either to surrounding structures or to regional lymph nodes. These are the obvious malignant nodules (c) A dominant apparent solitary nodule in a multinodular goitre sometimes discovered at operation.

INVESTIGATIONS

The objective of investigating patients presenting with thyroid nodules is to define the small number of malignancies requiring surgery.

Ultrasound of the thyroid gland: will determine whether the nodule is solid or cystic. It can also show the presence of other impalpable nodules, specifically it will provide evidence of multinodular disease. Most

cancers are solid while most cystic lesions are benign. Some papillary cancers are however also cystic[6]. Purely cystic lesions of less than 4cm. in diameter are almost never malignant[14]. However, studies by Loy and Sundvam[16] and also by Franklyn et al[17] have shown the poor specificity of ultrasound in the diagnosis of malignancy.

Fine Needle Aspiration Cytology: This is done by an 18-22 gauge needle to obtain a cellular aspirate for smear for cytological analysis. Advantages of the method are that it is simple, quick to perform (even in the outpatient department), is readily repeated, has excellent patient compliance and in experienced hands has a high diagnositic accuracy. The accuracy of fine needle aspiration cytology is about 80%. However false negative results have been obtained with the use of both core needle biopsies and fine needle aspiration cytology. False positive results are rare[7][9]. Papillary thyroid carcinoma is readly diagnosed. However the important limitations of this method are:-

- that it cannot distinguish between benign follicular adenoma and follicular carcinoma.
- you need an expert pathologist with skill to interpret the specimen[8]. This is a major limitations of this investigation in Tanzania where we do not have many skilled personnel.
- The absence of malignant cells in the aspirate does not exclude a malignancy because of the possibility of inaccurate targeting of the needle.
- The presence of malignant cells within the aspirate indicates the nature of the underlying disease but does not indicate the degree of malignancy,. for example it will not differentiate between carcinoma in situ and an invasive carcinoma. This type of information would be obtained if a core of tissue was removed by a Trucut needle biopsy[15]. In the present series 8 out of the 13 patients underwent fine needle aspiration cytology.

Among these, malignancy was ruled out in 6 patients while the diagnosis in 2 patients (25%) remained indeterminate by fine needle aspiration cytology (see table 6). The actual cytological reports are shown in table 7 versus the actual histological reports. As it can be seen, the diagnostic accuracy was above 80%. This diagnostic accuracy is comparable to the results from the Karolinska Hospital in Sweden where Lowhagen et al[9] recorded no false positives with respect to malignancy and a false negative rate of2.2% which later reduced to zero.

Isotope Scan: Is done with 123_I or ^{99}Tc. It will determine if the lesion is functioning (warm) hyperfunctioning (hot). or non-functioning (cold) and also whether the lesion is single or multiple(**18**). More than 50% of malignant nodules are cold[18]. Only 10-20% of cold nodules are malignant[18] and about 1-5% in multinodular - goitres[8,14]. A small proportion of cases of carcinoma of the thyroid have clinical evidence of hyperthyroidism (5.9% of cases in the Royal Mardsen Hospital series)[10]. However other authors believe that a solitary toxic nodule is usually a toxic adenoma and is never malignant[19].

Tumour markers. Calcitonin is produced by the parafollicular C cells. It can be used for the diagnosis and follow up of medullary carcinomas.

Studies of DNA Patterns or DNA Ploidy analysis: This is a new technique currently under evaluation. It can help in distinguishing follicular adenomas from minimally invasive follicular carcinomas[19].

Circulating thyroid hormone (T_4): This is important for patients who are thyrotoxic. This must be reversed before any surgery is contemplated.

Thyroid antibody analysis: Thyroid antibodies against thyroidglobin and thyroid follicular microsomes will indicate the likelihood of an autoimmune thyroiditis.

Histology of the surgical specimen. Will be able to determine

whether a nodule is malignant or not. An incisional biopsy is strictly contraindicated.

TREATMENT

The treatment of a solitary thyroid nodule varies according to whether one is dealing with a benign lesion or a malignant one and the histological type of carcinoma as well as to whether signs of thyrotoxicosis are associated. Thyroid nodules can be managed conservatively or surgically. The major indications for surgical removal of a thyroid nodule are[10]:

(1) When one suspects malignancy
(2) In the presence of pressure symptoms
(3) When there are signs of hyperthyroidism
(4) When the nodule produces cosmetic deformity and
(5) When there is substernal extension of the lesion.

Cyst - If the centre has got facilities for ultrasound, this will distinguish cystic from a solid lesion and will detect a multinodular gland. If an experienced cytologist is available, a fine needle aspiration cytology will determine with accuracy whether the lesion is benign or malignant. In places where such facilities are not available (no ultrasound, no cytologist), the history and physical findings may give an indication about the benign or malignant nature of the lesion. For example a long duration rate of increase in size, the smooth outline, mobility, no fixity to strap muscles or surrounding tissues. Solitary thyroid cysts of less than 4 cm. in size are almost always benign. Those above 4cm. are suspect and should be excised or referred to centres where fine needle aspiration cytology can be performed. A benign cyst is managed conservatively by observation and follow up. Otherwise it can be aspirated. If it recurs or if on aspiration haemorrhage is extensive surgical excision should be advised. Many of these benign lesions are removed for cosmetic and symptomatic reasons after consultation with the patient. In our series one patient had a 2cm. benign cyst

that was enucleated for cosmetic reasons. She was a young lady of 23 years.

Adenomas: A hyperthyroid adenoma will most probably be benign. Fine Needle aspiration however, will be able to distinguish the small proportion of malignant nodules. In individuals below 45 years antithyroid drugs are indicated followed by unilateral lobectomy. Patients with a toxic adenoma above 45 years may be put on radioactive iodine. Where the facilities of fine needle aspiration cytology are not available the history and physical findings consistent with a benign toxic adenoma will help one arrive at the diagnosis.

In the case of nontoxic adenomas (euthyroid nodule), the disadvantage of fine needle aspiration cytology here is that it cannot distinguish between benign follicular adenoma and follicular carcinoma. The majority of these benign nodules need no treatment. the patient can be reassured and followed up at intervals[2]. Many of them will remain static for years. Rarely, may they develop into toxic adenoma.

Eight of the patients in this series had non toxic adenomas. They were young patients below 30 years who underwent surgery. The indication for surgery was mostly cosmetic. Furthermore most of them had come to the centre as referral cases from upcountry hospitals where regular follow up would be difficult to maintain.

Dominant Nodule: If the patient is euthyroid and has a small asymptomatic goitre the treatment recommended is usually conservative. No special treatment is required. If the patient is euthyroid and has a large symptomatic or disfiguring goitre, surgery may be performed for therapeutic and cosmetic reasons[6]. A unilateral lobectomy is usually done on the side of the lesion including the isthmus. This is indicated for a simple multinodular disease involving one lobe only, the other lobe being normal[2]. A bilateral subtotal lobectomy is recommended for a bilateral nontoxic multinodular goitre[2]. In this case it is important to give thyroxine

after operation to suppress TSH stimulation of the apparently normal lobe[8].

If the patient is hyperthyroid, treatment is usually conservative either by antithyroid drugs or radioactive iodine[2]. The incidence of malignancy in a dominant nodule is considerably low such that surgical excision is usually not required unless one has got good clinical grounds to suspect malignancy[2,14].

Focal thyroiditis: We did not come across cases of focal thyroiditis. But when they occur these are usually associated with symptoms and signs of acute inflamation. The treatment is usually with appropriate antibiotics[2]. Rarely is a surgical drainage required. Hashimoto's thyroiditis is usually associated with a family history of autoimmune disease like pernicious anaemia, or autoimmune gastritis[14].

The recommended treatment is thyroxine replacement for life because these patients usually develop hypothyroidism later[2]. Operation is indicated for marked pressure symptoms, suspected malignancy and for cosmetics.

Malignant Nodule: The diagnostic limitations at the regional or consultant hospital level have been discussed under clinical diagnosis and under investigations (see above). This would mean that much of the decision and patient selection for surgery will be based mostly on clinical judgement. Clinical acumen is fundamental. The importance of a careful history and a complete physical examination cannot be over emphasized (see clinical diagnosis). A solitary nodule of short duration for example rapidly increasing in size in a child below 14 years or in the old is likely to be malignant. Fixity, to the strap muscles or to the surrounding tissue are features of malignancy. If facilities are available, and a skilled cytologist is available a fine needle aspiration is done. If a solitary thyroid nodule is found to be malignant on fine needle aspiration cytology or on strong clinical grounds the treatment of choice should be surgery[6]. If the lesion is

confined to one lobe and the contralateral lobe is uninvolved unilateral lobectomy can be performed. Many however, recommend total thyroidectomy as the treatment of choice for differentiated carcinomas[6]. It has been claimed that even when the contralateral lobe appears normal both on clinical examination and at operation lymphatic spread of the disease may have occurred. In addition the tumour may be multifocal in origin[6]. In situations where one does a lobectomy on an apparently benign thyroid nodule which later proves to be malignant at histology, repeat exploration is indicated. The absolute indication for total thyroidectomy are[10]:

(1) Presence of a palpable or visible nodule in the opposite lobe or in the isthmus
(2) Obvious multicentricity in the involved lobe
(3) Bilateral lymphnode involvement
(4) In follicular or medullary carcinoma where metastases may be present so that a diagnostic dose of radio iodine can be given after operation. Both papillary and follicular carcinomas are multicentric or multifocal in up to 13-23% (10) of cases.

Hormonotherapy with thyroxine (0.2-0.3mg/day) is given as replacement therapy to those who have undergone total thyroidectomy[6]. Thyroxine is also suppressant of TSH secretion thus reducing the incidence of recurrence of the primary tumour and also suppresses the growth of metastases[6].

Radiotherapy is given as adjuvant therapy following total thyroidectomy[6]. After operation the patient is deprived of thyroxine so as to increase the thyroid's avidity to iodine. Iodine (^{131}1) scan is performed 6 weeks postoperative. If treated the scan is repeated after 1 year. A further dose of radioiodine can be given for treatment. There after it can be repeated as necessary.

CONCLUSION

Clinically solitary thyroid nodules are mentioned to be

common in Western literature. Our experience with 13 patients in a 2 years period may not indicate a common clinical ocurrence. The main management difficulty in thyroid nodules is how to select the small percentage of patients requiring surgery. This is a major problem especially to the clinician working in developing countries like Tanzania where diagnostic facilities are far from ideal and the trained, skilled personnel scarce. A minimum of diagnostic facilities like ultrasound and fine needle aspiration cytology is essential in order to make a somewhat reliable diagnosis of a thyroid nodule. In the absence of these facilities the diagnosis and management of these lesions becomes very difficult, because the history and clinical findings can only be helpful but are not diagnostic. Patients with suspicious history and physical findings should be referred to a centre where these facilities are available. Otherwise one should use their judicious judgment as to whether or not to subject these patients to surgery. Follicular adenomas and carcinomas present particular challenging problems of differential diagnosis even in well equiped centres.

REFERENCES

1. Wade H. Solitary Thyroid Nodule Annals of the Royal College of Surgery of Eng. 55: 13-20, 1974.

2. Khan O, Fernando R. Solitary Thyroid Nodule Postgraduate Doctor (Africa) 10(10): 275- 1982.

3. Ellis H. The thyroid Nodule Postgr. doctor (Africa) 4(8): 248-252, 1982.

4. Robbins S.L. Cotran R.S. Kumar V. Pathologic Basis of Disease 3rd Ed. W.B. Saunders Co. pp.1203-1224,1984.

5. Wright H.K., Burrow G.N., Spaulding S., Sheahan G. Current Therapy of Thyroid Nodules. The Surgical Cl. N.A. endocrine Surg. 54(2): 277-288, 1974.

6. Wheeler M.H. Malignant Goitre Surgery 1(9): 200-6, 1984.

7. Schwartz S.I. Principles of surgery - 5th Ed. Mc Graw - Hill book Co. 1989.

8. Young A.E. Goiter, surgery 1(10): 233-37, 1984.

9. Bailey H, Love M. Bailey and Love's short practice of surgery, Eng, Lang. Book Society 20th Ed. 1988.

10. Smithers D. Tumours of the Thyroid gland. Vol. VI Pub. E & S Livingstone pp 55-70, 1970.

11. Warren S. and Meissner W.A. Tumours of the Thyroid gland, Atlas of Tumour Pathology. Section IV Fascicle 14 Washington D.C., Armed Forces Institute of Pathology, 1953.

12. Ingbar S.H., Woeber, K.A. Diseases of the Thyroid in Harrison's Principles of Internal Medicine - 7th edition. Mc Graw - Hill Book Co. pp. 482-484 1978.

13. Lazarus J.H. Lumps in the Thyroid Hospital Update 2239-46, 1988 Dec.

14. Clark O.H. Evaluation of thyroid Nodules in Current surgical diagnosis and Treatment - 5th edit. by Dunphy Publ. Lange Med. Publ. Los atlas California pp 237-238, 1981.

15. Thomas P. A solitary Thyroid Nodule British Journal of Hosp Med. 46: 179-181, 1991.

16. Loy T.J., Sundvam F.X. Diagnostic of Solitary Thyroid Nodules. Aann. Acad. Med. Singapore 18(6): 658-64, 1989.

17. Franklyn J.A., Sheppard M.C. Thyroid Nodules and Thyroid Cancer Diagnostic Aspects. Baillieres Cl. Endocr. and Metab. 2(3): 761-75, 1988.

18. Wheeler M.H. Investigation of the Thyroid. Surgery 1(7): 148- 49, 1984.

19. Grant C.S., Hay I.D., Ryan J.J. et al Diagnostic and Progressive utility of flow cytometric DNA measurements in follicular thyroid tumors. World J. Surg. 14(3): 283-9, 1990.

TRAUMATIC RUPTURE OF THE SPLEEN

SUMMARY

The following is a study of 24 patients who sustained splenic rupture following blunt abdominal trauma from various causes. All of them underwent splenectomy at the Muhimbili Medical Centre, over a one year period between October, 1989 and October 1990. Twenty were males while four were females. Their ages ranged from 5 to 61 years. The peak incidnece was between 10 and 40 years.

Nineteen were involved in motor traffic accidents, 3 were assaulted and 2 fell down from a height. The commonest clinical features were abdominal pain, abdominal tenderness, pallor, low blood pressure and high pulse rate. Associated injuries were present in 16 patients. The commonest ones were brain concussion and soft tissue lacerations. The commonest splenic injuries were lacerations and linear ruptures. Two of the removed spleens were found to be obviously enlarged. Twenty two were of normal size. All the 24 patients underwent emergency total splenectomy. The average hospital stay was 9 days.

INTRODUCTION

The historical development of the surgery of the spleen makes good reading. For a period of over 2000 years the spleen was viewed as being an organ of interest and perplexity by many authors both scientific and nonscietific[1]. Galen (130-200) for example regarded the spleen as an organ full of mystery (organum plenum mysterium). He ascribed to the spleen such interesting and imaginative functions as mirth, anger and even speed in running. In ancient Greece, physicians and philosophers such as Plato and Aretaeus suggested a more realistic role of the spleen. They thought the spleen was a filtering mechanism. They believed that the spleen "strained black blood or black bile" and helped keep the liver "bright

and pure"[1]. The first attempts to describe the anatomy of the spleen were done by Marcello Malpighi who in 1659, described some of the microscopic anatomy of the spleen including the splenic nodules which bear his name, the Malpighian corpuscles. Centuries earlier Aristotle had suggsted that the spleen was not a vital organ, that it was not nesessary for a healthy life. Experimental studies done by Barbette, Mathis, Morgani and Clark on the effects of splenectomy in the dog confirmed what had earlier been said by Aristotle.

Post-traumatic splenectomies may have been performed by military surgeons earlier but Quittenbaum is said to have been the first in the modern era to have done splenectomy in 1886 but for non-traumatic disorders. In 1921 Mynihan wrote extensively on splenectomy. Since then other techniques have been added to the techniques of management of splenic injury like splenorrhaphy and partial splenectomy.

CASE PRESENTATION

N.M. was a 26 year old male who worked at the local butchery. He was admitted to hospital four hours after he, as a passe-by, was knocked on the left side of the trunk by one of the colliding trucks. Shortly after the incidence he felt generalised abdominal pain more severe in the left upper quadrant, and was associated with abdominal distension. He was feeling "bad and dizzy". He denied any history of loss of consiciousiness, haematuria, or of bleeding from the nose or ear.

On examination at admission he was found to be a young man, in good nutritonal status, fully conscious and orientated in time and space. He looked ill, and pale. He had a clammy skin with cold extremitites. The blood pressure was 70/50mmHg with a weak, thready pulse of 120/min. There were no external signs of injury, lacerations or bruises. The pupils were central and equally reacting to light. The neck was not

tender. The trachea was central and the chest moved normally with respiration. The respiratory rate was 40/min with good air entry and the breath sounds were normal on both sides of the chest. The heart sounds were normal. There was no evidence of injury to the upper and lower limbs.

Examination of the abdomen revealed a grossly distended abdomen making it difficult to palpate the spleen and the liver. There was generalised abdominal tenderness more marked in the left upper guadrant where rebound tenderness could be elicited. Shifting dullness was present and the bowel sounds were absent. Digital rectal examination was normal. The abdominal tap was positive revealing non-clotting blood.

The patient was clinically diagnosed as having haemoperitoneum due to a ruptured viscus, most probably the spleen with impending shock.

Blood was taken for urgent grouping and cross matching and 3 units of blood were requested. An intravenous line was set with a wide bore cannula no.16 on both arms and the patient was started on crystalloids. Hartmanns solution was given rapidly while the patient was being prepared for a laparatomy. Two litres of Hartmann's solution were infused in the first hour and the BP of the patient rose from 70/50 to 110/70mmHg with a pulse of 90/min. within the next two hours.

A self retaining Folley's catheter was inserted to monitor urine output and a naso-gastric tube was inserted for decompressing the stomach.

The patient was taken to the theatre 2 hours after arrival to hospital. With the patient in the supine position and under general anaesthesia and transoral endotracheal intubation routine cleansing with antiseptics was done and the patient was draped with sterile towels. The abdomen was opened through an upper midline incision extended caudally. Massive haemoperitoneum was found. About 3 litres of blood were sucked from the abdominal cavity. The spleen was found to be enlarged. The spleen was found to be completely

transversely bisected. The left hand was passed over the diaphragmatic surface of the spleen which was rapidly lifted out from its bed. The pedicle was identified and cross-clamped close to the hilum with long artery forceps and then was devided between clamps in small sections at a time from below upwards. The splenic artery and vein were identified clamped and transfixed separatetly with silk no 2/0. The other peritoneal attachments were cut. The lieno renal and splenocolic ligaments were divided under vision. Blood vessels in the gastrosplenic and gastrocolic parts of the lesser ommentum and tributaries of the short gastric vessels were identified and ligated individually. The spleen was removed and delivered out through the wound.

After achieving haemostasis the remaining blood was sucked out and clots removed. A systematic examination was made for other visceral injuries, and for iatrogenic injury to the neighbouring organs. Having been satisfied that none of these were present peritoneal lavage with warm normal saline was done. The fluid was sucked out and the abdomen was closed in layers. Intra operatively the patient received one unit of iso-group, isoRh whole blood.

Post-operatively he received 2 more units of blood and 2500mls of Dextrose/saline solution in 24 hours. Intraumuscular injections of pethidine 100mg were given 6 hourly for 24 hours. Vital signs were monitored 1/4 hourly for 4 hours then hourly for 24 hours. These included blood pressure, pulse, and respiration. A strict input and output chart was kept.

The patient made an uneventful recovery. By the first postoperative day the vital signs were stable, the urine output was satisfactory and the general condition of the patient had obvisously improved. The nosogastiric tube and the urethral catheter were removed after the first 24 hours of surgery.

After 48 hours the bowel movements had re-established and the patient was allowed to take oral fluids, and was

mobilised. On the 7th postoperative day the wound was found to have healed and the stitches were removed. The patient was allowed home with an Hb of 9gm/dl on fersolate tablets. The patient was followed up for six months. His condition continued to remain good.

RESULTS

The following is a summary of findings in the 24 patients in the series.

Table 1: Sex Distribution

SEX	Number of Patients
Male	20
Female	4
Total	24

The M/F ratio was 5:1

Table 2: Age distribution

Age in Year	Number of Patients
1 - 10	1
11 - 20	9
21 - 30	5
31 - 40	7
41 - 50	0
51 - 60	1
61 - 70	1
Total	24

The age range was 5 - 61 years. The peak incidence was 10 -40 years.

Table 3: Causes of Injury

Cause of Splenic Injury	Number of Patients
1. Paedestrian knocked by care	7
2. Passenger "Daladala" overturned	7
3. Cylist knocked by car	3
4. Driver - care overturned	2
5. Builder fell off from roof at construction site	2
6. Assaulted by dandits or mob	3
Total	24

Motor traffic accidents were the major cause of injury.

Table 4: Time of Presentation to MMC after injury

Hours after injury	Number of Patients
0 - 6	17
7 - 12	1
13 - 18	1
19 - 24	2
25 - 30	0
31 - 36	1
37 - 42	0
43 - 48	2
Total	24

The majority of the patients (17/24) presented to hospital within the first 6 hours of injury. Two patients had delayed splenic rupture and presented to hopsital 48 hours after injury.

Table 5: Clinical features

Symptoms	Number of Patients
Abdominal pain	22
Abdominal fullness	4
Dizziness	6
Body weakness	5
Vomiting	3
Constipation	2
SIGNS	Number of patients
Abdominal tenderness	24
Palor	22
Shifting dullness	16
Abdominal distention	10
Rebound tenderness	4
Muscle guarding	7
Pulse rate > 100/min.	8
Fall in BP < 80mmHg. Sytolic	8
Restlessness	4
Absence of bowel sounds	7
Bruising on the abdomen	4
Positive abdominal tap	10

The commonest symptoms were abdominal pain, dizziness and abdominal fullness; while the commonest signs were abdominal tenderness, palor and shifting dullness. An abdominal tap was positive in 10 patients.

Table 6: Associated Injuries

Type of Associated Injury	No of Patients
Brain concussion	4
Laceration of liver	2
Small bowel injury (bruising, echymosis)	3
Lacerated scalp wound	2
Lacerated of humerus	3
Fracture of rib	1
Fracturer of pelvis	1
Retroperitoneal haematoma	2
Chemical burns	1
Skin laceration and bruising	3

Of the 24 patients 16 had other associated injuries apart from having splenic rupture. The commonest associated injuries were brain concusion, lacerated wounds, fracture of the humerus and bowel injury.

Table 7: Size of the spleen and appearance

Size of Spleen	No fo Spleen
Enlarged	2
Normal size	22
Total	24

At operation 2 of the removed spleens were obviously enlarged even though they could not be weighed. By estimation they were between 1kg - 2kg.

The rest of the spleen looked normal in size appearance and consistency.

Table 8: Type of Splenic injury

Type of Splenic injury	Number of Patients
Laceration	14
Linear rupture	4
Stellate rupture	2
Tear in fragments	1
Avulsion of pedicle	2
Complete transverse bissection	1

Lacerations were the commonest types of injury

Table 9: Complications

Complication	Number of Patients
1. Wound sepsis	1
2. Left pleural effusion	1
3. Incisional hernia	1

DISCUSSION

CAUSES OF SPLENIC RUPTURE

Table 3 summaries the causes of splenic rupture in this series. The majority (19 patients - 79%) were due to motor traffic accidents in various circumstances. Three were due to assault by a mob or by bandits. Two builders fell off from the roof during construction work.

Surprisingly, the spleen, despite being a well protected organ is one of the most frequenty injured intrabdominal organ.

Penetrating trauma to the abdomen or chest can be due to gunshot wounds. These usually present with significant

visceral injury in more than 90% of cases[2]. Stab wounds through the anterior abdominal wall may also reach the spleen. Splenic ruptures due to penetrating injuries may be single, but many times they are associated with other visceral injuries like those of the pancreas, the left kidney, stomach and the vascular structure caused by penetrating trauma.

Blunt abdominal (or lower thoracic) trauma is one of the major causes of injury to the spleen[3]. The mechanism of injury is by compression between the anterior abdominal wall and the posterior rib cage on the left or by being compressed on the vertebral column. Otherwise the spleen may be dislodged from its peritoneal attachments by sudden big force by the coup - contracoup mechanism[4]. The leading causes of splenic rupture due to blunt trauma are automobile accidents, fall from a height, direct blows incurred during contact sports, assault, and bicycle injuies[3,5]. The severity of trauma may vary. Sometimes the trauma may be so trivial that the patient gives little attention to it[5]. This reminds us of the importance of taking a detailed history because such cases could be interpreted as being spontaneous splenic rupture. Diseased and enlarged spleens for example in malaria, chronic leukemia, infectious mononucleosis and polycythemia vera, have been found to rupture more easily than normal spleens[3,4]. However,from Abdulmunin's[6] study of 128 patients with ruptured spleens in rural Egypt where a large number of the rural population have hepatosplenomegally due to chronic schistosomiasis and malaria the incidence of splenic rupture was not particularly high as one would expect. In this series 2 of the removed spleens were found to be grossly enlarged (8.3%).

Splenic rupture following blunt trauma may be an isolated event in 30% of cases or it may be associated with other organ injuries[3]. Other organs that may be injured in order of incidence include chest, rib fracture, kidney, spinal cord fracture, liver, lung, cranilcerebral structures, small

intestines, pancreas and stomach[3].

Table 6 shows the commonest associated injuries in this series. The commonest ones were head injuries and soft tissue injuries. Iatrogenic trauma to the spleen most commonly occurs during upper abdominal operations on adjacent viscera like the stomach, oesophageal hiatus, vagus nerve and the splenic flexure of the colon. According to Schwartz[3]. The spleen is injured in approximately 2% of the patients undergoing operations of the viscera of the upper quadrants while in another report operative trauma to the spleen accounts for about 20% of splenectomies in the USA[5].

The mechanisms of injury are usually avulsion of the splenic capsule by traction on peritoneal attachments and direct injury by a retractor placed against the organ in order to obtain exposure. Such injuries usually permit repair but may require splenectomy at times[3]. Certain procedures, like percutaneous splenic puncture in order to perform splenic pulp manometry and splenoportography may be associated with persistent bleeding[3]. This however is uncommon in patients whose platelet counts are above 70,000/mm^3 and whose prothrombin time is greater than 20% or more[3]. Spontaneous splenic rupture is rare in normal spleens. It is more common in diseased or enlarged spleens with various haematogical disorders[3] Traumatic rupture due to trivial trauma has been mistaken for spontaneous rupture[3].

PATHOLOGY

We speak of rupture of the spleen to describe the disruption of the splenic parenchyma, capsule or blood supply[3]. During blunt injury the spleen may be ruptured through the parenchyma and capsule, or it may be avulsed from its pedicle or disrupted beneath an intact capsule to produce a subcapsular (or contained) haematoma. The spleen being a highly vascular and friable organ bleeds profusely even when injured minimally especially if it is enlarged or diseased.

Table 8 shows the types of splenic injuries found in this study. Most of them (14 patients 60%) were lacerations of the spleen.

Spleen injuries vary from linear and stellate lacerations, capsular tears, puncture wounds caused by penetrating objects or fractured, ribs, avulsion of the organ from its vascualr pedicle, laceration of the short gastric vessels within the gastro splenic omentum, simple transverse tears of the perenchyma, transverse cracks of the hilum and longitudinal injuries. Most injuries result in transverse ruptures of the parenchyma. The direction of rupture is determined by the internal architecture of the organ which is arranged in a transverse plane[3].

Splenic ruptures may be acute, delayed or occult. Acute splenic rupture is manifested by immediate signs and symptoms of intraperitoneal haemorrhage[3]. It accounts for about 90% of cases from blunt trauma to the spleen[2]. There were 22 patients (91.7%) in this series with acute splenic rupture. Delayed rupture manifests itself days or weeks after injury. It accounts for about 5-15% of the cases of blunt trauma to the spleen[3,4].

There were 2 patients with delayed splenic rupture in this series (8.3%). The terms occult splenic rupture is applied to traumatic pseudocysts of the spleen when injury to the organ has not been diagnosed previously. It accounts for about 1% of cases of blunt trauma to the spleen[3]. None of the patients in this series had occult splenic rupture.

CLINICAL FEATURES

Table 5 summarises the clinical features of the patients in this series. Abdominal pain and abdominal tenderness were present in almost all the patients. Signs of intraperitoneal haemorrhage were present in a large percentage of the patients.

The clinical features of splenic rupture may vary

depending on a number of factors[3]:-

1. The severity of intrabdominal injury and haemorrhage.
2. The rapidity of intrabdominal haemorrhage
3. The presence or absence of other organ injuries
4. The interval between the injury and examination.

There is usually a history of trauma or a blow to the upper abdomen especially to the left flank or to the posterior rib cage on the left. The trauma may be of variable severity. Sometimes it may seen trivial as to be overlooked by the patient especially if he/she is a child. Penetrating trauma is of course obvious.

On admission some patients may present with signs and symptoms of severe hypovolaemic shock. Others may have minimal or no symptoms at all. Most patients will fall between these two extremes. Tachycardia is almost always present. There is usually a slight or marked reduction in blood pressure with orthostatic hypotension. Most patients complain of abdominal pain which is generalised but more severe in the left upper quadrant. In about 1/3 of the patients pain is confined to the left upper quadrant[3,5]. Signs of external violence to the abdomen like bruises, and echymosis may be present although a significant number of patients with splenic injuries have no external manifestastion of injury[6,7]. Kehr's sign (referred pain in the left shoulder or cervical region) is often postive in 50% of the patients[3]. It is an indication of diaphragmatic irritation. It can be ellicited by placing the patient in the Trendelenberg position or by palpation in the left upper quadrant. Mild nausea and vomiting may be encountered in some patients. Signs of peritoneal irritation (tenderness, guarding abdominal distension) may be present.

Even though abdominal pain and tenderness are usually present, peritoneal reaction to bleeding varies greatly. Some patients will have minimal findings, even when intraperitoneal bleeding is massive[5]. Shifting dullness is a sign of

excessive intraperitoneal bleeding. It is said not to be very common. The area of splenic dullness on percussion may be increased. Sometimes a mass may be palpable in the left upper quadrant. Tenderness over the 9th, 10th ribs on the left is an important early diagnostic sign and should arouse suspicion of the posibility of a ruptured spleen. It occurs in about 20% of the cases[5]. Tenderness of the left upper quadrant is a frequent finding. Balance's signs may be positive in about 25% of cases (elliciting a mass or percussible area of fixed dullness in the left upper quadrant) as evidence of subcapsular haematoma or omentum surrounding an extracapsular haematoma[7].

Rectal examination frequently reveals tenderness and sometimes a soft swelling due to blood or clot in the rectoversical pouch[7]. An unusual presentation of splenic rupture has been described by Tyler and Stahl[8] with haemoperitoneum which was forced through two abdominal wall hernias to produce potentially misleading ecchymosis of the upper midline and thigh. Abdominal paracentesis or the 4 quadrant tap gives a positive tap in about 50% of the cases[1]. Only 10 cases (41.7%) in this series yielded a positive abdominal paracentesis.

Diagnostic difficulties can arise. Physical examination has an accuracy of about 65%[2]. The presence of tenderness in the left upper quadrant and positive Kehr's sign are not always indicative of splenic injury particularly in the presence of lower left sided rib fracture[2]. Abdominal findings may be marked by several other associated factors like severe pain from associated injuries, sensory loss from spinal cord trauma, altered states of consciouness in head injuries, shock or intoxication. In a multiple injured patient hypotesion without other suggestive signs may be due to haemorrhage from noncavitary sources like external lacerations, pelvic or long bone fracture. Hypotension may be due to spinal cord trauma or cardiogenic shock. Abdominal

distension after blunt abdominal trauma may be due to gastric or colonic dilatation rather than intrabdominal bleeding. Warning has been given by Ward[9] on the use of epidural analgesia in controlling the pain from chest injuries because it may mask and delay the diagnosis of a splenic rupture.

INVESTIGATIONS

In a significant number of cases the diagnosis of splenic rupture is not straight forward. In such cases repeated examination and other laboratory tests are necessary.

The haematocrit may be reduced but the initial determination may be normal. It is not however an accurate reflection of blood volume deficit in the acutely injured, unresuscitated patient, as it can take 18-24 hours for transcapillary refill to occur[2,3]. It however serves as a baseline investigation. Serial determination of haematocrit are necessary and a fall in hametocrit suggests intrabdominal haemorrhage.

A raised white cell count of the order of 15000/cm^3 or more is also suggestive of splenic rupture[3] or liver injury. Radiographic evaluation is occasionally helpful. The spleen may be visible as a soft shadow in plain films of the upper abdomen. If these features are repeated at intervals then this is suggestive that the spleen is not ruptured. An enlarged splenic shadow is suggestive of rupture. Other features suggestive of splenic rupture include left rib fractures. About 20% of cases with fracture of the 9^{th} and 10^{th} ribs present with ruptured spleen[5]. Other features include elevation and immobility of the left haemidiaphragm, pleural effusion, and medial displacement of the gastric shadow with serration of the greater carvature due to dissection of blood into the gastro splenic omentum[2].

Blood gas analysis has been a useful indicator of ongoing intrabdominal haemorrhage[2].

An arterial base deficit of 3mEq/l suggests significant

haemorrhage in healthy trauma patients. Increasingly negative base deficits are more sensitive indicatiors of persistent bleeding than serial haematocrits[2].

Diagnostic peritoneal lavage(DPL) has been used in those situations in which there is an immediate need to know if haemoperitoneum, exists. Its accuracy in blunt abdominal trauma ranges from 92-98%[2]. There is a greater rate of false positives than false negatives. The procedure can be done under local anaesthesia.

Scanning of the spleen has been used for haemodynamically stable trauma patients. Ultrasound, scintigraphy and CT scans have been used for diagnosing splenic injury in stable patients. Arteriography has also been used to demonstrate splenic injury as well as to document persistent arterial bleeding. It may show disruption of the spleen, radiopacity in the peritoneal cavity or early filling of the splenic vein. In the present series diagnosis was reached by history taking and by physical examination. Ocassionally serial abdominal examinations were necessary. Abdominal tap was done in 10 patients. The sophisticated techniques discussed above like CT scan are not available at Muhimbili. The utrasound that is available is not regularly serviceable.

TREATMENT

All the patients in this series were managed by total splenectomy after resuscitative measures were done wherever called for. At this centre most of the decision to explore or not in blunt abdominal trauma is made on the history and on the physical findings.

Traditionally splenic injury has been managed by total splenectomy with the aim of eliminating the danger of haemorrhage. Total spelenectomy as a life saving procedure is still the standard, procedure in many cases in severely injured spleens[3]. In the past splenectomy was widely performed because splenic removal seemed harmless. Splenic

repair was considered dangerous because of the risk of subsequent haemorrhage[10].

If the patient's circulatory status deteriorates in spite of vigorous resuscitative measures then one has to operate immediately and bleeding stopped to save life. It is the opinion of this author that in our setting repeated observations and examination is one of the best guides to appreciating a patients condition and enables one to decide when to operate. Some surgeons would reimplant a slice of splenic tissue beneath the peritoneum or omentum[2,3]. They claim that they have demonstrated its viability. Some however, argue that the amount of tissue mass and the blood supply do not provide sufficient phagocytic clearance of the encapsulated bacteria implicated. Ludtke et al[11] advise that reimplantation should be performed only if spleen preservation procedures are not feasible.

Knowledge of the increased susceptibility to post-splenectomy infection and a better understanding of the surgical anatomy of the spleen have lead surgeons to adopt splenic salvage techniques. The decision to perform splenectomy or a splenic salvage prodecure is based upon a number of factors[2]:-

1. The condition ofthe patient
2. The condition of the spleen
3. The age of the patient

Splenorrhaphy has been found to be a safe procedure for the management of splenic traumal[2]. Splenorrhaphy is indicated in patients who are haemodinamically stable and those who have no other potential life threatening problems like head injury, thoracic trauma, or blood disorders[2]. It is also indicated in capsular tears, polar injury and in larger injuries that do not involve the hilar vessels[5]. It is far more likely to succeed when attempted soon after injury than after a period of failed observation[2]. The devitalized splenic tissue is debrided and the organ is repaired by suture technique with or

without omental patch or by partial resection when necessary. Splenorrhaphy has been found to be applicable in about 50% of the cases of splenic trauma[2] with only 1% incidence of rebleeding necessitating reoperation. Splenectomy has the same incidence of rebleeding.

Mabogunje[14] in Nigeria has succeeded preserving ruptured spleens in four men by using splenorrhaphy and partial splenectomy. Two of them had splenomegaly.

Partial splenectomy is another splenic salvage techniques. Recent studies suggest that it offers protection against preumococcal sepsis[2]. While this is so it remains a relatively rare operation because the spleen is a friable and highly vascular organ that can be difficult to mobilize once haemorrhage has began[2].

Splenic salvage by splenic Capping Method[12]. This is another method of splenic conservation. It consists in mapping around the entire surface of the injured organ with polyglycolic acid elastic mesh and allowing to heal. This technique is still experimental.

Nonoperative Management has also been applied. The low but definite risk of over whelming post-splenectomy infection (OPSI) has led surgeons to consider a more conservative management of splenic trauma. However this is only possible in centres equiped with facilities to monitor patients. The non operative management consists in admission to the ICU, bed rest, nasogastric tube, close monitoring, frequent physical examination, serial haematocrits and follow-up splenic imaging[2].

Non-operative management of blunt splenic trauma has been accepted in selected paediatric patients but has been controversial in adult patients[2]. However it is reported to be successful in over 90% of case in which it has been attempted(2). Possible candidates for non operative management must fulfil several criteria[2]. They must:-

- have suffered blunt trauma

- have an isolated splenic injury
- be sufficiently alert to comply with repeated physical examination.
- be haemodynamically stable.

The diagnosis of splenic injury and grading of injury is usually established by CT Scan and an assessment of the severity of injury is made.

It is believed that non operative management is successful in children because of the haemostatic properties of the splenic capsule which is thicker in children than in adults[2]. It also contains some myoepithelial cells which may have constrictive properties[13].

Non operative management has recently been advocated for adults. Cogbill[2] has reported a success rate of 83% in adults. He suggests that success is more likely in those patients who have grade 1 or 2 injury. Shackford et al[2] found out that approximately 12-15% of adults were elligible for non operative management with a success rate of approximately 70%[2]. Patients with coagulation defects are excluded. Based on the clinical presentation in the present series and later on the operative findings, perhaps none of the patients in this series could have been managed conservatively by nonoperative method. Most of the patients had major parenchymal injury (Table 8). Demonstration of signs of peritoneal irritation on repeated examination as it was in most of these patients (see table 5) would make non operative option unwise[2]. Some of the patients had more than one organ injured (table 6). In these situations the chances of successful non operative management diminishes and the risk increases[2]. Furthermore ICU monitoring, ideally requires more personnel (one-on-one nursing care) and around-the-clock laboratory, radiologic support and CT Scan control. These are some of the constraints currently prevailing at Muhimbili. They would make patient selection and management by this method at the centre rather difficult and unsafe. However,

the 4 patients who had linear splenic rupture with stable conditions and no associated injuries could have been possible condidates for splenorrhaphy. Most of the ruptured spleens at the centre are managed by junior doctors who see them first. They might not have the experience or courage to apply splenic salvage techniques single handedly.

COMPLICATIONS

Splenectomy is not without complications. These include rebleeding following splenectomy or splenorrhaphy. This may necessitate blood transfusion reoperation or both. Reoperation following splenorrhaphy or splenectomy is rare (1%). It was not encoutered in this series.

Related to the above are the risks of blood transfusion. The frequency of post transfusion hepatitis varies from 7-50%[2]. Chronic hepatitis develops in 20-50% of those infected and between 5-10% of these die of hepatitis related complication[2]. Furthermore in this era of the AIDS epidemic the risk of HIV infection through blood transfusion is very real and should never be taken lightly. Blood transfusions should be given only when absolutely necessary.

Five patients were transfused in this series - an average of 1.8 units each of iso group, iso Rh blood. Five others were auto transfused with an average of 2 units of autologous blood. No complications were noted after a 6 months follow up. This is a very short time to draw any reliable conclusion from. Longer follow up periods for HBV or HIV infection are required. The syndrome of over whelming postsplenectomy infection (OPSI) is a well documented complication. It is associated with septicaemia, meningitis or pneumonia, usually fulminant but not always fatal, occuring days to years after splenectomy. It is characterised by early systemic symptoms of nausea, vomiting and malaise that rapidly progresses to coma, hypotension and death within hours of onset. Its incidence is rare, between 0,28 to 1% per year[2,5] but the mortality is

reported to be high in the order of 50-80%. The risk is highest in young children, especially in the first 2 years after surgery (80% of cases) and when the disorder for which splenectomy was done was a disease of the reticuloendothelial system(5). It is less after splenectomy for trauma. The organisms most frequently responsible are encapsulated streptococcus preumoniae, diplococcus pneumoniae, H. influenzae, N. meningitidis. Other organisms include pseudomonas, B-Haemolytic streptococci(14). The cause of OPSI is thought to be specific immunologic defects which have been described in asplenic states and defective phagocytosis due to tuftsin deficiency, decreased properdin and IgM levels. The risk of septicaemia in asplenics has been estimated to be approximately 140 times that of the general population(2). However Luna and Dellinger(2) suggest that the quoted 1 to 2% incidence of OPSi in trauma patients may be 10-to-20 fold overestimation.

Opinions differ about the prevention of OPSI. Some authors strongly advocate prophylactic administration of pneumococcal, H. influenzae vaccines and antibiotics (penicillin) to provide protection against infection for the rest of ones natural life(15). Others are skeptical[15].

Other complications of splenectomy are atelectasis, pneumonia (especially of left lower lobe) and left pleural effusion. Left subphrenic abscess occurs in approximately 3-13% of cases(2). Thrombocytosis (with platelet count of more than 400,000 platelets/cm3) occurs within 2-10 days after splenectomy in as many as 50% of the patients. Thrombocytosis may lead to increased risk of thrombopthlebitis, deep vein thrombosis and pulmonary embolism sometimes leading to sudden death[2,5]. It has been reported that after splenectomy malaria attacks may increase or recrudescence of latent infection may occur[14]. Rare occurrence of rapidly fatal cerabral malaria

has been reported in West Africa nine months after splenectomy[14].

CONCLUSION

The spleen is one of the most commonly injured organ in blunt as well as in penetrating trauma to the abdomen and chest, despite its apparent protected anatomical position. Splenic rupture constitutes one of the most frequent surgical emergencies at Muhimbili Medical Centre.

In the last two decades the management of splenic injury has undergone a revolution from the traditional radical approach to a more conservative approach of splenic salvage by splenorrhaphy, partial splenectomy and other non operative methods. Splenorrhaphy has proved to be safe in both adults and children and it has become the standard method of surgical management of splenic injury in carefully selected patients in many centres. The post splenectomy immunological deficiencies and the incidence of OPSI have not been well documented in Tanzania. Similarly, literature from elsewhere in Africa is scarce. This subject might attract interest for thorough longtime follow up of all the patients undergoing splenectomy for trauma.

Splenic conservation by splenorrhaphy has been tried and worked elsewhere in Africa. It is quite feasible in this setting even at Muhimbili in well selected patients and its wider application at the centre would be a welcome endeavour.

REFERENCES

1. Sabiston D.C. Davis Christopher's Text book of Surgery. W.B. Saunders Co. pp1311, 1981.

2. Shackford S.R., Melinda M. Abdominal trauma: Management of splenic injuries. The Surg. Clin. N.A. 70(3):595-616, 1990.

3. Schwartz S.I., Shires G.I., Spencer F.C. Principles of Surg. Mc Graw Hill Book Co. New York pp.1446-1456, 1989.

4. Leonard R., Cohen J.L., Nancy T. injuries to the spleen S.C. N.A. 52:310, 1972.

5. Goldstone J. In Current Surgical Diagnosis and Treatment Edited by Dunphy T.E Publ. Lange Med. Publ Los Atlas California, pp.535-538, 1981.

6. Abdulmonim R.I. Rupture of the spleen in the rural parts of Egypt. Am J. Srg. 123: 310, 1972.

7. Bailey H., Love M. Bailey and Love's short Practice of Surgery. Publ. H.K. Lewis pp.940-942, 1988.

8. Tyler G., Stahl D.L. An unsual presentation of splenic rupture. J. Trauma 28(11): 1584-6, 1988.

9. Ward A.J., Gillatt D.A. Delayed diagnosis of traumatic rupture of othe spleen - a warning of the use of thoracic epidural analgesia in chest trauma. Brit. J. of Accid. Surg. 20(3): 178-9, 1989.

10. Leon P, Steven H., Frank S. Evolving concepts in splenic surgery. Splenorrhaphy versus splencectomy and post splenectomy drainage. Experience in 105 patients. Annals of Surg. 194:262, 1981.

11. Ludtke F.E., Mack S.C., Schuff-Werner P., Koth E. Splenic function after Splenectomy for Trauma. Role of autotransplantation and splenosis. Acta Chirurg. Scand. 155(10):533-9, 1989.

12. Takeda J. Hashimoto K., Tanaka M. Iwai H., Kakegawa T. Experimental and clinical evaluation of the splenic capping method in the treatment of injured spleens. Japanese J. Surg. 20(2):137-42, 1990.

13. Morgenstern L. Uyeda R.Y: Nonoperative management of injuries of the spleen in adults. Surg. Gynoecol Obstet. 157:513-518, 1983.

14. Mabogunje O.A. Splenic studies: Preservation of the Ruptured spleen. A pilot study. E.A.M.J. 62:579, 1985.

15. Jordan G.L. The Acute abdomen in Advances in Surgery Vol.14 Year Book Publ. INC Chicago, 1980.

ATYPICAL KAPOSI'S SARCOMA

SUMMARY

Twenty patients were admitted to the Muhimbili Medical Centre with an atypical presentation of Kaposi's sarcoma between July 1990 and July 1991. Sixteen were males, and 4 were females. The male to females ratio was 4:1. The age range was from 20 to 48 years with a mean age of 33.1 years. The peak incidence was between 26 and 45 years. The average age of the female patients (26.4 years) was approximately 10 years younger than the male counterparts (35.3 years). Nineteen of the patients were Tanzanians of indigenous African origin while one was a Tanzanian of Asian origin. The atypical presentation of the disease consisted of the disseminated nature of the lesions, its poorer response to treatment as compared to the endemic type of Kaposi's sarcoma, and its poor prognosis.

The commonest clinical presentation was the generalised nodular lesions or their location in unusual sites. All except one patient tested positive to the ELISA test for HIV. All the patients received half body radiation therapy at the Tumour Centre.

INTRODUCTION

Kaposi's Sarcoma has been recognised to be endemic in equatorial Africa for quite sometime[1], in 1922 the disease was described in West Africa and has subsequently been described in other African countries. In 1960 Deutz and Stout[1] described 1200 cases of Kaposi's Sarcoma. In the following year it was reported that endemic Kaposi's Sarcoma accounted for 9% of all malignant tumours in Eastern Zaire[1]. The annual incidence in Uganda was reported to be 0,7 per 100,000 population in Kampala[2].

In Tanzania one study published in 1983 reported that Kaposi's Sarcoma accounted for 17.6% of all malignancies of the skin[3].

In Africa the disease has similar clinical features in adults but behaves differently in children where it tends to be more aggressive and involves the lymph nodes[1]. The M:F ratio in children is 3:1. The clinical picture in adults is that of chronic indolent, (purplenodules) or plaques of lower extremities on an edematous background. It usually responds well to radiotherapy and spontaneous regression. All age groups are involved but occurs most commonly in the 4th and 7th decades with a dramatic male predominance[3]. The M:F ratio in adults is between 12:1 and 10:1. The mean duration of symptoms is approximately 30 months since the onset of symptoms[1].

Since the 1980's a new type of presentation of Kaposi's sarcoma began to be noted in some parts of Africa. In 1983 Bayley in Zambia reported a steady increase in an atypical form of Kaposi's sarcoma in Lusaka from 13 cases in 1983, 22 in 1984 and over 50 in 1985. It was atypical in the sense that it demonstrated features which were not previously seen: It was characterized by its greater aggressiveness, by its disseminated nature, the atypical appearance and location of the nodules, its early occurrence in lymph nodes and/or internal organs and its refractory nature to treatment.

She noted that as opposed to the endemic condition which occurred in elderly men, the patients were younger (mean age 28 years) and more likely to be women (M:F ratio 3:1). The patients were of better socio-economic status, had shorter histories and their symptoms started acutely.

In 1987 Casellas[4] working in North West Tanzania also recorded a clear pattern of changing type of Kaposi's sarcoma from 1983, onwards from the typical condition in elderly men to a more aggressive form in younger subjects affecting females as often as males.

This atypical form of Kaposi's sarcoma resembled AIDS-related Kaposi's sarcoma which was first described in 1981 in Western homosexual men[1]. The pre-AIDS annual incidence of

Kaposi's sarcoma in the US among all age groups was 2.9 cases per million men and 0,7 cases per million women per year[5]. The number has dramatically increased since the initial recognition of the syndrome[5].

We are currently seeing more and more cases of the atypical form of Kaposi's sarcoma at Muhimbili.

During a 30 month period between January 1964 and June 1966, 117 cases of Kaposi's sarcoma were recorded in the Muhimbili Registry[6], representing an average of 3 patients per month. During 12 month period between July 1990 and July 1991 73 patients with Kaposi's sarcoma were admitted to the surgical wards at the centre, most of them from upcountry stations. This is an average of 6 patients per month, which is a figure double that of the former period. Though said with some statistical caution, our experience of 73 patients in 12 months indicates a recent increase in incidence of kaposi's sarcoma.

CASE PRESENTATION

T:M was a 40 year old male who was referred to Muhimbili from an upcountry hospital complaining of nodular skin swelling on the body of 5 months duration. The nodular lesions were of insidious onset initially involving the feel, gradually increased in number and became generalized. They were associated with fever, diarrhoea and progressive loss of body weight. Over the 5 months his weight had dropped from 72 kilogram to 67 kilogram. There was also a history of cough but no night sweats. There was no history haematemesis or hemoptysis. He was a secondary school leaver who had worked as a civil servant for a number of years. He was married and lived with his wife and healthy children. He denied homosexual relations but admitted to promiscuous behavior without due protection. He had several admissions at several hospitals due to on and off fever which persisted despite courses of anti-malarial and 2 months prior to admission at

Muhimbili, he had an appendicectomy done. He denied history of tuberculosis contact or blood transfusion in the past. He denied having travelled out of the country in the past 5 years.

On examination he was found to be a young man who was moderately wasted and pale. He was febrile with an axillary temperature of 38.8oc. He was not dehydrated nor jaundiced. The blood pressure was 130/70 and the pulse was 84/minute. Palpable, non tender lymph nodes of variable sizes from 1.5-2cm diameter were found in the cervical, axillary and inguinal areas. Dark, bluish nodular swellings were detected on the face, neck, upper and lower extremities including the palms and the soles. Similar lesion were also found on the trunk. The nodules were hyper-pigmented with irregular edges. Similar lesions were found on the head palate and tongue. There was no oral thrush. Rectal examination revealed a normal anal verge. Palpable nodules were felt on the rectal mucosa on digital examination. No blood was detected on rectal examination. The respiratory and cardiovascular systems as well as the abdominal examination were normal.
A provisional diagnosis of atypical kaposi's sarcoma and AIDS was made. Investigations were carried out as follows:-
Full blood picture: Hb = 8.5 gm/dl
Red blood cell morphology - RBC morphology anisocytosis, microcytosis, macrocytosis.
ESR = 58 mn/first hour (westergren) Platelet count - reported adequate White blood cell count
Total 3.5. x 10^3 cell/mm^3
Differential: Neutrophils = 64%
Monocytes = 11.0%, lymphocytes= 25%, Eosinophil- 0 Basophil= 0
T_3 cells = 86%, T_4 cells = 1% = 20 cells/mm_3
T^8 cells = 81% = 1810 cells/mm^3
Ratio T_4/T_8 = 0,01
ELISA test for **HIV** = Positive

A skin biopsy was taken under local anaesthesia from one

of the skin nodules from the right leg. The histological features were those of Kaposi's sarcoma. A lymph node biopsy from the right groin was taken and the histology report confirmed Kaposi's sarcoma of the groin lymph nodes. The chest X-ray showed perihilar infiltration. The sputum smear for acid fast bacilli was negative three times.

A Mantoux test was requested but could not be performed for lack of reagents. With the investigations completed the patient was presented to the Tumour Board which recommended half body radiation at the Tumour centre, where he under went. His condition continued to deteriorate and he was eventually discharged home for terminal care. He died 6 months later at his home.

DISCUSSION

Aetiology

The aetiology of Kaposi's sarcoma is unknown. Though most researchers classify Kaposi's Sarcoma as a malignancy, some researchers have expressed skepticism to the historical classification of Kaposi's sarcoma as a malignancy[8] maintaining that endemic Kaposi's sarcoma for example has many unusual features distinguishing it from other types of human neoplasia. Several factors have been incriminated in the causation of Kaposi's sarcoma:-

Genetic factors: are suggested by a cluster or ethnic distribution that for example favours individuals of Italian, Jewish and Slavic ancestry[5]. Even though the disease has been described in some families in East Africa[9] familial Kaposi's sarcoma in these areas is extremely rare and probably not significant[8]. An increased incidence of HLA - DR5 has been found in both classical cases and AIDS Kaposi's sarcoma patients[5,9].

Various Oncogenic agents: AIDS patients have an increased incidence of Kaposi's sarcoma[5] because the HIV has been shown to have a selective cytopathic effect upon T^4 lymphocytes

macrophage and other cellular defence mechanisms. A breakdown in immune function is also seen in iatrogenicaly immune-suppressed patients; in patients receiving immunosuppressive treatment including renal transplant recipients. These individuals have a 400-500% greater incidence of Kaposi's sarcoma than the general population[10]. All these factors support the concept that alteration in immune surveillance contribute to the development of Kaposi's sarcoma though the trigger for this is not known. It is thought that perhaps impaired immune-surveillance may allow activation of an oncogenic virus[5]. Kaposi's sarcoma patients also tend to have high circulating levels of antibodies to several viruses like cytomegalovirus, herpes simplex hepatitis A and B, Epstein Barr Virus[11]. It has been suggested that cytomegalovirus may be a causative factor but there is no conclusive evidence[9].

In the USA, AIDS. Kaposi's sarcoma patients are mostly homosexual young men. In Africa, the homosexuality connection is not well established. We tend to find more heterosexuals who are promiscuous. In the current series all the patients were heterosexuals. None of them admitted to homosexual relations on interrogation. Fifteen of the patients were married (men and women) see Table 1. Two males admitted to have had multiple partner outside wedlock.

Table 1: Marital status

Sex	Married	Single	Divorced	Widow/widower	Total
Male	14	2	0	0	18
Female	1	1	2	0	4
Total	15	3	2	0	20

The majority of the patients were married individuals who admitted to promiscuous behaviour

Geographical and racial factors:- Earlier knowledge about classical Kaposi's sarcoma supposed that there was a geographical factor about the disease rather than racial. In Africa the frequency of Kaposi's sarcoma varied from place to place. Quenum in 1957[6] suggested that Kaposi's sarcoma increases infrequency as one approaches the equator, Whili Davies[6] in 1959 stressed, its frequency in dry sandy and greater frequency in most tropical area. There is no known geographical factor which correlates with the distribution of the tumour throughout the world.

In this study the largest numbers of patients were from Mwanza (5 patients) and Iringa (4 patients) (Table 2).

Table 2: Geographical distribution

Region	Number of patients
Mwanza	5
Iringa	4
Dar es Salaam	2
Kagera	2
Njombe	2
Kigoma	1
Mara	1
Morogoro	1
Singida	1
Tanga	1
Total	20

The largest number of patients came from Mwanza and Iringa regions.

These figures are comparable to those of Alexander[3] who found the highest incidence were from Mwanza, Iringa and Ruvuma. The ethnic distribution in his series was highest

among the Bende and Shira[3]. No ethnic or tribal relationship was studied in this series. This series is however too small for any strong conclusions. Kaposi's sarcoma was said to be rare in Black Americans and in non-Africans living in Africa. Chopra et al[12] reported in 1971 that although there is a large Asian population in East Africa, the tumour has not been reported in Asians. One patient in this series was a Tanzanian of Asian origin. He presented with an atypical Kaposi's sarcoma. He tested positive for HIV by the ELISA test. It was later revealed the he had an indigenous partner who was infected and has died of AIDS.

Environment, particularly repeated trauma, diet, infection and possibly occupational factors associated with race have been incriminated. A long history of walking barefoot has been incriminated by some authors[3].

The occupation of 12 of the 20 patients were analysed in order to find out if there was any casual relationship which may lead to identification of any aetiological agent. (See table 3).

Table 3: Occupation of 12 of the patients

Occupation	Number of Patients		Total
	Male	Female	
Peasant	4	-	4
School teacher	1	-	1
Petty tradder	3	-	3
Medical assistant	1	-	1
Digs in Privately owned gold mine	1	-	1
Soldier	2	-	2
Total	12	-	12

Peasant constituted the largest number of patients

This table indicates the increased susceptibility of the peasant which might be noteworthy. Its significance however is not clear especially in a small study group as this and in an agriculturally orientated country such as Tanzania where more than 90% of the population are peasants. It also shows the increasing involvement of patients of better socio-economic status in skilled professional and semi-skilled profession employment.

PATHOLOGY

The cell that gives rise to Kaposi's sarcoma is still not known and has been a matter of dispute[13]. It probably arises from the lymphonoreticular system. A recently reported study[14] concluded that Kaposi's sarcoma is of vascular endothelial origin. Two types of cells are related to the histogenesis of Kaposi's sarcoma.

(a) Endothelial cells - probably blood vessel endothelial cells and some lymphatic vessel endothelial lining.

(b) Spindle cells - the precise origin of the characteristic spindle cells is debated.

Both these cell types are displayed in varying proportions. General agreement exists as to the predominantly multicentric as opposed to metastatic nature of Kaposi's sarcoma lesion. Autopsy studies have shown that Kaposi's sarcoma is a multifocal disease affecting virtually every organ including the brain[1]. In endemic Kaposi's sarcoma visceral disease occurs late in adults. All tissues with a lymphatic supply have been found susceptible to the development of Kaposi's sarcoma[a(13)]. Vasoproliferation with spindle cells forming narrow vascular slits are the dominant histological features of Kaposi's sarcoma before the slits are obliterated by increased spindle cell growth[13].

A commonly used histological classification of Kaposi's sarcoma divides the lesion into 3 stages[9].

(1) Mixed cellularity - which characterises the classic and

endemic histologic pattern of patients with nodular disease. It comprises an equal mixture of spindle cells with interviewing vascular slits.

(2) The monocellular or monomorphic variants in which case spindle cell proliferation predominates and differentiation from fibrosarcoma becomes difficult.

(3) The anaplastic or pleomorphic variants are rare and resemble angiosarcomas (haemangio endotheliosarcoma). It has been demonstrated that the histologic features of Kaposi's sarcoma in immunodeficiency associated form are indistinguishable from those observed in classic or endemic forms[10]. The differences in the histologic picture have been related to the progression of lesions from patches to plaques and nodules.

The macroscopic lesions may be cutaneous presenting as nodules, plaques or florid lesions (table 4). Extracutaneous lesions can be lymphadenopathic or visceral. AIDS - Kaposi's sarcoma has a high degree of visceral involvement. While the classical Kaposi's sarcoma has about 10% incidence of extracutaneous involvement, the AIDS-related Kaposi's sarcoma has about 72%[15]. According to one report 8% of patients with AIDS - related Kaposi's sarcoma had no cutaneous involvement[16]. It has also been associated with previously unrecognised complications of unusual fungal, protozoal and bacterial infections like pneumocystitis carinii pneumonia.

About 2/3 of atypical Kaposi's sarcoma has opportunist infections when first seen[1] most often with oral candidiasis or balanitis.

CLINICAL FEATURES

Table 4 shows the age and sex distribution of the patients in this series.

Table 4: Age and sex distribution

Age of years	Male	Female	Total
20 - 25	1	1	2
26 - 30	4	2	6
31 - 35	3	1	4
36 - 40	4	-	4
41 - 45	3	-	3
46 - 50	1	4	1
Total	16	4	20

The youngest patients was 20 years old. While the oldest was 48 years old. The mean age was 33:1 years. The M/F ratio was 4:1. The youngest male was 24 year old while the oldest was 48 years. The mean age for the males was 35.3 years. The youngest oldest was 32 years. The mean age was 26.4 years for the female patients. This was almost 10 years younger than the male counterparts. The figures portray the relatively younger age group of atypical Kaposi's sarcoma patients as compared to the older age group of endemic Kaposi's sarcoma patients. Of the 20 patients in this series 12 were of indigenous African origin.

One was a Tanzanian of Asian origin. Although Kaposi's sarcoma is endemic and there is a large Asian population in East Africa, the tumour has not been reported in Asians(17). Oettle[6] has stressed that this immunity may be retained even when the immigrant population has been present in the community for over 3 centuries as in South Africa. Of his 66 cases of endemic Kaposi's sarcoma from Johannesburg, 63 were Bantu and only 3 were white people. None were Coloreds or Indians and he mentions that the latter are subject to the same type of housing conditions, are found in similar townships and attended the same hospitals. One wonders whether or not this is an indication that the disease is now

crossing the racial barriers. The M/F ratio of 4:1 found in this series is in great contrast to the M/F ratio of 12:1 or 10:1 or more, for the endemic Kaposi's sarcoma.

The duration of symptoms ranged from two months to 18 months. The average duration of symptoms was 8.3 months about 50% of the patients' symptoms were of less than 6 months duration.

Table 5: Type of Lesion

Type of Lesion	Number of patients
1. Cutaneous . Nodular	15
. Infiltrative	1
. Florid	2
2. Lymphodenopathic	2
3. Visceral	3*

*These patients had nodular skin lesions as well.

Table 6: Site of Lesions

Type of Lesion	Site	No of Patients
1. Skin nodules	Face	2*
	Lower limbs	18*
	Upper limbs	18*
	Trunk	18*
	Generalised	18*
	Genitalia	1*
2. Lymphodenopathy	Neck	1***
	Loin	1**
3. Mucous nodules	Palate	3*
	Tongue	3*
	Rectal	1*

*Some patients had nodular lesions in more than one location on the same patient.
**Two patients had lymphadenopathic Kaposi's sarcoma.

Fifteen patients presented with atypically localised cutaneous lesions which were generalised but found more dense on the trunk and on the proximal parts of the limbs. The characteristic lesions consisted of hyperpigmented skin nodules or plaques measuring 0,5 - 1cm in diameter with irregular edges. Some were localised on the face, genitalia and rarely on the distal parts of the limbs. Bayley, reporting from Zambia observed that nodules in atypical Kaposi's sarcoma had a centripetal distribution as opposed to the centrifugal distribution in endemic Kaposi's sarcoma[1]. Some of the cutaneous lesions were locally infiltrative or florid as well.

In two cases the initial presentation was symmetrical enlargement of lymph nodes other than the cutaneous lesions.

One had symmetrical involvement of groin nodes. The other one had symmetrical involvement of the cervical lymph nodes. Three patients had mucous lesions as the hard palate and tongue, one was a male the other 2 were females. They also had nodular skin lesions as well (See Table 4) and table 5). It is estimated that 34% of all patients suffering from AIDS develop Kaposi's sarcoma with an atypical presentation. 50-70% of these are in the oral cavity[18]. It is reported as benign the initial clinical manifestation in about 30% of the cases[18].

Oral Kaposi's sarcoma may present as oral nodules and patches or even ulcerated nodules or plaques of dark red or purple colour especially localized on the had palate, tonsil, pillars of the fauces and posterior pharyngeal wall or on the gums or less frequently on the tongue[1].

All sections of the gastrointestinal tract may be affected by lesions which can be haemorrhagic. Such lesions are seen in over half of all patients with atypical Kaposi's sarcoma[1] and predict the presence of nodules of Kaposi's sarcoma in the gastrointestinal tract, at any site from the oesophagus to the rectum[1]. Gastrointestinal disease may not

cause symptoms, but it can be associated with severe weight less or be responsible for intussusception needing urgent surgery[1].

All the patients who had oral lesions in this series were ELISA positive for HIV. According to this author a Kaposi's sarcoma patient with oral lesion is ELISA positive for HIV unless proved otherwise.

The two females and 1 male who presented with cervical and groin lymphadenopathy were histologically proven to be nodular Kaposi's sarcoma of the lymph nodes. Ass of them tested positive for HIV antibodies by the ELISA test. One of the female patient had a palpable epigastric mass. She was planned for endoscopy but she never came back for her appointment. In the endemic type of Kaposi's sarcoma lymphadenopathic type is almost exclusively seen in children less than 10 years and has a poor prognosis. Atypical lymphadenopathic kaposi's sarcoma has a poor prognosis[1].

One female patient had bilateral pleural effusion, which reaccumulated each time it was aspirated. About 25% of patients with atypical Kaposi's sarcoma have pleural effusion[1] which reacumulates rapidly after aspiration and unlike the rare pleural effusions of patients with endemic Kaposi's sarcoma fail to resolve after cytotoxic chemotherapy[1]. Patients develop a syndrome of respiratory distress which has not been described in endemic Kaposi's sarcoma[1]. Pulmonary complications are frequent in AIDS. Sometimes differentiation between these and pulmonary Kaposi's sarcoma is difficult by X-ray alone. AIDS patients have been associated with pneumocystis carini infection. There is also an increasing association of AIDS and tuberculosis pulmonary or extra pulmonary. In Africa 20-33% of HIV patients have pulmonary or extrapulmonary tuberculosis[19]. In order to confirm the diagnosis of pulmonary of Kaposi's sarcoma bronchoscopy with bronchial washing or biopsy of endobronchial or tracheo-bronchial lesions are indicated. Sputum for acid

fast bacilli may be taken. Kaposi's sarcoma patients may not have sputum or the sputum may be negative for tubecle bacilli. Where it is possible, isolation of pneumocystis carini should be carried out. The chest X-ray in pulmonary tuberculosis in AIDS patients may also be atypical and the patient may be associated with feature of Kaposi's sarcoma. Tuberculosis in AIDS tends to be milliary or extra pulmonary[19].

TREATMENT AND OUTCOME

All the patients in this series were presented to the tumour board after the histological diagnosis was made. In the absence of chemotherapeutic drugs at the centre radiotherapy was the only alternative recommended. All the patients received half body radiation at the tumour centre. **Table 7 shows the outcome of treatment at 6 months follow up.**

Table 7: Results of treatment

Outcome	No of patients
1. Completed remission	2
2. Temporary remission followed by recurrence	5
3. Died	2
4. Lost to follow-up	11
Total	20

At 6 months follow-up 2 patient showed fairly good response. Temporary response was followed by recurrences. As it frequently occurs with referral patients from upcountry most of our patients never came back before follow-up and were lost to follow-up. Two patients died.

Treatment of Kaposi's sarcoma should bear in mind that it is a systemic disease displaying multicentricity. The treatment should thus be addressed to the whole body otherwise local or regional treatment is bound to be followed by a

recurrence of disease elsewhere in the body sooner or later. In the African atypical Kaposi's sarcoma treatment is marked by short remissions followed by recurrences[1] after a few months.

In the AIDS-related Kaposi's sarcoma treatment is of the manifestations of HIV infection. There is no effective treatment of HIV infection available.

Systemic chemotherapy is the treatment of choice[1] for the systemic multicentric disease in the endemic type. Chemotherapy has been effective for endemic Kaposi's sarcoma patients with more disseminated disease[20]. A combination of Actinomycin D and Vincristine has been found effective[1].

Patients with Kaposi's sarcoma who are severely immunocompromised respond drugs[20] various combination have been experimented. Single agents such as VP-16 or vinblastine yield complete response rates of 20 - 40% with prompt relapse when treatment is stopped[20]. Though conventional cytotoxic therapy has been used. Some authors caution that these may be counter productive in that they may further impair cellular immunity in these already immunosuppressed patients favouring the appearance of opportunistic infections[20].

Endemic Kaposi's sarcoma is radiosensitive and has been treated with radiotherapy. Radiotherapy is the treatment of choice for localised nodular Kaposi's sarcoma. Most large series report complete response in rates of 40-85%[20]. Patients with atypical Kaposi's sarcoma have a poor response to treatment as compared to those with endemic Kaposi's sarcoma. Because the disease's multicentricity some authors have suggested its combination with chemotherapy[9].

Experience obtained at this centre indicates that patients with endemic Kaposi's sarcoma respond well to radiotherapy alone either by local, extended local or half body radiation.

It has been suggested that in the treatment of Kaposi's sarcoma in the immunocompromised patient the most important

determinant of outcome is restoration of immunocompetence. Several clinical trials of alfa inteferons, particularly recombinant interferon alfa-2a (rbe) have been reported in the USA[21]. High dosage regimens tend to produce the best responses[20][21]. Azidothymidine (AZT) is currently being used for treatment of AIDS. It functions by blocking viral replication. Its effectiveness in the treatment of atypical Kaposi's sarcoma is still to be evaluated.

The role of surgery in the treatment of atypical Kaposi's sarcoma is limited due to the multicentricity of the disease. However, if the lesions are unusually extensive or interfere with a bodily function such as ambulation then circumscribed surgery can be used to control local symptoms, for example surgical extirpation of a tumour on the sole of the foot so that the person can walk.
Surgical amputation of part of a limb can palliate the patient for some months but more often than not the tumour will develop in other parts of the body. Surgical application has been indicated in a few circumstances for example for tissue biopsy, to reduce tumour bulk, to remove a disease riddled limb or to deal with complications of the tumour for example peritonitis, intestinal obstruction.

None of the patients in this series was managed surgically except for a skin biopsy from the nodular lesion or lymph node biopsy. These were done under local anaesthesia.

CONCLUSION

More than 100 years have passed since the Hungarian dermatologist Moritz Kaposi's described what the called Idiopathic, Multiple Pigmented Sarcoma in 1872, describing it as being "curious and fatal". Even today Kaposi's sarcoma still remains a clinical curiosity and indeed a histogenetic and aetiologic puzzle around which many scientific papers are still written. Many questions still remain unanswered. How for example could a tumour with such a singular histologic

appearance occur in such diverse clinical circumstance? What is the reason for its unusual geographic, ethnic and demographic features?.

The recent description of still a new type of an atypical form of Kaposi's sarcoma complicates the situation still further. Along side with classical (European) and endemic (African) Kaposi's sarcoma which have been known for many years the advent of AIDS brought with it an atypical form of Kaposi's sarcoma in AIDS patients and in other immunosuppressed risk groups. The situation has been complicated still further by still another but similar atypical form of Kaposi's sarcoma described by Bayley as African Atypical Kaposi's Sarcoma. It would appear that the common denominator in AIDS related Kaposi's sarcoma and the (Africa) atypical Kaposi's sarcoma is the situation in which the body's immune function has been tempered with in one way or another.

The large percentage of seropositivity for HIV in this study group is noteworthy. Most patients with oral lesions should be considered ELISA positive for HIV unless proven otherwise. Perhaps vigorous control of the HIV infection and AIDS disease would lead to a significant containment of atypical Kaposi's sarcoma.

REFERENCES

1. Bayley A.C. Kaposi's sarcoma in African. Postgradu. Doctor (Afr) 9(5):147-42, 1984.

2. Bayley A.C. Aggressive Kaposi's Sarcoma in Zambia, 1983 The Lancet i:1318-1320, 1984.

3. Alexander G.A. Geographical Aspects o Cancer in Tanzania J. Nat. Med. Ass. 75(8):797-804, 1983.

4. Casellas F.S. Changing Patterns of Kaposi's Sarcoma in North West Tanzania. Trans. R. Soc. Trop. Med. Hyg. 81:841, 1987 (Short report).

5. Penneys N.S. Skin manifestations in AIDS. Publ. Martin Dunitz pp94-113, 1990.

6. Slavin G. Cameron, H.M. Singh. Kaposi's Sarcoma in Mainland Tanzania. A report of 117 cases, Brit. J.Cancer 23(2):349-57, 1969.

7. Bayley A.C. Aggressive Kaposi's Sarcoma in Zambia in 1983. Proceedings of the Associated E. African 7:189, 1984.

8. Brooks J.J. Kaposi's Sarcoma:A reversible hyperplasia. lancet 2: 1309-11, 1986.

9. Kyalwazi S. Kaposi's Sarcoma. The Ibadan Surgeon. 2:70-78, 1978.

10. Santucci M, Pmpinelli, Morettis, Giannotti B. Classic and Immunodeficiency - Associated Kaposi's Sarcoma. Arch. pathol. lab. Med. 112:1214-20, 1988.

11. Baughan C.A, Phillips R.H. the clinical presentation of AIDS Kaposi's Sarcoma and other tumours. Postgrad Doct (Afr) 8(9): 261-6, 1986.

12. Chopra S., Templeten A.C, Cancer in East Africa Indians Int. J. Cancer 8:176-83, 1971.

13. Owor R. Epidemiologic of Cancer in E. Africa. Postgrad. Doctoral (Afr) pp.15-22, Jan 1985.

14. Rutgers J.L. et al: The expression of endothelial cell surface antigens by AIDS - Associated Kaposi's Sarcoma. Am. J. pathol. 122:493-499, 1986.

15. Fauci A.S. et al. Acquired Immunodeficiency Syndrome: Epidemiologic clinical, immunologic and therapeutic considerations. ann. Interm. Med. 100(1):92-100, 1984.

16. Krigel R.L et al. Kaposi's sarcoma. A new staging classification cancer Trest Rep. 67(6):531, 1983.

17. Chopra S., Templeton A.C. Cancer 8: 176-83, 1971.

18. Kunz, A.A., Golderbom HR, Winkel T. Reichart PA. Ultrastructural findings in oral Kaposi's sarcoma J. Oral Path. 16:372-9, 1987.

19. Bigger R. Clinical Features of HIV in Africa. Brit. Med. J. 293: 1453-1454, 1986.

20. Ziegler J.L., Templeton and Vogel C.L. Kaposis sarcoma: a comparison of classical, Endemic and Epidemic forms. Seminars in Oncology 11(1):47-52, 1984.

21. Krown S. et al. Kaposi's sarcoma and acquired immune deficiency syndrome: Treatment with recombinant interferon alpha and analysis of prognostic factors cancer, 57 (suppl):1662-1665, 1986.

CARCINOMA OF THE OESOPHAGUS

SUMMARY

Twenty patients with carcinoma of the oesophagus were admitted and managed at the Muhimbili Medical Centre between April and November 1991. Fourteen were males and 6 were females. The M/F ratio was 2.3:1. The age range was between 30 and 73 years with a mean age of 55 years. The peak incidence was between 40 to 70 years. The commonest presenting symptoms were progressive dysphagia, regurgitation and weight loss. Most of the patients presented late with an advanced disease. Management consisted in palliation of the symptoms.

This monograph presents in detail one of these patients and analyses the results of the rest of the patients with a discussion of the results. A review of the current literature is made.

INTRODUCTION

For a long time appreciation of the pathology of the oesophagus had been obscured by the relative inaccessibility of the structure. With the designing of the first oesophagoscope by Kussmaul in 1863 and the subsequent development of more sophisticated methods of endoscopy and diagnostic radiology it has become possible to diagnose and study oesophageal pathology[1].

Despite of the tremendous advances made in the management of the disease, late presentation, high mortality and low rates of resection reported in various series[2] still qualify carcinoma of the oesophagus as a devastating silent killer disease in many parts of the world including Tanzania.

Dysphagia which is the most significant symptom that brings most patients to hospital is an almost invariable sign of advanced disease[3]. In such circumstances and since it is necessary to relieve the distressing symptom of dysphagia,

palliative treatment becomes an important consideration in our setting. Palliative irradiation, intubation or a combination of these are the commonest mode of treatment. Surgical palliation by a by-pass procedure is not often considered in view of the poor general conditions of the patients at presentation.

CASE PRESENTATION

A.M. a 42 year old African male who was employed as a watchman at one of the government departments at Morogoro, presented with a 3 months history of difficulty in swallowing. He had been experiencing progressive difficulty in swallowing, first with solids and now with fluids as well. Of late he was able to swallow water and porridge but with difficult. He regurgitated foods and occasionally these were foul smelling and might be bloody. He had lost considerable weight. There was no history of having ingested any corrosives in the past. There was no history of cough. He denied any history of trauma or any history of previous instrumentation to the throat. He had never changed residence. He had generally been healthy and this was his first admission to hospital. The bowel habits had been normal. He had no urinary complaints.

He admitted to have been a cigarette smoker for over 20 years consuming an average of one packet of cigarettes in a day or two. He denied history of alcohol consumption. He was married with 2 children who were alive and well. He lived with his family at Morogoro.

On examination he was a wasted young individual who looked ill. He was not dehydrated nor was he pale. There was no peripheral lymph-adenopathy. The pulse rate was 88 beats/minute and the blood pressure was 100/60mmHg. The Virchows node was not palpable. The respiratory and cardiovascular systems were normal. Abdominal examination was normal. A diagnosis of carcinoma of the oesophagus was made.

The following investigations were taken:-

Full blood picture:

Hb = 11.9gm/dl

WBC = total count - 6,800/ul

Differential - Neutrophils = 65%

Lymphcyotes = 25%, Eosinophils = 2%

Basophils = 3%, Monocytes = 10%

RBC morphology - normochromic, normocytic

Platelet count - "adequate"

Serum electrolytes - Na+ = 135mmol/l

K = 3.5mmol/l

Serum creatinine 71 umol/l

Stool - normal

Urine - normal

The chest X-ray was normal

Barium swallow showed a narrow segment of the distal third of the oesophagus with a ragged mucosal pattern with little passage of barium into the stomach.

Rigid oesophagoscope under general anaesthesia revealed a foul smelling pool of secretions and food debris in the oesophagus. After aspiration a fungating tumour was visualised at 34cm from the upper incisor teeth. A biopsy was taken whose histological report revealed moderately differentiated squamous cell carcinoma of the oesophagus. The patient was then presented to the Tumour Board which recommended radical radiotherapy at the tumour centre. The patient received a total of 6000 rads at a dose of 200 rads per day, 3 days per weeks. After completing the treatment the dysphagia improved considerably. He was discharged home for follow-up in 3 months. He was however readmitted to hospital 6 months later with recurrence of the dysphagia. He complained of dysphagia to solids as well as to liquids. He was coughing and choking on taking fluids and had episodes of haemoptysis. A repeat chest x-ray and barium swallow were done. The barium meal showed ragged proximal dilatation of

the oesophagus. There was passage of contrast through an oesophago-tracheal fistula. Intravenous fluids were given. Six days after admission he died.

RESULTS

Table 1: Age and Sex distribution

Age (years)	Nummber of patients		TOTAL
	Male	Female	
30 - 40	2	-	2
41 - 50	2	2	4
51 - 60	5	3	8
61 - 70	4	1	5
71 - 80	1	-	1
Total	14	6	20

The M/F ratio was 2.3:1. The age range was 30 to 73 years. The peak incidence was 40 - 70 years. The mean age was 55 years.

Table 2: Regional distribution

Regional	Number
Morogoro	6
Kilimanjaro	4
Tanga	3
Coast	2
Dodoma	2
Iringa	2
Ruvuma	1

Table 3: Presentation

Symptoms/Signs	No of Patients
Progressive dysphagia	20
Weight loss	18
Regurgitation	15
Pain	5
Cough	4
Haematemesis	1
Choking	3
Axillary lymphadenopathy	1
Hepatomegaly	1

The commonest presentation was progressive dysphagia and weight loss.

Table 4: Duration of symptoms

Time in months	Number of Patients
0 - 4	6
5 - 8	5
9 - 12	4
13 - 16	3
17 - 20	2
Total	20

The duration of symptoms was less than 12 months in 75% of the patients.

Table 5: Factors associated with Carcinoma of the Oesophagus

Associated factors	Number of Patients
Smoking	11
Local brew	9
Snuff	1
Occupational exposure	0

Approximately half of the patient had history of smoking and of consuming local brew.

Table 6: Site or level of lesion

Site/level of lesion	No of Patients	Percentage
Upper third	4	20
Middle third	9	45
Lower third	7	35
Total	20	100%

The middle third of the oesophagus was affected most frequently.

Table 7: Pathology

Histology	No of Patients	Percentage
. Squamous cell Carcinoma	18	90
. Squamous cell carcinoma and mucinous cell carcinoma on the same specimen	1	5
	1	5
Total	20	100%

The commonest histological type was squamous cell carcinoma.

Table 8: Treatment and results

	No of Patients	Results/outcome
Palliative radiotherapy	17	6 died before completing treatment
		7 completed treatment but lost to follow up, died?
		3 refused (treatment)
		1 absconded
Radical radiotherapy	2	2 completed course with relief of symptoms but had recurrence of tumour 6 months later.
To advanced disease medical palliation	1	Discharged home at relatives request

DISCUSSION

The most striking feature of carcinoma of the oesophagus is that it occurs in certain well defined geographical areas throughout the world[4]. The disease does not seem to be peculiar to any racial group[4] but in the US both Black males and females have a roughly 4-fold greater incidence than whites[5].

The highest incidence rates are recorded in China, Russia, Scotland, France and the Scandinavian countries[6]. High incidence have been reported in East, Central and South Africa where epidemic levels have been described in the Transkei region[2]. Low incidences have been reported in North and West Africa where Akinsanya et al[2] found a M/F ratio of 2.6:1. In Tanzania, Aziz[7], analysing cases from the Tanzania cancer registry found that carcinoma of the oesophagus constituted 1.8% of all tumours recorded. Hiza[8] found it to be the 4th commonest cancer of the gastrointestinal tract.

Mwakyusa[9] found carcinoma of the oesophagus in 4.9% of a total of 934 endoscopies performed at the Muhimbili Medical Centre. Mmuni[10] in a 10 year retrospective study at KCMC found a high incidence of carcinoma of the oesophagus among the Wachaga and the Wapare. The M/F ratios among the Wachagga and Wapare were 3.6:1 and 7:1 respectively. The incidence was 1.39%. Crech[2] however has reported a much higher M/F of 11:1 in Tanzania.

AETIOLOGY

Carcinoma of the oesophagus is one of the most interesting tumours due to the great diversity with regard to its regional incidence worldwide. The precise cause of these major regional differences is still not fully understood[4]. However the role of nutritional determinants and other environmental factors has received increasing support from data collected during the last decades[11].

Nutritional/Dietary Factors and Environmental Factors.

Heavy alcohol consumption and tobacco smoking have been incriminated[4]. Compounds like fusel oils, benzopyrene and volatile nitrosamines or their precursors (nitrates and nitrites)which are common contaminants of alcoholic beverages, have been shown to be carcinomagenic in experimental animals[4].

Aflatoxins have also been suspected of causing oesophageal cancer. They have been identified in mouldy and fermented foods contaminated by fungi due to faulty storage of cereals or resulting from their mode of preparation[12]. Other dietary agents include irritants, excessively spicy foods and chills. Occupational exposure to various substances like metal dust has been associated with carcinoma of the lower oesophagus[4].

Predisposing Conditions:

A particularly high incidence of oesophageal carcinoma has been associated with certain predisposing condition[3]. These

include chronic iron deficiency anaemia especially in women (Plummer-Vinson or Peterson-Kelly syndrome)

- In Achalasia the reported incidence of malignancy is 5-15%[3]
- Corrosive oesophagitis - these patients are 200 times more likely to develop carcinoma than the general population.
- Long standing Gastro-oesophageal reflux when associated with columnar lined (Barrett's) oesophagus or peptic stricture the incidence of oesophageal adenocarcinoma is 2.5 -10%[3,5].
- Other conditions include lye burns, tylosis and diverticula of the oesophagus.

In a 10 years retrospective study of gastrointestinal malignancy by Mmuni(10) at KCMC the Wachagga and Wapare were found to be the highest affected tribe by oesophageal cancer, the disease occurred at an even younger age groups compared to Western data(10). No underlying causes could be established in this area but unidentified dietary factors could not be ruled out.

Carcinoma of the oesophagus affects men more than women(4,7) except in Iran where the M/F ratio is reversed(13). This would suggest cultural factors in its aetiology. Alcohol consumption and smoking or chewing have been reported to be associated with high incidence of oesophageal cancer(4). Some authors blame the amount of alcohol others believe that it is heavy alcohol that matters(4). The combination of alcohol and smoking is said to provide a particularly strong predisposition to carcinoma of the oesophagus the increase reported being as high as 44-fold(4).
At least 50% of the patients in this series admitted to have smoked cigarettes and that they consumed alcohol.

In Africa oesophageal carcinoma has been associated with consumption of local spirits made from maize meal(12) but this may be related to the mode in which it is prepared. Areas in

which local beer is made from millet, sorghum, cassava and other cereals, have generally lower incidence of oesophageal cancer.

PATHOLOGY

The majority of the series(3,4,6) show that approximately 20% of the malignancies occur in the upper third. The middle third accounts for 50% of the tumours while the remaining 30% occurs in the lower third. Table 5 shows the oesophagoscopic locations of the tumours in the current series. Our figures are comparable to those mentioned in the literature.

Typical of oesophageal carcinomas is that they remain silent and asymptomatic during much of their early development. By the time they present symptomatically and are diagnosed they are usually 4-5cm or more in length, are nearly or completely circumferential and have penetrated deeply into the oesophageal wall(3),4). Depending on whether the growth is predominantly exophytic, three endoscopic types can be recognised(3,4): fungating, ulcerative and infiltrative. Table 6 shows the histological types of the 20 specimens in this series. It is reported that when cases of adenocarcinoma of gastric origin involving the distal oesophagus are excluded from the series, about 95% of primary oesophageal malignancies are squamous cell carcinoma(4) and they arise from the proximal 2/3 of the oesophagus. True adenocarcinomas of the oesophagus account for 3% -4% of primaries. These figures tally with the results in this series (see table 6). The remaining 1-2% of the oesophageal cancers are made up of a miscellaneous group of neoplasms of varied and of uncertain histologies(4). Oesophageal carcinomas can spread intraoesophophageally and extraoesophageally(4). Three major ways of spread have been described: Direct spread is the main way of spread and the most important to the surgeon. Blood spread is a late mode of spread and is uncommon. Lymphatic spread is responsible for malignant submucosal lymphatic

extension up and down the oesophagus. Lymphatic extension may lead to involvement of the 3 parallel and interconnected chains of nodes - the paraoesohageal, perioesophageal and the lateral oesophageal nodes(4).

Either cell type of the carcinoma can extend submucosally through lymphatic spread. Usually this microscopic cancer spread is more extensive than the macroscopic reach of the tumour. This fact has got important therapeutic implications when defining adequate clearance during resection(3,4). It is therefore advised that in order to reduce the risk of inadequate excision, the surgeon should excise a 7-12cm margin of macroscopically normal oesophagus beyond the upper limit of the tumour(3,4). The inferior limit of visible tumour is less extensive and it requires a 5cm margin of un-involved tissues distal to the tumour for adequate excision(3,4). Extension of malignant cells by the submucosal lymphatics can result in tumour emboli to sites 2cm or more from the main growth producing "skip" or satellite nodules(4) and rarely, so called double carcinomas separated by radiologically intact epithelium. The prognosis of these is usually poor(4).

Carcinoma of the oesophagus can extend outside the oesophagus penetrating the muscle layers to vital neighbouring structures even before the lumen becomes obstructed by malignant growth and before the patient complains of dysphagia. The prognosis for patients with extra oesophageal spread is usually grave(4).

According to the TNM classification carcinoma of the oesophagus has been classified as follows(11).

T classification

T0 = no evidence of tumour

T1 = tumour invasion of the mucosa or submucosa but not the muscle wall

T2 = Tumour invasion of the muscle wall

T3 = Tumour invasion beyond the muscle wall (perioesophageal structures).

T4 = Distant metastasis < lung liver

N classification:

N0 = no lymph node metastasis

N1 = unilateral regional lymph node metastasis

N2 = bilateral regional lymph node metastasis

N3 = extensive multiple regional lymph node metastasis

M classification:

M° = no evidence of distant metastasis

M^{1} = distant metastasis present

Staging for oesophageal carcinoma

Stage I: T_1 N_0 M0

Stage II: T_2 N_0 M_0

Stage III: T_3 N_0 M_0, any T any N, M_0

Stage IV: Any T, any N, M^1

CLINICAL FEATURES

There were 14 males and 6 females. The age range was 30-73. Table 3 shows the commonest symptoms and signs at presentation in the current series. Progressive dysphagia was the commonest symptom present in all the patient. It was of varying durations from one month to 20 months (mean 7 months). Other features were weight loss and regurgitation. Painful swallowing, cough and haematemesis were noted in patients who had very advanced disease. They died early before completing the course of palliative radiotherapy. One patient had axillary lymphadenopathy and another had hepatomegaly. The significance of these findings could not be determined as the patients died shortly in the ward of advanced disease and aspiration. At least 2/3 of the circumference of the oesophagus must be involved by tumour before obstructive symptoms develop[3]. Difficulty in swallowing is the definitive and usually the earliest symptom of carcinoma of the oesophagus. Dysphagia which is progressive in nature is a late symptom. The most unfortunate thing about this symptom is that it is usually tolerated by the patient for several months before presentation to hospital.

Some patients present only when they have difficulty in swallowing fluids. Once dysphagia is present its progress is rapid[3]. Other symptoms include intermittent retrosternal discomfort found in 10% of cases in one series[4]. Regurgitation usually accompanies weight loss-despite constant hunger. Frank haematemesis can occur but is rare. Pain is also a common symptom. It may be experienced in the neck, in the retrosternal region, in the interscapular area or in the epigastrium. The symptom of pain should be related to the act of swallowing. Pain precipitated by swallowing indicates only a lesion in the oesophagus, while constant pain, exacerbated by swallowing usually means that the cancer has extended through the wall of the oesophagus to excite a perioesophageal inflammatory response or to invade extraoesophageal tissue. A chronic cough may indicate persistent basal pneumonitis as a result of aspiration. Haemoptysis is uncommon but when present is evidence of tracheobronchial invasion. Voice hoarseness is most often caused by recurrent laryngeal nerve involvement or compression by the tumour process. Sometimes, however, it can be due to the laryngitis associated with chronic aspiration.

INVESTIGATIONS

All the patients in this series were investigated. The key diagnostic investigations apart from the history and physical examination were barium swallow, rigid oesohagascopy and biopsy plus a few baseline investigations. Barium swallow was one of the most important investigations done in every patient. In all the patients they showed features of oesophageal malignancy. The characteristic feature of malignancy is that of a narrow segment of the oesophagus with a ragged mucosal pattern[4,6]. The oesophagus above the lesion may appear dilated. In the fungating variety of the lesion the barium swallow will show multiple filling defects with ulcerated borders. These may involve one margin of the

oesophagus or the entire circumference, producing a "shouldering" effect on the barium column[6]. In early oesophageal cancer where the lesion is limited to the mucosa or submucosa, the radiological changes are often inconspicuous and are easily overlooked.

Oesophagascopy is complementary to barium swallow in diagnosis. Either rigid or flexible fiberoptic oesoghagascopy can be used. All the patients in this series were scooped using a rigid oesophagoscope. Biopsies were taken at different sites of the tumour for histologic confirmation in all the patients. Up to 8.7% of the biopsies of subsequently confirmed cases have been reported as negative[4].

Where repeated biopsies have been negative, cytologic examination of either blind or directed[3,4] oesophageal "brushings" (brush cytology) is indicated. One recent advance is the use of endoscopic ultrasound probe in the diagnosis of and for evaluation of the depth of involvement or spread of the tumour[14]. Blind abrasive cytology (BAC) is an outstanding technique for mass screening of populations at risk of oesophageal carcinoma[4]. Plain chest x-ray is important and can influence the selection of treatment. One may be able to detect the presence of aspiration pneumonia, lung abscess, pleural effusion or the presence of other diseases like extensive pulmonary tuberculosis that may compromise surgical options of management. Other investigations include CT scan, radioisotope scanning, baseline investigations like full blood picture, serum electrolytes and urea, principally taken for the assessment of the nutritional and hydration, status, renal function and haematological status of the patient. The role of carbohydrate antigen 19-9 as tumour marker of oesophageal cancer is still at experimental stage[15].

TREATMENT

The objective of treatment in oesophageal carcinoma is

firstly to restore normal swallowing, secondly to prolong life in comfort and thirdly if feasible to cure the patient. Ideally the chosen treatment should be both effective and safe with acceptable low mortality procedures.

The most outstanding features of the patients in this series were their late presentation, the very high mortality and the relatively low patient compliance associated with radiotherapy. These features reflect the general gloomy picture of the prognosis of carcinoma of the oesophagus and the common man's erroneous belief circulating around here that the decision to go to Ocean Road Hospital for radiotherapy ("mionzi") is tantamount to a sentence of death.

The majority of the patients in the series had advanced disease at presentation, many of them wasted and in very poor condition. Five patients had dysphagia to fluids at presentation, they were dehydrated and had to be rehydrated with intravenous infusions in the ward.

Since it was necessary to relieve the distressing symptom of dysphagia palliative treatment became a very important consideration in the management of these patients. Palliative radiotherapy was recommended to 17 of the patients. (see table 8). Two patients who looked stronger than the rest underwent radical radiotherapy.

In general, management of oesophageal carcinoma can be disheartening because the return on the therapeutic investment is poor. Concerning curative therapy, surgical resection remains the only therapy to be associated with prolonged survival and long term palliation[4,16]. It has been found to be more effective in controlling the local disease and in restoring function[4]. Much of the success however depends on careful staging, on patient selection and on the expertise of the surgeon[4,17] Preoperative preparations include physiotherapy and correction of nutritional deficiencies[4] not by gastrostomy[6] but by an in-dwelling Clinifeed tube or by modern regimes of intravenous feeding. For carcinoma of the

mid third of the oesophagus a two stage abdominal-thoracic approach is applied. Partial oesophagogastrectomy with oesophagogastric anastomosis is done above the aortic arch through a right thoracotomy (the Ivor Lewis operation). The operation is preceded by a laparotomy. The stomach is mobilised and the abdomen is explored to ascertain the absence of possible metastases. The operation may also be performed through a left thoracoabdominal approach. At oesohagectomy it is essential to obtain a 7-12cm clearance of the tumour to prevent recurrence[3,4]. Nakayama[2,3] has gone further to advocate total oesophagectomy with cervical anastomosis as the only curative form of surgery for operable tumours. Complications include suture line leakage and anastomotic stricture. Many ingenious surgical procedures were developed in the past. Some of them were elaborate multistaged operations. The trend today is to minimize the duration of the treatment period utilizing one-stage procedure[16]. Carcinoma of the upper third of the oesophagus is rarely diagnosed in its operable stage. If it does then the operation of choice is McKeown 3 stage oesophagectomy[16]. Otherwise the tumour is excised and continuity is restored by jejunal or colonic transposition. For carcinoma of the lower third of the oesophagus partial oesophagogastrectomy is done by a thoracoabdominal incision through the 8th rib bed, extended on to the abdomen[4,16]. Operative mortality has been reported to vary tremendously ranging from 98% by Akiyama to 57% by Franklyn[4]. Earlam and Cunha Melo[4] reviewed the results of treatment in over 83,000 cases of oesophageal cancer. The one, two and 5 year survival rates after successful resection were 70%, 29% and 18% respectively. With experienced surgeons, and with the tumour localised to the oesophageal wall, 60-90% the patients survived 5 years in the series[4].

According to one Chinese report of early cancer diagnosed by cytologic screening of a high risk population the

resectability of 170 cases of early oesophageal cancers was 100%. The 5 year survival rate wa 90%[16].

Curative Radiotherapy: Theoretically squamous carcinomas are radiosensitive. Two patients in this series who had squamous cell carcinoma of the distal third of the oesophagus looked stronger and in better general condition than the rest. They completed a course of radical radiotherapy, a total of 6000 rads, given at 200 rads per day, 3 days a week. They improved and were discharged home for follow up, only to be readmitted 6 months after for tumour recurrence. They died 1 and 2 weeks respectively after readmission. Like surgical resection curative radiotherapy is used for local control of the disease.

There are several theoretical advantages of radiotherapy[3]. Firstly, it is non invasive and in favouralable conditions it may achieve similar results as those of resection without the associated morbidity and mortality. Secondly, it is safe, the treatment can be stopped if the patients' condition deteriorates. Thirdly it may be therapeutic as well as prophylactic for invasion of metastasis in areas that are not approachable surgically like the superior mediastinum[17]. Careful evaluation and selection of cases as well as expertise with the techniques of radiotherapy can influence favourably the results of treatment. The results of treatment have been varied. In 2 large series the 5 year survival rates were 0% and 1%[4]. In a more suitable series for operation the one year and 2 year survival rates were 42% to 46% and 8 to 27% respectively. The average 5 year survival rate was 6%[4]. Pearson[18] reported an exceptionally good 5 year survival rate of 20%. According to Pearson[18] radiotherapy fails to control the growth in the oesophagus in 50% of cases. He noted that recurrence was usually evident within 6-12 months after treatment. This is because "in vitro" radiosensitivity of the tumour may not be exactly the same as the "in vivo" radio sensitivity[4]. Two patients in our series had a recurrence 6

months after treatment. Furthermore, failures result from presence of tumour outside the irradiated field(4,16). Radiotherapy can be associated with complications like radiation pneumonitis, myocardial damage, spinal cord injury. Absolute contraindications to radiotherapy include malignant infiltration of the aorta or bronchial tree by the tumour, or when the tumour is large as 10cm diametre or more(4,17).

Combined therapy: has produced encouraging results. Akakura et al(19) found a 5 year survival rate of 25% in 117 patients treated by preoperative radiation followed by surgical resection compared to a 13.6% five year survival rate observed in an earlier group of 229 patients treated by surgery alone. Huang, Guojun and others(20) reported a 31.6% five year survival rate by combination therapy compared to 27.6% by surgery alone.

Palliative Therapy includes radiotherapy, intubation and chemotherapy. Palliative radiotherapy was used in 17 patients in this series. Only 7 patients completed the course (table 8) with a relief of dysphagia and pain. They were however lost to follow up. They were probably dead!

The use of tubes for palliative intubation of carcinoma of the middle third of the oesophagus has been practiced for several years. Several tube modifications have been used. Each one has its merits and disadvantages. Das and John(2) found the Celestine tube to be superior to Monsseau-Barbin tube in terms of operative morbidity and mortality. Hiza[21] found the Celestin tube to provide good palliation. Akinsanya et al[2] expressed dis-satisfaction with intubation either because of lack of medical supervision when the patients left hospital to their villages or because of the bulky foods that tended to block the tube from time to time. This consideration led them to use oesophagogastrectomy as an alternative method of palliation for carcinoma of the thoracic oesophagus. Endoscopic intubation is considerably safer than surgical intubation. Intubation with a Celestine or Atkinson tube

performed at fibre optic endoscopy is reported to be the safest and most effective means of palliation[3]. A hospital mortality of 11-27% has been reported[3]. Endoscopic laser therapy used to open an obstructed oesophagus with minimal morbidity and mortality has made it a primary approach in palliation of many of these patients[16].

Chemotherapy: There are conflicting results about the efficacy of palliative chemotherapy[3,4] with either squamous cell or adenocarcinoma. The cytotoxic agents found to have some effect on squamous cell carcinoma of the oesophagus include methotrexate, 5-fluoro uracil, Adriamycin, Bleomycin, Mitomycin C, Cis platinum and vindesine. The most commonly used drug is bleomycin used alone or in combination with other cytotoxics or with radiotherapy[3].

CONCLUSION

Carcinoma of the oesophagus is a common malignancy with varying geographical distribution worldwide. No one so far has come up with convincing evidence of one aetiological agent, but the role of nutritional factors and environmental factors are receiving increasing support in the last few decades. The poor prognosis of the disease and the poor survival statistics despite the ingenious surgical procedures developed has partly been attributed to late presentation.

Cure of early cancer can be achieved frequently with current available treatment modalities as is evident from the Chinese reports in early cancer detected by cytologic screening of high risk populations. Further studies are required in Africa to substantiate the present aetiological theories. Perhaps after all carcinoma of the oesophagus can to a large extent be prevented by for example preventing food contamination by mycotoxins, modifying social habits of high risk groups.

REFERENCES

1. Sabiston D.C. Davis Christopher Textbook of Surgery. W.B. Saunders Co. 10th Ed. pp760-71, 1972.

2. Akinsanya B., Bassey O.O., Elebute E.A. A ten year study of carcinoma of the oesophagus at the Lagos University Teaching Hospital. Nig. Med. J. 4(1): 18-21, 1974.

3. Watson T. Carcinoma of the oesophagus. Surgery 1(13): 292-295, 1984.

4. Manell A. Carcinoma of the Esophagus in Current Problems in Surgery. 19(10):557-638, 1982.

5. Robbins S.L.., Kumar V., Cotran R.S. Pathologic Basis of Disease - 3rd Ed. W.B. Saunders Co. pp.804-6, 1984.

6. Bailey H., Love M. Bailey and Love's Short Practice of Surgery 12th Ed. English Lang. Book Soc. pp 848-854, 1988.

7. Aziz M.R., Hiza P.R. Carcinoma of the oesophagus in Tanzania. Proceed. Assoc. Surg. E.A. 3:92, 1980.

8. Hiza PR. Malignant Disease in Tanzania, E.A.M.J. 53:82, 1974.

9. Mwakyusa DH., Dolmans WM., Mbaga IM. Oesophageal Malignancies in Dar es Salam Trop. Geograph. Med. 34(3):257, 1982.

10. Mmuni K.A. Upper gastrointestinal malignancies as seen at KCMC 1973-82. Proceed of the 24th Postgrad. KCMC Seminar pp. 142-8, 1984.

11. Khojasteh A., Kraybill WG. Cancer of the oesophagus: The environmental connection. South Med. J. 81(7): 878-82, 1988.

12. Manshande JP. Lancet I 1499-1550, 1987.

13. Mahboudi E.O., Aramesh B. epidemilogy of oesophageal cancer in Iran with special reference to nutritional and cultural aspects. Pre. Med. 9:613, 1980.

14. Takemoto T., Ito T. Aibe T, Oluta K. Endoscopic Ultrasonography in the diagnosis of esophageal carcinoma with particular regard to stagin it for operability Endoscopy 18 supp. 3:22-5, 1986.

15. Mc Knight, A., Manell A., Shperling I. The role of cabohydrate antigen 19-9 as a tumour marker of oesophageal cancer. Brit. J. Cancer. 60(2):249-51, 1989.

16. Schwartz S.I., Shires G.I., Spencer FC. Principles of Surgery 5th Ed. McGraw Hill Co. pp.1138-1145, 1988.

17. Akiyama H. Surgery for carcinoma of the oesophagus in current problem in surgery 17(2):73, 1980.

18. Pearson J.G. The value of radiotherapy in the Management of Squamous oesophageal cancer. Br. J. Surg. 58:794, 1971.

19. Akakura I. et al: Surgery of Carcinoma of the esophagus with presentative radiation. Chest 57: 47, 1970.

20. Guojun H., Xianzhi G. et al. Combined preoperative irradiation and surgery in esophageal carcinoma: Report of 408 cases. Chin. Med. J. 94:73, 1981.

PENETRATING CHEST INJURIES

SUMMARY

The ten cases presented in this study were collected over a 2 year period from July 1990 to July 1992. All of them had penetrating chest injuries of various aetiologies. Three of the patients were diagnosed and treated by the author hence they represent his personal experience. The ages ranged from 6 to 34 years. The mean age was 23 years. Eight patients were males and two were females. Nine of the patients were managed in Firm II. The youngest patient was managed by the author while rotating in the paediatric surgery unit.

The most common type of penetrating injuries were stab wounds (7 cases) to the chest with various sharp instruments like knives. The most frequent presentation was haemopneumothorax and pneumothorax. Associated injuries included rib fractures and injury to the abdominal viscera. The initial hospital treatment consisted in tube thoracostomy alone plus antibiotics in 6 patients, exploratory laparotomy plus tube thoracostomy in 2 patients and tube thoracotomy plus primary wound closure in 2 patients. The commonest complication was post traumatic empyema in 3 patients. There was one death. The high rate of infection was attributed to prolonged thoracotomy tube time, and late presentation to hospital (more than 6 hours post injury) due to difficult means of transportation from the site of accident. This predisposed to wound sepsis.

INTRODUCTION

Chest injuries were first described in the Smith Papyrus written at around 300 B.C[1]. In 950 B.C. Homer recorded the result of various chest wounds inflicted during the siege of Troy. During the war between Biotians and Peloponnesians in 352 B.C, Epaminondas, the Biotian general, was described to

have received a precordial penetrating wound by a dart. When his physician told him that he would die as soon as the dart was drawn from his chest, he did not permit its removal until the battle was over. Then he enquired which side has won. When he was told that his army won, he said, "Now is the time to die". He then ordered the dart to be drawn out and on its removal, he died.

In the U.S., trauma is the leading cause of death in the first 4 decades of life and ranks fourth among the causes of death overall[3]. About 25% of all trauma deaths are due to chest injuries[3]. About 75% of traumatic deaths are contributed by respiratory problems[3].

All these statements are terrifying. In this country, as we approach the twenty first century, the growing magnitude of the problem of trauma and especially of chest trauma, is becoming more and more appreciable, as revealed by the increasing rate of injury and death from violence, and motor traffic accidents. Records at Muhimbili show that we admit between 15-20 cases of serious penetrating chest trauma patients requiring major surgery annually. It is very possible that the most severely injured patients (especially from the country side) never reach hospital due to poor evacuation systems for the injured. Penetrating chest injuries constitute an immediate threat to life. If they are not treated immediately or within a brief period of time death often rapidly ensues. They should thus be the concern of every practicing doctor.

CASE REPORT

J.L. was a 25 year old African male adult who was admitted to Muhimbili Medical Centre two hours after he wa stabbed in the back with a knife during a quarrel with his brother. Following the incidence he felt pain on the left side of the chest and was having difficulty in breathing. He had lost some blood from the wound but not much. There was no

history of loss of consciousness.

On examination he was found to be a young man who was well nourished. He was fully conscious and alert and was smelling of alcohol, but was not drunk apparently. He was in pain and moderately dypnoeic. He was not pale or cynosed. The blood pressure was 100/70 and the radial pulse was 94/minute regular and of good volume. There was no evidence of scalp injury. The trachea was central and the chest moved regularly and symmetrically with respiration. The respiratory rate was 36/minute. There was a transverse penetrating wound 3cm long along the 6th intercostal space on the left just lateral to the posterior axillary line. The was no active bleeding from the wound. The chest was hyperresonant on the left with reduced air entry on the same side. The heart sounds were normal. The abdomen wa scaphoid and soft without tenderness. The liver and spleen were not palpable. The external genitalia and the extremities were normal. A diagnosis of penetrating chest wound and left pneumothorax was made.

An urgent chest X-ray and haemoglobin were ordered. An intravenous line was established with normal saline to run slowly. The chest X-ray showed evidence of a pneumothorax on the left side. There was also evidence of a subdiaphragmatic gas collection on the right side. The haemoglobin was 10.5gm/dl. The patient was made aware of his injury and was requested to sign an informed consent for operation.

At operation under general anaesthesia and under aseptic technique the wound was explored. There was a 3cm transverse incisional wound involving the left chest wall and pleura along the 6th interspace just about the posterior axillary line. Part of the left lung could be visualised through the chest wound and it was normal. There was no foreign body in the pleural cavity and a minimal amount of blood was sucked from the pleural cavity. There was a 3cm long perforation of the dome of the diaphragm with the stomach herniating into the

thoracic cavity through the diaphragmatic defect. No rib fracture was detected. There was no injury to the stomach. Through the diaphragmatic wound the spleen and transverse colon were inspected and were found normal. There was no haemoperitoneum or soiling of the abdomen. The stomach was reduced back into the abdomen and the diaphragmatic wound was repaired in two layers using silk number one. Wound debridement was done to the chest wall wound and it was closed primarily after sucking all the blood from the pleural cavity. A left tube thoracostomy was done on the ipsilateral side at the 8th interspace along the posterior axillary line it was connected to an under water seal drainage.

Post operative a control chest X-ray was done and the vital signs were monitored. The patient was kept on Inj. Ampicillin 500mg and pethidine 50mg intramuscularly 6 hrly for the pain in the first 24 hours. Breathing and coughing exercises were encouraged. After 72 hours a second control chest X-ray showed good lung expansion. The chest tube was removed. The stitches from the chest wound were removed on the 7th day postoperative. The patient was discharged home in good general condition.

At the follow up clinic 3 months after the patient was healthy with no complaints.

RESULTS

Table 1: Age and sex distribution

PATIENT	SEX	AGE
1. M.M.	M	34
2. N.M.	F	6
3. J.L.	M	25
4. O.S.	M	26
5. P.J.	M	23
6. R.H.	F	23
7. A.L.	M	17
8. C.A.	M	25
9. S.M.	M	25
10. C.M.	M	27

There were 10 patients aged between 6 and 34 years. The mean age was 23.1 years. The M/F ratio was 4:1.

Table 2: Cause of Injury and Side Affected

Cause of injury	Number of patients		
	Right	Left	Total
1. Assault:	1	6	7
.Stab wound (chest)			
.Gunshot wound chest	1	-	1
.Gunshot wound (thoracoabdominal)	-	1	1
2. Road traffic accident: (knocked by moving trolley)	-	1	1
TOTAL	2	8	10

The majority of the injuries were stab wounds on the left side following assault.

Table 3: Cause of injury and agents

Injury	Agent	Number of Patients
.Stabs	Knife	4
.Stabs	broken bottle	1
.Stabs	unidentified sharp object	2
.Gunshot	gun/bullet	2
Knocked by moving trolley	moving trolley	1

Stabs with knife constituted the majority of injuries

Table 4: Presentation

Presentation	No of patients
Haemopheumothorax	6
Pneumothorax	3
Hemothorax	1

Most patients presented with haemopneumothorax

Table 5: Associated injuries

Associated injuries	No of patients
Perforation of diaphragm	1
Rib fracture	2
Perforation of stomach	1
Bruising of skin	

Associated injuries involved chest wall structures and abdominal viscera.

Table 6: Initial Hospital Treatment

Intial Treatment given	No of patients	Outcome	
		Good	Complication
Tube thoracostomy alone + antibiotics	6	3	3
Tube thoracostomy + Primary closure + antibiotics	2	1	1
Tube thoracostomy + Laparatomy (exploratory) + antibiotics	2	1	1

Most of the injuries were managed by tube thoracostomy

Table 7: Complications and their management (Second modality)

	Complication	No of patients	Treatment	Outcome
1.	Empyema thoracis with draining wound	2	Drainage, physiotherapy, antibiotic	Good
2.	Peritonitis-> Septicaemia	1	Antibiotics	Died
3.	Residual blood + Empyema	1	Decortication	Good
4.	Recurrent haemoptysis + bullet lodged in lung	1	thoracotomy and removal of bullet from lung	Good

The one patient who died had multiple bullet wounds including a thoracoabdominal wound with severe injury to the left diaphragm and stomach and soiling of the peritoneal cavity. He died from generalised peritonitis and septicemia 8 days after injury.

DISCUSSION

Aetiology:

Seven of the injuries in this study were stabs by various agents: 4 by knife, 1 by the sharp end of a broken bottle and 2 by unidentified sharp objects (table 3). Two were gunshot bullet wounds. One patient was knocked by a fast moving trolley from the back sustaining an open lacerated chest wound with multiple rib fractures. Most of the stab wounds were sustained in a quarrel or assault (Table 2). Chest injuries can occur as a result of:(a) Road traffic accidents (b) Assault (blunt injuries, stab and gunshot wounds, blast injury) (c) Industrial and domestic accidents (falls, industrial machinery and mining accidents) or they can be (d) Iatrogenic (following thoracotomy, chest wall resection) thoracoplasty or heavy handed external cardiac massage)

In civilian practice penetrating wounds of the chest are usually single[1], and are usually caused by knives, pistols, free flying objects such as pieces of wood, spears, bricks or metal. Penetrating injuries of warfare are usually caused by high velocity bullets or shrapnel. They are usually multiple and more fatal than the former because of the extensive tissue damage caused[1,4] to the structures adjacent to the wound. The magnitude of the injury is predominantly determined by the velocity of the missile[1]. Thus,

$$K.E. = M \times \frac{(V_1^2 - V_2^2)}{2g}$$

Where M = mass, V_1 = Striking velocity

V_2 = exit velocity and g= gravity - K.E. = Kinetic Energy.

PATHOPHYSIOLOGY

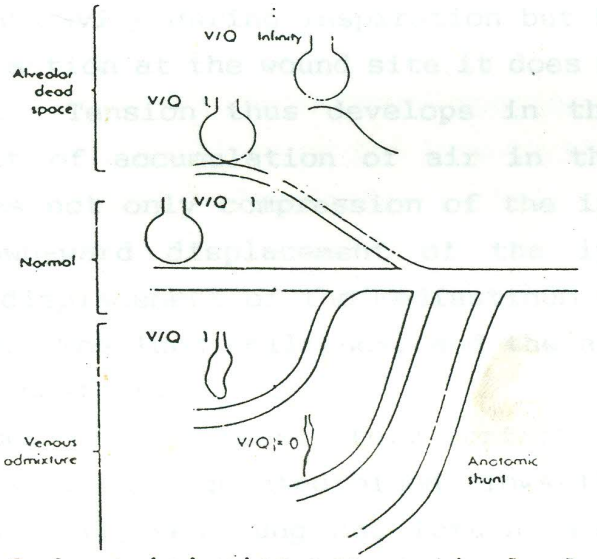
The most common traumatic problems which occur as a result of penetrating chest injuries are[5]:-

(a). Open pneumothorax (b). Tension pneumothorax (c). Cardiac tamponade (d). Massive haemothorax

Lung collapse as a result of pneumothorax or haemothorax will result in poor air distribution to the lungs leading to hypoxaemia and hypercapnia. These problems require immediate relief because they cause impaired ventilation and impaired circulation.

Gas exchange in any unit of lung tissue is determined by the ratio ventilation/perfusion or V/Q for that unit of lung[5] in the normal lung there is distribution of gas exchange which parallels the normal pattern of V/Q from apex to base. V/Q imbalance is the most common cause of reduced Pa O_2. Mismatch reduces the efficiency of gas transfer with an effect greater on O_2 than co_2 (Co_2 having a rate of diffusion 20 times greater than that of oxygen). Ventilation - perfusion inequalities of some degree occur in all normal individuals but these are not sufficient to cause significant decreases in arterial oxygen tension[5]. In pulmonary disease and traumatic injury states of the chest, the ventilation - perfusion imbalances are frequently aggravated to a degree to produce severe pulmonary inefficiency and varying degrees of hypoxaemia (Fig. 1).

Figure 1:



The consequences of chest injuries are particularly serious in patients with already impaired physiological reserves like the

aged, the very young, the obese, cardiac patients and those with chronic respiratory diseases like asthma, chronic bronchitis, emphysema.

Pneumothorax: is the accumulation of free air in the pleural space. It can be caused by either blunt or penetrating chest injuries. About 25% of penetrating chest injuries are associated with pneumothorax, according to one series[6]. Pneumothorax from penetrating chest injury can be open, simple, loculated or tension pneumothorax.

Open pneumothorax: is caused by an open sucking chest wound and occurs when there is disruption of the continuity of the chest wall in the absence of obliteration of the pleural space by adhesions. It results in a free communication of the pleural space with the atmosphere. The complications that follow are collapse of the lung on the side of injury followed by a concomitant partial collapse of the contralateral lung. The mediastinal structures are displaced toward the sound side. There may also be a mediastinal flutter.

Open tension pneumothorax: can result from a penetrating chest wound or from blunt injury. In this condition outside air enters the thoracic cavity during inspiration but because of a flap or valvular action at the wound site it does not escape during expiration. Tension thus develops in the pleural cavity as a result of accumulation of air in the pleural space. This causes not only compression of the ipsilateral lung but also down-ward displacement of the ipsilateral hemidiaphragm and displacement of the mediastinum toward the contralateral side. The lung collapses, and the alveoli and bronchiolus crowd together.

Lung collapse causes loss of respiratory surface. There is also a shunt effect as deoxygenated blood flows through the vasculature of the collapsed lung and returns to the left heart still deoxygenated. Sometimes neither air nor blood enters(6) the collapsed lung[6], so hardly any desaturated blood is shunted through it[6].

The shift of the mediastinum to the normal side during inspiration results in diminution of tidal volume (the volume of air entering and leaving the respiratory tract with each breath). Dead space air from the collapsed lung will move into the good lung on inspiration and back again to partially inflate the collapsed lung in expiration.

The resultant effect of these disturbances is to lower the PO_2, and raise the PCO_2. If the contralateral lung is normal this disordered physiology as a result of unilateral injury does not cause immediate threat to life(6).

In open pneumothorax depending on the size of the chest wall defect and the state of the underlying lung, the patient may either be free of symptoms or may be severely dyspnoeic. Small chest wall defects in patients with normal pulmonary function prior to injury and with minimal or no trauma to the lungs can be tolerated quite well for sometime. However, large chest wall defects with a diameter equal or greater than the diameter of the glottic opening of the trachea will mean that proportionately greater volumes of air will enter the thoracic cavity through the open wound during each inspiration rather than through the trachea. No gas exchange thus, takes place. A fatal outcome can rapidly result from cardiorespiratory failure. If the external opening is smaller than the glottic opening, collapse of the lung on the affected side results but no tension develops in the pneumothorax space. In this situation a proportionately greater volume of air enters the lung via the glottis and the cardiorespiratory disturbances are minimal.

Haemothorax: About 5% of cases of penetrating chest wounds are associated with hemothorax according to one series(6). Intrathoracic bleeding can be due to injury to the various organs and tissues in the chest or the chest wall. Bleeding from the pulmonary parenchyma tends to be self-limited(5). The exception is penetrating injuries near the hilar region, which may result in catastrophic haemorrhage. Continued

bleeding usually originates from injury to the chest wall (intercostal artery or internal mammary artery), the heart, the great vessels, the diaphragm or on rare occasions from an intrabdominal organ through a diaphragmatic wound(1). Haemothorax can be free or rarely loculated depending on the state of the pleural space, whether free or obliterated by adhesions. The amount and rate of the intrathoracic bleeding are dependent, on the site, size and duration of the bleeding. Haemothorax produces all the respiratory and circulatory disorders caused by pneumothorax (above). Besides, in this cases there is a depletion of the circulatory blood volume leading to diminished cardiac output as a result of the acute reduction in venous return to the heart. All these derangements contribute to the development of shock.

Haemopneumothorax: is estimated to occur in about 70% of cases of penetrating chest wounds according to one report(6).

Cardiac tamponade: can occur in penetrating chest wounds as a result of injury to the intrapericardial portions of the aorta, pulmonary artery or vena cava or in penetration of the heart itself. The presence of precordial wounds with shock should make one suspect of this condition(1). Blood is trapped in the pericardium causing compression on the heart and to the great vessels. There is a reduction of the venous return and of the cardiac output. Generalized vasoconstriction occurs which causes increase in peripheral resistance. This will maintain a near normal or even higher than normal blood pressure during the early stage of hemopericardium and tamponade, despite a progressively falling cardiac out put and a progressively rising venous pressure. This is why the blood pressure is diagnostically unreliable in this condition(5).

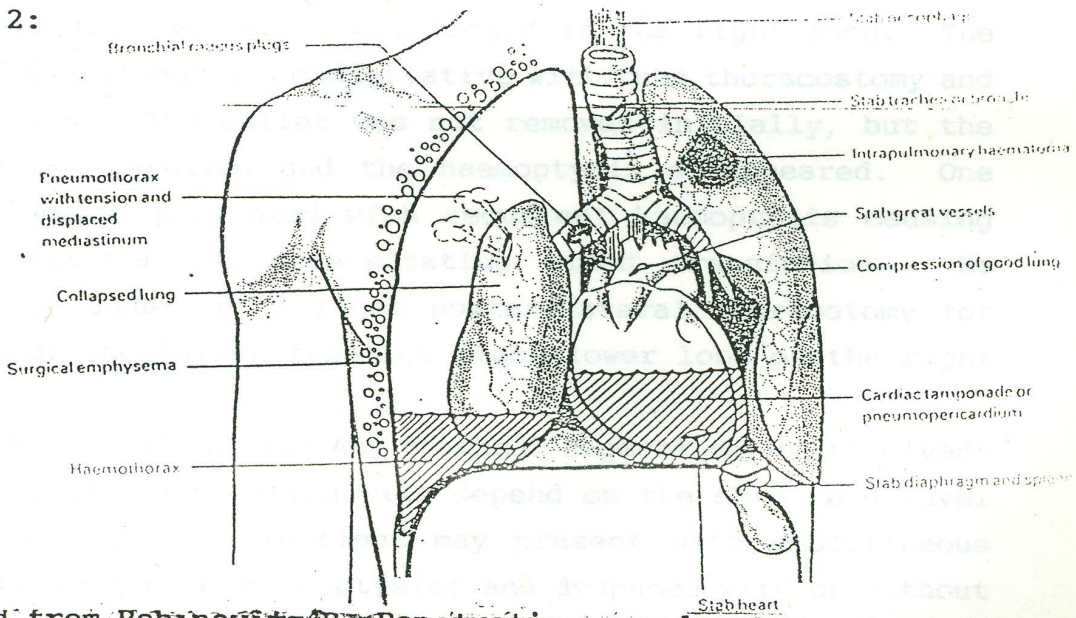
Secretional obstruction of lower airways: occurs practically in all patients who sustain serious penetrating or non penetrating chest wounds due to accumulation of secretions and blood in the lower air ways. Retained bronchial secretions

predispose to segmental and/or lobar atelectasis or massive lung collapse bringing with it further respiratory embarrassment and predisposing to pneumonic infection. Diffuse bronchospasm may also occur leading to obstructive dyspnoea[5]. Mediastinal emphysema can develop as a result of penetrating injuries to the hallow mediastinal structures like the trachea, bronchus, oesophagus. Pneumomediastinum is rarely harmful unless it originates from injury to the trachea, main stem bronchus or oesophagus. Mediastinal tamponade with serious interference with cardiac filling rarely occurs[5].

SPECIFIC INJURIES (see figure 2)

Penetrating trauma to the heart: The heart will be penetrated in about 15% of cases of stab wounds of the chest[7],[8]. Other cause are bullet wounds and missile wounds. The immediate mortality according to one report is 81%[9] and 76% in another[6]. The cause of death is usually exsanguination or cardiac tamponade. The remaining survive for varying periods of time so that many of them, if treated immediately and properly may recover[1].

Figure 2:



Adopted from Robinowitz B. Penetrating wounds of the chest in Modern Surgery in Africa.

Oesophageal injuries: Injuries to the oesophagus can be difficult to diagnose. They can be easily missed because patients are frequently asymptomatic initially. The injury usually becomes apparent 3-5 days after initial trauma[11]. Pain is the most common symptom. Its location will depend on the level of injury. Patients may present with mediastinal emphysema. Confirmation of injury can be done by endoscopy and barium swallow[1],[5]. The chest X-ray may reveal subcutaneous emphysema, widening of the mediastinal shadow (prevertebral shadow), hydrothorax or pneumothorax.

Injury to Pulmonary Parenchyma: Penetration of the lung will cause air leakage ("blowing Wound") as well as intrapleural, endobronchial and parenchymal bleeding[9]. Symptoms will depend on the amount of air or blood leaking into the pleural space and the presence or absence of adhesions between the parietal and visceral pleura. Patients with lung wounds caused by bullets will usually have haemoptysis shortly after or within a few days of injury as it was the case in one patient (case no 1) in this series. He had sustained a bullet chest wound one year previously with haemothorax and haemoptysis. The bullet was lodged in the right lung. The initial management was conservative with tube thoracostomy and antibiotics. The bullet was not removed initially, but the haemothorax resolved and the haemoptysis disappeared. One year later he presented with recurrent haemoptysis causing severe anaemia and necessitating blood transfusion. He ultimately underwent a right posterolateral thoracotomy for removal of the bullet from the right lower lobe of the right lung.

Tracheobronchial injury: Any tracheal segment may be involved. The clinical presentation will depend on the size, and level of the injury. The patient may present with subcutaneous cervical emphysema, haemoptysis, and dyspnoea with or without pneumothorax. A bronchoscopy is diagnostic. The chest X-ray will show mediastinal, cervical or whole body emphysema with

or without pneumothorax.

Injury to the Diaphragm: Diaphragmatic perforation occurs in about 13% of all stab wounds according to one series[6]. Herniation of abdominal viscera into the left pleural space occurs in about 90% in patients with severe penetrating injury according to one report[5]. This will usually lead to compression and collapse of the ipsilateral lung and a shift of the mediastinum to the contralateral side. Cardiorespiratory disturbances will vary according to the degree of lung compression, the extent of mediastinal displacemented and the nature of the diaphragmatic injury. Pulmonary re-expansion usually always follows completed reduction of the herniated contents and surgical repair of the diaphragmatic defect.

Clinical examination reveals replacement of the breath sounds by bowel sounds (if no ileus is present). The chest X-ray will show basal opacity which can be confused with pleural effusion[5] or basal consolidation. It may also resemble a pneumothorax[12]. Passage of a nasogastric tube or a barium enema may help establish the diagnosis, by demonstrating herniation of the stomach or colon[4]. Strangulation of the bowel may sometimes occur.

One patient in this study had a diaphragmatic injury. He has been presented in detail.

Thoracoabdominal and Abdominothoracic Injuries: These are wounds in which a single penetrating agent is responsible for simultaneous injury to the pleura, diaphragm and peritoneum. These types of injuries have the greatest lethal potential because of involvement of two major cavities as well as the diaphragm[5]. Abdominothoracic injuries have a worse prognosis than the thoracoabdominal injuries because in the former the penetrating agent first enters the abdominal cavity.. There is therefore a risk of soiling of the pleural cavity secondary to intrabdominal injury. The mortality rate varies between 10 and 100% depending on how much the diaphragm and the abdomen

have been wounded[5]. Most wounds are left sided in the ratio of 8:1[5] because of the face to face position of a right-handed assailant[10].

Missile wounds are especially more serious. Quite often it is difficult to predict whether or not there has been abdominal penetration with thoracoabdominal injury. About 30% patients may not present with diagnostic radiologic features, or the X-ray may reveal feature of pneumothorax, haemothorax or haemopneumothorax very similar to that of chest injuries[5]. One of the patients in this study had thoracoabdominal bullet wounds in which the left hemidiaphragm was perforated and the left crus of the diaphragm was shuttered. He also had multiple perforations of the stomach with soiling of the abdominal cavity. He died of severe generalised peritonitis and septicaemia 8 days after admission.

CLINICAL FEATURES

There were 8 males and 2 females in this study. The age range was between 6 and 34 years with a mean age of 23 years. Most patients sustaining injuries are young males[10] as was the case in this series. The most common types of injuries in this study were stab wounds following assault or quarrel. Six patients presented with haemopneumothorax, 3 had pneumothorax and 1 had haemothorax.

Gunshot wounds do also occur in civilian practice but they are uncommon[10,13]. There were two gunshot wounds in this study. Gunshot wound patients often have multiple organ injuries and few of them reach hospital alive[13].

Patients will usually present with pain with or without signs of cardiorespiratory embarrassment of varying degrees with or without shock. According to Berg et al[10] patients with penetrating chest wounds would usually present in well recognized clinical patterns:-

1. Patients who are not shocked
   . With penetrating wound in chest wall - with no

clinical signs, normal chest X-ray.

. With penetrating wound in the chest wall - with clinical signs (dull percussion note, absent breath sounds) and chest x-ray features of haemothorax and/or pneumothorax.

2. Patients who are shocked

. Hypovolaemic

- hemothorax
- extrathoracic injuries

. Cardiac tamponade

- pathognomonic triad of cardiac tamponade (Becks Triad): hypotension, raised central venous pressure (distended neck veins) and distant heart sounds.
- pulsus paradoxus (found only in 10% of patients)[6]. absence does not exclude cardiac tamponade; a raised central venous pressure may only be demonstrated when hypovolaemia is corrected.

3. Patients who are apparently dead.

. These are patients who have no discernible cardiac output but retain body heat. The clinical syndrome may be secondary to exsanguination, acute cardiac tamponade or both.

Most of the patients in this study presented to hospital in a stable condition. Only one was admitted in shock. He had sustained severe thoracoabdominal injury due to gunshot injury.

TREATMENT

Table 6 shows the initial hospital management given to the patients in this study. Six patients were managed initially by tube thoracostomy alone plus antibiotics. Two patients had

primary wound closure plus tube thoracostomy. Two had exploratory laparotomy and tube thoracostomy for thoracoabdominal wounds. The commonest complication was empyema thoracics which occurred in 4 patients.

The major objectives in the management thoracic injuries are[5]:-

(a). To restore normal cardiopulmonary function as rapidly as possible. (b). To ensure control of haemorrhage and (c). To prevent infection.

First aid management: The first aid management at the scene of the accident does not differ in principle from emergency treatment elsewhere. The standard principles of cardiopulmonary resuscitation are applied, for example by clearing the airways to ensure breathing, stopping acute blood loss by direct pressure and closure of sucking wounds by gauze or vaseline gauze taking care not to create tension pneumothorax. The patient should then be transported to hospital as soon as possible.

The initial Hospital evaluation and management: All the patients admitted with a history of penetrating chest trauma should be rapidly assessed. However the extent of initial assessment of the chest trauma victim and the time of performing the various diagnostic tests should depend on the patients physical condition[5],[10]. The airways are checked for their patency. It may be necessary to perform an endotracheal intubation. The patient is stripped for rapid re-evaluation. The site and size of external wounds are noted. The blood pressure and pulse are noted. A brief history can be taken from conscious patients enquiring about the length and width of the weapon used, and whether the patient had any loss of consciousness or has any associated injuries.

External chest wounds may appear deceptively innocuous, as the direction and depth of penetration cannot be estimated from external examination. Any ventilatory defects like open or tension pneumothorax are noted. Blood is taken for grouping

and cross-matching.

For patients in shock, resuscitation therapy for shock should be instituted through a large bore peripheral venous cannula initially with crystalloid infusions followed by whole blood where necessary and when available[9].

Some authors advocate the use of colloids rather than crystalloid solutions to reduce pulmonary oedema[4].

Pain should be relieved if present. Tension pneumothorax (evidenced by the clinical triad: absent breath sounds, mediastinal shift and respiratory distress) demands immediate insertion of a chest tube without waiting for an X-ray[13]. Otherwise it can be released by introduction into the pleural space of a large bore 14 gauge needle through the second or third interspace at the midclavicular line, or by making a rapid emergency stab incision into the chest cavity with a knife at the same site until insertion of a thoracostomy tube is possible.

Sucking wounds should be closed immediately. Most knife wounds have straight edges and can be cleaned,debrided and closed if less than 6 hours old. Most penetrating chest injuries can be managed conservatively[10]. Some patients require no treatment other than observation for delayed pneumothorax or cardiac tamponade which may occur[10]. All wounds that penetrate the chest and result into massive pneumothorax or haemothorax, require tube thoracostomy to drain the blood or gas and to re-expand the lung and allow any leaks to heal. Any intercostal space can be used to perform tube thoracostomy[5]. The site of puncture is determined by the physical findings and chest X-ray. The chest X-ray should be taken in the erect position as up to 1000ml of blood may be missed on a supine film[13]. Even on an erect film a collection of 300-500ml may be hidden by the dome of the diaphragm[13].

The thoracostomy tube can be inserted in any of the intercostal spaces 3, 4, 5, 6, 7 in the mid axillary

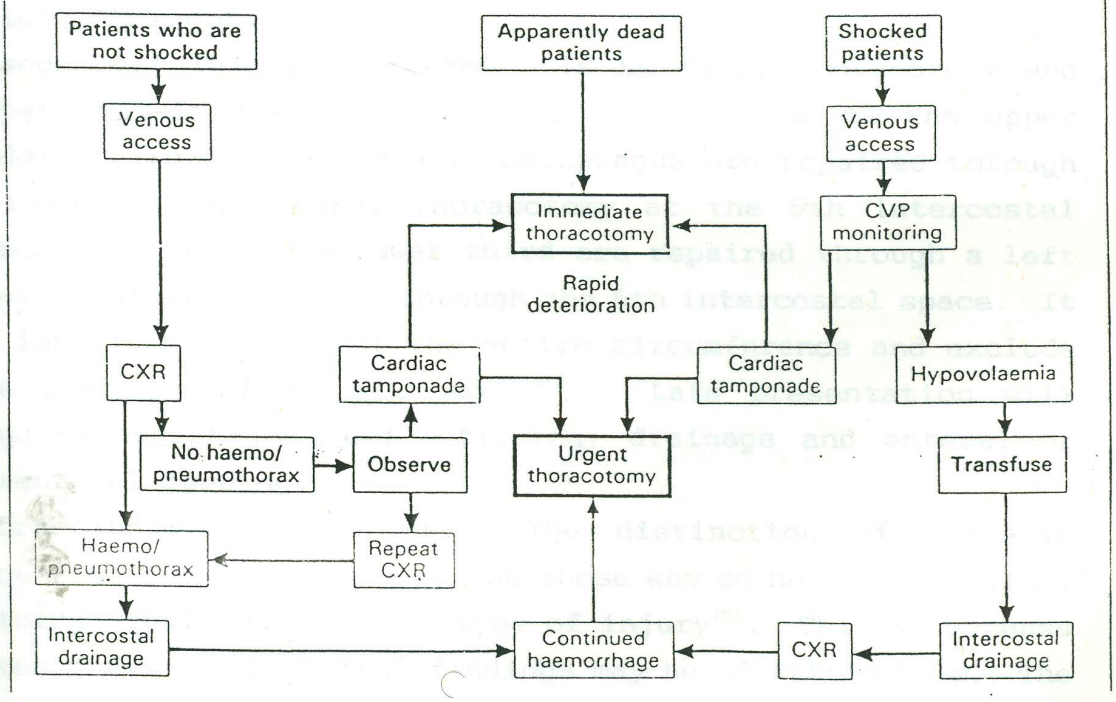
line[1],[5],[6] and is connected to an underwater seal drainage. This should be done under aseptic technique under local anaesthesia with the patient in the sitting or the supine position. This should drain both blood and air and is effective in about 85% of the cases[2].

The haemothorax must be completely evacuated through the tube. Subsequent blood loss can be assessed. Prophylactic broad spectrum antibiotics should be given as this reduces the incidence of infective complications and the period of hospitalization[13].

The indications for thoracotomy include:[13] continued bleeding 200-300ml in the first hour or undiminishing bleeding in continuous hours, uncontrolled massive air leaks, suspicion of injury to a major viscus: centrally situated wound or long - blades knife wound and a clotted haemothorax.

The following is a good flow diagram proposed by Berg et al[10] showing the management of penetrating chest wounds.

Figure 3:



Adopted from G.A. Berg, Hospital update Nov. 1987 pp 950.
CXR = Chest X-ray

TREATMENT OF SPECIFIC ORGAN INJURIES

Penetrating Injury to the Heart: The definitive management of penetrating injuries to the heart consists in resuscitation as in any other severely injured patient and emergency repair is performed through a left lateral thoracotomy. In patients with suspected cardiac tamponade, pericardiocentesis or subxiphoid pericardiotomy through an upper epigastric incision should be performed. However, surgery is the only effective and definitive treatment for penetrating cardiac wounds[1].

Tracheobronchial Injury: Confirmation of diagnosis is done by bronchoscopy. Treatment will depend on the type, size and site of the wound[5]. Small wounds without loss of tracheal tissue can be managed by orotracheal or nasotracheal intubation[1]. Large wounds should be repaired primarily as early as possible under general anaesthesia. Wounds of the thoracic segment and right bronchi are repaired through a right thoracotomy. Those of the left bronchi are done through a left thoracotomy[1].

Oesophageal Injury: Treatment is by early thoracotomy and repair within the first few hours. Injuries of the upper middle third of the thoracic oesophagus are repaired through a right posterolateral thoracotomy at the 5th intercostal space. Those of the lower third are repaired through a left posterolateral incision through the 6th intercostal space. It is important to inspect the entire circumference and exclude the presence of an exit wound[1]. Late presentation will require oesophageal defunctioning, drainage and enteral or parenteral feeding.

Thoracoabdominal Injuries: The distinction of patients requiring immediate surgery and those who do not is a constant clinical challenge in this type of injury[5]. This is because often times the abdominal findings may be of little help. The characteristic signs of peritoneal irritation are also present in patients with thoracic injuries without associated damage

to the intrabdominal structures. Sometimes the initial abdominal findings may be minimal or absent, or may appear 6-12 hours after injury. This clinical picture may be complicated by the presence of severe prostration, shock, toxicity or altered states of consciousness by the effects of alcohol or drugs[5],[6].

Treatment is by exploratory thoracotomy and/or laparotomy in all patients in whom there is strong suspicion of thoracoabdominal penetration even in the absence of positive clinical evidence of intrabdominal injury[5]. It is advised that when there are specific indications for exploratory thoracotomy, this procedure takes precedence over abdominal exploration. It is easy for one to approach thoracic injuries and wounds involving the diaphragm, spleen, stomach, transverse colon and the dome of the liver through the thoracotomy incision. Sometimes one finds it necessary to do a primary thoracotomy and to supplement it with a laparotomy if necessary. It is advised to do laparotomy preferably through a transverse incision when it is believed that abdominal exploration cannot adequately be performed through transthoracic, transdiaphragmatic incision.

There were two patients with thoracoabdominal injuries in the study, both of them following gunshot wounds. One of them had multiple gunshot wounds and multiple perforations to the stomach with soiling of the abdomen. Both of them underwent exploratory laparotomy. One of them died of generalised, peritonitis and septicaemia.

Diaphragmatic lacerations are best managed with interrupted mattress type suture using nonabsorbable material. There was one patient in this study who had a stab wound to the chest with injury to the left diaphragm, with herniation of the stomach into the thoracic cavity.

Complications of tube thoracostomy:

The following are the most common complications of tube thoracostomy reported in various series.

1. Post traumatic empyema: This complication was reported to occur in 10 - 25% of patients during World War II and the Korean War(14). There was a fall (6%) in the Vietnam War. The incidence of empyema in civilian practice is usually lower than in warfare practice, this probably reflecting the difference in civilian and military weapons.

   Nosocomial colonization of the respiratory tract in multiply injured patients and contamination at the time of tube thoracostomy have been mentioned as possible causes[14]. Graham and associates[14] reported a low incidence of 1.6% while Millikan and coauthors[14] reviewing 1,249 patients with predominantly penetrating chest trauma found the incidence of empyema to be 2.4%. They suggested that adequate drainage and prompt pulmonary re-expansion were important in preventing pleural infection. Others[14] have suggested that prophylactic antibiotics were important in reducing infectious complications following penetrating chest injuries although not all such studies have been conclusive. Infectious complication were quite high in this series (4/10 cases). This could be attributed to the severity of injury, late presentation to hospital (> 6 hours post injury) due to difficulty in getting transportation, and also due to prolonged tube-thoracostomy time.

2. Incomplete evacuation of infected clotted blood by tube thoracostomy (18%) has on occasions necessitated decortication for complete lung expansion or elimination of infection. In this series one patient had to undergo decortication following empyema formation from the first thoracostomy.

3. Recurrent pneumothorax following tube thoracostomy removal has been reported in about 24% of cases with penetration chest injuries[15].

Prognosis

At 3 months follow up 7 of the patients were in good general condition and had returned to their jobs. Two were lost to follow up.

CONCLUSION

A good percentage of penetrating chest injuries usually constitute surgical emergencies and must always be treated as such. With the current increase in violence, motor traffic accidents, and work accidents it is impossible to imagine that a hospital like Muhimbili catering for a city of more than 3 million people could admit only 20 patients with serious penetrating chest injuries in a year. With the present means of communication and transportation in this country it is likely and indeed possible that a good number of the serious penetrating chest trauma patients never reach our hospitals before they are dead. It is stressed that there is need to improve our ambulance and telephone services.

Improvement of communication and transportation will automatically mean that more and more severely injured chest trauma patients will reach the centre for treatment. Much of the success or failure of the definitive surgical management of pepetrating chest trauma patients depends on the previous initial management. There is need to improve our Casualty and Emergency Department at Muhimbili so that the necessary life saving and resuscitative procedures like pericardiocentesis for cardiac tamponade, thoracotomy for internal cardiac massage for severely shocked or apparently dead patients, can be performed pending major surgery in the operating theater. As for the practicing doctor in the up country hospital, mastery of chest tube insertion under aseptic technique is a simple but crucial procedure worth knowing, because most chest trauma patients can be managed by tube thoracostomy alone.

REFERENCES

1. Symbas PN. Cardiothoracic trauma in current problems in Surgery. 28(11):747-792, 1991

2. Oreshovich, MR. and Carico J. Thoracic Injuries in Sabiston D.C. Texbook of Surgery. 13th edition. W.B. Saunders & Co. pp300-306, 1986.

3. Shwartz S.I. Principles of Surgery. Mc Graw Hill Co. 5th Edt. 1989.

4. Locke T., Smith G. Thoracic Trauma. Surgery 6:1650-6, 1989.

5. Naclerio EA. Chest injuries. Physiologic Principles and Emergency Management. Grune and Stratton. New York 1971.

6. Robinowitz B. Penetrating wounds of the chest in Modern Surgery in Africa. The Baragwanath Experience by D. Pantanowitz.

7. Oparah SS, Mandal AK. Operative management of penetrating wounds of the chest in Civilian practice: Review of indications in 125 consecutive patients. J. thoracic Cardiovascular Surg. 77:162-88, 1979.

8. Elkin D.C. Diagnosis of and treatment of wounds of the heart. Review of 22 cases. JAMA 111:1750, 1938.

9. Platt MR. Penetrating chest wounds in Trauma of the chest. Edit. by William WG. Bristol: John Wright & Sons Ltd pp. 227-37, 1977.

10. Berg GA, Kirk AJB, and Reece I.J. Management of Penetrating chest wounds, Hosp. Update, pp.949-961 Nov. 1987.

11. Cummings, RG. Intensive Care Management of the trauma Patient in the Handbook of Surgical Intensive Care. Year Book Med Publ. pp 473-486, 1989.

12. Hurt RL. Thoracic trauma. Surgegy 16:381-87, 1985.

13. Moussall H. and Hooper T. Management of Chest Injuries: 1. Hosp Update pp 751-760, Oct. 1986.

14. Caplan ES., Hoyt NJ., Rodriquez A. et al: Empyema occurring in multiple traumatized patient. J. Trauma 24: 785-789, 1984.

15. Helling TS, Cyles NR., Eisenstein CL., Soracco CA. Complications following Blunt and Pen. Injuries in 216 Victims of Chest Trauma Requiring Tube Thoracostomy. J. Trauma 29(10): 1367-70, 1989.

BURNS IN CHILDREN

Summary

Among the 705 admissions to the paediatric surgery unit in the 4 months period between May and August 1992, 55 patients were admitted due to burns of various causes and degrees. This consituted approximately 8% of the total number of admissions to this unit during that period. Twenty nine of these patients were males while 26 were females. The M/F ratio was 1.1:1. The age range was from 2 months to 10 years (120months). The peak range was between 12 and 60 months. Eight percent (80%) of the patients were 5 years and below. Domestic accidents were the major causes of burns in 54 patients (98%). One was involved in a motor traffic accident. The majority of the burns (75%) were partial thickness burns. Scalds were the commonest type of burns in 40 patients (72.3%). The extent of burns was between 5 and 60%. Patients with less than 10% burns were admitted because the burns involved priority areas or had other associated disorders. Thirty five patients reported to hospital within the first 6 hours postburn. Twelve patients presented more than 36 hours post burn. The commonest complications were wound sepsis and shock. There were 7 deaths. All the patients were managed by the expossure method. Patients with 10% burns or more received intravenous resuscitation therapy and nutritional support.

INTRODUCTION

Burns in children constitute a major health concern[1,2] not only in Tanzania but throughout the world. Despite all this burns are still one of the worst and the least expertly managed type of injury[2]. In the developed countries extensive acute burn injury would optimally be managed in special burns units by trained personell and plastic surgeons. In developing countries like Tanzania due to

meagre resources and shortage of trained personell, the management of burns in children leaves a lot to be desired. However, though more efforts are required to improve the situation many more lives can be saved and misery reduced by using the available resources judiciously.

This study was carried out in order to present the magnitude of the problem at the centre, to analyse the aspects of management, especially the fluid resuscitation therapy and wound care in our environment, particularly during the early critical hours of burn injury.

CASE REPORT

H.K. was an apparently healthy 4 years old female who sustained burns 2 hours prior to admission to the Muhimbili Medical Centre. She was wearing a "khanga" which cought fire as she was passing near a kerosine lamp (kibatari). She sustained burns to the chin, lower lip, cheeks, right ear, upper limbs, left lower leg and the left side of the abdomen. The mother who was in the other room rushed to the child and quickly removed the burning khanga off the patients body. The patient was taken to the nearby district hospital from where she was referred to the centre. The patient was the third born from a family of 3 children. All were alive and well. There was no family history of epilepsy and this was her first admission. The father worked at a local butchery while the mother was a housewife. They lived together in one of the suburbs of Dar es Salaam city. The patient had had normal milestones and had completed her immunisation programme.

On examination the patient was fully conscious and in pain. The airways were clear. She was restless but not pale. The pulse was regular but weak with a rate of 120 beats/minute. The respiratory rate was 58/minute. The extremities were cold. She had partial thikness burns with blisterring over the chin, lower lip, cheeks, right ear,

upper limbs left leg and on the left side of the abdomen. Pin prick test revealed a very sensitive burn area. The respiratory and cardiovascular systems were normal. The abdomen was soft without any palpable abnormal masses. Using Evans chart the extent of the burn was calculaated to be 25%. The diagnosis made was partial thickness burns 25%.

A blood sample was immediately taken by venopuncture from the right cubital vein for baseline investigations estimation of the haematocrit, serum electrolytes, blood urea and serum creatinine.
The patient was weighed and found to have 16kg. Using a number 20 gauge cannula an intravenous line was established though the right cubital vein. Fluid resuscitation therapy was started using Ringer's lactate solution. The fluid requirement was calculated according to Parklands formula which provides for fluid replacement plus the normal metabolic fluid requirements. Ringers Lactate was given at 4ml/kg body weight/percentage burn for the first 24 hours. thus 4 x 16 x 25 = 1600mls/24 hours. 800mls of this fluid was given in the first 8 hours post burn, then 400mls were given in the following 8 hours and the remaining 400mls was given in the last 8 hours. For the second 24 hours 1200mls of fluid was given in the form of Dextrose 5% in 1/2 normal saline. An injection of pethidine 16mg was given intramuscularly stat for the pain. Thereafter 16mg of promethazine was given orally 8 hourly for the remaining 16 hours.

The burn wounds were then cleaned and debrided, removing the devitalized skin and debris with a dilute solution of savlon leaving the blisters intact. Pieces of sufratulle dressing were applied over the wounds. The patient was placed on clean, bed sheets under a craddle and a mosquito net. A booster dose injection of tetanus toxoid was given intramuscularly 0.5ml. stat. Meanwhile the investigation results were reported:

Sodium - 134 mmol/l
Potasium - 3.6 mmol/l
Chloride 98 mmol/l
Serum creatinine - 132 mmol/l
Blood urea - 35 mg/dl
Haematocrit - 40

The patient was allowed to take oral fluids as tolerated. the progress of the patient was carefully monitored every 4 hours noting in particular, the general condition of the patient, hydration status, the pulse and the urine output. The urine output was noted to be 250mls in the first 16 hours. The lungs were also checked to detect any signs of fluid overload or chest infection. By the end of the first 24 hours her vital signs were stable, her general condition was good and she was taking fluids orally. The urine output was 600mls ater the first 24 hours. The intravenous drip was discontinued after 36 hours. A high calorie and high protein diet was given. Daily antiseptic bath was given in lukewarm water followed by daily application of sufratulle dressing over the wounds.

Early physiotherapy was started to the left elbow and knee joints to prevent contractures. By the 25th day of admission H.K's condition had improved tremendously, the wounds had virtually healed. She was ambulant and had gained weight. she was discharged home in satisfactory condition.

Before discharge a strong point was made to the mother insisting that most fire accidents like this one were preventable if only parents like herself became more responsible to their children. They should be more careful of their children especially in their cooking premises.

The patient was seen again at the outpatient clinic four weeks after. Her general condition was good. She had gained weight, the wounds had healed with areas of minimal scarring but no contractures. She was happy.

RESULTS

Table 1: Age distribution

Age in months	No of patients	Percentage (appr.)
0 - 12	9	16.4
13 - 24	18	32.7
25 - 36	6	10.9
37 - 48	6	10.9
49 - 60	5	9.1
61 - 72	3	5.5
73 - 84	3	5.5
85 - 96	3	5.5
97 - 108	1	1.8
109 - 120	2	3.6
Total	55	100%

The age range was between 2 months and 120 months (10 years) The peak range was 12-60 months. 80% of the patients were below 5 years.

Table 2: Sex distribution

Sex	Number of patients
Males	29
Females	26
Total	55

The male: female ratio was approximately 1:1.

Table 3: Types of burns

Type of burn	No of patients	Percentage (approx.)
Scald	40	72.3
Flame	13	23.6
Electrical	1	1.8
Chemical	1	1.8
Total	55	100%

Table 4: Causes of burn injuries

Causes of burn injury	No of patients	Percentage
Domestic	54	98.2
Motor traffic accident	1	0.8
Assault	-	-
Industrial accident	55	100%

The major cause of burn was domestic accidents.

Table 5: Time elapsed from burn injury to the time of presentation to hospital

Time in hours	No of patients	Percentage (approx.)
0 - 6	35	63.6
7 - 12	2	3.6
13 - 18	1	1.8
19 - 24	1	1.8
25 - 30	1	1.8
31 - 36	1	1.8
> - 36	12	22.0
Total	55	100%

Most patients presented to hospital between 0-6 hours postburn (64%). Twenty patients (36%) presented to hospital more than 6 hours post burn. Twelve partients presented more than 36 hours post burn (22%).

Table 6: Distribution of burn "size"

Extent of body surface area burnt (%)	No of patients	Percentage (approx.)
0 - 10	10	18.2
11 - 20	26	47.3
21 - 30	8	14.6
31 - 40	6	10.9
41 - 50	4	7.3
51 - 60	1	1.8
Total	55	100%

The majority of the patients had burns extent of between 10 - 30%. Patients with less than 10% were admitted because they involved priority areas or had associated diseases.

Table 7: Distribution of burn depth

Degree of burn	No of patients	Percentage (appr.)
Partial thickness	41	74.5
Full thickness	2	3.6
Mixed	12	21.8
Total	55	100%

Most of the patients had partial thickness burns.

Table 8: Complication in burn patients

Complication	No of patients	Percentage (appr.)
Wound sepsis	20	36.4
Shock	5	9.1
Contractures	2	3.6
Curlinng's ulcer	1	1.8
Palalytic ileus	1	1.8
Tourniguet effect	1	1.8
Septicemia	4	7.3

Wound sepsis was the commonest complication.

Table 9: Organisms isolated from infected burn wounds in the 20 patients.

Organisms isolated	No of patients
Staphylococcus aureus	13
E.Coli	3
Pseudomonas	2
Klebsiella	2

The commonest microrganism isolated was staphilococcus aureus. They were sensitive to cloxacillin and erythromycin.

Table 10: Analysis of the 7 deaths

Patients No.	Age	Sex	Type of Burn	% BSA	Depth of Burn	Time of Presentation	Cause of death	Time of death
1. S.A.	14	M	Scald	20	Partical thickness	35hrs	Shock, B/S+	12hrs
2. M.K.	72	M	Flame	60	Mixed	1hr	Septicemia	12th day
3. A.S.	2	M	Flame	30	Mixed	40hrs	Septicemia	22nd day
4. S.K.	108	F	Flame	50	Mixed	3hrs	Septicemia	20th day
5. J.R.	36	M	Scald	33	Mixed	35hrs	Septicemia	10th day
6. A.A.	24	M	Flame	35	Mixed	2hrs	Shock	1st day
7. M.K.	23	F	Scald	34	Partical thickness	12hrs	Shock	2nd day

CONCLUSION

The contention that burns are a common occurrence and yet one of the worst and least expertly managed condition resulting in immense iatrogenic misery and loss of life is a bitter fact to swallow. It consititutes a major health problem that needs to be addressed to in its proper degree of gravity.

Most burns can be prevented if due care is taken. While efforts to treat the burned patient are sustained, much more attention should be directed towards public health education on how to prevent burns; and how to give first aid to a burned patient.

Mothers, adults should be educated on how to prevent burns accidents in their homes. "Such burn education could be incorporated into the MCH programmes for mothers. While the establishment of burns units as it is in the developed countries provided the ideal conditions for the management of extensive burns, the meagre resources make this possibility a far fetched ideal. Many more lives can be saved and missery reduced by using the available resources judiciously.

REFERENCES

1. Curreri W.P., Luttermann A., Burns in Principles of surgery.Schwartz S.I. 4th edit. McGraw-Hill Edit. pp. 269-87, 1985.

2. Artz C.P., Yarbrough D.R. Burns in Davis - Christopher Textbook of surgery 11th Ed. Philadelphia, pp 295, 1977.

3. Shija J.K. Burn injuries in Paediatric surgical care for developing countries Publ. Heinemann, Kenya pp 63-64, 1989.

4. Swenson O. Burns in Pediatric Surgery 3rd Ed. Publ. Appleton - Century-Crafts pp. 997,1969.

5. Loefler I.J.P. Burns symposium Proceedings of the Assoc. of Surgeons of E.A. 1:38, 1978.

6. Sharma B.C. Postburn contractures in Children. Proceedings of Assoc. of Surg. of E.A. 3:172, 1980.

7. Shija J.K., Omar O.S. Trauma as a Major Paediatric Surgicalproblem in Dar es Salaam. Proceedings of the ass. of Surg E.A. 3:169, 1980.

8. Kakonde I. Thermal injuries in Mulago Hospital, Kampala, E.A. Med. J. 55: 236-41, 1978.

9. Blocker T.G., Wash Burn W.W. Lewis S.R., Blocker V. A statistical study of 1000 burns patients admitted to the plastic surgery service of the University of Texas medical Branch. J. Trauma 1: 409, 1961.

10. Sinha P. Epidemiology of burns. Proceedings of the Assoc. of Surgeons in E.A. 1: 40, 1978.

11. Bailey H., Love M. Burns and Scalds in Bailey and Loves Short Practice of surgery 20th Ed. English Lang. Book Soc. pp 147, 1989.

12. Jacson D.G. Burns in Hamilton. Bailey Emergency Surgery HAF Dudley (ed) 10th ed. 75:91, 1984.

13. Trunkey D.D. Hunt T.R.B. and Way L.w., Burns and other thermal injuries in Current Diagnosis and Treatment - 5th ed. Chief Edit. Dunphy J.E. Publ. Lange Med Publ. 17 pp. 203 - 211,1981.

14. Blank I.H. The skin as an organ of protection against the external envirnment. Dermatol 102-106, 1979.

15. Robbins S.L., Cotran R.S. Kumar V. Thermal burns in Pathologic Basis of Disease 3rd Ed. W.B. Saunders Co. pp. 462, 1984.

16. Kemble J.V.H. the early Management of burns. Surgery 1(3):52-56, 1983.

17. Parks D.H., Carvajal H.F., Larson d.L. Management of burns. Surg Cl N.A. 57(5): 875-894, 1977.

18. Moncrief J.A. Burns. New eng. J. Med. 288(9): 444, 1973.

19. Pruitt B.A. Fluid and electrolyte replacement in the burned patient Surg Cl. N.A. 58(6): 1291, 1978.

20. Daynes R.A. What is the Relationship between the status of apatient - host defense mechanism, his Metabolic Response and his Ability to Respond to Injury? J. Trauma 24(9) 84-100, 1992.

21. Curreri P.W., Luttermann a. Nutritional support of the burned patient Surg. Clin. N.A. 58(6): 1151, 1978.

22. Wilmore D.W., Avuck L.H. Metabolic Changes in burned patients. Surg. Cl. N.a. 58(6): 1173, 1978.

23. Principles of Burns Management - Burns symposium held in Lusaka, Zambia Medicine Digest 3(3): 6-7, Dec. 1977.

24. Smith I. Acute Management of thermal burns in children. The Surg. Cl. N.A. 50(4): 807-814, 1970.

25. Udekwu F.A.O. Letters to the editor on Burns symposium held in Lusaka Medicine digest 4(8): 64 Oct. 1978.

26. Clarke J.A. The treatment of burns Postgrad. Doctor (Afr.) 5 (3): 111-119, 1983.

TUBERCULOSIS OF THE SPINE IN CHILDREN

SUMMARY

Six children with tuberculosis of the spine were admitted to the paediatric surgery unit between May and October 1992. Their ages ranged from 3 to 10 years. Four were males and two were females. The M/F ratio was 2:1. The commonest mode of presentation was back pain, back deformity, and weight loss. One of the patients was paraplegic. The duration of symptoms was between 1 and 12 months with an average of 7.3 months. Four patients had a positive BCG scar. Two vertebral involvement was found in 3 patients. One patient had 3 vertebrae involved. The most frequently involved vertebra was T_8. All the patients were managed conservatively with good response. Recovery was slow in the paraplegic patient.

In this study one of the patients is presented in detail and the results of the study are analyzed in a tabular form and discussed.

INTRODUCTION

Tuberculosis still remains one of the major health problems in the developing countries[1] like Tanzania. In these countries tuberculosis of the spine is essentially a disease of childhood and adolescence[2]. This is exemplified by data from the Medical Research Council (MRC) series where in Korea (MRC 1973a), 78% of the patients were under the age of 10 years. In Hong Kong 59% were under 25 years[3]. In Konstam's series of 210 cases of tuberculosis of the spine 65% were children[4]. In these countries tuberculosis of the Osteo-articular system accounts for the highest incidence of crippledom among children[3]. When neglected tuberculosis of the spine can lead to paraplegia. It affects the cord leading to paraplegia in between 10 and 25% of cases[2,5,6,7]. It is the commonest cause of non traumatic paraplegia in

developing countries accounting for over 50% of non traumatic paraplegia[5]. Such patients spend many weeks and months in hospital. Jones,[3] analyzing his collection of 276 cases of spinal tuberculosis among Nigerians who attended Igbobi Hospital between January 1949 and June 1955 found that of these, 115 at one time or another suffered from paraplegia and paraparesis. Between 1963 and 1964 there were respectively 31 and 37 admissions due to tuberculosis of the spine at Muhimbili, Dar es Salaam[8].
The advent of potent chemotherapy has altered the course of the disease in general to a great extent but its morbidity and mortality is still high even after prolonged treatment.

CASE REPORT

A.N. was a 3 years old female child who presented to hospital with a progressive painful swelling at the back for 6 months duration. The pain had increased gradually and she had become unable to walk properly for the past one month. She could stand up normally but the deformity on the back and the pain made walking difficult. She was not gaining weight and occasionally she had fever in the evening for the previous one month. She had no cough but a year earlier she was admitted to the paediatric ward for one week due to cough and fever. She denied any history of trauma or night sweats. She denied any tuberculosis contact. The child was able to pass urine and was continent of stool. Her mother died earlier when she was 1.5 years old and she was under the care of grand mother who had brought the patient to hospital.

On examination she was found to be a young girl who was afebrile, moderately wasted and pale. She had a BCG vaccination scar on the right deltoid area. The body weight was 11kg. The pulse was 100 beats/minute and regular. She had no oedema. She had discrete palpable mobile, painless lymph nodes in the cervical, axilla, and inguinal area. The

respiratory, cardiovascular systems were normal. The abdominal examination was normal. The liver and spleen were not palpable. She was fully conscious, alert and well orientated. She walked in a stooping position with an antalgic gait. The cranial nerves were normal. The upper limbs had normal power and tone. The sensation and tendon reflexes were normal. She had a kyphosis in the lower thoracic spine with a tender area. No fluctuation or sinuses were detected. The lower limbs were not wasted but she had reduced muscle power in the lower limbs (grade 4) with normal sensation. The knee and ankle jerk reflexes were brisk but with no clonus. The Babinski sign was absent. Rectal examination revealed good anal sphincter tone.

A provisional diagnosis of vertebral tuberculosis was made. Investigations done revealed the following results:-

Full blood picture: Hb = 8.7gm/dl, ESR = 80mm/first hour(Westergren)
WBC- Total = 11,000. Differentials: Neutrophils = 23%, Lymphocytes = 69%, Monocytes = 5%, Eosinophils =1%, Basophils = 2%.

The plain chest X-ray was normal. The Spine X-ray - Anteroposterior and lateral views of the thoracic spine showed destructive lesion and collapse of the 12th thoracic vertebra and a destructive lesion of the first lumbar vertebra with evidence of fusiform paravertebral shadow around T_{12} and L_1.
The patient was started on antituberculous chemotherapy - namely as a combination of intramuscular injections of streptomycin 30mg/kg/day and thiazina tablets, 15mg/kg/day.

At the end of the first 2 months of chemotherapy there was considerable improvement. The pain had subsided, the patient could walk without support, she had gained weight 0.5 kilogram. She had regained power to the lower limbs and

the knee and ankle reflexes were normal. The control spine X-ray showed considerable resorption of the paravertebral abscess. After completing 60 injection, of streptomycin the patient was allowed home to continue with thiazina tablets daily for a further 16 months.
At the follow up clinic 3 months after discharge, the patient was ambulant, had gained weight and was doing well.

DISCUSSION

Incidence

Tuberculosis in general is still a world wide problem. The WHO statistics report of 1977 estimated it to be among the commonest communicable disease of priority in the world[9]. According to this report tuberculosis causes about 3 million deaths/year, causes 4-5 million contagious new cases/year plus 4-5 million other cases of less serious or less contagious forms of tuberculosis.

In Africa it ranks among the 13 priority communicable diseases[9]. The annual tuberculosis infection rate is between 2 and 5% in 18 countries compared to 1-3% in the developed world. The estimated incidence of new infectious cases in most parts of Africa and Asia is 200-350 cases/100,000 population.

In Tanzania, the exact magnitude is not known. But according to some tuberculin surveys so far done in Tanzania the annual tuberculosis infection rate ranges between 0.76% - 2.13%. (Newala, Masasi, Mtwara).

AETIOLOGY

Girdlestone and Somerville[10] have reported that before the era of pasteurisation bovine tuberculosis (M.bovis) accounted for the majority of cases of tubrculosis of the spine in children below 10 years. Now the human type M. Tuberculosis is by far the most important of the mycobacterial pathogens accounting for almost all cases of

tuberculosis[10]. In this study tubercle bacilli were not isolated from the sputum specimen taken. Negative specimen do not rule out tuberculosis. Needle biopsy from the spinal lesions were not done.

PATHOLOGY

Tuberculosis of bones including the spine is usually a metastatic manifestation of a primary focus. This focus may be known or unknown, active or latent either in the lungs or in the lymph nodes, in the gastrointestinal tract or in the genitourinary tract. In more than 50% of cases the primary focus is intrathoracic[3]. In a large number of cases active pulmonary lesions are detected. Collins[11] found that 80% of children with tuberculosis of the spine had an intrathoracic lesion. Similar observations were made by Falk[12]. One of our patient had radiological evidence of active pulmonary tuberculosis though no organisms were detected on the sputum smear. One patient had generalized lymphadenopathy which was histologically proved to be tuberculous.

Haematogeneous spread occurs from the primary focus usually whenever there is a lowering of the patients resistance, to give rise to secondary skeletal lesions.

Tuberculosis of the spine can affect any vertebral body[3]. The highest incidence is however found in the lower thoracic and thoraco-lumbar regions. This observation compares with the results from this small study. Almost all the lesions in this study were localized in the thoracic spine, only one patient had a lesion of T_{12} and L_1. No conclusive explanation has been given for this particular predisposition of this area of the spine apart from the fact that this area is prone to twists and strains and minor trauma which may help the organisms to lodge[3]. Tuberculosis of the cervical and sacral segments are relatively rare. One or more verterbral bodies may be involved or there may

be a generalized tuberculosis of the spine[7]. According to Kemp[2] tuberculosis of the spine is essentially a two body disease, 70% of the cases having 2 vertebral bodies involved and 3 or more bodies in 20% of cases[2]. We obtained comparable results from our study group. Half of the patients in this series (3 out of six) had 2 segments involved. One patient had involvement of 3 segments (table 5).

Invariably tuberculosis of the spine is a combination of tuberculosis arthritis and osteomyelitis. Through the haematogenous route the organisms reach the joint tissue or the spongiosa of the vertebral body. The perivertebral plexus of veins may also play a role in the haemategenous spread of tuberculosis[7].

Usually the organisms spread from one involved area to an intact area beneath the anterior longitudinal ligaments, but they may also advance directly by necrosis of one vertebral body to the next. Sometimes however a normal vertebral body or joint may intervene between two foci of infection. The tubercle bacilli would lodge on to the subchondral bone, the joint capsule or the synovial membrane, a process which is usually preceded by bacillaemia.

According to Girdlestone[6] the original foci may be anywhere in the vertebral body, but in children are most often close to the upper or lower epiphyseal plate[7]. Ordinarily the infection would commence at the anterior superior or inferior margin of the vertebral body near the intervertebral disc. The focus of infection is then infiltrated by plasma cells, lymphocytes, and monocytes with formation of Langerhans giant cells. The infection develops slowly and tubercles are formed in the marrow and they grow by expansion and coalescence. This is followed by destruction of the body by lysis of bone, infarction and necrosis. This mechanism of bone destruction by ischaemic

necrosis has been described by Cleveland and Bosworth[7] and also by Girdlestone.

Following these processes bone destruction occurs (usually starting on the anterior aspect of the vertebral body). The destructive process is faster in the case of a child than in an adult and is greater in the vertebral body whose major function is to transmit body weight. Collapse and deformity will therefore occur on the anterior elements of the vertebral body. The integrity of the arches and the interarticular processes prevent similar collapse posteriorly. The spinal canal is therefore usually preserved[7]. It is however possible for the process to extend posteriorly to involve the pedicles or the articular facets[2]. When it does the spine becomes dangerously unstable. This situation is found in approximately 5% of cases[2]. As a result of the destruction of the discs and bodies there is shortening of the trunk by collapse of the vertebra and discs and by interfering of the growth centres. Severe distortion of the normal spinal curves and alignment occurs. Necrosis at the anterior portion of the vertebra produces a kyphotic angulation or kyphosis. The normal antero-posterior curves increase to compensate for the angulation.

The earliest visible X-ray changes are the diminution of the joint space. This is not due to destruction of the disc but it occurs as a sequel to herniation of the attenuated disc through the destroyed vertebral plate into the substance of the affected body[3].

The after result of the destructive process is that the joint is destroyed. A tuberculous granulomatous abscess is formed in which the remnants of the disc persist. This abscess maybe compressed between healthy vertebrae above and below. Compression of the spinal cord will cause paraparesis or paraplegia. Contrary to the old belief that the coverings of the spinal cord provided a barrier to the

tuberculous invasion[13] clinical and histopathological observations by Michaud in 1871 and by many others[14] indicate that vertebral tuberculous infection does in some cases make its way across the dura, giving rise to focal meningitis and meningo-myelitis. It is also possible that in those patients where no myelitis occurs, the inflammatory swelling of the dura and adjacent soft tissues may produce pressure in the spinal cord and perhaps by itself or in conjunction with other pressure effects, be a significant factor in the pathogenesis of Pott's paraplegia. Spread of the infection to the surrounding soft tissues will lead to the formation of a "cold abscess". Hodgson[17] et al proposed that the causes of Pott's paraplegia can be divided into two types:-

(1) **Extrinsic causes:**

(a) In active disease - abscess (fluid or caseous) granulation tissue, sequestrated bone and disc, pathological subluxation and dislocation of vertebrae. The latter could have been the cause of paraplegia in one of the patients in this study because there were radiological changes suggestive of dislocation at T_{12} and L_1.

(b) In healed disease: - transverse ridge of bone anterior to the spinal cord, fibrosis of the dura.

(2) **Intrinsic causes:**

Passage of the tuberculous inflammation across the dura to involve the meninges and eventually the spinal cord with associated tissue destruction.

The risk of paraplegia developing is related to many factors. According to Guirguis[15] the following appear to be the most important:-

(a) Inadequate care - by unqualified by personnel at unqualified centre (b) Late diagnosis (c) Incorrect treatment (d) Premature cessation of treatment (e) Development of drug resistance (f) Level of disease -

in the thoracic spine, the spinal canal is narrow and the cord occupies most of it; so that even a small abscess or sequestrum may produce cord pressure. Natural drainage of pus is impossible in this area.

Pott's paraplegia can be reversible (oedema of cord, compression by fluid or semi-fluid collection under tension, compression from sequestra or remnant of disc, compression from collapsed vertebrae) or it can be irreversible where changes are permanent (associated with degeneration of the spinal cord and may be secondary to vascular changes). Reversible paraplegia can change to irreversible paraplegia if neglected.

Pus from the cold abscess may track up and down underneath the anterior common ligament. As this pus is under pressure it will cause erosion and excavation of the anterior surfaces of the vertebral bodies but not on the fibrocartilage of the disc as these are resistant to such pressure. As a result concave erosions appear on the anterior surfaces of the vertebral bodies[7] a condition likened to the "aneurysm phenomenon"[7].

Further development of the cold abscess will depend on the region of the spine affected. Abscesses can track and dissect along fascial planes and come to the surface in the para-spinal region. In the cervical region pus can drain away and a retro-pharyngeal abscess may be formed just under the prevertebral muscles. It may attain a significant size as to cause difficulty with swallowing and breathing[7].

Affection of the upper thoracic spine may present as an abscess in the posterior triangle of the neck[7]. A cold abscess localized in the middle thoracic spine cannot drain away early and occasionally may rupture into the pleura or into a bronchus[7],[15]. In the lumbar and lower thoracic regions abscesses dissect along the fascial sheath of the psoas muscle to appear in the groin or on the medial aspect of the thigh, and may be confused with a hernia.

Spontaneous rupture of the abscess causes the formation of a chronic sinus which may lead to amyloid disease or death from toxaemia if secondary septic suppuration takes place. Sometimes these abscesses may produce large fluctuant subcutaneous mass that persist for a long time. These can be sites of an extensive precipitation of calcium salts if they remain uncontaminated.

Sometimes sinuses may rupture through the dura and drain into the spinal canal leading to a tuberculous meningitis which is usually fatal. It has been shown that infection usually stimulates a considerable growth of granulation. Sometimes this granulation tissue may sufficiently narrow the canal to produce a transverse myelitis[12].

Repair is usually by fibrous tissue. When several vertebral bodies are destroyed invasion of the granuloma by fibrous tissue and new bone occurs, when the gap is large, repair by fibrous tissue results and bony ankylosis does not occur. It has been suggested that collapse of the vertebral column with consequent deformity may narrow the gap and thus favouring the process of bony ankylosis. However it has been found that while this may be true in the majority of cases, the presence of a granuloma prevents bony ankylosis in that a slow growing disease persists causing more vertebral destruction with the development of a vicious circle resulting in more deformity. The vicious circle is only relieved by the removal of the granuloma.

CLINICAL FEATURES

The age range of our patients was between 3 and 10 years with an average of 7.3 years. The M/F ratio was 2:1. (Table 1).

Tuberculosis of the spine can occur at any age but the highest preponderance of cases affect the prepubertal child[3]. Males and females are more or less equally

affected. Some authors have found a slightly higher incidence in males[3]. The significance of the M/F ratio of 2:1 in this study is difficult to interpret because the study group is too small. Some authors have found out that most of the patients come from the lower economic strata[3] which was also the case in all the patients in this study.

Back pain was a constant symptom in all the patients in the study. Its duration varied from one month to twelve months (Table 1). Deformity in the back at differing levels was also a constant observation in all the patients. Weight loss with or without gross malnutrition, anaemia and growth retardation was observed in the majority of the patients. Three patients had on and off fever. Two patients had cough, one of them had radiological changes suggestive of active pulmonary tuberculosis. One had generalized lymphadenopathy. Night sweats were not a constant finding. One patient was paraplegic with involvement of sphincter control.

Pain is one of the early symptoms which is followed by gradually increasing stiffness and deformity of the back. Back pain in children especially when persistent is rare in contrast to the adult where back pain is a common symptom. When a child presents with back pain the possibility of tuberculosis of the spine should be considered and ruled out early. The back pain may be of an aching character. Muscle spasms, weakness of a limb or girdle pain are also early symptoms and indicate the effects of pressure and irritation by the tuberculous granuloma on the spinal nerves. In children suffering from malnutrition the disease may be associated with systemic manifestations like evening pyrexia, cachexia and anorexia[2].

Clinically the signs may be absent or minimal in early cases. In late cases however the presence of kyphosis or gibbus is an important finding synonymous with established disease. The majority of the patients in this study were

late cases; already with back deformities and systemic manifestations. Stiffness of the back is a prominent sign and is due to the spasm of the erector spinae muscles. It can be easily detected when the child stoops with a straight back to pick up an object off the floor.

Occasionally a child may first present with a psoas abscess in the inguinal area which can be mistaken for a hernia. Besides tracking down from the lumbar vertebrae, abscesses which present in the right iliac fossa may also rise under the iliacus fascia from the sacroiliac joint or from tuberculous mesenteric glands[7]. An abscess from a tuberculous hip may also present above Poupart's ligament.

Sometimes children may present with weakness of one foot, dragging of the legs or complete paraplegia all of which are due to cord compression.

Paraplegia has been graded according to the severity of the deficit in the following categories, according to Konstam[4].

grade I - Spastic, but able to walk

grade II - Unable to work, but voluntary movements present

grade III - Paraplegic in extension, no voluntary movement

stage IV - Paraplegic in flexion, no voluntary movements

stage V - Flaccid paraplegia

RESULTS

Table 1: Presentation

Case	Sex	Age	Presentation	Duration Months	Level of spinal lesion	Duration of para-plegia	Comments
1. A.M.	F	3	Swelling at the back -Back pain -Inability to walk -I muscle power -Weight loss -Evening fever -Generalized lymphadenopathy -Gibbus, antalgic gait	6	T_{12} L_1	-	BCG Scar+ ESR =80mm/hr Hb 8.7gm/dl WBC = 11 000 Lymph = 69% BWT = 11.0kg CXR -NAD Lymh node Bx = TB
2. A.S	M	7	Back pain -Progressive back deformity -Night sweats -Weight loss, Gibbus -Intermittent fever	9	T_{11}, T_{12} paravertebral abscess	-	BCG Scar+ ESR=50 WBC=6700 Hb=9gm/dl CXR - NAD
3. S.Y.	M	7	Swelling at the back - Fever -Back pain -Weight loss -Loss of power in Lower limbs -Loss of sensation of legs -Urine retention -Loss of sphincter control	3	T_{12} subluxation or dislocation at T12/L1	3/52	BCG Scar+ ESR =100mm/hr Hb = 8.6gm/dl WBC =11,100 CXR-NAD
4. M.A	M	8	-Back pain -Back deformity -Weight loss -Cough	1	T_7, T_8, T_9 paravertebral abscess	-	BCG Scar+ ESR = 85 WBC = 6,300 Hb = 12.1 CXR-?PTB
5. B.M.	F	8	-Swelling at the back -Weight loss -Back pain -Discharging (R) sinus high -Growth retardation -Malnutrition -Fever (38) -Gibbus	12	T_8	-	BCG Scar- ESR= 65 Hb = 7.5 WBC= 9500 CXR-NAD

6. J.L.	M	10	Swelling at the back -Back pain -Cough -Weakness of lower limbs -Growth retardation -Weight loss	8	T_3, T_9 paravertebral abscess	-	BCG Scar- ESR= 120

Table 2: Region of Spine Affected

Region of Spine affected	Number of patients
Cervical	0
Thoracic	6
Lumbar	1
Sacral	0

Table 3: Number of Vertebrae affected in individual patients.

Case number	1	2	3	4	5	6
No of vertebrae affected	2	2	1	3	1	2

(a) The majority of patients had more than one vertebrae involved.
(b) Vertebrae affected in the paraplegic patient was T_{12}.

Table 4: Results of treatment last follow-up 3 months after discharge

Improved, walking normally, no back pain, weight gain	5
Slow recovery with paraplegia	1

. Five of the patients had good response to treatment.
. Recovery was slow in the paraplegia patient

INVESTIGATIONS

All the patients in this series were subjected to a battery of investigations. The results are shown in table 1. The following investigations are important when investigating for tuberculosis of the spine.

X-ray of the spine: - both anteroposterior and lateral views are taken. All patients in this study had late

radiological changes of a tuberculous process at different levels of the spine. The thoracic spine was involved in all the patients. Only one patient had L_1 involvement. None had cervical or sacral spine vertebral involvement (table 2). Four patients had more than one vertebral involvement (table(3). Half the patients had evidence of paravertebral abscess. One had dislocation of T_{12}/L_1 with paraplegia.

The typical radiological evidence of tuberculosis of the spine are areas of bone destruction associated with vertebral collapse and wedging. There may be a paravertebral shadow indicating the presence of paravertebral abscess. There may be evidence of spinal deformity like kyphosis or kyphoscoliosis. These radiological changes are late signs of tuberculosis of the spine. In early disease the radiological changes may be absent minimal or inconclusive. In such cases the value of tomography is important especially in lateral projections. The early radiological changes may take the form of reduced disc space without destruction or demineralization of adjacent bone surfaces. Occasionally in 5% of cases presentation can be totally atypical so that a precise radiological diagnosis cannot be made.

According to the first report the MRC Working Party on tuberculosis of the spine, radiographic activity of tuberculosis of the spine can be categorised in the following manner[16].

(a) Active disease - in which the radiographic features show:-
   - Loss of the thin cortical line
   - Rarefaction of the affected vertebral body

(b) Quiescent (inactive) disease in which radiologically there is:-
   - Bony fusion of the affected vertebral bodies
   - Scoliosis of the contiguous surface of the affected vertebrae with reduction or

disappearance of the intervening disc space.

(c) Disease of doubtful activity which appear as marginal sclerosis where there was much destruction of vertebral bodies. Cavitation of the vertebral body or sequestrum formation may be evident.

Chest X-ray: In this study, chest X-rays did not produce much positive findings in the majority of the cases. Only one patient had evidence of active pulmonary tuberculosis. Routine chest X-ray is however important to detect primary foci. In more than 50% of cases the primary focus is intrathoracic and in a large proportion of cases active pulmonary lesion are detected[3].

Full blood picture: - is of little diagnostic value except perhaps in ruling out other haematological disorders, that may influence the line of management of the patient or that need correction for better response to treatment of the patient. The ESR may be normal in 15% of the cases with skeletal tuberculosis. It is however important when raised initially as it serves as an invaluable index for monitoring the patients response to chemotherapy. The ESR in the current study were raised between 50 - 120mm/first hour Siller[17] reviewing on 30 cases of tuberculosis of the spine found the ESR to be the only consistent laboratory test. The white cell count maybe raised with a lymphocytosis.

Immunological Test: No immunological test like Mantoux test could be performed in this study because there were no reagents for the test at the time of the study.

(a) **The Mantoux and Heaf tests** are frequently used investigations. They are just aids to diagnosis. They are of considerable value in populations where the positivity rate is low. Hence a spinal lesion with strong tuberculin test is highly suggestive of tuberculosis of the spine. Vaughan et al[18] found a high rate of non specific

tuberculin reactivity and cautioned on the possible presence of cross reactivity. A higher cut-off point has been suggested (10-12 mm diameter) for our environment. Negative tuberculin skin tests (Mantoux) have been reported in patients with immunodeficiency - AIDS/Tuberculosis[19],[20].

(b) **BCG test:** - Udani et al[21] suggested that BCG vaccine, apart from its utility as vaccine against tuberculosis, can also be used for a diagnostic test. When BCG vaccine is given subcutaneously to a child suffering from tuberculosis, a local lesion appears within 24 to 72 hours and varies from redness and induration to necrosis (Koch's Phenomenon). The test has been found to be superior to tuberculin test[21]. However in previously immunized children the test has been found to have a strongly accelerated BCG reaction (children under 5 years) and this cannot be used as a diagnostic test in these children[22].

<u>Needle Biopsy</u> of the radiologically involved tissue is the method of choice in spine tuberculosis. With the introduction of the image intensifier it can be performed quite safely. It provides material for histological and bacteriological studies. Needle biopsy was not done in this study.

Bacteriological studies (i) Specimen from the sputum, pus from the abscess, or sinuses, gastric washing, should be taken for Ziehl-Nielsen staining. M. tuberculosis appear as Acid-fast bacilli. Positive acid fast reaction can also be given by Narcadia infections. They should be differentiated by culture results. In this study, three sputum specimen were taken for Ziehl-Nielsen Staining from the 2 patients who had cough. None were positive for tubercle bacilli. A negative Ziehl-Nielsen stain test, however does by no means

rule out tuberculosis infection. (ii) Culture and sensitivity test for M. tuberculosis takes a long time (6-8 weeks) and may thus be of less value when deciding early therapy on these patients.

Konstam and Blesovsky[1],[4] proposed the following diagnostic criteria for the diagnosis of tuberculosis of the spine:

(1) A radiograph typical of tuberculosis. (In East Africa the diagnosis based on radiological changes alone was subsequently found to be inaccurate in 10% of cases(1)
(2) Typical pulmonary lesions
(3) Positive bacteriological or histological evidence
(4) Clinical and radiological improvement after treatment with antituberculous drugs.

In this study the history, physical findings, the typical radiological changes of late tuberculosis of the spine and the clinical and radiological improvement with antituberculous drugs were thought of as being enough diagnostic criteria.

TREATMENT

All the patients in this study underwent conservative treatment with chemotherapy using the standard antituberculous regime of a combination of intramuscular injections of streptomycin 30mg/kg/day for two months and tablets of thiazina 15mg/kg daily taken orally. All the patients were kept in the ward for two months till they completed 60 injections of streptomycin, after which they were allowed home to continue with thiazina for 16 months further. Significant improvement was noted in 5 patients by the end of the second month of treatment. The back pain subsided or diminished, five of them could walk without support, and had favourable radiological changes. Results

of treatment were assessed at the follow-up clinic by clinical assessment of the general condition of the patient, radiological changes and the ESR (table 4). Patients were kept in the ward for completion of the 60 streptomycin injection because according to our experience ambulant treatment outside hospital has revealed several management problems; (1) Drugs are not constantly available at the district or regional level. (2) Default as soon as the patients improve (3) Supervision and diet is more assured and is better in the hospital and help in a speedy recovery of the patients.

Principally there are two major treatment modalities:-

(1) Conservative treatment by chemotherapy alone or

(2) Surgery in combination with chemotherapy.

Chemotherapy: The aim of chemotherapy is to eradicate the infective organism and thus to arrest the destructive process. The chemotherapeutic regimen is basically similar to that used in the treatment of pulmonary tuberculosis[2]. The standard regime currently in use in Tanzania is a combination of streptomycin injections 30mg/kg/day given intramuscular for a 60 day period and thiazina tablets 10-20mg/kg/day given orally for 60 days in combination and then thiazina alone for a further 16 months.

Other combinations have been used either on continuous administration or on intermittent basis. Intermittent short course antituberculous regime has not received wide application in this country because the availability of the drugs is erratic and when they are available, they are too expensive to afford.

Surgery: The main principle of operative treatment is to drain the abscess and remove diseased tissue as early and as thoroughly as possible. Surgical treatment has two major roles in the treatment of spinal tuberculosis:-

(a) Treatment of complications, the commonest ones being paravertebral abscesses, sinuses and

progressive paresis or paraplegia.

(b) In patients in whom there is extensive destruction or any evidence of mechanical instability[2], progressive deformities especially those of the thoracic spine that impair functioning of the heart and lungs.

According to Guirguis the major indications for surgery are as follows[15].

(a) Progressive paraplegia

(b) Compression of the cord but with no tense abscess shadow to be seen radiologically

(c) When the cause of paraplegia can be suspected radiologically to be sequestra or the posterior border of a collapsed vertebra especially when the canal appears narrow and angulated.

(d) In patients with no improvement after decompression of a tense abscess by other means such as aspiration or costo-transversectomy.

(e) For neglected paraplegia

(f) When there is no radiological cause of compression - exploration of the cord at suggested site of compression may show a tense membrane around the dura or a sequestrum not seen by X-ray.

(g) When there is severe gibbus with narrowing of the canal at its apex.

The operations usually carried out are:-

- Simple debridement, antero-lateral decompression with/without anterior spine fusion for stability of the spine.

A number of risks can be associated with the operation[15] of anterolateral decompresion:-.

(a) Weakening of the vertebral column (b) Vertebral collapse after operation (c) Tuberculous meningitis (d) Tuberculous empyema.

(e) Injuries to the intercostal nerves (f) Injury to the cord.

Increase in paraparesis or complete paraplegia after operation may be caused by: Concussion, oedema of the cord, pressure by a haematoma or due to organic injury to the cord.

The choice of what treatment modality to follow will depend upon the environment in which the clinician finds himself and the clinical condition of the patient when he presents for treatment[2]. Patients who present with active disease can present with or without paraplegia.

There had been no unanimity before, on the best form of treatment modality of spinal tuberculosis, a fact which prompted the Medical Research Council (MRC) to conduct comparative controlled treatment trials of tuberculosis of the spine in different countries. The findings were stipulated in the MRC report MRC 1973a, 1973b, 1974a, and 1974b(2). According to the MRC 1974b trial report it was established that the spinal disease is extremely responsive to chemotherapy[2]. It also became obvious that ambulatory treatment with appropriate chemotherapy was logically the method of choice especially for third world surroundings[4,16]. Other researchers however, have alleged that complete healing with sound bony union cannot be guaranteed without surgical debridement and that only operative stabilization will prevent deformity[1].

Dickson[1] and also Konstam[4] recommend that patients without paraplegia can be safely treated ambulant as outpatients with streptomycin and thiazina for 3 months and then thiazina alone for the remaining 15 months. The method had been shown to be successful in early cases. However, this method of treatment although simple in plan and requiring few facilities, may fail if the thoroughness and attention to detail are lacking. Good follow up by clinical assessment and radiology is essential and interpretation of

radiographs requires experience.

Admission of patients to hospital depends upon local resources. Dickson[1] suggested the following priorities as guidelines:-

(i) Very ill children (ii) Paraplegics (iii) All cases of extensive disease (iv) All patients not clinically well after 3 months or radiologically arrested after 6 months (v) All patients with paravertebral abscesses (vi) Patients presenting with advanced disease and much vertebral body destruction who appear to be less suitable for ambulant treatment.

As for the patients with paraplegia, they should be treated in hospital. They can be treated with chemotherapy alone (although their progress is often slow) or with chemotherapy and surgery. In those in whom the prognosis with conservative treatment is bad, operation is essential[15]. Konstam[4], one of the main proponents of conservative management of spinal tuberculosis with paraplegia by chemotherapy unsupplemented by any other measure has claimed to have excellent results.

Assessment of results: Irrespective of what modality of treatment is used, progress should be assessed by assessing the general condition of the patient, by radiographic controls and the ESR changes. Favourable features are shrinking of paravertebral abscesses and recalcification of the affected bone. Konstam and Blesovky[4] divised a simple geometric presentation by which the severity of kyphosis could be measured. This was later modified by Dickson[1] who used to measure the internal gibbus angle. This is a good objective index of progress. Five of the patients in this study had good response to conservative treatment (table 4). Recovery was slow in the paraplegic patient.

Prophylaxis for tuberculosis: Four out of the six cases in this study had a positive BCG scar on their right deltoid area signifying that they had BCG vaccination (in infancy or

childhood) against tuberculosis.

The effectiveness of neonatal BCG vaccination in preventing tuberculosis is uncertain and some findings have expressed doubt about BCG vaccination effectiveness.

In 1979 the Indian Council for Medical Research in collaboration with the W.H.O. conducted a study involving about 260,000 individuals of whom 47,954 were aged 1 month to 5 years. The first 7.5 years of follow up yielded no evidence that BCG vaccine had a protective effect[24]. A number of arguments were given to explain this lack of effect including the high prevalence of atypical mycobacteria and the prevalence of a variant of M. tuberculosis, the South Indian Variant. In the Manchester[25] series, the suggested level of protection was above 75%.

A study on the efficacy of BCG vaccination in 3 Tanzanian regions conducted by Mkerenga[26] found that BCG vaccination had no significant effect in the prevention of bacillary tuberculosis.

Shortly after the results of the Chingleput report, the WHO study group in 1980 reviewed the available evidence on the effect of BCG vaccination and came to the conclusion that there was no reason to change the current vaccination policies[27]. In Tanzania, the Ministry of Health still believes that BCG vaccination should still be considered as being highly effective in the prevention of tuberculosis in infants and children.

Tuberculosis and AIDS

The HIV epidemic is causing a huge increase in the number of people with tuberculosis either as a reactivation of latent disease or as a new infection and the association of tuberculosis and AIDS has been described by various authors from different countries. In Tanzania there has been a dramatic increase of 15.8% of new cases of tuberculosis since 1983[28]. In the USA, in addition to an

increase in tuberculosis cases since 1986, a number of drug-resistant outbreaks of tuberculosis have occurred[29].

A high susceptibility of developing tuberculosis has been observed in AIDS patients as a result of immunodeficiency[30]. In Africa, tuberculosis involves about 20% of HIV infected patients[31]. In Zaire about 33% of tuberculosis patients were HIV seropositive in 1985 according to one report from Kinshasa[32]. HIV positive tuberculosis patients are more likely than HIV negative tuberculosis patients to have[28,29]: (a) tuberculosis in uncommon sites (usually extrapulmonary like lymph nodes milliary, pericarditis, central nervous system) (b) a negative sputum smear (c) a negative tubercillin test (d) an atypical chest x-ray (e) a greater danger of relapse (f) more serious adverse reactions to anti tuberculous drugs particularly to thiacetozone (up to 20% of HIV positive tuberculosis patients).
The incidence of HIV infection in patients with tuberculosis of the spine in Tanzania could not be documented.

CONCLUSION

Tuberculosis of the spine is still a major health concern in Tanzanian children as well as in other third world countries. It is a cause of considerable morbidity and mortality. with the current trend of rising incidence of pulmonary tuberculosis in the country, tuberculosis of the spine will continue to prevail for many years to come The identification of 4 cases with positive BCG scars and with advanced tuberculosis of the spine from such a small study group is another sign pointing to the uncertainty of the effectiveness of the BCG vaccination. Further controlled studies at a more extensive level are required to evaluate the present questionable effectiveness of BCG vaccination. The advent of AIDS has changed the clinical aspects of tuberculosis.

REFERENCES

1. Dickson J.A.S. Spinal tuberculosis Nigerian children. A review of Ambulant Treatment. J.B.J.S. 49B:682, 1967.

2. Kemp H. Tuberculosis of the Spine Brit. J. Hosp. Med. 15:39, 1976.

3. Ganguli P.K. Radiology of bone and joint tuberculosis. Asia Publ. House New York, pp.3, 1963.

4. Konstam P.G, Blesovsky. The ambulant treatment of Spinal Tuberculosis. Brit. J. Surg. 50:26, 1962.

5. Howlett W. Paraplegia: Incidence, causes and diagnosis. Proceed of the 25th Postgrad. KCMC seminar pp47, Oct., 1984.

6. Girdlestone G.R. Tuberculosis of bone and joint. Ed.2 New York, Oxford Univ. Press 1952.

7. Heaf, FRG, Symposium of Tuberculosis. Cassell and Company Ltd. London pp.538, 1957.

8. Michell, R.M.: Anterior fusion in spinal tuberculosis. EA. Med. J. 43:119, 1966.

9. Bulla A. World Health Statistics Report. Tuberculosis Patients: How many Now Bull. Int. Un. Tuberc. 52:35-40, 1977.

10. Girdlestone, G.R. and Somerville E.W. Tuberculosis of bones and joints Oxford Med. Publ., London, New York, Toronto, 1952.

11. Collins, D.H. Pathology of Bone tuberculosis, Butterworth, London pp216, 1966.

12. Falk, A. Results of Long term chemotherapy for spinal tuberculosis: A follow up study of 235 patients. Am. Rev. Resp. dis. 95; 1967.

13. Lichtenstein, L. Diseases of bone and joints 2nd ed. The C.V. Mosby Company Saint Louis pp61, 1975.

14. Hodgson A.R. Skinsnes OK, Leong CY. The Pathogenesis of Pott's Paraplegia J.B.J.S 49A:1147, 1967.

15. Guirguis A.R. Pott's Paraplegia. J.B.J.S. 49B:pp 658, 1967.

16. M.R.C. - First Report:
J.B.J.S 55B:678-696, 1973.

17. Siller T.N., Wittshire. Bone and Joint Tuberculosis. J.b.J.S. 57B:532, 1975.

18. Vaughan J.P., Lindquist K, Brooke, D., Doyle, R.F. Tuberculin Sensitivity in Tanzania. E.A. Med. J. 48:307, 1976.

19. Chaisson R.E, Schecter, G.F., Theuer, C.P. et al. Tuberculosis in Patients with acquired immunodeficiency Syndrome. Clinical Features, Response to therapy and Survival. Ann. Rev. Resp. Dis.

20. Pitchenile A.E., Burr J., Suarez M. et al. Human T-Cell lymphatropic Virus III Seropositive and related Disease among 71 consecutive points in whom tuberculosis was diagnosed. A prospective study. Am. Rev. Resp. Disease 135:875-79, 1987.

21. Bhandari N.R., Bhambal SS, Beohar V. Diagnostic Value of BCG test in childhood Tuberculosis. Indian Pediatrics 21(7): 555-59, 1984.

22. Myint TT, Yin Y., Yi MM., Aye HH. BCG test reaction in previously BCG vaccinated children. Ann of Trop. Paed. 5:29-31, 1985

23. Goel M.K. Treatment of Pott's Paraplegia by Operation. J.B.J.S. 49B: 676, 1967.

24. Tuberculosis Prevention Trial. Trial of BCG vaccines in South India for tuberculosis prevention: first report Bull Wld. Hlth. Org. 57:819-27, 1979.

26. Mkerenga - Thesis DPH University of Dar es Salaam, 1980.

27. WHO Technical Report Series No. 654, 1980.

28. Pallangyo K.J. Clinical Presentation of HIV infection and AIDS in Tanzania. Tanzania Med. J. 6(2):49-51, 1991.

29. AIDS action - The intern. Newsletter on AIDS and control. Publ. Appropriate Health. Resources of technologies Action Group Ltd. Issues 18 Sept. 1992.

30. Editorial:
Morbidity and Mortality Weekly Report (MMWR). Centre for Disease Contol 36:378-794, 1987.

31. Mindel A. in AIDS a Pocket Book of Diagnosis and Management- 1st Ed. Edward Anald Publ. pp 1 - 239, 1990.

32. Biggar R. Clinical Features of HIV in Africa. Br. Med. J. 293: 1453-1454, 1986.

CHRONIC OSTEOMYELITIS

SUMMARY

Fourteen patients with chronic osteomyelitis of various aetiologies were managed during the 4 months rotation in the Orthopedic and Trauma Department between August - December 1991. 13 of the patients were males and one was a female. Their ages ranged from 11 to 51 years with an average of 25.1 years. Nine of the patients were victims of trauma: 5 had sustained open fractures following motor vehicle accidents, 3 had fractures during a football match and one sustained an open fracture following a fall from a coconut tree. In 4 of the patients the infection was spontaneous. One patient was a diabetic.

The commonest presentation was local pain in 11 patients and the presence of a discharging sinus in 12 patients. The duration of symptoms varied from 3 months to 10 years (average 8.8 months). The commonest site of infection were the femur in 5 patients, the tibia in 4 patients and the fibula in 3 patients. the commonest organism isolated was staphylococcus aureus.

All the patients underwent sequestrectomy and saucerization. The wound cavities were left open to be filled by granulation tissue and to heal by secondary intention

INTRODUCTION

Chronic osteomyelitis can be defined as one or more foci in bone that contain pus, infected granulation tissue, sequestra, a draining sinus and resistant cellulitis[1]. The inflamatory foci are surrounded by sclerotic bone with poor blood supply and are covered by a thick, relatively avascular periosteum and scarred muscle and subcutaneous tissue.

Chronic osteomyelitis is a very old disease as opposed

to acute haematogeneous osteomyelitis which appears to have been understood in the last six decades or so[2]. It is often a sequel of acute haematogeneous osteomyelitis from childhood carried forward to early adulthood[3]. Because of this close relationship one is inevitably obliged to talk about certain elements of the acute form when talking about chronic osteomyelitis. Chronic osteomyelitis can also occur following open fracture or after bone surgery. Speaking of the management of osteomyelitis following open fractures Hippocrates said: "One should especially avoid such cases if one has reasonable excuse, for the risks are great and rewards are few[1]. With the advent of antibiotics the incidence of chronic osteomyelitis complicating acute haematogeneous osteomyelitis has decreased tremandously[4]. Chronic osteomyelitis, though less common now in some parts of the world is still a scourge in many parts of the developing countries like Tanzania and presents real problems of management.

The problem is compounded by the increase in the number of open fractures associated with the present increase of motor traffic accidents brought about by the modern era of fast moving motor vehicles.

CASE PRESENTATION

R.K. was a 28 year old male adult who was referred to Muhimbili Medical Centre from a district hospital in a West Lake region of the country with a long standing history of recurrent pus discharging sinus in the left upper humeral area associated with local pain for 3 years. Initially the lesion was preceded by a spontaneous local pain in the upper arm associated with fever. This was followed by a spontaneous formation of a pus discharging sinus. There was no history of trauma and there was no history of tuberculosis, diabetes mellitus or sickle cell disease in his family history or in his past medical history. No

history of diarrhoea or weight loss could be ellicited. He had been diagnosed as a case of chronic osteomyelitis and he had been operated on thrice - once at a district hospital and twice at a consultant hospital and had received several antibiotics. The wound on the left humeral area had healed following the third surgery.

Three months prior to admission to the hospital he developed swelling and local pain and eventually a discharging sinus on the lateral aspect of the right upper thigh. This was the reason for the referral to Muhimbili Medical Centre.

Physical examination revealed a young man in fair nutritional status, not wasted, mildly pale, not jaundiced, afebrile. No lymph nodes could be palpated and no skin or oral lesion could be ellicited other than the scar on the left humeral area and the discharging sinus on the right thigh. Systemic examination was normal.

Local examination revealed a healed ovoid scar in the lateral aspect of the proximal third of the left upper arm. There was a pus discharging sinus in the lateral aspect of the proximal third of the right thigh. The surrounding skin was of normal appearance and consistence. The immediate skin and tissue surrounding the sinus was indurated and slightly swollen. There was no fluctuancy detected and the ipsilateral hip and knee joints were free. A provisional diagnosis of Chronic Osteomyelitis was made. A number of investigations were carried out as follows:-
The full blood picture was normal. He had an Hb of 15.2gm/dl, the total WBC was 4,700, ESR = 10mm/hour. The fasting blood sugar was 4.1 mmol/dl and the ELISA test for HIV was negative. The culture and sensitivity of a pus specimen obtained by deep aspiration from the discharging sinus on the left thigh revealed unidentified coliforms sensitive to Tetracycline, Cotrimoxazole, Chloramphenicol and Gentamycin and resistant to Ampicillin. A plain X-ray

of the right femur revealed osteomyelitic changes of the proximal third of the right femur with clear evidence of involucrum and sequestrum formation. The patient was put on Chloramphenical 500mg intramuscularly six hourly for 4 days, this as a preparation for surgery.

Informed consent was obtained from the patient. At operation, the patient was placed in the supine position. Under general anaesthesia and under aseptic conditions the discharging sinus on the thigh was excised right up to the bone and saucerisation was done removing as much as possible of the necrotic tissues, sequestra and debris. The wound was then irrigated with hydrogen peroxide and with copious amounts of normal saline. The wound was left open for daily Eusol dressing. Daily Eusol dressing was continued for two weeks. Gradual mobilisation and exercise were started and on the 14th day the patient was discharged home with a healed wound. Oral antibiotics were continued for a period of four weeks. The patient was seen again at our folow up clinic six weeks later, the wound had healed completely with normal leg function. On the day of discharge from the clinic it was explained to the patient that though the results looked excellent recurrences could not be ruled out in the future.

DISCUSSION

INCIDENCE

Osteomyelitis is mainly a disease of children and young adults. This was also reflected in our small series where the peak range was between 10 and 30 years. Waldogel[3] found that 85% of the patients were less than 16 years. The morbidity seems to be worse in the lower socio-economic groups[5]. Males are affected more frequently than females in the ratio of 2:1[5]. In the present serie the M/F ratio was 13:1. Most of the patients were trauma patients and the series is probably too small to draw any firm conclusions.

Figures at the Mayo clinic in the years 1951 to 1961 indicate that approximately 44% of cases of osteomyelitis of the femur and tibia had a history and clinical course consistent with osteomyelitis in childhood that persisted into adult years[6]. During the 14 years of 1967 to 1980. The mean incidence in this type of osteomyelitis had decreased to 28%[6].

Acute haematogenous osteomyelitis is uncommon in healthy adults. However, it has been encountered in drug addicts and in patients who are immunologically incompetent[6] because of serious systemic disease. Even here figures indicate that the incidence of acute haematogenous osteomyelitis has decreased[6]. Jones et al[7] also reported the declining number of patients with chronic bone infection after inadequate treatment of acute infectious. However an interesting feature he noted was the high incidence of diabetes with chronic osteomyelitis of the foot secondary to diabetic neuropathy. Miller and Miller-Faber[7] reported post-operation sepsis in 3% of the 1176 femoral fractures treated by intramedullary fixation.

AETIOLOGY

The most frequent cause of osteomyelitis in this study was open fracture which occurred in various circumstances (see tabe 1). Five partients had open fracture following motor traffic accidents, 3 patients had open fractures during a football match and one fell from a coconut tree. Three patient had chronic osteomyelitis arising from acute haematogenous osteomyelitis in childhood. One was a diabetic.

Staphylococcus aureus was the most frequent organism isolated in the 8 patients tested (table 2). Staphilococcus Aureus has been found to be the cause in up to 80-90% of cases in most series[2]. Even though most studies have previously shown that staphilococcus aureus was the

commonest etiological agent more recent studies show that it is not the cause in approximately 30 to 50% of patients[2]. That is why it remains important to search for other organisms as well.

Table 1: Underlying causes of chronic osteomyelitis (n= 14 patients)

Underlying Cause	Male	Female	Total
1. Open fracture following MTA	5	-	5
2. Open fracture during footbal match	3	-	3
3. Open fracture following fall from coconut tree	1	-	1
4. Spontanous - from acute haematogeneous osteomylitis	3	1	4
5. Spontaneous in eldery diabetic	1	-	1
Total	13	1	14

The most common cause of osteomyelitis in the study was open fracture following trauma.

Several other organisms such as gram-negative bacteria like E.Coli, fungi, pseudomonas, klebsiella, salmonella and proteus have been isolated in increasing frequency. Other aetiological organisms include streptococci, coliform organisms, pneurmococcus, gonococci, staphylococcus albus and brucellosis. Pseudomonas has previously been isolated frequently in drug addicts[6]. For some obscure reasons salmonella has been found to be the commonest organism in sicklers (up to 75% in some series) and it is unusual in that it is often multifocal[8] and will often involve the

diaphysis of long bone rather than the metaphyses[5]. In non-sicklers less than 1% of chronic osteomyelitis is due to salmonela[19]. The thrombosed, infarcted and necrotic bone may be a predisposing factor, but the reason for the predisposition to a particular organism is still obscure. In infants the commonest organism is haemophilus influenzae[2]. However infants suffer more from septic arthritis rather than acute osteomyelitis[2]. Anaerobic organisms, first described by Rist in 1898 are being reported with increasing frequency[10]. At the Mayo Clinic the anaerobes accounted for 25% but accounted for 61.5% with failure rate in the treatment against the average of 20%[10,11]. However, the actual significance of anaerobic organism is still controversial and it has been suggested that the rarity has been due partly to inadequate technique in our surroundings for collection and transportation of specimen, inadequate laboratory facilities for their growth and isolation and also probably due to the lack of awareness on the physician's part. Mixed aerobic/anaerobic osteomyelitis has been documented and it has a poor prognosis[1] Studies have also emphasised the role that aerobic and anaerobic bacteria play in diabetic foot in diabetic patents and that antimicrobial therapy must be based on this fact[12]. Eikenella Corrodens which is a fastidious gram negative rod that is part of the normal human flora, is known to be one of the complex flora that can cause osteomyelitis of the foot in diabetic patients[13] especially when it acts as a synergistic pathogen with streptococcus viridans[14]. This organism is usually isolated in mixed cultures and is easily overgrown and missed on routine (aerobic) cultures[15]. In the present series results of culture and sensitivities were obtained in 8 patients. During that period only aerobic cultures could be performed at the centre. Because of unavoidable constraints anaerobic cutures could not be performed. The

results are shown in the following table.

Table 2: Microrganisms isolated (n=8)

Organisms isolated	Number of patients
- Staphylococcus aureus	5
- Unidentified coliform	1
- Proteus species	1
- E. Coli	1

The commonest microrganism isolated was staphylococcus aureus.

PATHOLOGY

A relevant account of chronic osteomyelitis can hardly be made without touching on some aspects of acute haematogeneous osteomyelitis because in the majority of cases the latter is usually a prerequisite of the former, that is chronic osteomyelitis is a sequel of acute haematogeneous osteomyelitis. The other forms of chronic osteomyelitis are post traumatic where you have chronic osteomyelitis complicating open fractures. Iatrogenic osteomyelitis is a complication of surgical operation. The acute stage of osteomyelitis tends to follow a different course in infants, children and adults due to the diffrence in the anatomical structure of the bone and the peculiar vascular arrangement of the growing bone[2]. Acute haematogeneous osteomyelitis is usually secondary to haematogeneous spread from a primary septic focus which may be insignificant. The site of entry of the organism to the blood stream often cannot be traced. In about 25% of the cases there is a history of a recent infectious process (respiratory, aural or cutaneous[2]. About 50% the patients give a history of trauma[2] but the relationship is usually

not clear. A lowered general resistance has been incriminated as well[2]. There is a transient subclinical bacteriemia followed by organisms entering through the nutrient or metaphyseal vessels commonly of the long bones (femur, tibia, humerus). In the newborns the umbilical cord is a potential portal of entry for bacteria[2]. As the invading bacteria reach the metaphyseal veins and multiphy, the initial host response is an infiltration of polymorphonuclear leukocytes. There is an inflamatory exudate with increased vascularity and oedema. The combination of bacteria and leukocytes constitutes an abscess. Oedema in the area increases causing an increase in the intramedullary pressure and as there is no room for expansion between the rigid walls of metaphyseal bone, the swelling of the acute inflamation leads to thrombosis of nutrient vessels leading to ischaemia, capillary distruction, loss of circulation and subsequent death or necrosis of the trabecular bone within the abscess. The infection rarely spreads down the medullary cavity of the bone but rather will gain egress to the subperiosteal space in the metaphyseal area first. This is the path of least resistance because the cortex in this area is parous, like a maze and the inflamatory response blocks spread within the bone.

Within 24 to 48 hours the increased intramedullary pressure due to oedema and abscess formation, forces the fluid and organisms through the Harvesian and Volkmanns Canals[2] and elevates or lifts the loosely attached periosteum stripping it from the bone along with the blood supply of the underlying cortex thus inteferring with vascular connections to the cortex, that is, the inner cortex becomes ischaemic by virtue of periosteal lifting. the cortical bone dies leading to the formation of a sequestrum if the inflamatory process is allowed to proceed. The osteomyelitis is hence more of ischaemic in nature

rather than of infection. A subperiosteal abscess is thus formed, within the first 24 to 48 hours. Periosteal elevation is painful and since the periosteum is inelastic, the inflamatory exudate deep to it is under tension. As a consequence the patient rapidly develops marked pain and toxic signs. Subperiosteal pus tracks longitudinally and circumferentially around the bones stripping the periosteum and interrupting the periosteal vessels. Thus progressively larger areas of cortex become infarcted and involved in the inflamatory process. In the absence of treatment pus finally bursts through the periosteum and tracks through the muscles to present subcutaneously. Eventually the skin breaks down and pus discharges from a sinus which connects, the bone with the skin surface. The isolated necrotic bone within the abscess cavity is called a sequestrum. The periosteum which maintains its blood supply will begin to generate new bone which is responsible for the appearance of the periosteal new bone or the involucrum. In places where pus has broken through the periosteum to form a defect in it, sinuses develop which are represented in the involcrum by holes known as cloacae.

In cutaneous and subcutaneous infective foci, clinical evidence of abscess formation depends upon a finite mumber of bacteria per unit volume (10^5/mm)[2]. It is likely that similar quantitative factors apply in a focus of osteomyelitis. The fate of metaphyseal abscess resulting from this inflamatory process is dependent upon the virulence of the invading organisms and the resistance of the host[2] It may subside, become chronic or it may spread. The transition from acute to chronic inflamation is gradual without sharp clinical or histological dermacation[3]. In the absence of treatment death of some of the physis and metaphyseal bone occurs after organisms lodge in the medullary vessels. The dead bone seeded with bacteria can then act as a permanent reservoir for bacterial growth and

invasion and the formation of involucrum by primitive mesenchymal tissue of the periosteum further isolates the dead and infected bone. No antibiotics nor antibodies can penetrate the depth of the sequestrum and hence cannot eradicate the infection at this stage[2].

Some antibiotics may reach such tissue by diffusion[1]. The sequestrum may be reabsorbed by phagocytic cells in the granulation tissue. This process is slow and bacteria from necrotic bone cause the inflamation to flare up from time to time. More bone is destroyed and a vicious cyle develops. Spread of infection from the metaphysis involves bone resorption which takes place by both osteoclastic and non osteoclastic processes. Dekel and Francis[2] suggested that staphilococcus aureus might be able to synthesize prostaglandins that accelerate bone destruction. Rapid spread of infection along the entire length of the bone and absence of large sequestra and involucrum are typical in the adult because the periosteum is so firmly attached to the bone that it cannot be easily separated, as opposed to that in the child where periosteum is loosely attached.

The natural course of acute haematogenous osteomyelitis not treated with antibiotics will hence lead to the following course. First there is rarefaction of bone in which the bone becomes less dense. There is osteoporosis with periosteal reaction. These features appear radiologically only after the first 7 or 10 days of infection[2]. Secondly there is destruction of bone structure, sequestration and periosteal reaction with involucrum (newbone) formation. Lastly there may be attempts of the bone to heal. This results in ossification hypertrophy and osteosclerosis.

Several morphological forms of chronic osteomyelitis have been described. The common ones are sequestrum - involucrum type usually with sinuses, the Brodies abscess and Chronic sclerosing osteomyelitis of Garre. Bjorkstem et

al[16] have described a variety of chronic osteomyelitis which they named chronic recurrent multifocal osteomyelitis. The disorder is characterized by an insidious onset of fever, local swelling and pain in the affected bones and radiological abnormalities suggestive of osteomyelitis (osteolytic lesions). No organism was isolated both on aerobic and anaerobic cultures.

Special Clinical Forms of Chronic Ostemyelitis

Besides aerobic, anaerobic and sickle cell ostemyelitis which have already been mentioned, other clinical forms of chronic osteomyelitis include typhoid, brucella and fungal osteomyelitis. Spinal osteomyelitis has been reported in heroin addicts and in patients following minor procedures like cystoscopy[17].

Osteomyelitis in immune deficient patients has been documented in sporadic cases[17]. Osteomyelitis in these patients is not associated with open fractures, operative intervention or childhood osteomyelitis. This can be associated with chronic diseases like chronic gastrointestinal diseases such as regional enteritis, or it can be associated with heroin addiction, alcoholism or in diabetes mellitus[17]. It is possible that there might be an abnormality in phagocytic activity and suppression of the immune system. One of the patients in the present series had diabetes mellitus and presented with osteomyelitis of the tarsals and metatarsals with autoamputation of the toes. Osteomyelitis in AIDS has also been documented[18]. This underlines the need for orthopaedic surgeons to be aware of an increasingly prevalent disease.

DIAGNOSIS

Usually the clinical diagnosis of chronic osteomyelitis does not present major problems. In the present series the diagnosis on admission was the same as the final diagnosis in all the patients. The commonest symptoms in our series

were discharging sinus in 12/14 patients, local pain in 11/14 patients (see table 4). At the Mayo Clinic, the presence of an active sinus tract or fistula, has been associated with poorer prognosis[17]. In the present series the presence of an active sinus was the rule rather than the exception (12 patients out of the 14). Any age group can be affected depending on the cause.

Table 3: Age and Sex Distribution n = 14

Age	Number of Patients		Total
	Male	Female	
10 - 20	6	-	6
21 - 30	5	-	5
31 - 40	-	1	1
41 - 50	1	-	1
51 - 60	1	-	1
Total	13	1	14

The M/F = 13:1

Table 4: Presenting Symptoms

Symptoms	Number of patients
Discharging sinus	12
Local pain	11
Local swelling	4
Pyrexia	2
Limb deformity	3
Impairment of limb function	3
Autoamputation of toes	1

The commonest presentation were a discharging sinus and local pain.

Table 5: Site (n = 14)

Site	Number of Patients
Tibia	5
Femur	4
Fibula	3
Humerus	1
Foot	1

The commonest sites were the femur and the tibia.

Chronic osteomyelitis arising as a complication of acute haematogeneous osteomyelitis usually affects children or young adults. This was also reflected in the present small series where the peak age was between 10-30 years. Waldogel[3] found that 85% of patients were less than 16 years of age. Interestingly the majority of patients with chronic osteomyelitis are men. At the Mayo clinic men outnumbered women in a ratio of 2:1, during 1981[17]. In the present series most of the patients were men[13] (Table 3). All bones can be affected but the most commonly affected are the femur and tibia. The majority of the patients in this series (9/14) presented with a history of trauma and tibia. Since major fractures occur more frequently in the lower extremities[17] it is not surprising perhaps, that post traumatic osteomyelits affects predominantly the tibia and femur (see table 5).

The humerus is the third most commonly involved site of osteomyelitis. Other bone involvement accounts for about 20% of cases or less[11]. Osteomyelitis of the foot is also found, but this is ordinarily a consequence of puncture wounds in children or of ischemia or neuropathy in the elderly, often as a complication of diabetes[19,20]. In the present series there was one such diabetic patient with similar complications. Osteomyelitis of the hand or fore

arm almost universally is post trauma[17].

Typical of chronic osteomyelitis is its chronic course characterised by recurrent exacerbations. According to Hughes[21] chronic osteomyelitis may present in one of the forms:

(1) Recurrent flares without sinuses: in which case the original wound heals, the inflamatory process of the bone becomes quiescent for some time, only to reoccur at an interval of months or years without forming sinuses. An example is given when some men wounded in the 1914 - 1918 World War had their first recurrence as long as 25 years afterwards[21].

(2) Persistent sinuses without flares in which case the acute inflamatory process becomes quiescent leaving a permanently discharging sinus. the surrounding skin remains fairly healthy. The discharge is minimal.

(3) Persistent sinuses with flares. Here there is an infection of bone that is continually active and flares up every now and then every after few months as acute or subacute exacerbations.

This pattern of chronicity was also noted in our series. The duration of infection ranged from 3 months to 10 years with on average of 8.8 months (see table 6).

Table 6: Duration of symptoms at admission

Duration of Symptoms (months)	Number of patients
> - 1	2
> - 6	5
12	1
24	1
36	4
120	1

The majority of patients had symptoms of more than 6 months duration. A few had had several operative procedures. The duration of infection prior to admission was longer in patients with chronic osteomyelitis resulting from acute haematogeneous osteomyelitis than in patients with traumatic osteomyelitis. A similar pattern of chronicity was found at the Mayo clinic in which the duration of infection prior to admission was found to be longer in patients with haematogeneous osteomyelitis and in patients with fracture union than in patients with fractures with non union[17]. This is in contrast to the pattern of chronicity noted in Denmark[22] where the range was 3 to 540 months for the duration of infection. The duration of infection prior to admission was longer in patients with traumatic osteomyelitis than in patients with haematogeneous osteomyelitis. Long hospital stays and repeated admissions were common as it was in our series. Perhaps this feature of chronicity and lack of response to therapy with antibiotics has distinguished chronic osteomyelitis from other infectious diseases. Other authors believed that chronic osteomyelitis could hardly be cured, that it could only be controlled, and patients were warned that recurrence could occur any time in the future[21].

INVESTIGATIONS

All the patients in this series were investigated. Investigations included X-ray of the affected area, pus aspirate for culture and sensitivity. Full blood picture and sickling test. ELISA test for HIV was performed in 3 patients. Table 7 summarises the investigations done in this series.

Table 7: Investigations done (n=14)

Laboratory		No of patients
1. Blood test	- Raised WBC count1	1
	- Raised ESR = 155mm.hr	1
	- Low Hb - <10 gm/dl	6
	Sickling test - Positive - Negative	 1 1
	- ELISA test for HIV neg (n=3)	3/3
2. Bacteriology (results obtained in 3 pts)	- Staph. Aureus	8
	- Unidentified coliform	5
	- Proteus species	1
	- E. Coli	1
3. Radiological changes (n = 14)	- Sequastrum-Involucrum formation	12
	- Brodies abscess	1
	- Bone sclerosis	1
4. Biopsy = Chronic osteomylitis		1/1

Bactetiology: Every effort must be made to isolate the aetiologic agent. Results from the 8 patients whose specimen results were recovered showed that staphilococcus aureus was the commonest organism. Correct collection of specimen, culture technique and result interpretation are needed. The causative organism may be obtained by culture of pus found at operation, culture of an aspirate from the affected area, sinuses, or from culture from venous blood.

Waldvogel and Vasey[17] argue that direct bone aspiration or surgical biopsy is still strongly to be considered in patients with negative blood cultures, although Cole, Dalziel and Leitl[17] did not think this necessary. Specimen taken from a tract or from the sinus orifices may be misleading because skin contaminants are likely to be present[2]. Some advocate bacterial examination from several levels of the sinus tract. Both aerobic and anaerobic cultures and sensitivities should be performed. Haematological tests are not diagnostic and are inconstant, but may be supportive. The erythrocyte sedimentation rate is usually raised as is the white cell count which generally shows a neutrophil leucocytosis. Anaemia may be present. Jones et al[7] found that the erythrocyte sedmentation rate was frequently raised in all forms of osteomyelitis. We did not find it diagnostically reliable. Radiological investigations are valuable diagnostic aids. Plain Xrays are imprtant for the appreciation of the extent of the disease. They may show sequestra, involucrum, evidence of Brodies abscess or bone sclerosis. Tomograms may be helpful in identifying deep areas of bone destruction or for localisation of sequestra. Sinography can aid in the localization of the site and direction or course of the sinus or fistula tracts. This can help in the planning of the surgical approach.

Bioptic specimens should be taken to enable sort out problems of differential diagnosis. In this study a bone biopsy was done by the open method in one patient because he had an atypical presentation with rapid bone destruction. At operation no pus was found. A bone biopsy was taken for confirmation of the diagnosis and for ruling out a more sinister pathology. The histology however revealed features consistent with chronic osteomyelitis.

Bone scaning is also increasingly being used in bone infection. It will be able to localize occult infection.

TREATMENT

The objectives of treatment of chronic osteomyelitis have been:

(1) Elimination of all the dead bone, soft tissue and all the foci of infection
(2) Administration of appropriate antimicrobials
(3) Immobilisation of involved bone to prevent dissemination of infection and to allow healing to take place.
(4) To attain adequate skin coverage of the affected area.
(5) Rehabilitation of the limb.

All the patients in this series were managed surgically by complete debridement of the dead bone and soft tissue under the cover of antibiotics. The resulting cavities and the soft tissue defects were left open to heal by secondary intent. The cavity was gradually filled by granulation tissue and later covered by epithelial tissue growing in from the edges of the wound. Daily Eusol dressing was ensured. Antibiotics were continued for at least 4 to six weeks. Immobilisation of the limb was achieved by external fixation or more simply by a back slab. A few patients required preliminary preparations prior to surgery. This consisted in improvement of their nutritional status and correction of anemia by hematenics, blood transfusion and treatment of hookworm infestation whenever was necessary. All the patients had a good response. The average hospital stay was 3 weeks. A lot of delay however, was encountered in the preparatory period, either there was no available blood in the bank or the laboratory results took too long to come.

This method of management was found preferable to other methods because it is simple, cheap, attains good wound drainage, and allows for continuous inspection. One drawback of this method is the scar that might occur. This

is usually covered by thin epithelium which is easily damaged and offers no help to the revascularisation of the area so important in the prevention of recurrences - especially in the tibia.

Chronic osteomyelitis should not be regarded as merely an infection of bone. The disease also involves the soft tissue around (the skin, muscle, fascia, periosteum)[23]. The diseased bone is ischaemic or dead and is surrounded by sclerotic bone, such that even the most potent antimicrobials cannot reach the affected tissue[23,24,25]. That is the reason why successful treatment depends so much on complete removal of the ischaemic bone and tissues.

Some workers have advocated conservative management[21] in certain stages of chronic osteomyelitis; but it is generally agreed that the mainstay of treatment of chronic osteomyelitis is surgical, consisting in complete debridement of dead bone and soft tissue and appropriage antibiotic therapy. Immobilisation has also been emphasised to minimize chances of dissemination of sepsis, deformity and pathological fracture[26].

It is proposed that certain preliminary preparations of the patient should be fulfilled before any surgical intervention is contemplated. Chronic osteomyelitis is a long standing disease. The patients may be wasted or anaemic because of the longstanding infection[27]. Some form of general measures may be required to improve their nutritional status, to correct the anaemia before surgery is undertaken. The patients should be fully investigated. Preoperative antibiotics may be required. Many surgeons advocate the administration of antibiotics one day or 4 days prior to surgery to provide an effective blood level during operation; and their continuation in the post-operative period for several weeks. The operation itself will consist in complete excision of all dead bone, necrotic tissue and scar tissue till good bleeding margins are obtained taking

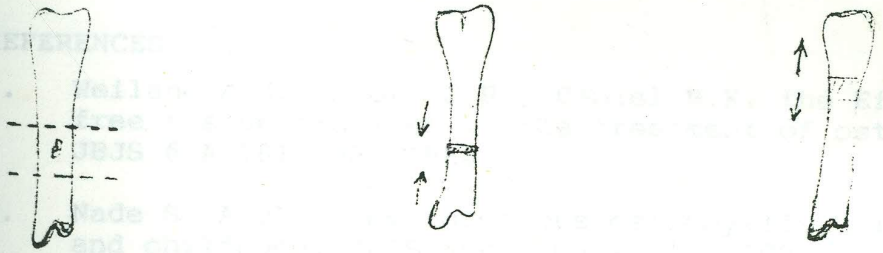
care not to weaken the remaining bone. If after debridement a cavity remains, several methods have been employed to obtain obliteration of the cavity for example by insertion of autologous or homologous bone grafts to the debrided cavity[11,28,29]. Other workers have obliterated the cavity by using transfer of a pedicle of skeletal muscle[11]. These methods have not been used at our centre. If the defect that results does not have a large dead space and if the surrounding soft tissues are free of scar and pliable then it may be possible to achieve primary delayed closure[11]. Debridement and delayed primary closure followed by closed irrigation and suction of antibiotic or of isotonic solution have been used[11,27]. Implantation of gentamycin Pemma beads into the saucerised cavity has also been employed with reported success[11]. The commonest cause of unsuccessful treatment has been failure to remove all the involved tissue and poor surgical technique[11].

Since chronic osteomyelitis is often times a sequel of acute haematogeneous osteomyelitis prevention of chronic osteomyelitis will consist in early detection and timely treatment of acute haematogenerous osteomyelitis by adequate doses of the correct antibiotics. It is recommended that the antibiotic therapy should continue for a period of 4-6 weeks.

Current Treatment of Chronic Osteomyelitis:

Up to until a few years ago the management of chronic osteomyelitis had been removal of the diseased bone and tissues; antibiotic therapy, skin grafting and so on. It has become apparent that removal of the diseased bone and soft tissue by debridement, sequestrectomy and saucerisation as done in the past did not attain complete removal of the affected bone in some cases[30]. That is why it was said in the past teaching that complete cure of chronic osteomyelitis could never be guaranteed[21]. The disease could flare up months or years after apparent successful

treatment[1.21]. The current recommended most effective mode of treatment for chronic osteomyelitis is radical surgical excision of the deseased bone[30]. The excision of the diseased bone should be "as radical as in tumour surgery" until good bleeding margins are obtained[30]. In well equiped centres the level of excision can be determined by scintigraphy. This radical excision may require the removal of a significant bone segment. In this case if the bone defect is small, it is reconstructed by compression. If the bone defect is larger reconstruction is done by compression followed by gradual distraction to correct the shortening (see diagram)



Resection of dead bone **Compression** **Distraction**

Bony instability is controlled by external fixation by various types of external fixators. The Ilizarov external fixatior has gained acceptance in a number of countries. It has got the advantages that the patient can start ambulating quite early; and allows for easy continuous inspection of the wound. Proper pin care is howerver, essential. Soft tissue coverage can be obtained by skin grafting. Most literature emphasises the importance of excision of sclerotic bone, dead bone and the involved soft tissues.

CONCLUSION

Chronic osteomyelitis still remains a scourge in many developing countries like Tanzania. It present real management problems because of late presentation to hospital or because of improper initial management of the acute phase of acute haematogeneous osteomyelitis.

The management of chronic osteomyelitis has changed over the years though the objectives of treatment have remained the same. The current approach now is more radical than before. Hence the excision of the diseased bone should be "as radical as in tumour surgery", sometimes requiring the removal of a significant bone segment.

REFERENCES

1. Weiland A.J., Moore J.R., Daniel R.K. the Efficacy of free tissue transfer in the treatment of osteomyelitis. JBJS 66A:181-193, 1984.

2. Nade S. Acute haemotogeneous osteomyelitis in infancy and childhood. JBJS 65B: 109 - 119, 1983.

3. Waldogel F.A., Medoff G. et al. Osteomyelitis, a review of clinical features, therapeutic considerations and unusual aspects. New Eng J. Med 282: 198 - 206, 1970.

4. Kennedy w.E. The prognosis of acute haematogeneous osteomyelitis with and without chemotherapy Surgery 16: 477 - 84, 1944.

5. Sabiston D.C. Davis Christopher Textbook Surgery 13th Ed. W.B. Saunders Co. Philadelphia, 1984.

6. Gerszten E. Allison M.J. Dalton H.P. An epidemiologic study of 100 consecutive cases of osteomyelitis. South. Med. J. 63: 365 - 67, 1970.

7. Jones N.S., Anderson D.J., Stiles P.J. Osteomyelitis in a General Hospital a 5-year study showing an increase in subacute osteomyelitis JBJS 69B: 779-83, 1987.

8. Adevokumu a.A., Hendricks R.G. Salmonella osteomylitis in childhood, a report of 63 cases seen in Niverian children in whom 57 had 57 sicckle cell anemia. Arch. Dis. Child 55: 175 - 84, 1980.

9. Huckstep R.L. The management of Osteomyelitis in East Africa. E.A. Med J. 45: 429-62 1968.

10. Finegold S.M. Anaerobic bacteria in human disease. New York Acad. Press, 1977.

11. West W.F., Kelly P.J., Martin W.J. Chronic Osteomyelitis 1- Factors affecting the result of Treatment in 186 patients. JAMA 213: 1837 - 1842, 1970.

12. Fierer, Joshua, Daniel, Dale and Davies Charles The Fetid foot. Lower extremity infections in patients with diabetes mellitus. Rev. Infect. Dis 1: 210 - 217, 1979.

13. Konugres G.S., Linda L., Goldstein EJC, Wallace S.L. Eikenella Corrodens as a cause of osteomyelitis in the feet of Diabetic patients JBJS 69A, 940 - 941, 1987.

14. Brooks G.F., O'donoghur J.M., Rising J.P., Soapes, Kenneth and Smith J.W., Eikinella Corrodens. A recently recognised pathogen. Infections in Medical Surgical patients and in Association with Methyl phenidate abuse. Medicine 53: 325-342, 1974.

15. Goldstein E.J.C., Barones M.F., Miller T.A., Eikenella Corrodens in Hand infections J. Hand Surg. 8: 563-567, 1983.

16. Bjorkstein B., Gustavson K.H., Ericson B., Lindholm A, Nordstroms. Chronic Recurrent Multifocal Osteomyelitis and Pustulosis Palmoplantaris. J. Paediatrics 93: 227-231, 1978.

17. Hughes S.P.F., Fitzgerald R.H. Jr. Musculaskeletal Infectious. Year Book Med. Publ. Inc. pp 136-150, 1986.

18. Crawfurd E.J.D., Baird P.R.E., An orthopedic Presentation of AIDS. JBJS 69B: 672-73, 1987

19. Fitzgerald R.H., Jr, Landells D.G., Gowan J.D.E., Osteomyelitis in children: Comparison of haemiotogeneous and secondary osteomyelitis. Can Med. Assoc. J. 122: 166-9, 1975.

20. Johanson P.H., Pseudomonas infectious of the foot following puncture wounds JAMA, 204: 170-172, 1968.

21. Hughes S. Sweetnam R. The basis and Practice of Orthopedics Heinnemann, 1980.

22. Damhott V.V. Treatment of chronic osteomyelitis. A

prospective study 55 cases treated with radecal surgery and Primary wound Closure. Acta Orthop. Scand. 53: 715-720, 1982.

23. Winters J.L., Cahen I. Acute haemotogenous osteomyelitis, JBJS 42A: 691-704, 1960.

24. Lack W., Bosch P, Arbes H. Chronic osteomyelitis treated by Cancellous homografts and fibrin adhesion. JBJS 69B: 335 - 337, 1987.

25. Daoud A., Bouaouina A.S. Treatment of sequestra, pseudoarthrosis and defects in the long bones of children who have chronic haemotogeneous osteomyelitis. JBJS 71A: 1448 - 1467, 1989.

26. Esterhai J.L., Sennett B., Gelb H. Treatment of chronic osteomyelitis complicating Nonunior and segment defects of the Tibia.

27. Clawson D.K., Stevenson J.K. Treatment of chronic osteomyelitis surg., Gynaec. Obst. 120: 59 - 69, 1965.

28. Rawling D.E. The positive approach to chronic osteomyelitis JBJS 41B: 681 - 688, 1959.

29. Wilson T.E. The treatment of chonic osteomyelitis Surg. Gynae. Obst. 98: 73 - 83, 1954.

30. Prof. Kinzyl. Lecture on Management of chronic osteomyelitis at Muhimbili Medical Centre. Novemer 1992.

OSTEOSARCOMA

SUMMARY

The six osteosarcoma patients presented in this study were admitted to Muhimbili between October 91 and April 1992. Five of the patients were referral cases from upcountry hospital. Only one came from Dar es Salaam. The ages ranged from 7 to 26 years. Average - 17 years. Four were males and 2 were females. The male to female ratio was 2:1. Three patients came from Iringa. The distal femur was affected in 5 patients. One patient had the tumour localised in the distal humerus. The presenting symptoms were pain and swelling. Surgery was the mainstay of management. Four patients underwent limb amputation: 2 "early amputees" were alive at 3 months follow up with no evidence of lung or local recurrences. Two "Late amputees" had radiological evidence of lung metastases at 3 months follow-up but had no local recurrences. One patient refused amputation. The other one had advanced disease and was unfit for surgery. the fact that six patients with osteosarcoma were observed in a 6 months period does not mean that osteosarcoma is common at Muhimbili. Five of the patients were referral cases from upcountry hospitals. Only one patient was a resident of Dar es Salaam.

INTRODUCTION

Osteosarcoma is a malignant tumour arising from mesenchymal cells of bone. The malignant proliferative cells produce osteoid tissue. The importance of osteosarcoma lies in several factors. Firstly, it is reported that bone tumours account for roughly 1.5% of all malignancies. Osteosarcomas are the most frequent tumours (30%) (1) according to one report, second only to multiple myeloma. Secondly, as opposed to multiple myeloma which affects older patients osteosarcoma affects young innocent

young men in their teens and early twenties. The distress caused to the patient and family members is enormous. Thirdly the tumour has got a very lethal biological behaviour with poor prognosis. It is this lethal biological behaviour that makes the management of this tumour one of the most challenging subject to the surgeon. Fourthly, osteosarcoma is a composite tumour arising from about nine cell types. It thus presents a variability of histological characteristics making it one of the most challenging subjects in terms of clinical and morphological diagnosis.

CASE REPORT

E.K. was a 19 year old female who was referred to the Muhimbili Medical Centre from Iringa, with a 2 months history of a painful swelling in the proximal part of the right leg. The pain was spontaneous,and was of insidious onset, dull in character and intermittent. It gradually became worse with time and was worse at night. Later it was no longer intermittent but continuous. In a months time there was an obvious swelling in the proximal part of the leg and the pain had become incapacitating. The knee joint was not affected. There was no history of trauma or fever. History of cough and bony pains were denied by the patient. She admitted to have lost some weight but not much.

Clinical examination revealed a young girl, who was not wasted, afebrile . She was neither pale nor jaundiced. The blood pressure was 120/70 and the pulse was 88/minute, regular and of good volume. She was not in severe pain. No peripheral oedema or lymphnodes could be detected. The respiratory and cardiovascular systems were normal. The abdominal examination was normal. The liver and spleen were not palpable. The inguinal lymph nodes were not palpable. Local examination revealed a fusi form swelling in the proximal third of the right leg. The overlying skin was of normal appearance. There were no distended veins or any

ulcerations. The right knee joint was not swollen. The joint movements were not impaired. The swelling was firm in consistency and was tender on palpation. The temperature of the surrounding skin was equal to that of the equidistant area on the contralateral leg. The muscle circumference was 5 cm. larger than the contralateral leg at equidistant level. No bruit or hum was detected.

A provisional diagnosis of osteosarcoma was made. The following investigations were ordered. A plain Xray of the right leg including the knee joint showed evidence of a bone forming tumour involving the proximal metaphyseal part of the right tibia. A characteristic sunray. appearance was noted. The tumour was extending into the soft tissue. No typical features of Codmans triangle could be determined on the Xray. The knee joint was not involved. the chest xray anteroposterior and lateral views both in inspiration and expiration did not show evidence of metastatic lesions or any other abnormality.

The full blood picture showed:

Haemoglobin = 12.6 gm/dl

Total white blood count = 5.700

White cell count = differentials =

Neutrophils = 65% lymphocites 21%

Basophil 1% monocytes = 10%

Eosinophils = 3%

Serum alkaline phosphatase - 196 u/l

proteins 57.1 g/l

- Serum alanine Aminotransaminase = 4 1.u/l
- Serum aspartate Aminotransminase = 10 i.u/l

An incisional biopsy was taken from the tibial bone tumour under general anaesthesia in the theatre. The histological results confirmed the diagnosis of osteosarcoma.

In the abscence of chemotherapy at Muhimbili at that time an above knee amputation was recommended as the only

mode of treatment available. However consultations with the patient and relatives proved futile as the patient refused amputation and requested to be discharged.

Three months after the patient was readmitted to hospital and she had come to terms with the question of amputation. However, the lesion had dramatically increased in size. This time it was involving the right knee joint with radiological evidence of tumor extension to the knee. There was impairment of movements at the knee joint.

An above knee amputation was performed. The post operative period was uneventful and the wound healed primarily. The patient was discharged home on the forteenth postoperative day.

The patient was seen again at the follow up clinic three months after surgery. She was in fair general condition. She had no signs of local recurrence but the chest Xray showed two suspicious 1 cm round opacities in the middle lobe of the right lung. These were highly suspected to be metastatic secondaries to the lung. She was however not complaining of cough or chest pain.

DISCUSSION

INCIDENCE

Most Western literature indicates that the incidence of osteosarcoma is about 0,2 per 100,000 persons[2]. In the United States the incidence of osteosarcoma is reported to be 2.8 in 1000,000 of the population[3]. In Kenya available data indicates that the incidence is about 0, 17 per 100,000 persons per year[2]. No reliable data is available for Tanzania but Charles[4] observed an incidence of 0,2 and 0,29 cases per 100,000 population per year. Whites and blacks are equally affected[5]. In this series all the patients were black indigenous Africans. Males are more often affected than females. The reported male to female ratio is

2:1 in Western countries[5]. Other series have reported a male to female ratio of 1.3:1(3). It seems that before puberty both sexes are equally affected.
Male predisposition becomes apparent past puberty with active bone growth[6]. In this series 4 of the patients were males and 2 were females. The male to female ratio was 2:1 Osteosarcomas tend to occur during the period of active skeletal growth. During childhood and adolescence as is well shown by the present series. They usually affect young persons in their second decades before the epiphyses have closed. They are rare before 10 years of age. However, any age can be involved. The youngest patient in this series was 7 years old.

Table 1: Age Sex Regional Distribution

Patient	Sex	Age	Domicile
1. N.M.	M	26	Kilimanjaro
2. E.M.	F	19	Iringa
3. N.R.	M	15	Mbeya
4. S.M.	M	13	Iringa
5. D.N.	F	12	Dar es Salaam
6. D.K.	M	7	Iringa

The M/F was 2:1, The age range was between 7 and 26 years, average 17 years. The largest number of patients came from Iringa.

Various researchers from different countries have found a slightly higher incidence of osteosarcoma in urban populations than in the rural population[4]. Charles reviewing a series of 100 cases in Tanzania found that the highest incidence was in Kilimanjaro and Iringa region[4]. The majority of our patients came from Iringa (3 cases). There is no known reason for the apparent predilection of these areas.

AETIOLOGY

The aetiology of osteosarcoma is still unknown. Several hypotheses and theories have been put forward. Geschickter and Copeland[7] in 1949 postulated the theory of Arrest of Histiogenesis to explain the origin of various bone tumours among which osteosarcoma is but one. He postulated that primitive mesenchymal cell rests (from which bone originates) differentiate into specialized cells like chondroblasts, fibroblasts and osteoblasts which are responsible for bone formation, regeneration and repair.. He said that interruption of histogenesis at any one stage would bring about the occurrence of bone neoplasm. It was believed that this theory might probably explain the high early peak incidence during the active phase of skeletal growth (in the second decade of life).

Several other factors have been incriminated. These can be grouped into predisposing factors and precipitating factors[8].

Predisposing factors: Age, sex and certain anatomatical sites (more than 90% of the primary tumours arise in the metaphyses of major tubular bones) may be predisposing factors[9]. No reason has been found to explain the particular predisposition of the bones around the knee area. A genetic predispotion has been incriminated in individuals with genetically determined retinoblastoma, multiple enchondromatosis and multiple osteosarcomatosis[10,11,12]

Precipitating factors: Various RNA and DNA containing viruses have been incriminated. DNA viruses include the FBT mouse virus, SV40 and the polyoma virus. The RNA viruses include the Harvey and Maloney mouse sarcoma viruses and C-type RNA virus. They have been found to induce bone tumours in animals[9]. Presently no epidemiological evidence is available that these are responsible for human osteosarcoma. Exposure to radiation especially of actively proliferating cells ("osteoprogenitor cells") which are most sensitive to

radiation has also been thought to cause osteosarcoma[9]. Clinically it is uncommon to find sarcomatous lesions developing within an irradiated field following treatment for benign or malignant conditions but a long time follow-up of these patients would be recommended.

Cohan et al[13] described 11 cases in whom osteosarcoma developed 6-12 years after radiotherapy. X-emitters were more oncogenic than B-emitters. Strong B particles are more oncogenic than weak B-particles. Radio muclides with a long biological half life are more oncogenic than those with short half life[9]. Metal and plastic bone implants have also been thought to cause osteosarcoma but no conclusive proof is available[14].

Various organic and inorganic chemicals like cholanthrene and beryllium compounds have been implicated in experimental animals[9]. The spleen is thought to have tumour preventing effect[9]. Trace elements or their chemical compunds in air, food and water may play an important role in the causation of osteosarcoma in man. Immune mechanisms may be implicated as well[6]. None of these aetiological factors could be demonstrated from the history and physical findings in this series. No further attempts were made to pursue thesubject. It is interesting however to note that the majority of our patients (3/6 cases) came from Iringa Region. In his series of 100 Tanzanian osteosarcoma cases Charles[5] noted that a good percentage of them came from Kilimanjaro and Iringa regions. Unidentified environmental factors cannot be ruled out in these regions.

PATHOLOGY

It has been demonstrated that osteosarcoma is actually a composite tumour consisting of up to 9 cell types[15] probably arising from a common multipotential mesenchymal tissue. These cells are osteoblasts at different stages of

differentiation, fibroblastic cells, anaplastic cells, angioblastic cells, hystiocyte - like cells, myofibroblastic cells, Ewing's sarcoma like small cells and lipoblastic cells.

Characteristic of osteosarcoma cells is their ability to produce a matrix containing substance "osteoid" at least in somefoci. Also histochemically the cells are capable of elaborating demonstrable abudant alkaline phosphatase in their cytoplasm. Osteosarcomas have been subclassified on various bases[6]:

1. On the basis of their clinical setting in which they appearas:
   (a) Primary osteosarcoma
   (b) Secondary osteosarcoma
2. On the basis of their presumed point of origin in relation to bone, they can be:
   (a) Conventional (or medullary)
   (b) Parosteal (juxta cortical)
   (c) Periosteal
   (d) Intracortical
   (e) Extraskeletal (arising in soft tissue)
3. On the basis of their dominant histomorphological feature you can have:
   (a) Classical Osteoblastic osteosarcoma
   (b) Classical chondroblastic osteosarcoma
   (c) Classical fibroblastic osteosarcoma
   (d) Telengiectatic osteosarcoma

The majority of these tumours arise de novo in the apparent abscence of underlying bone desease or recognised carcinogenic influences[6].

Secondary Osteosarcomas: These develop against a background of preexisting bone pathology or previous exposure to some potentially carcinogenic influences like.- Paget's Disease of bone, chronic osteomyelitis,- Multiple enchondromatosis, multiple osteochndroma, fibrous dypplasia, bone irradiation,

infarcts and fractures. Secondary osteosarcomas rarely arise in childhood or in young adult life especially when the neoplasms are super-imposed on Pagets disease. Association of Pagets disease with osteosarcoma is usually noted in elderly individuals[2].

In the present series all the tumours arose from locations which showed no apparent background pathology in the bone (de novo) and in relatively young patients. However, this is a rather small study group to draw any reliable conclusions. Furthermore Pagets disease is a rare entity in Tanzania[5]. Charles noted that out of the 100 Tanzanian osteosarcoma cases 94% of the primary lesions arose de novo. In 5 cases two primaries arose from fibrous dypplasia and two from osteochndroma[4]. In their series, Huvos et al[15] recorded 85% of osteosarcoma primaries arising de novo in the intramedullary space. In the United States although the risk of developing a neoplasm with Paget's disease is only 1%, the frequency of this skeletal disorder in older individuals accounts for the fact that approximately 10-15% of all osteosarcomas arise in persons over the age of 30 years[5]. In 10% of the cases of Pagets disease sarcomotous changes occur and the prognosis in this disease is one of the poorest. Osteosarcoma in Paget's disease has got the lowest rate of survivals of any of the tumours of the sarcoma type series[5].

Location: Most conventional osteosarcomas arise in the medullary cavity of the metaphyseal end of the long bones of the extremities. the following bones are affected in a decreasing order of frequency[3].

- The distal end of the femur, 52%
- the proximal end of the tibia - 20%
- the proximal end of the humerus - 9%

Other authors say that the humerus is a preferable site second to the femur. The radius, ulna, ilium and the scapula are less usual sites of primary osteosarcoma. The

short bones are rarely affected. However in persons over the age of 25 the incidence in flat bones and long bones is almost equal[2]. Table 2 shows the anatomical locations of the primary lesion in the present series.

Table 2: Anatomical location of tumour (n=6)

Location/Site	Number of patients
Distal femur - right	2
left	3
Distal Humerus - right	1
left	0

The distal femur was affected most commonly.

Macroscopy: Osteosarcomas show diverse forms and character depending on the amount of the osseous component. When it forms abundant tumour bone it is called sclerosing sarcoma[5]. When it forms little bone it is called osteolytic[5]. Regardless of its histological variablility an osteosarcoma of any type presents a typical clinical pattern:

Usually 50% of the tumours show heavy ossification containing sufficiently large amounts of osteoid to merit the name of osteoblastic or sclerosing osteosarcomas[5]. The tumour tissue here may be hard and have a gritty feeling. About 25% of these tumours show moderate degree of ossification showing predominant chondroid differentiation. The remaining quarter or 25% show relatively little ossification reflected radiologically. They are largely fibroblastic and have a more typical fish-flesh sarcomatous appearance. The tumour penetrates the cortex and produces a triangular new bone formation which lifts the periosteum for a short distance and produces the characteristic Codmans triangle seen on the X-ray. This is the angle between the

plane of the outer surface of the cortex and the elevated periosteum. Also new bone formation may be arranged in the form of specules radiating perpendicularly from the periosteum giving the "Sunray appearance" visible on radiography. These radiographic features are characteristic of osteo-sarcoma though not pathognomonic. Radiological features in this series was revealed both osteosclerotic and lytic lesion almost in equal proportions. Some features of Codman's triangle and sunray appearance were detected (see table 4).

Microscopy: It has been mentioned earlier that osteosarcoma is a composite tumour consisting of at least nine cell types probably all of common origin[14]. These cells present a diverse degree of pleomorphism which is most marked in the extremely anaplastic tumour.

About 37% of all osteosarcomas are osteoblastic that is, present with osteoid as an important element in samples taken from most areas of the tumour[16,17]. About 18.5% of the tumours are chondroblastic osteosarcoma, that is, they present with predominantly malignant chondroblastic cells producing chondroid matrix besides the presence of malignant osteoid. Another 18.5% of tumours are fibroblastic osteosarcomas. These ones show predominantly malignant fibroblastic cells and fibrous matrix except for small foci of malignant osteoblasts prodicing osteoid.

Therefore classic or conventional osteosarcomas are high grade malignant tumours arising fron no obvious previous bone pathology. the three major types are osteoblastic, chondroblastic and fibroblastic and they constitute about 75% of all the variants of osteosarcoma put together[18].

Spread: Osteosarcomas can spread by the haematogenous route, by the lymphatic spread and by local spread.

Haematogenous Spread: Is the mojor route by which ostesarcomas can spread to the lungs and to other osseous sites. Though they are not detectable in early stages,

secondary metastases to the lung occur quite early. In fact extensive micrometastases are already present by the time the primary becomes manifest[19]. This point of view indicates the main problem as regards to the management of osteosarcoma. However, early the diagnosis is made by the available diagnostic methods, osteosarcoma is already a systemic disease in the majority of cases. The prognostic outcome of the tumour as a disease is dependent upon the quality and quantity of these systemic micrometastatic cells. The primary tumour per se plays a minor role. Approximately 20-30% of patients have demonstrable metastases when first seen, and more than 90% of those who die of neoplasm have metastases to the lung[5]. Late metastases to other bones occur in about 15% of cases[20]. Two of the patients in this series had radiologically detectable lung metastases at presentation (see table 4).

Synchronous multifocal osteosarcoma metastases: This is the simultaneous development of primary osteosarcomas in different bones. Other authors think that this is due to a rapid development of multiple skeletal metastatic lesions from the primary tumour.

Metachronous multifocal osteosarcoma: This is the later development of primary osteosarcomas in different bones.

Lymphatic Spread: Though this is rare it is another mode of spread Lymphatic spread to regional lymphnodes is possible and denotes a poor prognosis. "Skip" metastases can also occur. The significance of the palpable inguinal lymph nodes noted in one of our patients could not be determined for certainity. Local spread is also possible by local invasion.

Staging: Bone tumours are not yet classified according to the TNM system[1]. Enneking et al[22] have described a system of staging - The Surgical Staging System (S.S.S.). This was later adopted by the Musculoskeletal Tumour Society (USA). This system takes into consideration the grade behaviour,

local extent of the primary tumour and the types of metastases, whether local or distant. According to this system a tumour is assessed by the following factors:

(1) Surgical grade (G)

Go = Benign tumour

G1= low grade malignant tumour and

G2= high grade malignant tumour

(2) Local extent of primary tumour (T)

To= intracapsular tumour

T1= extracapsular intracomportmental;

T2= extracapsular extracompartmental

(3) Regional or local metastases (M)

Mo= no metastases detectable

M1= Regional or distant metastases detectable

Stage - Each stage is sub-divided into:

A = intracompartmental

B = extracompartmental

Stage IA - G1 T1 Mo - a low grade (D1) extracapsularintracompart-mental (T1) sarcoma without metastases (Mo).

Stage IB - G_1 T_2 M_o

- A low grade (G_1), extracomportmental (T_2) sarcoma without metastases (M)

Stage IIA - G_2 T_1 M_o

- A high grade (G_2) malignancy,
- extracapsular, intracompartmental (T_1) without metastases (Mo)

Stage IIB - G_2 T_2 M_o

- A high grade (G_2)
- Extra capsular extracompartmental (T_2) - without metastases (Mo)

Stage III - G_1 (or) G_2 T_1 or T_2 M_1

- A sarcoma of either grade G_1 or G_2 or eitheranatomical setting T_1 or T_2 with distant metastases (M_1).

CLINICAL FEATURES

Table 3: Shows the clinical presentation of the patient in this series

	Pain	Swelling	Wt loss	Fever	Anaemia	LN	TM
1. N.M.	+	+	+	-	+	-	-
2. E.K.	+	+	+	-	-	+	-
3. NR	+	+	-	+	+	-	+
4. S.M.	+	+	+	-	+	-	-
5. D.N.	+	+	+	-	-	-	-
6. D.K.	+	+	+	-	-	-	-

LN = inguinal lymph nodes

TM = Therapeutic marks

Pain: is probably the most common presenting symptom. At first the pain is intermitent, usually more severe at night and later becomes continuous and persistent. It is caused by periosteal irritation. Occasionally these tumours remain entirely silent and are called to clinical attention by the sudden fracture of the involved bone[5]. Intermitent pain which is more severe at night is typical of osteosarcoma. Other authors say that a pathological fracture is not typical of osteosarcoma since pain and swelling keep the patient from walking. Sometimes there is some initial pain and effusion into the nearest joint but movement is usually free and painless. Tenderness and swelling of the affected parts are also common, sometimes with local heat and venous engorgement. Tenderness and swelling were ellicited in all of the patients in this study. Occaasionally one may be able to elicit a "bruit" or "hum" in the telengiectatic type. Rapid increase in size due to rapid growth causing progressive expansion and enlargement of the limb is characteristic. The overlying skin may become stretched and glossy as it exhibits distended veins. Usually it does not

ulcerate local inflamatory signs and general constitutional signs are usually absent.

Site: (see also location under pathology above) Almost 70% of all these tumours arise close to the knee[3]. In a series of 100 Tanzanian osteosarcoma cases Charles found involvement of the distal femur in 46% of cases and proximal tibial metaphyses in 27% (- total 73% of cases). The proximal humerus was the next most common site (6%).

Duration: The duration from the onset of symptoms to the time of diagnosis of the tumour may be variable from a few weeks to several months[22] depending on several factors like severity of pain, rapidity of increase in size of swelling, availability of medical facilities the educational level of the patient and the index of suspicion of the attending doctor. In present series the patients presented with a 2-8 months of symptoms.

Onset: is usually spontaneous and gradual. Occasionally trauma precipitates symptoms. Infrequently one may find a pathological frature. Up to 50% of patients give a history of trauma but its significance is questionable[4,23]. One of the patients claimed to have a trivial trauma to the leg which precipitated the symptoms.

INVESTIGATIONS

The investigations done in this series included Xray of the primary tumour and chest Xray, Alkaline phosphatase, Haemoglobin and biopsy for histological confirmation of the lesion (see table 4). The Xray findings are an important aid in the diagnosis of osteosarcoma.

A plain Xray of the primary tumour as well as a chest Xray, both preferably in the anteroposteior and lateral views may give important information about the location and extent of the primary tumour and its overt metastases in the lungs. Although pulmonary metastases are invariably present as micrometastases they are rarely demonstrable when the patient is initially seen. Characteristic of the primary

tumour but not pathognomonic are the following radiological changes:

The Codman triangle which is a radiological feature due to tumour extension. (see pathology above). This anatomical feature is visible on the Xray. It is characteristic of osteosarcoma though not pathognomonic because this is actually normal bone formation, not orignating from malignant osteoblasts and can be caused by any condition that leads to periosteal reaction. The "sun-ray" appearance: Especially in advanced cases of osteoid producing osteosarcomas spicules of mineralised (malignant) osteoid are formed perpendicular to the surface of the bone giving rise to a "whisker-like" and later a "sun-ray" appearance. A plain Xray can also identify evidence of pathological fracture.

Skeletal survey can be done to determine metastatic lesions.

Computerised tomography - is an extremely valuable method to evaluate the tumours, their reactive zones and their metastases.

Tomography of Primary Tumour and lung: Will show the primary tumour in detailed perspectives and may reveal tumour nodules in lungs not exposed by chest Xray.

Isotope Bone Scan: Can reveal solitary or scattered skeletal metastases.

Radioactive Scan with technitium Poly phosphate: Can give details about the extent of primary tumour and its reactive zones. "Hot" scan indicates increased vascularity in the tumour or around the tumour; or increased activity in malignant cells actively forming osteoid.

Regional Angiography: May show neovascular formation in tumour and its reactive zone. It will accurately define intraosseous and extraosseous extend of the tumour. Open biopsy for histological examination. This is an important investigation inorder to make a definitive diagnosis. The specimen must be taken from the most representative tissue.

Enneking et al[24] have suggested that open biopsy should be done at the same sitting as the definitive surgical procedure. Proponents of this opinion argue that this reduces the chances of dissemination of the tumour. However, such precautions have shown no advantage[25]. Liver function tests will determine whether or not hepatic metastases are present.

Needle aspiration Biopsy. Is useful especially in inaccessible sites such as the vertebral column. Other tests include: histocytochemical studies to determine alkaline phosphatase, immunological tests to determine osteosarcoma associated antigens, determination of acid and alkaline phosphatase, and Bence Jones proteins for multiple myeloma.

TREATMENT

Finding an effective treatment of osteosarcoma has been one of the most challenging problem to the surgeon. Treatment should aim for cure whenever feasible, control of the primary tumour and prevention of metastases. But in most patients palliation is the only option[4,26] either because of late presentation or because of the lethal biological behaviour of the tumour itself. The treatment modalities available are: surgery, chemotherapy and radiotherapy.

Surgery was performed in 4 patients in this series. Two underwent above knee amputation, one had a hip joint disarticulation, and the other one had amputation of the humerus. One patient categorically refused amputation, and in the absence of an alternative therapy she was discharged home. The 6th patient had an advanced disease involving the whole thigh with radiological evidence of multiple lung lesions. He was discharged home for medical palliation at the request of the relatives. A combination therapy of surgery and chemotherapy was considered optimal for most of

these patients, but was not available for any of the patients at the time of the study. The biggest problems encountered in the study were that one had to spend much time trying to persuade the patient or the relatives to accept amputation. This resulted in unnecessary delays, as it occurred in the patient presented in detail (see case report). Secondly there is also considerable diagnostic delays especially when a biopsy is to be repeated. Thirdly availability of chemotherapy is unreliable and quite erratic. When it is available often times it is too expensive for the patients to afford.

There is still controversy about the level of amputation[2]. High amputation has been associated with stamp recurrence in 4-16% of cases (average 10%)[2] before the era of adjuvant chemotherapy. As some of the patients do not survive the 5 years and beyond, this figure could be even higher had the patients survived the 5 years and beyond. Disarticulation is thought to be a rather multilating operation though it is associated with negligible recurrences. But of course the subsequent disability is a great deal more, should the patient survive. Opinions were equally divided between the advocates of disarticulation and those who preferred leaving a stump accepting the small risk of recurrence in order to provide a more suitable limb for the survival.

Principally radical amputation should be done proximal to the tumour, leaving the least amount of residual tumour. Theoretically it would appear that extensive resection including the entire affected bone would appear to offer the greatest chance of eliminating the disease. Other surgeons argue that the 5 years survival rate is not affected regardless of the level of amputation[4].

With the introduction of chemotherapy the chances of stump recurrence is likely to be reduced even further. In these circumstances many surgeons advise that the femoral

stump should be preserved when possible. Disarticulation should be reserved only for the less common larger and more proximal tumours where it is impossible to devide the femur high enough above the growth and yet leave the minimum acceptable length of femur for limb fitting which is 10cm. in adults, measured from downwards from the ischial tuberosity[2]. Such was the case in one of the patients in this series who underwent hip joint disarticulation. In the case of distal femoral lesion for example many advocate high transfemoral amputation[2] at the junction of mid third and proximal third, in order to avoid medullary extension of the tumour). The level of amputation from the tumour is made to pass at least 10cm (4 inches) above the level of the most proximal extension of the tumour as dermacated by CT scan when available. Wound margins and proximal marrow are histologically checked for the presence of tumour infiltration.

Radical amputation should be followed by adjuvant therapy like chemotherapy or radiotherapy with the aim of controlling microresidual systemic tumour load[4]. Cade's humane method[2] (initial full course of radiotherapy to the pirmary tumour followed by amputation six months later only in patients still free of metastases) is no longer acceptable because it carried the risk of local recurrences causing severe pain, ulceration and loss of function in those patients spared of amputation. The result was that the patients suffered from pain and more misery than would have had with primary amputation. The other problem with Cade's humane method was the difficulty of persuading parents and patients to accept the necessity of amputation when the primary tumour appeared to have responded to radiotherapy and the lungs remained clear of metastases. **En block or local radical resection** was advocated as a limb saving procedure. Its major disadvantage is that it almost invariably leaves behind microresidual tumour seedlings

especially in form of "skip metastases. Hugues[2] is of the opinion that except for the juxtacortical (parosteal) or low grade endosteal variety, there is no place for local resection in the treatment of osteosrcoma[2].

Local resection may have some role in the treatment of tumours involving the axial skeleton (e.g. osteosarcoma of the skull, pelvis). In these circumstances local resection might be the only surgical option. The surgical procedure should be followed by adjuvant radiotherapy and chemotherapy.

Pulmonary Resection: There is substantial evidence in support of increased survival rate and cure by thoracotomy with removal of lung metastases with or without chemothrapy[27]. Thoracotomy plus wedge resection of the nodules followed by chemotherapy has been indicated when the metastatic lesion is single and does not appear until a year after the definitive surgical procedure for the primary tumour or following stoppage of adjuvant chemotherapy.

Chemotherapy: Some of the patients in this series could have benefited from adjuvant chemotherapy. However chemotherapy was not available at the time of the study. Formerly osteosarcoma was regarded as a tumour refractory to chemotherapy. This was due to the then existing antitumour agents. Presently chemotherapy has been used as adjuvant therapy with some success[8]. The rationale of adjuvant chemothrapy lies in the fact that by the time diagnosis is made osteosarcoma is already a systemic disease with micrometastases to the lung[19]. Chemotherapy is aimed at having a systemic effect for an already systemic disease. Many chemotherapeutic agents have been used: Methotraxate, vincristine, cyclophosphamide, adriamycin, cisplatinum are but a few examples.. Various combination protocols are available. Methotrexate is probably the most popular of these. In high doses it inteferes with the intracellular metabolism of folic acid. High doses of methotrexate are

used (6,000mg - 12,000mg/m^2) in combination with citrovorum factor (reduced folic acid to protect the normal cells). The dose is 40mg/24 hours in portions of 10mg given 6 hourly i.v. or orally. Vincristine 1.5mg/m^2 also may be alternated with Doxorubicin 70mg/m^2 and cyclophosphamide 200mg/m^2 at 2 weekly intervals. Other workers have used a lower dose of methotraxate 200mg/m^2 with citrovorum factor rescue[26]. It may be alternated with Doxorubicin. Goarin et al[28] obtained a 5 year relapse free rate of 44% with high dose methotrexate and citrovorum factor rescue; that with adriamycin alone was 39%, while a combination of two drugs gave a 5 year relapse free rate of 59%. When Rasen et al[29] added 3 more drugs (bleomycin, cyclophosphamide and dectinomycin - BCD) to their regimen, the 5 year relapse free rate approached 70%. Chemotherapy is effective as adjuvant therapy when the bulk of the primary lesion has been controlled by surgery because it is effective against small tumor load and is more likely to eradicate occult rather than overt secondaries[4]. Preoperative chemotherapy has been advocated[20]. The rationale is to achieve a higher disease free rate in osteosarcoma through the use of early systemic treatment. The major drawbacks of chemotherapy are:

(1) Toxicity - whose manifestations include nausea vomiting, and transient alopecia.

(2) Cost the high costs of these drugs make their use difficult especially in poor and developing countries.

(3) Carcinogenesis - the possibility of carcinogenesis is not yet well documented[4].

Radiotherapy

Radiotherapy was not employed in this series. Osteosarcoma has previously been regarded as being radiation resistant[8]. However, with the use of megavoltage

radiotherapy it is now possible to cause destruction of tumour cells with minimal effect upon uninvolved parts[8]. Radiotherapy alone is however, less likely to bring about survival rates comparable to total ablation of the tumour. It is however an important method of treatment of primary tumour in locations which are not accessible to surgery like the spine, axial skeleton or skull[4] as compared to 20-25% with surgery alone.

In a series of 33 patients, Chamber et al[30] using preoperative radical dose radiotherapy followed by radical surgery have obtained a 5 year survival rate of 73%. Prophylactic whole lung irradiation (1750 rads over 12 days) has been found to prevent subsequent development of lung metastases in some patients and may improve long term survival.

Combination therapy with surgery, chemotherapy and radiotherapy has produced an overall survival rate of 65-80% at 3 years[26] In patients with no evidence of macrometastases.

PROGNOSIS AND RESULTS

Osteosarcoma hss a poor prognosis. The gloomy prognostic picture is also depicted by the results of treatment in this series (see table).

Table 5: Results

Results	No of patients
"Early immedicate amputee" Alive and on follow up for 3 months had no distant metastases or local recurrence	2
"Late amputee" Alive and on follow up for 3 months had rediological metastatic lesions to right lung. No local recurrences	2
Lost to follow up	1
Had advanced disease discharged on medical palliation at the request of relatives	1

The gloomy prognosis is even complicated by late presention and the use of a single therapeutic modality by surgery alone in this series.

The patient lost to follow up was the one who had categorically refused amputation.

CONCLUSION

Osteosarcoma is a bone tumour with a lethal biological behaviour. Its prognosis is poor and finding an effective treatment of this tumour has been one of the most challenging subjects to the surgeon especially in developing countries where the therapeutic options are limited.

Since surgery, by radical amputation of early tumours, is the treatment of choice in Tanzania, early detection of the tumour is essential in order to offer the greatest chance of eliminating the diseases.

REFERENCES

1. Clinical Oncology - Edit by Committee on Professional Education of UICC. International Union Against Cancer. Publ. by Springer - Verlag B (1973) pp 256.

2. Hughes S, Sweetnam R, The basis and Practice of Orthopedics. Publ. by Heinemann pp 96-99, 1980.

3. Schwartz S.I. Principles of Surgery - 5th Edition pp 2007,1989.

4. Bovil E.G. Jr., Kungu A, Bencivenga a, Jeshrani, M.R.,Mbiridyo B.S., Hedr P.M. An Epidemiological study of Osteogenic sarcoma in Kenya. International Orthopedics Vol.9 No. 1 pp 59-63, a985.

5. Charles .D.: Thesis Osteasarcoma - The Theoretical Basis its Management 1990

6. Robbins S.L., Cotran R.S., Kumar V. Pathologic Basis of Disease. - 3rd edit. W.B. Saunders Co. Philadelphia, pp 1337, 1984.

7. Sabiston D.C. Textbook of Surgery - 13th Edd. W.B. Saunders & Co., 1986.

8. Geschickter C.F. and Copeland M.M. Tumours of bone. Philadelphia, Lippincott (1949).

9. Turck S.L. Tumours of bone in Orthopaedics Principles and Their Application. J.B. Lippincott Co. Philadelphia. pp 557-564, 1977.

10. Abramson D.H., Ellsuorth R.H., Zimmermann L.E. Non ocular cancer in Retinoblastoma survivors. Trans. Acad. Opthalmol-otoloryngol 81: 454-7, 1976.

11. Dryja T.P. Rappaport J.M., epstein J. Chromosome 13 nomozygosity in osteogenic Sarcoma without retinoblastoma AM. J. Hum. Genet. 1986.

12. Swaney J.J. Familial osteogenic sarcoma. Clin. orthop. and Related Research 97: 64, 1973.

13. Cohan W.G. Woodard H.Q., Hiqinbotham N.L. Sarcoma arising in Related Bone. Report of 11 cases Cancer 1:3-29, 1948.

14. Penman H.G., ring P.A.. Osteosarcoma in association with total hip replacement J. B. J. S. 66(5): 632 - 634, 1984.

15. Roessner A., Vase B., Rautenberg J., Immenkamp, M. and Grundman E. Biological characterisation of human bone tumours. J. Cancer Red. Clin. Oncol. 106: 234-239,1983.

16. Huvos A.G., Butter A. and Bretsky S.S. Osteogenic Sarcoma in the American Black. Cancer 52(10): 1959 - 63, 1983.

17. Dahlin D.C. et Pathology of Osteosarcoma. Clin. Orthop. and Related Research 111: 23-32, 1975.

18. Dahlin D.C. Bone Tumours, second Edit. Springfield Thomas, pp 157, 1967.

19. Dahlin D.C. and Unni K.K. Osteosarcoma of bone and its important, Recognisable varieties. The Am. J. Surg. Path. 1(1):61-72, 1977.

20. Carter S.K. and Friedmauth. Osteogenic sarcoma. Treatment overview and some comments on interpretation of clinical Trial data. Cancer Treat Report. 62(2): 199-204, 1978.

21. Mail J.t., Cohen M.D., Minkin L.D., Pravisor A.J. Response of Osteosarcoma to Preoperative intravenous High Dose Methotrexate Chemotherapy C.T. Evaluation A.J.R. 144: 89-93, 1985.

22. Erneking W.F., Spier S.S. and Goodman MA. A system for Surgical staging of Musculoskeletal Sarcoma. Clin. Orthop. and Rel. Research 153:106-120, 1980.

23. Lichtenstein L. Bone tumour 4th edition Publ. C.V. Mosby Co., 1972.

24. Oyemade g.A., Juniad T.A. Osteosarcoma of bone: Clinico. Pathological features of 69 cases in Ibadan, Nigeria. Int. Surg. 67(4): 553-555, 1962.

25. Enneking W.F., Springfield D.S. Osteosurcoma, Orthop. Clin. N.A. 8(4): 785-805, 1977.

26. Sweetnam R. Osteosarcoma. Br. J. Resp. Medicine 28(2) 112-121, 1982.

27. The "Lancet" Osteosarcoma Lanat 1985: ii 8447: 131-33.

28. Simon M.A. Causes of increased survival of patients with osteosarcoma. Current controversies. J B J S. 66A(2):306-310, 1984.

29. Goarin A.M., Abelson H.I., Frei C. Osteosarcoma, Fifteen years later. The New Engl. J. Med. 13(26): 1637-1643, 1985.

30. Rosen G., Marcore R.C., Huros A.G. Primary osteogenic Sarcoma 8 years Experience with adjuvant Chemotherapy. J. Cancer Res. Cl. Oncol. 106 (suppl.) 55-67, 1983.

31. Chambers R.G. amd Mahoney W.D. Osteosarcoma of Mandible: Current Management. Am. Surg. 36:463, 1970.

9. Bailey H, Love M, Bailey and Love's short practice of surgeryPubl. Eng. Long Book society 20th ed. pp 304-306, 1988.

10. Watson - Jones R. Fractures and joint injuries. 4th Ed. Vol. 2. Edinburgh Ltd. pp 664, 1960.

11. Gratton E, Hobbis S. Injuries to the hip joint in vehicle occupants. Road research laboratory. Ministry of Transport. RRL. Report LR 126.

12. Thompson VP, Epstein H.C. Traumatic dislocation ofthe hip, a survey of 204 cases covering a period of 21 years. JBJS 33A: 746-778, 1951.

13. Campbell W.C. Posterior Dislocation of the hip with fracture of the Acetabulum. JBJS 18:8842-850, 1936.

14. Knight, R.A., and Smith M. Central fractures of the Acetabulum JBJS 40A:1-16, 1958.

15. De Palma AF. The Management of Prognosis of fractures of the acetabulum JBJS 43A:30, 1961.

17. Hubbard Zeonard F. Computerised Tomography in Orthopaetidics S.C.N.A. 53(3):589, 1983.

18. Sarungi P.M. Traumatic dislocation of the hip in a child. A case report. E.A.M.J. 51(14):364-369, 1974.

19. Watson and Jones Fractures and Joints Injuries - 6th Ed. Publ. Churchill Livingstone pp 895-934, 1982.

20. Crenshaw A.A. Campbells operative Orthopaedics - 5th ed. 1971.

21. Hunter G.A. Posterior dislocations and fracture dislocations of the hip. JBJS 51(1):38-44, 1969

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