A DISSERTATION OF TEN SURGICAL CONDITIONS

SUBMITTED BY

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IN PARTIAL FULFILMENT FOR THE DEGREE OF MASTER OF MEDICINE(SURGERY) OF THE UNIVERSITY OF DAR ES SALAAM.

1991

DECLARATION

I do declare that this dissertation is my own work based on patients I was privileged to study and that this work has not been submitted for a similar degree in any other University.

Date 19:11:91

Signature .....

This is to certify that the Ten Surgical conditions presented in this book were studied by Dr. K.A. Mteta during his postgraduate training in Surgery under my direct or delegated supervision.

Date 19th NOV. 1991

Signature .....

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ABSTRACT

This dissertation forms part of the requirement for the award of master of medicine degree in surgery. It consists of ten surgical conditions studied by the author and is divided into three parts. I must admit that it was not an easy task for me to choose the conditions to write on. In a word I wanted an original work, which will add new knowledge in our present understanding of surgery.

In part one five general surgical conditions are described. Thoracic trauma which ranks high among causes of mortality is the first, followed by the empyema of the thorax, portal hypertension, cholelithiasis and goitre which are common surgical conditions.

In part two, three urological conditions are included. A challenging case of Cushing's syndrome, a review of cryptorchidism and a case of true hermaphroditism.

Part three includes two orthopaedic and trauma conditions. Knee injuries and supracondylar humeral fractures, which are some of the most serious orthopaedic emergencies.

Apart from the statistical data which reflects the size of the problem, a case report is presented for illustration and a review of relevant literature is discussed in each condition.

TABLE OF CONTENTS.

Declaration.....ii

Copyright 1991.....iii

Acknowledgement.....iv

Abstract.....v

Part I General Surgery:.....

1. Thoracic Trauma.....1

2. Empyema of the Thorax in MMC.....18

3. Portal Hypertension.....39

4. Cholelithiasis in MMC.....80

5. Goitre in MMC.....116

Part II Urology:.....

6. A case of Cushing's syndrome.....143

7. A review of 26 cases of Cryptorchidism.....167

8. A case of True Hermaphroditism.....180

Part III Orthopaedic/Trauma:.....

9. The pattern of Knee injuries in MMC.....200

10. Management of Supracondylar humeral fractures....218

ABBREVIATIONS.....235

PART I: GENERAL SURGERY.

1. THORACIC TRAUMA:

ABSTRACT

A review of 41 patients with thoracic injuries attended in the Surgical Department of Muhimbili Medical Centre from July 1987 to July 1990 is presented. Males accounted for 95% of the cases. The ages ranged from 16-64 years with a mean of 29.8 years. Road Traffic accidents were the leading cause of thoracic trauma accounting for 63.4% knife stab wounds were second leading cause 14.6%.. 63.4% of the patients had blunt chest injuries whole 36.6% had penetrating trauma. Rib fractures occurred in 50% of the patients. Hemopneumothorax in 53.8% followed and pneumothorax occurring in 22% of the patients. The commonest associated injury was a ruptured spleen. Tube thoracostomy was the commonest surgical treatment given in 78.1% of the patients. Active observation and resuscitation was considered adequate in 14.6% of the patients while thoracotomy was done in 7.3% of the cases. Hospital stay ranged from 4-15 days with a mean of 7.7 days. The duration of tube thoracostomy ranged 2-12 days with a mean of 4-3 days. There was a 9.7% mortality.

Better diagnostic facilities to asses intrathoracic injuries are recommended.

Keywords: Thoracic Trauma: causes, treatment, morbidity and mortality.

INTRODUCTION:

The pattern of chest injury vary from place to place and also with time(1).. In the United States one fourth of civilian deaths due to trauma result from thoracic trauma and the majority of these are due to automobile accidents(2).

It is the aim of this paper to study the causes and manifestation of thoracic injuries as well as associated injuries outside the thoracic region. The treatment, morbidity and mortality associated with thoracic injuries are also reviewed.

CASE REPORT

E A. (IP 61 7683) was a 22 year old male admitted on account of a left upper front chest stab wound nine days previously. E.A. who is jobless and single reports to have been assaulted by unknown "hooligans" at Morogoro Railway station while on transit from Mwanza where he resides to Dar es Salaam. He denied history of coughing up blood, difficult in breathing or fainting attacks. He further narrated that he was being treated in a Private clinic with daily dressing of the stab wound but developed fever and chills two days before admission.

On examination E.A. was ill-looking, febrile with a temperature of 38.C, pale but not cyanosed. He was dyspnoeic with a respiration rate of 35 per minute. He has

a granulating stab wound on the left upper anterior chest second interscostal space, midclavicular line. The wound which was 2x2cm. had no air leakage of suction.

The trachea was central. The left hemithorax was hyperesonant on percussion above while the lower zones were dull. The tactile vocal fremitus was diminished and there was no breaths sounds heard on the left hemithorax. The right hemithorax was normal.

He had a pulse rate of 96 per minute regular full volume, a blood pressure of 130/80 mmHg, the apex beat in the normal location and heart sounds were normal. No other abnormality was detected in systemic examination. Chest X-ray showed a left sided lower zone homogenous opacity with air-fluid level. Thoracocentesis of the left hemithorax revealed a hemothorax. The diagnosis of left hemopneumothorax was reached.

The patient had an emergency left tube thoracostomy, with an underwater seal. 500 mls. of blood were drained from the left hemithorax on the operating table. Control chest-X-ray a day later showed excellent lung re-expansion.

Two days after insertion of the tube the patient was still febrile but less dyspnoeic. He had breath sounds on the left and the tube was no longer draining and it was thus removed. On the sixth day of admission of the patient was afebrile, up and about. A control CXR taken on the Ninth

day showed remarkable improvement from the previous one. The patient was discharged in good condition, on the tenth day for outpatient follow-up where he was seen two weeks later with normal chest findings.

MATERIAL AND METHODS

Patients:

The 41 patients reported here were consecutive patients admitted to the surgical department of Muhimbili Medical Centre and attended by the thoracic unit from July 1987 to July 1990. The criterion for inclusion in this study was the diagnosis of chest injury of any kind.

RESULTS:

Age and Sex.

The study series had 39 males making 95% of the sample with only five percent females. Their ages ranged from 16-64 years with a mean of 29.8 years.

Cause of Injury (see table I).

Road Traffic accident is the leading cause of thoracic injuries accounting for 63.4%. Knife stab wounds were the second leading cause of thoracic injury 14.6%. 63.4% of the patients had blunt non-penetrating chest injuries while the rest 36.6% had penetrating chest injuries.

Manifestations of thoracic trauma (see table II).

Hemopneumothorax, the commonest manifestations of thoracic trauma, accounted for 53.8% followed by pneumothorax 22%. Open pnemothorax and ruptured diaphragm were seen in 7.3 and 4.9% respectively.

Associated Injuries (see table III).

Ruptured spleen was the commonest associated injury occurring in three patients. There was a case each for bowel injuries ruptured kidney, head injury, fracture thoracic spine and brachial plexus injury.

Type of treatment given (see table IV).

The commonest form of surgical treatment given was tube thoracostomy and underwater seal drainage done in 78.1% of the patients. Active observation and resuscitation was done in 14.6% of the patients while thoracotomy was done in 7.3% of the cases.

Morbidity.

Hospital stay ranged from 4-15 days with a mean of 7.7 days and the duration of tube thoracostomy ranged 2-12 days with a mean of 4.3 days.

Mortality.

There were four deaths i.e. 9.7% in the series.

Summary:-

Case I: A 24 year old policeman shot by a bandit had thoracoabdominal injuries. He had pneumothorax, bowel perforation and laceration of the larynx. He died suddenly on the second post operative day, cause of death not established..

Case II: A male adult involved in a Road Traffic Accident had a ruptured left diaphragm, ruptured spleen, ruptured left kidney and Head injury died of

Multiple organ failure three days after the accident. He developed respiratory and renal failure.

Case III: A male 21 years old involved in a road traffic accident had Multiple bilateral rib fractures with bilateral pneumothoraces and undetermined intrathoracic injuries died on the admission day.

Case IV: A 17 year old male stabbed with a knife on the left upper shoulder had injury to the brachial plexus and laceration of the axillary artery died of exsanguination two hours after admission.

Table I: Causes of thoracic trauma.

Causes	Blunt	Penetrating	Total	%
Road traffic Accid.	24	2	26	63.4
Stab knife wounds	0	6	6	14.6
Gunshot injuries	0	4	4	9.6
Others	2	3	5	12.6
Total	26(63.4)	15	41	100.0

63% of the patients had blunt chest injury while 36.6% had penetrating chest injury. Road traffic accidents were the commonest cause of injury.Of the five patients in "the others" group, four were peasants who fell from trees while working in their fields while one was a hunter attacked by an injured elephant.

Table I A: Cause versus manifestation of injury:

Manifestations	CAUSES					
	RTA	Stab wounds	Gun shots	Fall	Animal attack	Total
H/Pneumothorax	16	3	3	0	0	22
C/Pneumothorax	6	1	1	1	0	9
O/Pneumothorax	0	1	0	2	0	3
R/Diaphragm	1	0	0	0	1	2
L/Contussion	3	0	0	1	0	4
Axillary A.I.	0	1	0	0	0	1
Total	22	6	4	4	1	41

The leading cause of injury was Road traffic accidents (RTA) and the most likely manifestation of injury was haemopneumothorax (H/Pneumothorax). This was followed by open (O/P) and closed (C/P) pneumothorax.

Table II: Manifestations of thoracic trauma.

Manifestation	Number	%
Hemopneumothorax	22	53.8
Pneumothorax (closed)	9	22.0
Open Pneumothorax	3	7.3
Ruptured diaphragm	2	4.9
Others	5	12.2
Total	41	100.0

Hemopneumothorax was the commonest manifestation accounting for 53.8% followed by pneumothorax 22%.

Table III: Associated injuries.

Bowel injuries	1
Ruptured spleen	3
Ruptured kidney	1
Head injury	1
Fracture spine (thoracic)	1
Brachial plexus injury	1
Total	8

Ruptured spleen was the commonest associated injury.

Table IV: Type of treatment given.

Treatment	No.	%
Tube thoracostomy	32	78.1
Active observation + resuscitation	6	14.6
Thoracotomy	3	7.3
Total	41	100.0

Tube thoracostomy was the commonest form of treatment 78.1% while thoracotomy rate was 7.3%.

DISCUSSION

Cause of Injury:

In this series, Road Traffic accidents accounts for 63.4% of the causes of thoracic injury. Road Traffic accidents is also the commonest cause of thoracic trauma in the United States [2]. Rasmussen from Denmark also reported falls and Road Traffic accidents as the commonest cause of thoracic trauma [3].(see below).

Manifestations of thoracic trauma

The Rasmussen's series cited above[3] was a report of 93 patients referred for specialist management of thoracic injuries in a five year period 1973-1978. Penetrating trauma of the thorax had been sustained by 18 patients and blunt trauma by 75 patients. Rib fractures were the most frequent lesion. Hemothorax and or pneumothorax was found in four-fifth of the patients. The diaphragm was torn in five cases. Three patients had a tracheal lesion. Hallen from Uppsala Sweden reviewed 765 patients treated for thoracic injuries between 1956-1969[4]. As in this Muhimbili series, the commonest lesions were hemopneumothorax and hemothorax. The Hallen's series also included 129 cases of pulmonary contussion - a diagnosis which is difficult to establish with certainty.

Treatment

The basic pathologic mechanism in high speed accidents is the abrupt application of a shearing force to fixed and non-fixed contiguous intrathoracic structures as the patient rapidly decelerates.

The mechanism of low speed accidents is the application of more localized type of injury to the thorax. The consequences of shearing and crushing associated with non-penetrating thoracic trauma are less commonly seen in patients under the age of seven years due to the greater elasticity of the childs thorax. Stab wounds and other high speed missiles afford direct penetrating injury.

The commonest surgical treatment given was tube thoracostomy in 78% of the cases followed by active observation and resuscitation in 14.6%. The thoracotomy rate was 7.3% in the Rasmussen(3) series reported previously, closed pleural drainage was the most common treatment and the thoracotomy rate was 10.3%. In the Hallen's (4) series above thoracotomy was performed in 5% of the cases while active conservation measures were found adequate in most of the cases.

Eighty to ninety percent of all chest injuries, whether due to blunt or penetrating trauma are adequately treated by insertion of one or more chest tubes and do not require open thoracotomy (5). Whether the injury produces

hemothorax or pneumothorax a chest tube is required to allow drainage of the intrapleural blood, monitoring rate of further bleeding and evacuation of any are present. A posterior tube effectively drains both fluid and air and is therefore universally effective.

The principal indication for thoracotomy are cardiac tamponade, aortic arch disruption, massive hemothorax and un controlled pulmonary air leaks. Less common indications are perforation of the esophagus, trachea or major bronchi. An aggressive approach that utilises Emergency Room thoracotomy for"lifeless" or decompesating patients has been decried by some and advocated by others as an important factor in lowering mortality."Lifeless" are those patients arriving in hospital with cardiac arrest in the ambulance.

Sergio[6] of the University of Maryland Hospital reported that twenty one (57%) of 37 patients undergoing Emergency room thoracotomy survived; most of the deaths occurred in patients arriving "lifeless" from gunshot injuries. He thus advises the surgical team to concentrate on rapid resuscitation and insertion of an endotracheal tube, chest tubes and intravenous lines as needed and immediate thoracotomy in the agonal or rapidly deteriorating patients.

Washington[7] in a four year review of emergency room thoracotomy in Detroit reported 15% survival. He therefore advocates early thoracotomy in the management of patients who have cardiac arrest in the ambulance or emergency department.

Knottenbelt and Spuy[8] while reviewing cases of traumatic pneumothorax in Cape Town, South Africa reported a thoracotomy rate of 1.24%. Mean hospital stay was 22 hours for straightforward cases and 49 hours for those with a continuing leak. The mean hospital stay for this Muhimbili series was 7.7 days. In the Cape Town series the mean duration of intercostal drain was 24 hours, while the mean duration for the Muhimbili series was 4.3 days. The Cape Town series was, however, designed for rapid patient turnover. In the Cape Town series a clinical review and chest radiograph were carried out six hourly in those patients not drained and two hourly in those drained until progress in lung expansion had been achieved.Lack of portable X-Ray facility in MMC during the study period was a big handicap in the management of drained patients.

Mortality

There was a 9.7% mortality in this Muhimbili series. In Rasmussens (3) series the mortality was 10% while it was 2% in Hallens (4) series.

RECOMMENDATIONS

Prolonged thoracostomy drainage times are associated with increased infection rates and morbidity, take up valuable resources in terms of bed and staff and delay return of patient to normal life. The mean duration of 4.3 days in Muhimbili should be shortened to enable patients to return to normal activities earlier.

In this series it was difficult at times to ascertain presence and extent of intrathoracic injury due to lack of diagnostic facilities. It is recommended therefore that the Hospital should strive to acquire better diagnostic facilities such as CAT Scan and facilities for aortograms.

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2. THORACIC EMPYEMA IN MUHIMBILI MEDICAL CENTRE

ABSTRACT

A review of 114 thoracic empyema cases attended in the thoracic unit of the Muhimbili Medical Centre from July 1986 to July 1990 is presented. 87.7% of the cases were males. Their ages ranged from 9 to 79 years with a mean of 32 years. Tuberculosis was the major underlying disease accounting for 63.2% of all the cases. 7.9% of the cases were ELISA positive for HIV. 52.6% of the patients underwent either open chest drainage, decortication or thoracoplasty. The duration of hospital stay ranged from 2-8 months with a mode around 3.5 months. A 7% mortality was noted. To control thoracic empyema it is recommended to intensify efforts to control tuberculosis and adequate treatment of pneumonia. Thoracoscopy and irrigation of the empyema cavity where applicable should be employed to reduce the lengthy hospital stay as well as avoiding surgical procedures of bigger magnitude.

Key words: Thoracic Empyema: Morbidity, mortality and treatment.

INTRODUCTION

Empyema thoracis is defined as presence of purulent fluid in the pleural cavity. The collection of purulent material may be localized or it may involve the entire pleural cavity.

Thoracic empyema is a disease entity that has been recognized throughout recorded medical history. Hippocrates recognized empyema of the thorax and described its diagnosis and treatment. He recommended drainage but the difficulty of making an accurate diagnosis and lack of practical surgical methods made recognition and successful treatment rare. Graham and Bell of the United States Army's Empyema Commission placed the treatment of this entity on rational basis in 1918 [1]. Their observations of the natural history of empyema resulted in logical use of open and closed drainage.

Adebonojo in a retrospective study in Ibadan reported a study of 1150 consecutive patients with thoracic and cardiovascular diseases over a period of five years. He showed that 42% were treated for suppurative disease of the lung and pleura notably empyema thoracis, lung abscess and bronchiectasis. Of these 70% were treated for empyema thoracis. Adebonojo, therefore concluded that suppurative condition of the lung and pleura remain the greatest challenge to the thoracic surgeons coupled by inadequate facilities, lack of drugs, illiteracy poverty, superstitious beliefs and poor environmental hygiene. [2].

It is with this background that this paper seeks to study the age, sex, underlying disease and treatment given to empyema thoracis patients in Muhimbili, Dar es Salaam and the treatment outcome.

Empyema thoracic is known to be a chronic and often debilitating disease. This paper also analyses morbidity in terms of duration of hospital stay and duration of tube thoracostomy. Finally mortality is assessed as well as association with HIV infection.

CASE REPORT

M.K. (IP 609495) was a 44 year old female referred from Iringa Regional Hospital with two months history of right sided chest pain associated with a dry cough. Initially she had fever associated with difficulty in breathing. At Iringa she was diagnosed as having an Amoebic liver abscess and was treated with a course of Metronidazole. She later developed a right sided pleural effusion treated unsuccessful with closed drainage. She had no other complaint and denies history of TB contact. She is a peasant, mother of six ~~peasant~~ children from Iringa.

On examination, she was middle aged afebrile not wasted, not dyspnoeic, not cyanosed and no finger clubbing. She was not pale and she had no palpable superficial lymphnodes. She had no obvious chest deformity and her respiratory rate was twenty per minute. The trachea was

central she had a dull percussion note on the right hemithorax with impaired tactile vocal fremitus on the same side. Breath sounds were markedly reduced on the right hemithorax. The left hemithorax was normal. Hepatic dullness was normal. The liver spleen, and kidneys were not palpable and the cardiovascular system was normal.

A clinical impression of right pleural effusion was reached and a chest X-ray showed a homogenous opacity lower and middle zones right hemithorax. Right chest aspiration revealed thick pus and a diagnosis of right sided empyema of the thorax was confirmed. Other investigations carried out revealed the following:-

1- Hb 11.8g/dl

2. WBC 6400 N66; E3; L 28; M3

3. ESR 70

4. Mantoux positive:20mm

5. ELISA for HIV-NEGATIVE

6. Sputum direct smear and ZN stain for AFB was negative.

7. Sputum for mycobacterium culture showed no growth.

8. Right chest aspiration revealed pus with the following.

   WBC 18,750/mm3

   RBC 1200/mm

   N 76% POLYMORPHS 24%

Gram stain: Many pus cells, no bacteria

ZN stain: No AFB seen.

Aerobic culture: no bacterial growth.

SUMMARY OF TREATMENT PROCEDURES

15.5.90 Tube thoracostomy inserted and drained about 500 mls of pus.

25.5.90 Tube not draining even though control chest X-ray still showed homogenous opacity with air fluid level. The tube was extubated and found to be blocked with fibrin clots.

26.5.90 Right chest aspiration done, 750mls of pus drained. Fluid however reaccumulated.

25.6.90 Right tube thoracostomy was reinserted.

30.6.90 Tube not draining, therefore it was removed. Control chest X-ray still showed multiple loculations. The patient was worked up for thoracoscopy.

25.7.90 The patient underwent right thoracoscopy though a lateral transverse incision.

Findings

1. Big cavity containing thick pus.
2. Thickened parietal and visceral pleura.
3. No bronchopleural fistula.

Procedure:

Through the above incision the seventh rib was identified. The periosteum was elevated and rasped. 4cm. segments of the seventh and sixth ribs were resected. The trocar passed into the pleura cavity and pus was sucked out. Loculations were broken down using the tip of the laparoscope and suction nozzle. Specimens were taken for pyogenic and mycobacterium studies. Pleural biopsy was also done. Lavage with normal saline till clear fluid was obtained was done. A chest tube was introduced into the empyema cavity under direct vision through a stab incision. PUS FROM THE CHEST: DIRECT SMEARS WERE NEGATIVE FOR AFB HOWEVER MYCOBACTERIUM CULTURE WAS POSITIVE IN TWO COLONIES. THE PATIENT WAS STARTED ON STEPTOMYCINE INJECTION ONE GRAM DAILY FOR SIXTY DAYS AND THIAZINA TABLETS 450 mg.DAILY FOR FIFTEEN MONTHS.

4.8.90 The thoracostomy tube was removed since the lung had expanded fully and there was no more air or fluid being obtained.

17.8.90 Control CXR showed reaccumulation of fluid in the right hemithorax the patient was planned for open chest drainage.

26.9.90 Right open chest drainage.

FINDINGS

1. Medium size empyema cavity with 1000-2000 mls of Pus
2. Very thick cortex and pleura
3. No bronchopleura fistula

PROCEDURE

1. Routine cleaning and draping
2. Transverse lateral chest incision
3. Three rib segments resected
4. Pus taken for pyogenic and mycotb c/s
5. Pleural biopsy taken
6. Window constructed

POST OP

1. Given caps cloxacillin 500mg six hourly for two days.
2. Pethedine IM Inj 100mg 3 Doses.
3. Care as after general anaesthesia

Pleural biopsy: Showed extensive areas of fibrosis with inflammatory cells such as plasma cells lymphocytes, hystiocytes and neutrophilis were seen. Chronic inflammatory process was concluded.

The final diagnosis was right sided thoracic empyema secondary to tuberculosis. The patient was discharged on 20/10/90, six months after admission for review after three

months. She is to continue with anti TB therapy and dressing of the chest window in the regional hospital.

METHODOLOGY

While rotating in the thoracic Unit which incorporates General Surgery Firm II, I had the opportunity to attend to eighteen patients with empyema thoracis. I also reviewed the thoracic registry and obtained data on patients treated for empyema thoracis between July 1986 and July 1990. The patients files were retrieved from the Medical records to obtain more information. In this way, case notes of 114 patients with empyema thoracis were studied. The age, sex, underlying disease and treatment given was recorded. The duration of hospital stay was calculated as a measure of the morbidity. The ELISA for HIV status was also recorded. Where death occurred the cause of death was recorded.

RESULTS

Most of the thoracic empyema patients were young with 43.9% of the total being in the 21-30 year age group. The next largest group (see table I) was the 31-40 year group which contributed 25.4% of all the patients. These two group if combined that is 21-40 years group make 69.3% of the patients. There was an obvious male preponderance of 87.7%.i.e,100 out of the total 114 patients.

The most common underlying disease was pulmonary tuberculosis contributing 63.2% of the patients. Empyema

following pneumonia contributed 8.8% and chest trauma contributed 7% of the cases. Most of the trauma cases were chest stab wounds following assault. Blunt chest trauma leading to empyema was encountered in three cases only. Three other cases making 2.6% were due to ruptured liver abscess into the chest. There were two iatrogenic cases making 1.8%. One of these had recurrent thoracic empyema following transdiaphragmatic migration of a drain left, in an abdominal operation twenty years prior to presentation. In the second patient thoracic empyema followed thoracocentesis of a pleural effusion. In 16.7% of the cases the cause of thoracic empyema was not immediately established (see table II).

In this Muhimbili series all the patients had an initial closed tube thoracostomy. 52.6% of the patients required subsequent open chest drainage, decortication or thoracoplasty. 47.4% of the patients did not require any additional surgical treatment other than the tube thoracostomy and under water seal drainage.

1. MORBIDITY

   The duration of the thoracostomy ranged from 3-90 days with an average of 33 days. The duration of hospital stay ranged from two to eight months with a mean of three and a half months.

2. Relationship with HIV infection.

There were nine patients (7.89%) who were ELISA positive for HIV Of the nine patients, six had underlying pulmonary tuberculosis while in three cause of the empyema was not immediately established..

3. Mortality.

There were eight deaths in this series making an operative mortality of 7%.

Summary of cause of deaths:-

Case 1: Empyema thoracic patient with pulmonary tuberculosis died on the day of admission after tube thoracostomy cause of death advanced pulmonary tuberculosis.

Case 2: Empyema thoracic patient undergoing open chest drainage, died on the operating table (DOT). Cause of death not established.

Case 3: Patient with empyema thoracis secondary to tuberculosis died, several weeks after open chest drainage due to overwhelming TB involving both lungs.

Case 4: Empyema thoracic patient, pleura biopsy showed suppurative inflammation died three weeks after open chest drainage cause of death: over whelming pulmonary infection.

Case 5. Tuberculosis empyema thoracic patient developed fever, jaundice and dyspnea and died six months

after open chest drainage, due to overwhelming pulmonary tuberculosis.

Case 6: Traumatic empyema thoracic patient who under went thoracotomy for decortication died on the first day post operatively of aspiration preumonitis.

Case 7: ELISA for HIV positive patient died on the operating table while undergoing open chest drainage. Cause of death, flooding of the opposite lung. Since he had a broncho-pleural fistula and a double human endobronchial tube was not used in this case.

Case 8: Tuberculosis empyema thoracic patient, died three weeks after tube thoracostomy: cause of death; overwhelming pulmonary infection.

TABLES AND COMMENTS

Table I: Age and sex distribution.

Age group	Sex		Total	%
	Male	Female		
0 - 10	1	0	1	0.4
11-20	7	1	8	7.1
21-30	39	11	50	43.9
31-40	28	1	29	25.4
41 50	14	1	15	13.2
50+	6	0	6	5.3
Unknown	5	0	5	4.4
Total	100(87.7%)	14(12%)	114	100

Most of the thoracic empyema patients were young with 43.9% of the total being the 21 to 30 year age group. The second largest group was the 31 to 40 years age group which had 25.4% of all the patients. There were 100 males making 87.7% of the cases.

Table II: Underlying Disease.

Underlying disease.	Number	%
Pulmonary tuberculosis	72	63.2
Pneumonia	10	8.8
Chest trauma	8	7.0
Liver abscess	3	2.6
Iatrogenic	2	1.8
Not immediately established	14	16.7
TOTAL	114	100.0

The common underlying disease was pulmonary tuberculosis contributing 63.2% of all the patients. Empyema following pneumonia contributed 8.8% and chest trauma contributed 7% of all cases.

Table III: Treatment Given.

Treatment	Number	%
Tube Thoracostomy alone	54	47.4
Open chest drainage	53	45.6
Decortication/Thoracoplasty	8	7.0
TOTAL	114	100.0

All the patients had an initial closed tube thoracostomy. Sixty patients (52.6%) required subsequent open chest drainage, decortication or thoracoplasty. 47.4% of the patients did not require any additional surgical treatment

other than the tube thoracostomy and under water-seal drainage.

DISCUSSION

Most patient in this series were young, the age ranging from 9 to 79 years with a mean of 32 years. This differs from that reported by Lema et al (3) where the age ranged from 24 to 79 with a mean of sixty years. This was a series of patients in the U.K. It appears therefore that empyema thoracic is a disease of younger patients in Muhimbili.

There was an obvious male preponderance in those afflicted. This concurs with the study by Lema (3) reported above. In another series Lema et al (4) while studying empyema thoracic in Dar es Salaam noted a male preponderance of the tune of 82.5%. This is comparable with the 87.7% noted in this series. Empyema of the thorax is therefore seen here as a disease of young males.

Etiology

Tuberculosis was noted to be the commonest underlying disease contributing 63.2% of the patients. This does not differ significantly from the 62% reported by Lema et al (3) above. Pneumonia was the second most common underlying disease. Prior to the introduction of Sulfanilamide drugs in the 1930's and the development of antibiotics in the 1940's pleural empyema was a relatively common complication

of patients who survived a bout of pneumonia. These agents proved to be remarkably effective in the treatment of pneumonia and in reducing the incidence of post pneumonic empyema [5].

Before the antibiotic era it was a generally accepted fact that empyema could be expected to develop in approximately 10% of patients who survived pneumonia. In Finland's series of 3131 patients with pneumonia, empyema developed in 7%. Maes, Veal and McFetridge [5] reported empyema in 11.2% of 6050 patients with pneumonia observed in a New Orleans Charity Hospital in the 1930s.

Before the introduction of antibiotics approximately 60-75% of the cases of empyema of the thorax were post pneumonic in origin. Other causes of empyema included lung abscess, bronchiectatic abscess, penetrating wounds of the thorax, pulmonary emboli, perforations of the esophagus and pleurocentesis performed to aspirate fluid from the thorax.

Post pneumonia empyema is thought to develop when pulmonary lymphatics become blocked by inflammatory debris, which result in an outpouring into the thoracic cavity of lymphatic fluid contaminated by bacteria.

Penetrating and blunt thoracic trauma may result in empyema especially if foreign material is retained in the thorax.

Subdiaphragmatic infection may present as an empyema as

organism move up the lymphatic to the thorax. Three such cases were observed in this series.

Treatment:

Empyema thoracis have been divided into three phases based on its natural history. These phases are useful in the planning of therapy. The earliest phase is called the exudative stage and is characterized by thin pleura fluid with a low white blood cell count. Untreated the empyema rapidly progresses into a fibropurulent or transitional stage, which is characterized by thicker, more turbid fluid and the appearance of fibrin on the pleura surface. The lung begins to be trapped by a restrictive fibrin peel during this phase, known as the chronic phase. The peel become organised with ingrowths of capillaries and fibroblasts. Organisation may begin as early as 7 to 10 days after the onset of disease. The time course of these events varies with the different offending agents however by the fourth to sixth week the process has usually entered the chronic phase.

The treatment of acute empyema should have as its goal the following: to save life with complete elimination of empyema and the avoidance of recurrent empyema pockets; to return respiratory function promptly to normal by re-expansion of the trapped lung and restoration of mobility of the chest wall and diaphragm; to eliminate

complications, or chronicity and thereby any need for a crippling or debilitating surgical procedure, to reduce hospital stay.

A chest tube placed early in the course of an empyema in the acute exudative or early fibropurulent phase is frequently curative. 47.4% of patients in this series were treated, as such. Closed tube drainage is also useful to drain an empyema in the toxic patients. Drainage may not be accompanied by expansion of the lung but will allow stabilisation of the patient so that definitive therapy can be undertaken.

Further along in the fibropurulent stage and in the chronic stage, simple evacuation of the infected empyema material is not effective. At this stage the lung and chest wall have become relatively fixed by a fibrous peel and therefore more aggressive surgical procedures are necessary. Open chest drainage by rib resection is indicated when multiple loculations are present and the cavity is not drained by simple closed tube drainage. Forty five percent of patients in this series were treated by open chest drainage. Lema et al[3] above described a method of treating this stage of empyema by thoracoscopy and irrigation to avoid rib resection and decortication.

Formal decortication of the lung and, parietal pleura is indicated in patients with a major portion of the lung

entrapped. Lateral thoracotomy through the fifth or sixth interspace provides exposure to all aspects of the pleura cavity and is the incision of choice. The thick fibrous peel is removed from the lung, diaphragm mediastinum and pleural surface of the chest wall. Mayo (6), argues that empyema conventionally treated by thoracocentesis, thoracostomy drainage, and antibiotics have unacceptably high rate of morbidity and mortality. He therefore advocates early open thoracotomy to eliminate the empyema with decortication of the fibrinous peel to allow reexpansion of the lung. Occasionally thoracoplasty is needed to obliterate the pleural cavity when lung reexpansion is impossible [7,8]. Eight patients in this series were treated with decortication and or thoracoplasty.

Morbidity:

Empyema thoracis is a chronic and often a debilitating disease. The mean duration of hospital stay was three and a half months. Some patients spent up to eight months in Hospital undergoing multiple surgical procedures. This is quite taxing to the patient as well as the attending surgeons in terms of work hours lost. In the series reported by Lema[3] above the mean duration of hospital stay was 4.8 weeks. This series was from a developed country (U.K.). For a developing country like Tanzania

where the health budget is meagre it is very expensive to treat chronic empyema this way with a very long Hospital stay.

Relation with HIV infection:

It is difficult to comment on the 7.89% HIV prevalence in the sample. This should be compared with the community HIV prevalence to see if there is any significant difference - seroprevalence of HIV amongst healthy blood donors in Muhimbili Dar es Salaam in 1990 was 8-9%.

Mortality

Of the eight deaths making a 7% mortality in the series three deaths were either death on the operating table or a perioperative death. Critical review of the anaesthesia techniques used in empyema thoracis surgery is probably indicated. The rest of the patients died of advanced tuberculosis or overwhelming infection. These are patients who reported very late to hospital.

Conclusions and recommendations:

Adebonojo [2] cited above had concluded that infectious disease of the lung and pleura remain the greatest challenge to the thoracic surgeons in tropical developing countries. The surgeon is often frustrated by inadequate facilities, lack of drugs illiteracy, poverty, superstitious beliefs and poor environmental hygiene. The

leading cause of empyema thoracic in this series was pulmonary tuberculosis.

It would be logical to conclude that if tuberculosis was controlled in Tanzania, cases of empyema thoracic would be greatly reduced.

The National TB and Leprosy Control programme should be intensified. Pneumonia was the second leading cause. Adequate treatment of pneumonia is recommended to reduce empyema thoracis. The same can be said of amoebic liver abscess. Strict aseptic technique is reemphasized during thoracocentensis as one case of empyema thoracic followed aspirations.

To avoid the lengthy hospital stay open chest drainage and thoracotomy one would recommend use of thoracoscopy and irrigation of the empyema cavity.

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3. PORTAL HYPERTENSION

Abstract

A review of six cases of portal hypertension seen at Muhimbili Medical Centre and KCMC is presented. Five of the six patients were males and their ages ranged from 15 to 50 years with a mean of 27 years. Hematemesis was the commonest presentation and all six patients had esophageal varices at endoscopy. Four of the six patients had post infectious hepatitis cirrhosis and two had schistosomal liver fibrosis. Two patients underwent devascularisation procedures, two had splenectomy alone, one had proximal splenorenal shunt and one died before surgery. Two of the six patients were dead at one year of follow-up.

INTRODUCTION

Portal hypertension is not uncommon and is life threatening due to massive upper gastrointestinal hemorrhage from bleeding esophageal varices. In Zimbabwe bleeding from varices is the leading cause of upper gastrointestinal hemorrhage and it is three times as common as bleeding from peptic ulcers. In Johannesburg hemorrhage from varices is still in advance of ulcers of the stomach and duodenum. However in Nigeria duodenal ulcers are the most common cause of upper gastrointestinal hemorrhage[1].

The common causes of portal hypertension are theoretically preventable. Many viruses are associated with liver

disease although hepatitis A, hepatitis B and non-A non-B hepatitis account for the majority of cases. The diagnosis can usually be established on serological grounds. Whilst acute hepatitis usually resolves spontaneously, chronic hepatitis often requires therapeutic intervention to prevent progression to cirrhosis, portal hypertension and hepatocellular carcinoma. In chronic hepatitis B infection, persistent viral replication is an indication for antiviral therapy, whilst corticosteroids may be indicated for a proportion of those without viral replication[2,3]. In one control community based study in West Africa exposure rate to HB virus was 64 percent[1]. Hepatitis B vaccination should be considered for all high risk individuals (for example health care workers). Vaccination of newborn children may be needed to prevent transmission of the virus in highly endemic areas such as Africa and the Far East. Immediate protection can be given by combining vaccination with an injection of hyperimune serum[4]

Human schistosomiasis is the commonest cause of portal hypertension world wide and it is related to contact with water. Essentially a rural disease, it is now being increasingly seen in many urban and peri-urban areas where stagnant streams and drains provide focus for transmission. In many countries the disease spreads as agricultural

development increases; hopefully, socioeconomic progress will enable control to be undertaken on a wider scale. This will require planning and cooperation between a number of government departments including health water development, power, agriculture, mining and education. Chemotherapy is a short term tool, in control but needs a health infrastructure for diagnosis and treatment; environmental sanitation and snail control are more long term measures. Research is continuing into the immune complex mechanisms concerned with immunity which it is hoped will lead to the development of an effective protective vaccine[5,6].

Alcoholic cirrhosis which is common in the Western world is potentially preventable by alcohol abstinence; like wise portal vein thrombosis secondary to umbilical sepsis can be prevented by improved baby delivery care.

CASE REPORT

P.C. IP 482059 a 25 year old female from Korogwe was admitted on account of vomiting blood. This was a spontaneous painless episode and she reported that she vomited about a litre of altered blood. She had two previous episodes of vomiting blood necessitating admission and blood transfusion in her district hospital. There was no previous history of abdominal pain or passage of black stools. About five years previously she had developed

yellow colouration of urine and eyes associated with fever, malaise and right upper abdominal discomfort. She had no history of taking alcohol and no history of hematuria.

On examination she was pale, not icteric and was well oriented. Her blood pressure was 110/60mmHg and she had tachycardia of 95 per minute with cold clammy extremities. There was no fetor hepaticus, spider naevi, skin hyperpigmentation, palmar erythema or Dupuytren's contractures. She had no caput medussae though the abdomen was slightly distended with positive shifting dullness but not fluid thrill. Bowel sounds were normal. The spleen was palpable about 10cm below the left costal margin and the liver was not palpable. Rectal examination revealed malena stools. A provisional diagnosis of portal hypertension with bleeding oesophageal varices was made. Blood grouping and cross match was done and an intravenous cannula was set up and 2 litres of Hartman's solution was given rapidly. A nasogastric tube was inserted and aspiration revealed altered blood. Lavage with cold saline was done till clear fluid was obtained. A urethral catheter was also passed and 300mls of concentrated urine was drained. The patient was sedated with 10mg of intravenous diazepam, Neomycin 1 gram and Magnesium Sulfate suspension 30mls, were given through the nasogastric tube. Six hours later she was stable with a blood pressure of

120/80mmHg and a pulse of 78 per minute and normal urine output. She had received two units of blood transfusion and 2.5 litres of Hartman's solution.

Investigation	Value
Hemoglobin (g/dl)	8g/dl
Serum creatinine umol/l	125
ALAT (IU)	30
ASAT (IU)	25
Alpha-Fetoprotein	Negative
HBs Ag	Positive
Serum albumin mg/dl	38
Serum Bilirubin umol/l	20
Prothrombin time delay(s)	2

Utrasound revealed a coarsened hepatic echos without focal lesions and a normal biliary tree. Superior mesenteric vein was prominent, splenic vein was difficult to asses. These features were suggestive of liver cirrhosis. Endoscopy revealed prominent lower third esophageal varices, with normal stomach and duodenum. The patient was classified as Childs group A. Sclerotherapy facility was not available and the patient was planned for elective shunting procedure. Anemia was corrected preoperatively with blood transfusion and four units of fresh blood were cross-matched for the operation.

A left paramedian incision was done for the Splenorenal shunt procedure. A grossly enlarged spleen (33 x 18 x 12 cm) was found, the liver looked normal and there was minimal yellowish ascitic fluid. Engorged mesenteric veins were noted. Canulation of the left gastroepiploic vein for

manometry revealed portal pressure of 30cm of normal saline. Splenectomy followed by mobilisation of the splenic vein was done. The left renal vein was identified after dissection of the renal pedicle. End to side anastomosis of the splenic vein to the left renal vein was done with a double swaggered nylon number 7/0 suture. Hemostasis was achieved and when the vascular clamps were removed the portal pressure dropped to 19.7cm of normal saline. The abdominal cavity was mopped and closed in layers after a corrugated rubber drain was left in situ.

Post operatively the patient had half a litre of normal saline, 2.5 litres of dextrose 5% and one unit of blood in the first twenty four hours. Input-output chart and pulse rate, blood pressure and respiratory rate were monitored half hourly for six hours then 4 hourly for 24 hours. Intramuscular pethidine 100mg was given 8 hourly for the first 24 hours. The vital signs remained stable in the first 24 hours. The patient opened bowels on the third post operative day, and started taking oral sips of clear fluids and could sit up in bed. The patient then made an uneventful recovery with stitches being removed on the 9th post operative day.

Liver biopsy showed enlarged portal tracts with lymphocytic infiltration and fibrous tissue around them. The liver architecture was intact. Chronic infectious hepatitis was

concluded. The spleen was normal histologically. Endoscopy two months after surgery showed significant reduction of the esophageal varices. The patient continues to attend the outpatient follow-up clinic and has no haematemesis or features suggestive of encephalopathy one year after surgery.

RESULTS

Table I: Presenting symptoms in six patients.

Symptom	Frequency
Hematemesis	5
Left upper abdominal swelling	6
Black stools	2

Table II: Signs noted in the six patients

Sign	Frequency
Splenomegaly	6
Anemia	6
Ascites	3
Hypersplenism	1
Varices at endoscopy	6

Table III: Child- Pugh classification of the cases.

Grade	Number of patients.
A	4
B	2
C	0

Table IV: treatment given, (Surgical)

Surgery	Number of patients.
Devascularisation	2
Proximal Splenorenal Shunt.	1
Splenectomy alone.	1

Four of the six patients had possibly post infectious hepatitis liver cirrhosis, three of whom were still HBsAg positive at time of surgery suggesting chronic infectious hepatitis. Two patients had post schistosomal liver

fibrosis. Two of the six patients died within one year of follow-up.

REVIEW OF CASES

CASE I:

B.P. IP 552738 a 50 year old male admitted on account of vomiting blood and who on examination was found to have gross splenomegally and endoscopy confirmed bleeding esophageal varices. He was Childs' class B. A lymphovenous shunt was done and a portalcaval anastomosis was attempted but failed for technical reasons. Splenectomy was done. The patient was discharged for out patient followup and was readmitted for recurrent esophogeal bleeding and died three month after the surgery from terminal liver disease.

CASE II

M.I. IP 523564, A 30 year old patient admitted with hematemesis and on examination found to be grossly pale. Endoscopy confirmed massive esophageal varices. He was Child's group B. He was successfully resuscitated only to die of recurrent bleeding one year later. He was HBsAg positive.

CASE III

A.I. IP 587962 a 21 year old male from Kondoa complained of a left upper abdominal swelling for five years. On examination he had gross splenomegally with ascites. Endoscopy revealed non-bleeding esophageal varices. Peripheral blood smear and Bone marrow studies revealed hypersplenism. He was planned for splenectomy and esophagogastric devascularisation. He ended up having splenectomy alone. The liver was macronodular and histology revealed cirrhosis and HBsAg was positive. The patient was discharged in good condition and planned for control esophagoscopy. Blood picture showed that the hypersplenism had been reversed.

CASE IV

M.J. IP 549292 a 24 year old male patient from Moshi presented with vomiting blood. Endoscopy confirmed esophageal varices. He was Child's class A. He had lymphovenous shunting done on 9/1/89 and Hassab's decompression procedure on 7/4/89. He had no recurrent hematemesis at one year of followup and endoscopy showed disappearance of varices.

CASE V

A 15 year old female from Mbuguni a schistosoma endemic area presented with hematemesis at K.C.M.C. Endoscopy

confirmed bleeding esophageal varices. She was Child's group A. Conservative treatment failed to control the bleeding. Emergency surgery was planned. She had truncalvagotomy, pyloroplasty, extensive esophagogastric devascularisation and splenectomy. She was discharged in good condition and planned for esophageal transection and anastomosis if the varices persist at followup.

DISCUSSION

Aetiology

Venous blood from the gastrointestinal tract, spleen and pancreas drain eventually into the portal vein and passes through the liver before entering the inferior vena cava via several hepatic veins. The normal adult liver is perfused by about 1500ml blood per minute. Approximately two thirds of hepatic blood flow and one half of the oxygen supply is provided by the portal vein; the remainder is derived from the hapatic artery[7] Normally the pressure in the portal vein is quite low (10-15cm saline), since the vascular resistance in the hepatic sinusoids is low. Little is known about the factors which control splanchnic and thus portal flow, and the increase which occurs after meals appears to be proportional to the increase in cardiac output. Portal hypertension is defined as a portal pressure of over 30cm saline when measured at surgery.

12cm Normal 5-10 mm H_2O

1.5 l/min

Hypertension within the portal vein and its tributaries may accompany hepatic disease or disturbance in the anatomy of the extrahepatic vascular system. As a consequence of this elevated pressure or in association with it congestion of collateral pathways is established and may be manifested by esophagogastric varices, ascites, hypersplenism or encephalopathy.

Increased hepatopedal flow is an infrequent cause of portal hypertension. Hepatic arterial-portal venous fistula has been reported and the diagnosis is established by aortography. Successful treatment has been effected by ligation of the hepatic artery or aneurysmorrhaphy. Splenic arteriovenous fistula is also a relatively uncommon lesion. This has a predilection for females between the ages of twenty and fifty and may be symptomatic during pregnancy. Calcification in the left upper quadrant is suggestive and aortography may be diagnostic. Resection of the fistula is therapeutic. An increase in forward blood flow in the portal venous system has also been proposed as the cause of portal hypertension in patients with Boeck's sarcoid, Gauchers disease and myeloid metaplasia. The pathophysiology is intrasplenic arteriovenous fistula.

A small group of well-documented cases of bleeding varices with portal hypertension in the absence of demonstrable intrahepatic or extrahepatic obstruction may be related to

a similar intrasplenic pathology. These include tropical splenomegaly, and represent the rare circumstances in which splenectomy alone may be therapeutic. Splanchnic hyperaemia is recognized as part of the generalised hemodynamic disturbances which gives rise to a hyperdynamic splanchnic and systemic circulation. The hyperdynamic circulation develop as a consequence of enhanced levels of endogenous vasodilators in conjunction with a reduced vascular sensitivity to vasoconstrictors. In addition to the proposed gut hormones, opiod peptides, prostanoids (GABA), Glucagon has received particular attention since it is a potent vasodilator that may attenuate catecholamine induced vasoconstriction(8)

Since the hepatic veins constitute the sole efferent vascular drainage of the liver, obstruction or increased pressure within these vessels or their radicles results in an increased sinusoidal and portal pressure. This outflow obstruction (Budd-Chiari) syndrome is most frequently associated with an endophlebitis of the hepatic veins, which may be isolated or part of a generalized thrombophlebitic process. A web in the suprahepatic vena cava has been reported to cause the syndrome in the Japanese. The clinical picture depends on the rapidity and degree of venous obstruction. With sudden and complete obstruction, the presentation is that of an abdominal

catastrophe with severe abdominal pain nausea, vomiting and rapid enlargement of the abdomen by ascites. This rarely occurs. Most commonly obstruction to the hepatic venous tracts appears to be gradual and is associated with mild to moderate abdominal dicomfort and ascites. Portal hypertension secondary to impaired flow in the extrahepatic portal venous system is unique in that the hypertension is not usually complicated by hepatocellular dysfunction. Congenital atresia or hypoplasia, or an extension of obliterative process of the umbilical vein and ductus venosus is rare. More commonly, there is cavernomatous transformation of the portal vein which probably represents organisation and recanalisation of thrombi within the vessel. The most common etiologic factor in the development of extrahepatic portal venous obstruction in childhood is some form of infection. Bacteria may be transmitted via a patent umbilical vein, but a history of neonatal omphalitis is rarely obtainable. Extrahepatic obstruction may also be secondary to trauma and extrinsic compression caused by adhesions, inflammatory processes and tumors. Isolated splenic vein thrombosis, usually a consequence of alcoholic pancreatitis, may cause esophagogastric varices. In this case splenectomy cures the portal hypertension.

The overwhelming majority of cases of portal hypertension are related to an intrahepatic obstruction. This accounts for 90 percent of patients with portal hypertension. A variety of hepatic diseases have been implicated, but no single explanation of pathogenesis has proved totally satisfactory. The implicated pathogenic factors include: hepatic fibrosis with compression of portal venules, compression by regenerative nodules, increased arterial blood flow, fatty infiltration acute inflammation, and intrahepatic vascular obstruction. The hepatic diseases associated with portal hypertension include nutritional cirrhosis, postnecrotic cirrhosis, biliary cirrhosis, hemachromatosis, Wilson's disease, congenital hepatic fibrosis and infiltrative lesions.

Schistosomiasis is the most common cause of portal hypertension. Although this condition is rare in North America and Europe it should be remembered that the prognosis is usually good with correct treatment. The acceptable options for treating hepatic schistosomiasis in patients with portal hypertension are an extensive esophagogastric devascularisation operation with splenectomy, or a distal splenorenal shunt[3] The ova of Schistosoma mansoni elicit granulomatous and fibrotic reactions along the portal tracts, the results of a delayed hypersensitivity reaction modulated by an antigen-antibody

immune-complex is therefore, noncirrhotic ("pipestem") fibrosis, which produces progressive hypertension. In advanced cases sinusoidal portal hypertension may be seen. True cirrhosis may be present in some cases, but nutritional deficiencies and other factors eg. hepatitis B infection contribute to this process. Clinically, the signs of portal hypertension predominate and variceal hemorrhage is the usual presentation.

The liver and spleen are moderately enlarged. Hepatocellular function is usually normal, but jaundice and serum alkaline phosphatase elevation may be noted.

Nutritional cirrhosis is also common and in the western world it is frequently associated with chronic alcoholism. As is true with all intrahepatic lesions, with the exception of congenital hepatic fibrosis and schistosomiasis, the major resistance to the flow of portal blood is located on the hepatic venous side of the sinusoid (post-sinusoidal). Post necrotic cirrhosis accounts for 5 to 12 percent of the cases and represents progression of an acute viral hepatitis or toxic hepatic injury. Advanced portal cirrhosis is an almost invariable feature of hemachromatosis. Wilson's disease (hepatolenticular degeneration) is characterised by alteration of hepatic function and structure and by mental deterioration. Congenital hepatic fibrosis is related to cystic disease of

the liver. Clinical features include gross enlargement and firm consistences of the liver, accompanied by manifestations of portal hypertension. Hepatic function is not disturbed.

Clinical features

Many patients with portal hypertension of intra-or extra hepatic origin have symptoms or signs related only to the primary disease and portal hypertension is tolerated without incident for years. Four major clinical consequences of portal hypertension may, however, lead to its recognition: The development of extensive portal systemic venous collaterals, with gastrointestinal hemorrhage; the appearance of congestive splenomegally with hypersplenism; the outset of episodic stupor or hepatic encephalopathy and; the development of ascites. The development of collateral channels between the portal and systemic venous beds is the most characteristic consequence of portal hypertension. Major sites of collateral flow involve cardioesophageal junction (esophagogastric varices), retroperitoneal space, falciform ligament of the liver (periumbilical or abdominal wall collaterals) and dilated veins around the rectum (hemorrhoids). Although hemorrhoids bleed frequently and varices of the bowel rupture occasionally, massive hemorrhage from thin-walled varices in the upper stomach and lower esophagus is the

major complication of portal hypertension. Variceal bleeding occurs without obvious precipitating cause and presents usually as painless massive hematemesis or malena. Abdominal wall collaterals are helpful clinical signs of portal hypertension and appear as slightly tortuous epigastric vessels (caput medusae) that radiate from the umbilicus toward the xiphoid and rib margins. Vascular bruits may be heard over the upper abdomen. Splenomegaly, the result of passive congestion,fibrosis, and siderosis, is present in most patients with significant and longstanding portal hypertension, because splenic size correlates poorly with the level of portal pressure. Splenic enlargement may be accompanied by abnormal sequestration and destruction of circulating blood cells (hypersplenism). Patients with portal hypertension and with extensive portal-systemic shunting may experience bouts of hepatic encephalopathy or coma. These episodes are often precipitated by gastrointestinal hemorrhage and a moderate to high protein diet. Ascites is not a feature of portal vein obstruction or other forms of presinusoidal portal hypertension but is a common occurrence in portal hypertension associated with cirrhosis or hepatic venous blockade.

The diagnosis of portal hypertension is usually based on indirect evidence such as the presence of esophageal

varices, abdominal collateral veins, hypersplenism or ascites in a patient with cirrhosis. Esophageal varices are visualized in the majority of cases by a careful barium swallow or fibreoptic endoscopy.

Direct measurement of portal venous pressure by transhepatic catheterisation is technically difficult and is not usually performed except during surgery. In certain patients with suspected portal vein thrombosis or previous porto-caval shunt surgery it is useful to obtain measurement of portal pressure and visualization of the portal system prior to operation. Splenoportography, which requires percutaneous needle puncture of the spleen is not widely used because it is technically difficult and hazardous. Mesenteric and hepatic angiography are frequently obtained prior to shunt surgery to visualise the hepatic arterial bed as well as the portal system during the venous run off phase. This allows the surgeon to plan the operative approach and also provides valuable information regarding the presence of unsuspected lesions such as hepatocellular carcinoma or Budd-Chiari lesions. Rectal snip will exclude schistosomiasis. Where ultrasonography facilities are available this should be done to assess the liver' the portal and splenic vein diameter should also be recorded.

Variceal bleeding is the unifying pathway to the diverse causes of portal hypertension. Although 30 percent of all patients with cirrhosis have esophageal varices only 30 percent will ever bleed from them. The interval between diagnosis and initial bleeding varies, but almost all hemorrhages occur within two years of diagnosis. The degree of portal hypertension has no predictive value in determining who will or will not bleed. The size of the varices, however, is directly related to the risk of bleeding. The inciting event erosion versus explosion is unknown, although food, gastric acid and salicylates have been implicated. Initial bleeding in cirrhotics results from esophageal varices in 60 percent of patients (20 per cent each for ulcers, Mallory-Weiss and gastritis) and carry a 50 to 70 per cent mortality if untreated. Furthermore, once bleeding is controlled 60 percent will have a second major hemorrhage within one year. Thus the control of bleeding must be rapid, with an attempt toward stabilisation and performance of a definitive procedure at a later date.

Treatment

Most commonly patients with portal hypertension present with hematemesis and initial stabilisation must be accomplished as for any major hemorrhage of the gastrointestinal tract, but certain factors must be given

special consideration in cirrhotic patients. Adequate blood replacement is administered but overtransfusion must be avoided. Fresh frozen plasma and platelets are used as necessary and saline solution is avoided because it may increase ascites in the presence of cirrhotic hyperaldosteronism. Spironolactone is sometimes used to prevent overload of sodium. Placement of a Swan-Ganz catheter and monitoring in the intensive care unit are often required. Because of the high frequency of associated peptic ulceration and gastritis in alcoholic cirrhotics cimetidine may be given. Sedation is avoided because of impaired metabolism of drugs by cirrhotic liver but chlordiazapoxide hydrochloride, Heminervrin or Chlormethiazole may be given if delirium tremens is suspected. Lactulose and neomycin are appropriate when hepatic encephalopathy is present. Lactulase containing becteria convert lactulose into acetic and lactic acids, which decrease colonic pH and transform absorbable ammonia (NH3) into its nonabsorbable, ionised form (NH4+). By eliminating NH4+, the amount of serum ammonia decreases and encepalopathy improves. Lactulose also distends the colon, thus promoting diarrhoea and assisting the evacuation of blood from intestines. Neomycin destroys urease-producing bacteria and digestion of protein (hemoglobin). A nasogastric tube is usually inserted initially to lavage

the stomach to detect any continuation or recurrence of bleeding.

If ascites presents a serious problem, paracentesis is appropriate to decrease intra-abdominal pressure. The aforementioned resuscitative measures aim at rapid stabilisation of the patient within one to two hours after admission, so that an accurate diagnosis can be made using endoscopy.

Most centres have a sequential management policy, employing the simplest and least invasive method first. One of the major factors in determining the patients immediate prognosis is deterioration in liver function. In those with poor liver function measures to stop bleeding and prevent early rebleeding are very much less successful. The most important prognostic indicator for survival of cirrhotics, who have bled from their varices, is the degree of hepatic failure which is present(9).

Endoscopy is the primary diagnostic method in acute variceal bleeding. Hemorrhage must, however, be slowed to permit adequate visualisation. In general patients with amounts of albumin greater than 3 grams per decilitre and mild or moderate ascites, encephalopathy or malnutrition are catergorized into child's A or B (good risk patients). This method of categorisation has been shown to be a useful predictor of operative survival and prognosis in cirrhotic

patients. Knowing the Child's classification for a patient can help determine which therapeutic options are most appropriate in the future management of the patient.

Vasopressin has been shown to vasoconstrict the splanchnic bed, decrease cardiac output by 14 percent and decrease the heart rate by 11 percent. These effects secondarily decrease hepatic blood flow by 44 percent and wedged hepatic vein pressure by 11 percent, thus decreasing the gradient between portal and systemic pressures by 23 percent and reducing the extent of variceal hemorrhage[10]. Administration of vasopressin is begun after the patient is stabilized. The initial dosage is 20 units per 100 millilitres of 5% dextrose in water over 20 minutes, followed by a continuous infusion of 0.2 to 0.4 unit per minute, to 0.8 units per minute if necessary. If bleeding is not severe a continuous drip alone may be necessary. The drug should be continued for three days or until bleeding ceases and then tapered by 0.1 units per minute at intervals of six to 12 hours. Twenty four hours are usually required before complete discontinuation. Vasoconstriction of the coronary artery leading to myocardial ischemia is one of the untoward side effects of vasopressin. If vasopressin is given with sublingual nitroglycerin to patients at risk, the effects of portal pressure are maintained while the deleterious cardiac

effects are reduced. Vasopressin also has antidiuretic hormone effects that may cause dilutional hyponatremia with decreased urinary output. Diuresis however occurs with cessation of the drug. Vasopressin also causes peripheral vasoconstriction, which may produce abdominal cramps and occasionally, Raynaud's symptoms, thereby limiting its effectiveness. The use of an analoque such as triglicyl vasopressin is probably safer than the parent vasopressin but there is no clear cut evidence of increased efficacy.

Somatostatin is a 14 aminopeptide which is of value in controlling acute variceal hemorrhage by reducing portal pressure. Its current high cost prevent wider use. It appears to be as effective as vasopressin but does not result in serious complications.

Propranolol is a non-selective beta-blocker that has been shown to decrease the risk of recurrent esophageal variceal bleeding in patients with portal hypertension. It decreases the wedged hepatic vein pressure, cardiac output and hepatic blood flow in alcoholic patients with cirrhosis, for whom the dosage is adjusted to decrease the heart rate by 25 percent.

Endoscopic injection sclerotherapy was first reported by Crafoord and Erencker in 1939[11,12]. There was initial interest but this was largely supplemented by the newly introduced portacaval shunting operations. Today it is the

most commonly used method for managements of patients with esophageal varices.

Although sclerotherapy is largely performed by physicians, usually gastroenterologists or hepatologists, the procedure is part of the surgical armamentarium in a number of units There has been an almost complete shift from the original rigid endoscopic techniques to the use of the flexible fiberoptic endoscope. Two main technical variants exist, i.e, intravavariceal and paravariceal technics. The concept of intravariceal sclerotherapy is that the variceal channels will be thrombosed and obliterated thereby controlling acute variceal bleeding and preventing recurrent variceal bleeds. The concept of paravariceal injection in acute bleed setting is that this procedure produces a bleb or swelling that compresses the adjacent varix and stops bleeding. In long-term management it produces thickening of the overlying mucosa, thereby theoretically reducing or preventing recurrent bleeds. A number of sclerosants are in use including ethanolamine oleate, polidocanol, absolute alcohol and sodium tetradecal sulfate (STD).

Most results suggest that between 80 and 90 percent of acute variceal bleeds can be controlled with endoscopic injection sclerotherapy. Rebleeding ranges from 2.9 to 66 percent at two years (mean 40%)[9]. Data in reports of

several controlled studies show significantly lower rates of early death of patients with acute bleeding managed by sclerotherapy compared with conventional methods. The effect of sclerotherapy on longterm survival, however is not clear cut.

In a randomised study, conventional medical therapy was compared with endoscopic sclerotherapy in the longterm management of 75 patients (37 controls and 38 treated with ES) who had esophageal bleeding. The ES patients had fewer episodes of recurrent bleeding than did the controls (43 versus 73 per cent), and a life table analysis revealed that there was no difference in survival rates between the two groups at five years. This suggest that, although ES may improve early survival rates, its effect on longterm survival is uncertain.

In a prospective, randomised trial reported in 1986 endoscopic sclerotherapy (ES) was compared with distal splenal renal shunt (DSRS). The 26 months follow-up study involved 71 patients, 36 of whom were treated by ES and 35 by DSRS. Alcoholic cirrhotic patients composed 61% of the group and non alcoholic cirrhotics, 39%. A total of 56% of the patients with cirrhosis were classified in Child's A or B category and 44%, Child's C. Patients in whom ES failed (total of 12) underwent DSRS, thus creating a bias in the protocol favoring the ES group. Rebleeding was

significantly reduced in the group with DSRS (3 versus 53 per cent), while the two year survival rate was significantly improved in the ES group (83 versus 59%). Moreover, portal perfusion and galactose elimination capacity (an indirect measure of hepatic function) both improved significantly at one year in the ES group, compared with the group that underwent DSRS. Encephalopathy, however, occurred with equal frequency in both groups. ES is associated with a higher rebleeding rate than DSRS with one third of the patients failing treatment and requiring surgical intervention. When it is successful, ES allows significant improvement in hepatic function. The combination of initial ES and back-up and surgical intervention may be more beneficial than sclerotherapy alone for patients with acute variceal bleeding, while, the DSRS may be a more suitable surgical technique for patients having previous ES. In general, the results of most studies show that survival curves after acute variceal bleeding are parallel, no matter how the bleeding is managed. The major difference between ES and DSRS is that approximately 80 per cent of ES patients leave the hospital while only 58 per cent survive shunting or conservative therapy(10,12). The complications of ES are primarily local and minor, with perforation rarely

occurring, and dysphagia, superficial ulceration and retrosternal pain in the chest being common.

Balloon tamponade is a temporising technique to control bleeding while other modalities of management are being arranged. It is most effective especially when used within 6 hours of onset of hemorrhage[13]. One of the three types of tubes is the Blakemore-Sengstaken apparatus, which contains a gastric tube as well as an esophageal and gastric balloon. A nasogastric tube is positioned in the esophagus to prevent aspiration.

Another is the Edlich-Minnesota tube, which is similar to the Blackemore-Sengstaken but has an esophageal tube for continuous aspiration of salivary secretions obviating the need for a separate nasogastric tube. The third type of tube is the Linton-Nachlas, which is similar to the Edlich-Minnesota but has no esophageal balloon. Once the gastric balloon is placed in the stomach, its position is checked by roentgenography after instillation of 100 millilitres of air.

If the gastric balloon alone controls the bleeding, it is left in place for 24 hours before deflation. If both the esophageal and gastric balloons require filling, they are also left in place for 24 hours, after which the esophageal balloon is deflated, and if bleeding does not recur, the tube is left in place for one more day before removal. If

bleeding recurs, the procedure is repeated while more definitive action is planned. Serious complications occur esophageal rupture or necrosis on both as well as tracheal obstruction secondary to migration of the balloon have also been reported. Moreover, areas of necrosis in the naris secondary to pressure from traction may occur.

In general, 80 per cent of variceal bleeding will cease with the use of the Sengstaken-Blakemore tube but rebleeding recurs shortly after deflation of the balloon in 24 to 52 percent of patients and in 35 percent it cannot be controlled. Moreover, the incidence of another episode of bleeding after a second period of control is 40 per cent so that 80 percent of the originally controlled 80 percent eventually rebleed. With such poor results, the use of this Method of Management must always be followed by a more definitive form of therapy.

Percutaneous transhepatic embolisation (PTE) was first introduced by Lunderquist and Vang in 1974[13]. The coronary vein is usually the largest inflow tract and is selectively embolised by a variety of occlusive agents, such as spring coils, absorbable gelatin sponges and thrombin, Microfibrillar collagen, sodium tetradecyl sulfate, bucrylate, and detachable ballons, have been inserted into this channel in an attempt to occlude esophageal varices. Initial control is achieved in 45 to

90 percent of patients, but in all series, there is a high rate of rebleeding of 25 to 86 per cent within days to months. Moreover the complication rate is high, with a mortality rate of 5 to 20 per cent and an intra-abdominal bleeding rate reported as high as 20 percent. Embolisation of the occluding material has been reported and thrombosis of the portal vein occurs in 20 to 30 percent of patient, making future portalcaval shunting impossible. Transhepatic embolisation is currently used as a temporising step in very poor-risk patients who continue to bleed despite the use of Pitressin, ES and balloon tamponade. Endoscopic electrocautery as well as laser photocoagulation has been tried. Endoscopic spraying of topical clotting factors such as thrombin with fibrinogen, cryoprecipitate, collagen and the cyanoacrylate polymer glues have been used in gastrointestinal bleeding but not in esophageal varices where they would probably be ineffectual(13).

A good-risk patient (Child's A or B) who continues to bleed after receiving pitressin and ES is a candidate for operation before excess transfusion and fluid increase operative risk. Patients with amounts of Serum glutamic oxaloacetic transaminase greater than 300 units per litre, total bilirubin greater than 6 milligrams per decilitre, ascites, hypoalbuminemia or other determinants of a Child's

classification are associated with a mortality rate after surgical intervention near 100 percent and maximum non operative management should be used for these patients[19].

NON SELECTIVE SHUNTING

Angiography to define suitable venous anatomy should be done before surgical treatment. These studies delineate the specific anatomy of the given area, asses portal hemodynamics and determine the direction of portal flow (hepatopedal versus hepatofugal). The information is critical in determining the appropriate shunting procedure to be used. The liver receives 75 percent of its blood supply from the portal vein and 25 percent from the hepatic artery: yet each of these vessels carry 50 percent of the hepatic oxygen supply. Portacaval Shunts (PCS), by directing 50 per cent of the oxygen from an already impaired liver, leads to further hepatocellular damage resulting in hepatic encephalopathy (HE). Four prospective randomised, controlled clinical trials in the United States and France have been done to determine the efficacy of elective PCS[13]. The operative mortality ranged from 8 to 19 per cent (mean of 15 percent), while the rebleeding rate was 9 per cent in the group treated with shunts and 70 percent in the medically treated group. Hepatic Encephalopathy occurred equally in both groups (approximately 35 percent), while if was usually mild and

easily treated in the medically treated group, in the group with shunts it was usually severe and chronic. Moreover the five year survival rate was not significantly improved in the shunted group (approximately 40 to 60 percent) when compared with the medical treated group (approximately 10 to 50 percent) in either study. The results of the four studies show that PCS will prevent recurrent bleeding if they remain patent, but the survival is not improved and the quality of life is impaired because of the degree and chronicity of HE.

H-grafts are physiologically and functionally identical with side to side PCS. Gortex (expanded polytetrafluoroethylene) or Dacron (polyestre fiber) 14 to 20 millimetre shunts are used but some investigators have tried 10 millimetre grafts. In doing so, however, patency is sacrificed to increase hepato pedal flow. Indeed, the shorter and wider the graft, the better the patency but the lesser the portal perfusion. The overall mortality rate and incidence of rebleeding and hepatic encephalopathy in mesocaval shunt is identical with that of conventional PCS; however with the collective incidence of thrombosis of the graft being 9 to 14 per cent, the application of this procedure in the elective situation is limited except in certain instances, such as ascites, periportal fibrosis, portal vein thrombosis, previous operation in the right

SELECTIVE SHUNTING

Distal splenorenal (Warren) shunt. The goal of a Warren shunt is the selective transsplenic decompression of gastroesphageal varices through the short gastric veins with simultaneous maintenance of hepatopedal portal perfusion and intestinal venous, hypertension[18]. In this way recurrent variceal bleeding is prevented while progressive damage to the liver and Hepatic encephalopathy is not enhanced. The main components of this procedure include the division of the splenic vein as it joins the superior mesenteric vein together with anastomosis to the superior aspect of the left renal vein, gastric devascularisation with preservation of the short gastric veins coronary-azygous disconnection, and ligation of the left adrenal and inferior mesenteric veins (optional). The aims of DSRS are reached only after gastric devascularisation with preservation of the short gastric veins and coronary-azygous disconnection are performed. DSRS has been compared with PCS (mainly mesocaval shunts) in six randomised trials[19]. In general, no difference in survival rates was seen after either operative approach. HE, however is significantly reduced after selective shunting, and long term patency and control of bleeding are better after selective than after non-selective shunting.

Splenopancreatic disconnection (SPD) is a recent modification of DSRS. Conventional DSRS leaves the splenic vein embedded in the body of the pancreas and 60 percent of alcoholic patients have large portal to splenic vein collaterals develop by one to two years postoperatively. The collaterals may enlarge and decompress the portal system, thereby creating the so called pancreatic siphon, which carries blood from the portal vein by way of the pancreas to the low pressure shunt. The pancreatic siphon may contribute to the loss of portal perfusion in 50 to 60 percent of alcoholic cirrhotics and to the loss of pancreatic hepatotrophic factors in all patients, leading to measured reduction in the volume of the liver at one year. SPD which involves the complete separation of the splenic vein from the pancreas during DSRS, was designed to interrupt the pathway of this transpancreatic siphon. DSRS-SPD has significantly improved maintenance of portal perfusion and survival in patients with alcoholic cirrhosis requiring selection shunt for variceal bleeding when compared with standard DSRS. In this population DSRS-SPD is the procedure of choice. In patients with non-alcoholic cirrhosis DSRS-SPD has not shown advantage over standard dsrs[20].

The low operative mortality rate and low incidence of HE and hepatic failure as well as improved survival rate in

non alcoholics, make DSRS the gold standard against which other methods of definitive control of variceal bleeding must be compared. Sometimes DSRS is not feasible as it is contraindicated in the presence of ascites, hepatofugal flow, portal vein occlusion, and after splenectomy or left nephrectomy. Central splenorenal (Linton) shunting involves a splenectomy with a proximal splenic vein to left renal vein end to side anastomosis. It is technically demanding and has a low patency rate secondary to low flow through the small diameter of the splenic vein. They are frequently complicated by thrombocytopenia, although this is rarely detrimental to the patient.

Devascularisation and Transection Operations

A wide variety of devascularisation and/or transection procedures involving the lower oesophagus and upper stomach have been described. The variceal bleeding is usually situated in the lower 4 to 5 cms of the esophagus. Conceptually this area is disconnected from the high pressure, intra-abdominal portal venous systems by dividing in flow venous connection as part of an esophagogastric or esophageal or gastric devascularisation procedure while the venous blood flow in the deeper layers of the stomach or esophagus is interrupted by division and anastomosis of either organ. Today most workers divide the lower

esophagus immediately above the esophagogastric junction using a staple gun for the anastomosis.

Various transabdominal devascularisation procedures have been described. One of the most widely used in the past was the Hassab procedure, which is an upper gastric devascularistation and splenectomy without transection. Excellent results were obtained in schistosomiasis. An alternative and very similar procedure is the Womack operation that devascularises the upper stomach and also includes splenectomy and oversewing the varices but without transection. Current evidence suggest that a devascularisation procedure alone without a transection is insufficient in Western patients presenting with cirrhosis.

The Suguira procedure of esophageal transection has its goal to reduce the gastro esophageal venous pathway of portal hypertension. Its main components consist of trans-thoracic esophageal transection and devascularisation, trans-abdominal esophagogastric devascularisation, splenectomy, sparing of the coronary azygous communication and, finally, fundoplication and pyloroplasty. The esophagus and stomach are thoroughly stripped of all venous collaterals from the left pulmonary vein to the upper one-half portion of the stomach. It is done in two stages, depending on the condition of patient. If two stages are chosen, suguira recommends that the abdominal portion be

done transdiaphragmatically through the thoracic cavity during the first operation. Because portal hypertension is maintained after the Sugiura procedure, hepatic function is preserved. Excellent results of the Suguira procedure has been reported in Japan and elsewhere, but results in the West where the patient material is different have been less favourable. A study in New York concluded that although the procedure stops acute bleeding reliably and prevents early rebleeding, it is tedious, time-consuming operation with a high complication rate and a high rebleed rate. They point out that similar results can be achieved with simpler procedures[9].

Conclusion

The management of both acute recurrent variceal bleeding continuous to be a significant challenge to the clinician. The cause and pathogenesis of portal hypertension has been described. A patient who presents with variceal bleeding must be rapidly stabilized with fluid resuscitation and general measures such as the use of vasopressin and balloon tamponade, must be instituted to control hemorrhage so that endoscopy can be used to establish the diagnosis. Sclerotherapy achieves a high rate of success in acute situation, but if hemorrhage cannot be controlled, back up surgical devascularisation or shunting should be planned, Endoscopic injection sclerotherapy is not currently

available in Muhimbili Medical Centre, however such a facility when available may go a long way to improve patient care. Preventions of portal hypertension by controlling hepatitis, schistosomiasis and alcoholism has been mentioned.

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4. CHOLELITHIASIS IN MUHIMBILI MEDICAL CENTRE

ABSTRACT

A review of 31 patients who underwent cholecystectomy in the Department of Surgery Muhimbili Medical Centre from July 1988 to July 1990 is presented. Eighty seven percent of the patients had cholelithiasis. 87% of the patients were females. Their ages ranged from 26 to 76 years with a mean of 38.6 years. Three patients (9.7%) developed post cholecystectomy complications. One patient had superficial wound sepsis while two had post operative jaundice requiring relaparotomy and billiary duct exploration. The duration of hospital stay after surgery ranged from 4 to 16 days with a mean of 11 days. Operative mortality was zero.

Key Words: Cholelithiasis: treatment, morbidity, mortality: M.M.C.

INTRODUCTION

The epidemiology of gallstones is both exciting and frustrating. Exciting because it holds the key to the aetiology and frustrating because accurate information on who gets gallstones is hard to come about. The incidence varies widely in different parts of the world and is reported to be increasing. To most people of the last generation and to many now, the epidemiology of gallstones is summed up in that all too memorable phrase! Female, fair, fat, fertile and forty or fifty, while some would

even add "flatulent". Most of these features are not particularly associated with gallstones[1].

Gallstones remain undetected during life in a large proportion of cases. This is because most gallstones are assymptomatic. Consequently the prevalence of gallstones cannot be deduced from clinical records. Such records, however are useful in indicating gross differences in prevalence and detecting a changing trend in the incidence of biliarycalculi. The ideal way of determining prevalence of gallstones would be graphic survey in randomly selected samples.

American, Indians have the highest known prevalence of cholelithiasis. A cholecystographic survey of 596 Pima Indians in Arizona revealed gallstones in no less than 73 per cent of women aged 25-34. In the entire sample which was made up of about equal numbers of male and females and in each decade from 15 to 74, the prevalence was 48.6 percent[1]. In North America generally gallstones are common. The actual prevalence of gallstones in the U.S.A. is not known. In a very large autopsy survey at Philadelphia, covering the period 1920 to 1949 gallstones were present in 16.8 per cent of women and 7.8 per cent of men aged over 20[1]. The prevalence in Negroes is less than half that in whites.

In Western Europe too cholelithiasis is also a common disease. In Leeds stones were present in (autopsy series) about 15 per cent of autopsied women in the 40-49 age group, whereas in London and Birmingham the figures are 7 per cent and 6 per cent[1]. In the older age group (76yrs) the overall prevalence may rise to 30 per cent and the sex ratio is less marked[2].

In France and Germany, gallstones are probably at least as common as in Britain. Jorgensen studied the prevalence of gallstones in a random sample of 540 men and women aged 70 years in the Western part of Copenhagen by ultrasonography. He found prevalence of gallstones to be 30.2% and 18.8% among women and men respectively being significantly higher among women[2]. Other Western-type societies where gallstones are common include Israel, South Africa and Australia.

In the orient, gallstones are relatively uncommon. In Thailand cholelithiasis is present in only 3 per cent of autopsies on women aged 40 to 49. However in Singapore 6 per cent of such women were found to have stones. A postal survey in China in the 1920s revealed that biliary calculi accounted for only 0.23 per cent of laparotomies compared with 9.3 per cent at the Mayo clinic. Characteristically many if not most oriental gallstones are rich in bile pigment and are located in the common bile duct rather than

the gallbladder. Thus in Hong Kong Chinese, 74 per cent of gallstones were found at laparotomy to be in the bile ducts and 88 per cent contained only minor quantities of cholesterol. By contrast in the same study, stones from the USA were present in the gallbladder in 92 percent of cases and 88 percent were composed chiefly of cholesterol. Africa provides the best documented example of populations which are almost free of cholelithiasis. This is however not the case in Johannesburg where ten percent of Bantu females in their forties have been found to have gallstones at autopsy. In Cairo, one per cent of surgical admissions are due to biliary calculi. In rural sub-saharal Africa the great majority of Africans are almost free of gallstones. For instance in Kampala, Uganda, large surveys shown gallstones to be present in only 1.35 per cent of autopsied subjects aged 35 to 54 and to account for only 15 out of 6,000 hospital admissions. Moreover most of the calculi found were pure pigment stones probably associated with chronic hemolysis(3). Similar figures has been obtained in West Africa. Thus in the first five years of Ibadan University Hospital only 27 patients were admitted to the Surgical wards on account of gallstones and in Accra, Ghana only 0.4 per cent of the general surgical admissions were with symtomatic gallstones. More strikingly, no gallstones at all were found in a series of

4395 autopsies performed in Ghana from 1923 to 1955[1]. These Epidemiological facts accord well with the findings of Biss et al (1971) that the bile of East African Maasai tribesmen is very undersaturated with cholesterol[4]. In Tanzania in 1971, Hiza, while reviewing Surgical Admissions to the Muhimbili Medical Centre recorded only six cases of cholecystitis among 26700, admissions[5].

There is a wide spread impression that gallstones are increasing in frequency both in developed nations and in those which are rapidly developing. These impressions are hard to confirm or disprove because of lack of sufficient statistics. Japan has provided impressive evidence of a changing pattern of gallstone incidence. In Fukuoka, the composition of 20 stones removed in 1927 was compared with that of 20 stones removed in 1967 to 1968. In 10 of the 1927 stones, bilirubin predominated over cholesterol, whereas it was true of only two of the 1967 to 1968 stones[1]. In autopsy series, overall gallstones incidence rose from 4.4. per cent in 1955 to 1961 to 8.3 per cent in 1966 to 1967. It seems likely that in the last two decades there has been an increase in cholesterol stones and probably a fall in pigment stones. At the same time, the typical Western preponderance of females has replaced the previously equal sex incidence.

The steady increase of gallstones prevalence with advancing age has been clearly demonstrated by many autopsy studies. Some workers have calculated that the rate of rise in gallstone prevalence is progressive in males but reaches a peak in the forties in women. Clinically, the commonest age for gallstones to present or at least to be operated upon it the fifties or sixties. It is not clear whether the reduced number of cholecystectomies in the seventies and eighties is due wholly to the smaller size of the population at risk or partly to a lesser tendency of gallstones to cause symptoms in the elderly.

In all countries where cholesterol-rich gallstones are common the prevalence is substantially higher in women than in men. This is true of both autopsy surveys and of clinical experience. The female to male ratio is usually about 2. In Japan the last 40 years have seen the female to male ratio rise from 0.8 to 2.3.

The literature on clinical associations of gallstones is full of unsupported assertations and inadequate statistics. The only completely non-controversial associations (apart from that between pigment stones and hemolytic states) are those with gall bladder cancer and pancreatitis. Both of these disease may be caused by the presence of gallstones. There are also two disorders which are known to alter the composition of bile namely hepatic cirrhosis and terminal

ileopathy, which are undoubtedly complicated by cholelithiasis. In cirrhosis, the autopsy prevalence of gallstones is more than doubled, and most of the calculi are pigment stones. In patients with resection or extensive Crohns disease of the terminal ileum, the cholecystographic prevalence of stones is at least three and possibly five times higher than expected, probably because of bile salt deficiency resulting from interruption of the enterohepatic circulation.

It is generally accepted that gallstones are commoner in women who have been pregnant than in nulliparae; but there is disagreement as to the effect of multi-parity. Most autopsy and clinical survey agree in showing obesity to be commoner amongst gallstone patients than in the general population.

Contrary to previous beliefs, serum cholesterol levels are no higher in gallstone patients than in general population. However it is probable that in populations where gallstones are common, the normal range of serum cholesterol is higher than in those where gallstones are rare. For example, the mean serum cholesterol in rural Japanese is 146mg/100ml and in East African Maasai it is 135mg/100ml[4].

There is probably no association between peptic ulcer and gallstones. Cholelithiasis as a complication of vagotomy and gastric resection has been much discussed, but without

a firm conclusion being reached. Even the "Saint's triad" of gallstones, hiatus hernia and diverticulosis coli has not been adequately documented.

CASE REPORT

G.C (IP. 593624) a 26 years old female sought medical attention on account of eight months. History of recurrent upper right abdominal colicky pain. The initial colicky pain lasted about one hour to be replaced by a dull ache. She also complained of associated frequent belching nausea and occasional vomiting. She often had decreased appetite and she denied history of fever and chills. She was a petty trader, married with one child. On examination she was apparently healthy, not obese, afebrile with a tinge of jaundice. The cardiovascular system was normal with a radial pulse of 80 per minute and regular. The blood pressure was 110/70 mmHg and the Heart sound were normal. The respiratory system was normal. There was tenderness in the Right upper quadrant though Murphy's sign was negative and the gallbladder was not palpable. The liver spleen and kidneys were not palpable and there was no evidence of ascites. She had normal gynaecological findings.

Her hemoglobin was 15.8gm/dl and the total white cell count was 4000/mm3. The platelet count was 294000/mm3. The total serum bilirubin was 28 umol/l and direct bilirubin of 8 umol/l. The prothrombin time was initially 39% of the

control plasma, this later rose to 66% and finally 100% of the control after Vit K injections. The The HBs Ag and alfa-fetal protein were negative. Oral cholecystogram demonstrated multiple calculi. Ultra sonography revealed normal hepatic echogenicity multiple gallbladder stones, thickening of gallbladder wall and echogenic lesion within the common bile duct which was 8mm wide. The diagnosis of Cholelithiasis, choledocholithiasis with obstructive jaundice was made and the patient was planned for cholecystectomy and common bile duct exploration. Preoperatively the patient was given Vitamin K. injections and serial PT were done. Blood grouping and Crossmatching of two units was done. The patient was put in supine position and under general anaesthesia the lower thoracic region was supported by the "bridge" of the operating, table. A right subcostal incision was made since the patient had a wide subcostal angle. Careful examination of the stomach, duodenum and the colon did not reveal any abnormality. Palpation of the gallbladder revealed multiple calculi. The common bile duct was also identified and adhesions noted. A hand was passed over the liver to admit air to enter the subphrenic space and allow the liver to descend. The first abdominal pack was placed in the lower part of the wound, displacing downwards the duodenum, transverse colon and the small intestine, a second pack was

placed medially to cover and retract the stomach. A third pack was placed laterally to fill the right kidney pouch. Deep retractors were placed in position and held by assistant so as to give best exposure. A sponge holding forceps was applied to the infundibulum of the gallbladder and the bladder was gently drawn forwards and to the right. The junction of the cystic and common ducts was then displayed by snipping of the overlying peritoneum by a combination of blunt and sharp dissection. A silk ligature was passed around the cystic duct and tied close to its junction with the common bile duct. An artery forceps was applied 1 cm from the ligature; a small swab was placed behind the cystic duct in order to absorb any leakage and the duct was divided close to the forceps. By gentle traction of the forceps and by gauze dissection the cystic artery was displayed as it was branching from the right hepatic artery to reach the upper surface of the gallbladder neck. It was above and posterior to the cystic duct just below the liver. The artery was divided between silk ligatures. The neck of gallbladder was then drawn forward away from the liver and a finger was insinuated between the two.

The finger was then gently worked up ward separating the gallbladder from its bed. As the separation progressed the peritoneal reflections on each side of the bladder were

divided with scissors leaving a fringe of peritoneum on each side with which to cover the gallbladder bed. No abnormal blood vessel was encountered and the separation of the gallbladder was completed and the peritoneal fringes were apposed with catgut to cover the gallbladder bed. The common bile duct was then opened by a one cm. longtudinal incision after applying two catgut stay sutures. Several pigment stones and sludge were milked out. The stones were 0.4 to 0.5 cm. diameter. The common bile duct was then flushed with normal saline and was noted to be patent. A 0.4cm T-tube was inserted at the choledochotomy site after it was nipped several times on the side opposite the T to make drainage holes. A two cm. length of tubing was left on either side of the T. The tube was brought out laterally through a stab skin incision. Operative cholangiogram was not done due to lack of portable X-ray machine. A corrugated rubber drain was left in situ at the gallbladder bed and the incision was closed in layers.

Post operatively the patient had injection Ampicillin 500mg IV six hourly for 24 hours. She had 3.5 litres of fluids (Normal saline and Dextrose 5%) in the first 24 hours. No excessive drainage was noted from the corrugated rubber drain or the Nasogastric tube. The Nasogastric tube was removed on the second post operative day and the patient

started oral fluids and light porridge diet. The corrugated rubber drain was removed on the third post-operative day, and skin stitches on the tenth post operative day. The patient remained afebrile and no jaundice developed. On the fourteenth post operative day a T-tube cholangiogram was done. Free flow of contrast into the duodenum was demonstrated and there was no leak at the choledocotomy site, or stones noted in the common bile duct. The T-tube was left to gravity drainage and then pulled out the next day.

The patient was discharged in good condition for follow-up in the Surgical out patient clinic where she was seen two weeks later free of jaundice fever and pain. Three months after cholecystectomy the patient was completely free of pain, and had resumed her normal activities fully. Gallstone analysis revealed Calcium carbonate stones and the gallbladder bile was sterile.

RESULTS

Table I: Sex distribution.

Sex	Number	%
Female	27	87.1
Male	4	12.9
Total	31	100.0

The majority of the patients were females accounting for 87.1% of the cases. M:F = 1:7.

Table II: Age distribution.

Age	Number	%
20-29	5	16.1
30-39	17	54.9
40-49	4	12.9
50-59	1	3.2
60+	4	12.9
Total	31	100.0

The age ranged from 26 to 76 years with a mean of 38.6 years. Most of the patient were young females 54.9% of them being in the 30-39 year age group.

Table III: Operative findings.

Operative findings	Number	%
Gallbladder stones	27	87.1
Adenocarcinoma	1	3.2
Acalculous cholecystitis	1	3.2
Hydrops cystica	1	3.2
Periampullary tumor	1	3.2
Total	31	100.0

The commonest indication for cholecystectomy was gallbladder stones seen in 27 (87.1%) patients. There was one case of superficial wound sepsis and two cases of post-operative jaundice requiring relaparotomy and biliary duct exploration(6.5%). The duration of Hospital stay after surgery ranged from 4 to 16 days with an average of 11 days.

Operative mortality was zero.

DISCUSSION

Traditionally it is taught that there are three basic types of gallstones: Solitaire cholesterol stones, pigment stones and Mixed stones. Solitaire stones usually contain in excess of 95% crystalline cholesterol, while the majority of mixed stones contain over 70% cholesterol. The easily recognizable, small friable black pigment stones on the other hand contain little cholesterol. All gallstones contain a small amount of protein matrix which acts as a skeleton and their other main constituents are cholesterol, calcium carbonate (valerite aragonite) calcium phosphate (apatite and whitlockile), calcium polocite and calcium bilirubinate. Little is known about why patients form stones containing the calcium salts of carbonate, phosphate, or palomitate. It is plausible to assume that at the time the stones are forming their respective solubility products in bile must be exceeded.

One popular theory held that infection in the wall of the gallbladder led to exfoliation of inflammatory cells which served as a nidus for the precipitation of cholesterol into stones. This view supposed that infectious agents from the bowel reached the gallbladder by lymphatic or hematogenous spread. Since the stones could also be found in the absence of demonstrable infection, others regarded gallbladder infection as an effect of gallstones rather than their cause. This school of thought maintained that gallbladder stasis initiated formation of stones by allowing gallbladder bile to layer out into a colloidal solution that permitted cholesterol to precipitate [6]. Investigators began to pay more attention to the physico-chemical properties of biliary lipids. The three major classes of lipids in bile are cholesterol, bile salts and phospholipds. Cholesterol by itself is insoluble in water and so in lecithin which is the major phospholipid in bile. However when mixed together, these three lipids associate into complex aggregates known as micelles in which the lipophillic portions, of the three molecules aggregate in the centre of the structure, while the hydrophilic portions are oriented peripherally thus allowing solubility in the acqueous environment. These micelles have the polar groups of the bile acids molecules oriented outwards into the molecular, aggregate. Thus the centre of the micelle can

be conceived as having fat-dissolving properties and it is for this reason that the bile salt molecule is able to dissolve cholesterol.

Cholesterol solubility is determined by the ratio of cholesterol to bile acids and phospholids rather than absolute amounts. An elegant method of depicting the relationship of these three components is to plot them on triangular coordinates. In this way, each bile sample can be plotted as a single point on the graph and it becomes easier to make comparisons between biles of different composition. In constructing such a tertiary phase diagram the relative quantities of cholesterol, phosplodipids, and bile acids in mmo/l are expressed as a percentage on a triangular diagram. The lithogenicity of bile may thus be expressed as a single point on the triangular coordinates. When samples of gallbladder bile were plotted graphically on these coordinates, Admirand and Small noted a fairly clear separation between patients with cholesterol gallstones and a control population without stones. Furthermore when gallbladder and hepatic bile from patients who formed stones were analysed in this fashion, it was found that both were supersaturated with cholesterol implying that the liver was the source of lithogenic bile in these patients [7,8]. It appears therefore that, the pathogenesis of cholesterol gallstones requires three

stages namely, secretion of supersaturated bile, precipitation of microcrystals and growth into a macroscopic stone.

The incidence of cholesterol cholelithiasis is increased in obesity because of excessive rates of cholesterol synthesis, and secretion into bile. Virtually all the cholesterol in bile is synthesized de novo by the hepatic Microsomes from acetate, the enzyme which limit the rate of cholesterogenesis being 3-hydoxy-3methyl glutaryl-CoA reductase (HMGCoAR). Although it is known that high caloric intake predisposes an individual to stone formation by increasing the synthesis and secretion of endogenous cholesterol the effects of ingestion of excessive cholesterol in less clear. The effects of a diet high in polyunsaturated fats are unclear but at least some individuals seems to respond to such a diet with increased secretion of cholesterol in bile. The use of clofibrate to lower serum by increasing the biliary secretion of cholesterol.

In a few cases supersaturation result from a relatively pure deficiency in the secretion of bile salts. This occurs as in interruption of the enterohepatic circulation such as by T-tube fistula after common bile duct exploration, creates a situation in which the ability of

the liver to synthesize new bile acids is exceeded by the rate of loss of these acids.

As a result of this, the bile salt pool and the rate of secretion of bile salts can progressively decrease and bile becomes super saturated with cholesterol. Normally bile salts are reabsorbed actively in the terminal ileum. If this reabsorption is inpaired by malabsorption such as in regional ileitis or cystic fibrosis with pancreatic insufficiency, on resection of the terminal ileum, the bile acid pool will diminish, resulting in supersaturation in bile. Similarly ileal by pass results in increased lithogenicity.

In the examples cited above one can indict either increased secretion of cholesterol or decreased secretion of bile salts to the formation of gallstones. In the majority persons who form stones, however bile supersaturation appears to result alterations in the enterohepatic cycling of bile salts which secondarily results in changes in the synthesis of biliary lipids and their secretion in liver. Even "normal" subjects.without gallstones intermittently secrete supersaturated bile. These periods of lithogenicity can be temporally related to the interruption of enterohepatic circulation that normally occurs during fasting. After a meal about 98% of the bile salts entering the intestine are reabsorbed and returned through the

portal vein to the liver, where they are cleared and resecreted into canaliculi. During the following fasting period most of the bile salt pool becomes, sequestered in the gallbladder and in effect is removed from the entrohepatic circulation until the next meal. As a result, the return of bile salt to the liver, decreases and the same happens to the secretion of bile salts from the liver. The rate of synthesis of bile salts is determined by a feedback mechanism so that the amount produced equals the small amount lost in feces, but much details of this feedback mechanism are unclear.

Schaffer and Small examined the improved solubility of biliary lipids that occurs after cholecystectomy in many patients. In grossly obese patients they found abnormally increased secretion of cholesterol pre- and postoperatively and little improvement in the solubility of biliary lipids after surgery. The majority of those who formed cholesterol stones, however, were characterised by normal rates of secretion of cholesterol before and after surgery. In this group, removal of the gallbladder resulted in more rapid cycling of the bile pool-(since it could no longer be sequestered in the gallbladder between meals) and a corresponding improvement in the solubility of hepatic bile(9). It has been suggested that dietary fibre decreases the synthesis or absorption or both of

deoxycholic acid in the colon. Although the overall pathogenic importance of these observation is unclear, gallstones have been shown to form in several species fed with fiber-depleted carbohydrate. Furthermore, this phenomenon may account for the low incidence of cholesterol gallstones in populations in which the diet is rich in fibre (4).

Toshinao has recently demonstrated that gallbladder bile was characterised by high concentrations of Prostaglandins and hexosamine and a high cholesterol saturation index. It is postulated that the high concentration of prostaglandins may be responsible for the high hexosamine concentration in supersaturated bile because prostaglandins are synthesised from arachidonate, which is released from gallbladder membrane phospholipase and cholesterol activate lipase in the gallbladder membrane. Toshinao further speculated that a component of lithogenic bile such as cholesterol or lecithin stimulate hypersecretion of gallbladder postaglandins. In turn prostaglandins stimulates mucin hypersecretion and the high concentration of mucin accelerates nucleation of cholesterol monohydrate crystals (10). Gallbladder mucin has drawn attention as a nucleating agent due to reports of it accelerating the precipitation of cholesterol supersaturated artificial biles and also because the structure of gallstones have

been shown to contain a mucopolysacharide matrix (11,12). Nilsell in a recent study of bile acid pool and gallbladder storage capacity found that storage capacity especially volume maybe a determinant of bile acid pool size. This finding gives some support to the hypothesis that the diminished bile acid pool size seen in gallstone patients is secondary to a reduction of the gallbladder storage function rather than a primary cause of the production of lithogenic bile. Gallbladder volume in gallstones disease reduced by fibrosis and shrinkage of the gallbladder. Loss of ability to concentrate bile acids may also contribute to reduced storage capacity (13).

Pigment stones, both black and brown, arise when there is excess unconjugated bilirubin leading to the precipitation of calcium bilirubinate. Unconjugated bilirubin may precipitate out when the total amount of conjugated bilirubin is increased as in hemolytic and cirrhotic stones, even though its proportion remains quite low; precipitation occurs when the solubility of calcium bilirubinate is exceeded. Brown pigment stones usually develop in the presence of B-glucoronidase promoting bacteria such as Escherichia coli, Bacteroides spp. and clostridia spp. Infection also enhances B-glucoronidase activity due to a decrease in ambient pH (14).

In summary therefore current evidence favours the concept that the liver produces bile which is chemically abnormal in the majority of patients with cholesterol gallstones. This is because of a reduced bile salt and phospholipid content relative to cholesterol and probably reflects a reduced bile acid pool in these patients but increased cholesterol secretion may also be responsible. Thus hepatic bile may be considered to be saturated or supersaturated with cholesterol. This chemical abnormality may be intermittent. The gallbladder must also play an important role in the formation of gallstones by providing the necessary nucleation sites for the precipitation of cholesterol crystals. Furthermore, the gallbladder, by acting as a reservoir, aids in the growth of gallstones. Infection in the gallbladder can enhance this process either by providing nucleation sites or by inducing an unstable chemical environment. It appears that gallstone formation is a complex process that depend upon the interaction of a number of variable factors which must involve both the liver and the gallbladder.

Biliary colic is probably the most common way for gallstones to present. The patient experiences severe pain in the epigastrium or right upper quadrant of the abdomen; it frequently radiates through to the tip of the right scapula. There is often associated sweating, nausea and

vomiting. The patient is usually quite unable to get comfortable and tends to roll about on bed or work around the room. The waves of pain lasting 30 to 40 minutes without relief, quite unlike the short spasm of intestinal colic. An attack of biliary colic may merge into an episode of acute cholecystitis as the impacted stone initiates the inflammatory process by causing extreme bile concentration.

Acute cholecystitis is nearly always associated with gallstones. The inflammation is usually sterile at the onset. The cystic duct become blocked by a stone or mucus. The bile in the gallbladder then becomes increasingly concentrated until it acts as a chemical irritant and an inflammatory response develops in the gallbladder wall. Secondary bacterial invasion occurs unless the cystic duct obstruction is relieved eg. by stone falling back into the gallbladder. The commonest organisms found are Escherichia coli and Streptococus fecalis rarely Clostridia welchii or even Salmonella typhi may be observed. Occasionally gas forms in the gallbladder wall and within the lumen; this is known as emphysematous cholecystitis. It is commoner in males then females and also commoner among diabetics. Acute cholecystitis may develop in patients without gallstones, usually as a result of bacteraemia from another

site which settles in the gallbladder. Acute cholecystitis usually resolves but it may suppurate or become chronic. Chronic cholecystitis almost always develops in association with gallstones. It usually presents clinically as recurrent bouts of biliary colic or repeated bouts of acute inflammation. Other symptoms such as belching, fullness after food, nausea, heartburn and an inability to take normal-sized meals have also been attributed to the presence of cholecystitis. Such symptoms are usually made worse by fatty foods. Sometimes the complications of gallstones produce other presenting symptoms eg. acute pancreatitis, carcinoma of the gallbladder, gallstone ileus or obstructive jaundice. In chronic cholecystitis stones are present in the lumen of the gallbladder and may be single or multiple. The multiple ones are usually faceted and there may be two or three different generation of stones, each generation being of comparable size. Repeated attacks over many years may lead to calcification of the wall, the so called "porcelain gallbladder". Sometimes the gallbladder becomes grossly shrunken by repeated books of inflammation, so that it may be difficult to find at operation and the wall is so thickened that the lumen becomes occluded. Although carcinoma of the gallbladder is rare, it almost invariably develops in the presence of gallstones and chronic

cholecystitis. The histologic features of chronic cholecystitis confirm the presence of fibrotic tissue as the cause of thickening of the wall. Areas of mucosal ulceration are often found probably as the result of trauma by stone in the lumen and endarteritis obliterans in the vessels in the wall of the gallbladder is a prominent feature.

Gallstones are sometimes discovered by chance when they are apparently causing little or no symptoms. Recent investigations have now clarified the position so that rational advise can be offered to the patients regarding surgery. A comprehensive review of 1501 patients was reported from Malmo. The patients fell naturally into three groups, Group A 128 patients in whom immediate surgery was performed. Group B, 534 patients operation being undertaken within a year of stones being found because of symptoms. Group C, 839 patients of whom 13 were not traced and 38 died from other diseases leaving 781 patients who were followed-up over a period of eleven years. Of the 781 patients, 49% (383) remained symptom free up to the time of follow up, 33% (254) developed severe symptoms, mostly recurrent biliary colic and these required operation and 18% (144) developed serious complications usually requiring operation. In this latter group of 144 patients 3 developed carcinoma of the

gallbladder, 42 developed mild to moderate cholecystitis, 39 developed severe cholecystitis and in this group 7 patients died. 59 patients developed jaundice with or without pancreatitis. The complication rate was significantly greater in patients over the age of 60 years. Over the eleven years of followup the annual incidence of patients developing complications showed no tendency to decrease although they had been completely free of symptoms from the time of the stones being first diagnosed until the day the complications developed. Operation was more complicated in the patients in Group C than in Group A and B operative cholangiography was used as a routine in the management of all these patients. In Groups A and B, exploration of the common bile duct was necessary in 16 per cent of the patients but in Group C, it was necessary in 27 per cent a highly significant difference. In almost every large review of biliary tract surgery the morbidity and mortality rate is significantly increased by the necessity of exploring the common duct; for example in a group of 4916 patient undergoing biliary tract surgery in the town of Malmo the overall mortality rate for elective cholecystectomy was 0.22% but for patients undergoing exploration of the common duct in addition to this it was 2.60%. Thus it would appear that the correct advice for patients with symptomless gallstones provided they are

otherwise fit is that they should be treated (15). The treatment should not necessarily be cholecystectomy. Alternative treatment such as chemical dissolution therapy (oral or local) and extracorporeal lithotripsy can be used. Although many gallstones do not cause symptoms and need not be treated gallstone disease in a major cause of hospitalisation for surgical treatment. Since Langenbuch performed the first cholecystectomy in Berlin in 1882 operative removal of the gallbladder has become the standard therapy for patients with symptomatic gallstones. Cholecystectomy has an overall mortality rate of 0.6% to 1.3% increasing from 0.2% in patients younger than 70 years to 5% in patients older than 70 years. A safe and effective treatment for many patients, the morbidity rate of cholecystectomy is 10% to 33%, it is uncomfortable to patients and causes invalidity for one month (16). To reduce the morbidity and mortality rates, as well as the cost several non operative therapeutic alternatives have become available for both cholelithiasis and choleducholithiasis.

Shock waves cause high pressure amplitudes to increase within noneseconds and to respond to the known physical laws of accoustics. If water is used as the transmission medium, focused shock waves spread through the body evenly without several damaging the tissues because the

accoustical impedence of most body tissues is close to that of water. At its focus point, the wave impact against the stone liberates short-term high energy mechanical stresses because of the abrupt change in acoustical impedance. This causes tear-and-shear forces, which, eventually lead to the disintegration of stone. The original waterbath lithotriptor developed spark that are condensed in the second focus. Positioning of the stone in this second focus occurs with a fluoroscopic guidance system.

Second generation ESWL are more compact in design operate at lower costs, require simpler patient positioning and many have the potential capability for treating gallbladder stones. The selection of cases is usually determined according to inclusion and exclusion criteria as follows.

INCLUSION AND EXCLUSION CRITERIA FOR ESWL OF GALLBLADDER STONES.

Inclusion criteria:

1. History of biliary pain.
2. Solitary radioluscent gallbladder stones less than 30 mm. or up to 3 stones with similar total volume.
3. Gallbladder visualisation by oral cholestography.
4. Stone positioning possible in shock wave focus.
5. Shock wave avoid lung and bone.

Exclusion criteria:

1. Acute cholecystitis, cholangitis or pancreatitis.

Conclusions and recommendation

Goitre is a common condition in Tanzania and in this Muhimbili series as in other series it is noted to be mainly a disease of young females. Efforts to control endemic goitre in Tanzania has started by use of oral iodine capsules,. depot iodine injections and also distribution of iodinated salt to endemic areas. Thyroidectomy is a major surgery not without complications. In this Muhimbili series of 76 patients mortality was zero.

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PART II: UROLOGY.

6. A CASE OF CUSHING'S SYNDROME

ABSTRACT

A case of Cushing's Syndrome treated surgically in Muhimbili Medical Centre is presented. Since the advent of cortisone replacement therapy in the 1950s made adrenal surgery possible, it has gained the reputation of being full of medical as well as technical difficulties. It is emphasized in this discussion that with good cooperation of endocrine physicians, anaethesiologist and sound surgical expertise, good results are obtainable even in developing countries like Tanzania.

Keywords: Cushing's Syndrome, Adrenalectomy, Muhimbili Dar es Salaam.

INTRODUCTION

Harvey Cushing's in 1912 and 1932 described the syndrome of "pituitary basophilism" which he characterized as abdominal obesity with thin extremities, cutaneous striae, hirsutism, amenorrhoea, osteoporosis, hypertension and often diabetes mellitus[1]. A small basophil adenoma was found in all but one of the patients in whom the pituitary had been examined adequately, and this finding led Cushing to describe the condition as "pituitary basophilism". He regarded it a pluriglandular syndrome in which the pituitary adenoma

played the main part, though he realized that a similar clinical state might arise from disease of the adrenal cortex- "Cushing's syndrome" as the clinical condition is now called has been universally recognized, and striking advances have been made in our knowledge of its etiology and pathogenesis of its clinical and metabolic features. Whereas formerly the disease progressed rapidly to a fatal termination, many patients can now be treated effectively and supported in good health for years.

In the past it was customary to refer to "Cushing's disease"

when the pituitary gland contained a basophil adenoma. Such a distinction is not always possible on clinical grounds and since a pituitary tumor may develop after successful treatment of Cushing's syndrome, the distinction seems artificial(2). It is therefore simpler to retain the term "Cushing's Syndrome" and to apply it to all patients who exhibit the clinical features described by Cushing.

Since the advent of cortisone replacement therapy in 1950 made adrenal surgery possible, it has had the reputation of being fraught with medical and technical difficulties. However, with good technical expertise, excellent results are obtainable (3).

CASE REPORT

M.S. I.P. 478955 a 17 years old Sukuma male patient was first seen at the endocrine clinic with a three year history of excessive weight gain, body weakness and easy fatigue. His parent had noted three years previously that the young man was rapidly gaining weight and developing a potbelly. They also noted that he was developing a puffy face and easy fatigue - He also complained of bony and joint pains. He denied history of nausea or vomiting. He declined history of increased frequency of micturition.

He had suffered from breast abscesses in childhood, which healed spontaneously. He had no history of prolonged medication. He was the seventh born in a family of ten. Birth and growth were normal before the illness. Other siblings are normal. No consanguinity in the parents who are currently separated. The patient interrupted school in 5th grade with the onset of illness.

On examination, he was a young man with gross truncal obesity, moonface, fish mouth and thin limbs. He was not anemic had no finger clubbing and no peripheral lymphadenopathy. Multiple striae were noted all over the body. He had a childs voice. He had a pulse of 76 per minute, regular and normal volume with a blood pressure of 120/80mm Hg. Heart sounds were normal. No thyroid enlargement noted. The respiratory system was normal.

He had gross obesity with protruberant abdomen, no organomegally and no palpable masses. He had normal pubic hair distribution with infantile external genitalia. He had normal muscle power. Visual fields and Fundoscopy were normal.

The clinical diagnosis of Cushing's Syndrome with a differential of Nephrosis and Adrenogenital syndrome were entertained :-

- INVESTIGATIONS:

1. Urine:
   Culture & Sensitivity: Sterile
   Trace Protein
   24 hr. protein: Not significant
   Sugar: Nil.
2. Fasting Blood Sugar: 2.9 mmol/l.
3. GTT: Normal curve.
4. Cortisol levels: 8 am 710nmol/l (9 am 145.610)
   11 am 704 nmol/l (12mn <170)
5. ACTH < 5 ug/l
6. Electrolytes: K^+ 4.4. mmol/l; Na^+ 144 mmol/l.
7. Serum creatinine: 100 nmol/l
8. Skull X-ray: Normal sella turcica,
   no other significant findings.
9. I.V.U. Noted generalized osteoporosis
   kidney & collecting system - normal

rather high positions both kidneys, though the right was higher than the left!

10. Ultrasound - No abnormality was detected in the region.

DIAGNOSIS: Cushing's Syndrome.

The patient was then worked up for Exploration of the Adrenals. The main problem preoperatively was to get adequate Replacement therapy. An anterior, transabdominal approach was chosen to allow inspection of both adrenals.Surgery was performed on 1/12/89 through a high transverse abdominal incision- At laparotomy both adrenals were noted to be characterized by multiple pigmented nodules on the cortex. The adrenals were not particularly enlarged- A right total adrenalectomy and a left subtotal adrenalectomy was carried out- In the rest of the abdomen nothing abnormal was detected. The post-operative period was uneventful and the patient was up and about on the tenth post operative day.

DISCUSSION

I. Etiology of Cushing's Syndrome.

Cushings Syndrome with primary pathology in the pituitary or adrenals is a relatively rare condition, found approximately once in 1000 autopsies, occurring far more frequently in females than in males in a ratio of about 4:1. (4). Functional adrenal adenomas occur in the male - The peak incidence among males and females is in the third and fourth decades, but the disease has been known to occur in persons younger than one year and older than 70 years.

Since the isolation of ACTH and cortisol, much has been learned of their physiology and of the effects of their administration to man. It was soon noted that if they were given in excess for long periods all features of Cushing's Syndrome might develop. It is now clear that the naturally occurring syndrome is the result of an excessive endogeneous production of cortisol and possibly to a smaller extent, of other adrenal steroids. Theoretically this might arise from lesions in the hypothalamus, which stimulate the pituitary to secrete an excess of ACTH, from, disorders of the pituitary itself, from tumor or functional disturbances of the adrenal cortex or tumors of ectopic adrenal tissue particularly in the ovary.

While Cushing's attributed the syndrome to pituitary basophilism, it is more commonly associated with either

bilateral hyperplasia of the adrenal cortex without any clinically obvious pituitary tumor or with adrenocortical tumors. It may be due to a malignant tumor of non-endocrine tissue, notably bronchus, thymus, pancreas and in recent years features of "mixed" Cushing's Syndrome have been observed in the malignant form of Conn's Syndrome.

When a functioning adrenocortical tumor is present, the clinical picture can be attributed to over production of steroid hormones by the tumor. When the syndrome is caused by an anterior pituitary tumor the clinical features are attributed to the liberation of trophic hormnone (ACTH) which in turn lead to overproduction of steroid hormones by the adrenal gland.

Most of the pituitary tumors causing Cushing's Syndrome have been basophil adenomas and since evidence points to this type of cell as the source of ACTH an explanation of Cushing's Syndrome due to basophilism is reasonable. However, the syndrome may result from a chromophobe adenoma and such tumors have developed in Cushing's patients following bilateral adrenalectomy when high concentration of ACTH is found in the blood.

Although Cushing's Syndrome can result from pituitary tumors it is worth remembering that such tumors may exist without any endocrine effects. Post-mortem surveys show

that "non-functioning basophil adenomas occur in about 3 to 4 percent of pituitaries[5].

Most patients with Cushing's Syndrome have bilateral adrenal hyperplasia and there is still some controversy whether this lesion in an intrinsic adrenal defect or the result of ACTH stimulation in the absence of a clinically demonstrable pituitary tumor. Recent observations favour the view that this form of Cushing's Syndrome is a defect in the corticotrophin-secreting mechanism. The development of pituitary adenoma in Cushing's patients following bilateral adrenalectomy could mean that a latent pituitary tumor clinically unrecognisable is present. The cause of the adrenal hyperplasia has been the subject of much discussion. While the logical explanation is an increase in the blood ACTH, early reports failed to demonstrate this increase, due mainly to the unsatisfactory technical method used to assay the hormone.

In bilateral adrenal hyperplasia causing Cushing's Syndrome unassociated with a pituitary tumor, a defect or disorder seems to exist in the hypophyseal - pituitary-ACTH secreting mechanism whereby greater than normal amounts of ACTH are produced and the feedback mechanism is unresponsive to the rise in blood cortisol. Little evidence exists at present on the nature of the hypothalamic disorder or its exact location. A number of

years ago investigators indicated that atrophy of the paraventricular nuclei occurred in Cushing's Syndrome[6]. While this was thought to be etiologically significant other workers from animal experiments, thought these atrophic changes in Cushing's Syndrome were due to the high level of circulating cortisol.

II. PATHOLOGY

Cushing's Syndrome may result from a pituitary tumor, bilateral adrenal hyperplasia with or without a clinically obvious pituitary tumor, adrenocortical tumors or may be associated with malignancy of non-endocrine tissues. The incidence of adrenal lesions vary in different series. While it is generally accepted that 75% of patients have hyperplastic glands and 25% either a single or malignant tumor, the cases reported by Neville and Symington (1967) indicate that 85% are due to hyperplastic adrenals and only 15% due to tumor. The reason for this difference is the recognition of "adenomatous" or nodular hyperplasia which accounts for 15.1% hyperplastic adrenal.

1. Pituitary tumor

The pituitary tumors are mostly basophil adenomas though chromophobe tumors have been recorded in patients particularly following bilateral adrenalectomy. Eosinophil adenomas have been noted especially when the syndrome is associated with acromegally:-

2. Bilateral adrenal hyperplasia.

This may take several forms:-

(a) Diffuse hyperplasia accounts for 72% of adult patients with bilateral hyperplasia(8). At operation the adrenal is yellow and the edges are full and round. On section the cortex is wider than normal. On microscopic examination there is a prominent compact cell layer which occupies the inner third of the cortex and a clear outer layer of the zona fasciculata.

(b) Primary nodular adrenocortical hyperplasia (NAH) accounts for about 10% of cases of bilateral adrenocortical hyperplasia[3]. It is characterised by multiple pigmented nodules in both adrenal cortices, ranging from a millimetre to several centimetres in diameter. These nodules may secrete cortisone and/or aldosterone in variable amounts causing either Cushing's or Conns Syndrome. ACTH production is suppressed by cortisone production in these nodules. NAH is after associated other condition such as myxomas, spotty pigmentation, and endocrine overactivity. Bilateral adrenalectomy cures patients with cushing's Syndrome in patients with NAH. The Histopathological and macroscopic description of NAH befits the index case presented here.

3. Adrenocortical tumor

While adrenocortical tumors are reported as responsible for Cushing's syndrome in 25% of patients and account for most recorded examples of this syndrome in childhood, they were found in only 15% of the series reported by Neville & Symington (1967). This is due to the recognition of nodular hyperplasia which in the past has masqueraded as on adenoma:-

(a) Adrenocortical adenomas:

These may secrete cortisone, aldosterone, sex hormones or non-functional precursors of these hormones- encountered. The cortisone-producing tumor account for 10% of cases of Cushing's Syndrome, and the aldosterone-producing adenoma for 90% of cases of primary aldosteronism. The treatment is usually unilateral adrenalectomy.

(b) Adrenocortical tumors:

This is rare (1 per 1700 million population) aggressive malignancy which is functioning in 40% of cases. The functioning tumors account for about 10% of cases of Cushing's syndrome. Most tumors reach a large size before diagnosis with metastases therefore only some 40% of patients are candidates for surgical resection - by unilateral adrenalectomy with or

without nephrectomy and wide excision of the tumor bed. Adjuvant Radiotherapy to the tumor bed and chemotherapy with the adrenal poison Ortho, para-1, 1-di-chloroethane (0,p'DDD) have been tried with some success. Recently streptozocin has been used in conjunction with 0, p' DDD with some success.

(c) Pheochromocytomas, Ganglioneuroma and neuroblastomas occur in the adrenal gland but these do not lead to Cushing's Syndrome.

III. CLINICAL PRESENTATION

1. SYMPTOMS AND SIGNS DUE TO EXCESS CORTISONE

Centripetal fat distribution without necessarily any marked weight gain is common. Redistribution also leads to fat pads in the supraclavicular regions, on the back (buffalo hump) and in the cheeks (moon face and fish mouth) but characteristically spares the extremities which appear unduly thin when contrasted with the pendulous abdomen. The skin is of fine texture, almost paper-like and the face is red because of transparency of the subcutaneous vasculature and polycythemia. Purple striae appear over the abdomen thighs and arms. Poorly healing wounds often infected are present. Bruisability is easy and numerous echymoses develop. As wasting of muscles progresses, marked weakness occurs. Backache is common and severe. Frequent compression fractures of the vertebrae, rib

fractures and dorsal kyphosis ensures because of marked osteoporosis. Arteriosclerosis develops rapidly leading first to systolic hypertension and later diastolic hypertension. Latent diabetes mellitus occur in 80% of the cases but only in 20% is there overt diabetes mellitus. Great liability of mood often occurs ranging from depression to mania, and psychoses are not infrequent.

2. SYMPTOMS & SIGNS DUE TO EXCESS 17-KETOSTEROI(ANDEROGENS)

Muscle size increases markedly. Hirsutism vary from patient to patient. Acne is of variable amount. Baldness and lateral recession of the hairline occurs in both females and males. Impotence or amenorrhoea occurs the latter often preceded by a period of metrorrhagia. Clitoral enlargement can be attributed to excess androgens, however all the other signs and symptoms are also found when excessive amounts of cortisol are given.

3. SYMPTOMS DUE TO EXCESSIVE ALDOSTERONE

Only hypertension and hypokalemia with diabetes insipidus like syndrome would suggest an excess. Hypokalemic alkalosis is found in about 15%. If all cases may be attributed to an excess cortisol, curticosterone and perhaps deoxycorticosterone since an elevation of aldosterone is very rarely found.

CLINICAL COURSE

The onset of Cushing's Syndrome is usually insidious and the relatives of the patient may simply attribute the changes observed to increasing obesity. The disease has transitory remissions and exacerbations and these are usually associated with increased psychic stress. As the disease progress the patient becomes weaker and weaker and finally bedridden because of loss of muscle strength and multiple vertebral fractures. If the disease is untreated, death ensues within a five year period from general debility, vascular accidents, or, rarely diabetic coma.

IV. DIAGNOSIS & INVESTIGATIONS

The diagnosis of Cushing's Syndrome is made primarily on clinical grounds and great reliance should be placed on a careful appraisal of the physical signs. Laboratory tests are essential to answer the following questions: Is the adrenal cortex overactive? If so, is this due to adrenocortical hyperplasia, to an adenoma or to an adrenocarcinoma? If due to hyperplasia what is the source of the excess ACTH.

LABORATORY FINDINGS SUGGESTIVE OF CUSHING'S SYNDROME

* A low relative lymphocyte count - less than 15% of the total leukocyte count.
* Neotrophilia, leukocyte count from 10,000 to 20,000 per cubic mm.
* Polycythemia with packed cell volumes of 50% or more
* Elevated Fasting blood sugar.
* Abnormal glucose tolerance test
* Hypokalemic Hypochloremic alkalosis (K^+ 2.8 Cl^- 95) normal values being K^+ 4.5, Cl^- 105 mmul/l.

LABORATORY FINDINGS "DIAGNOSTIC" OF CUSHING'S SYNDROME

The increased secretion of cortisol causes characteristically a raised level of cortisol in blood and an excess of 17 -oxogenic steroids and 17-hydroxysteroids in urine. In patients with hyperfunctioning or hyperplastic glands the increase is usually moderate, the response to ACTH is exaggerated and pituitary suppression is effective unless very large doses of dexamethasone are used. In those with adrenal carcinomas the increases are great but there is no response to ACTH no fall after suppression, even with large doses. An immunoassay for ACTH and cortisol is now available in some centres and accurate diurnal charting can now be done. These is usually total absence of the 50% diurnal variation between early morning and late afternoon usually seen. The index

case presented with cortisol level 710 nmol/l at 8 am and 704 nmol/l at 11 pm showing the values.

LOCALIZATION OF ADRENAL TUMORS PRODUCING CUSHING'S SYNDROME.

Tumograms may show depression of the kidney shadow on the side of tumor. This is a helpful sign by no means always present. Little is gained from Intravenous urogram. With CT scanning more and more tumors can be accurately localised. Retroperitoneal pneumography is no longer fashionable.

X-ray studies of the sella turcica should be made in all cases of Cushing's Syndrome, particularly those with pigmentation. The pituitary fossa may show enlargement in 10% of cases however this may sometimes be attributed to osteoporosis. Osteoporosis of the vertebrae gives the typical picture of codfish vertebrae formed as the vertebral bodies are compressed by the nucleus pulposus; often the skull is thinned too.

V. DIFFERENTIAL DIAGNOSIS

One occasionally finds a Schizoid individual with marked mania revealing mixed symptoms of Cushing's Syndrome. Simple obesity should also be differentiated from Cushing's Syndrome on clinical as well as laboratory grounds. Essential Hypertension does not have many features of Cushing's Syndrome as a rule. Diabetes mellitus is usually

associated with normal steroid excretion. Post-menopausal and senile osteporosis only rarely involve the skull, whereas Cushing's Syndrome often does. The sequel of Viral Hepatitis the often fatal cholangiolytic hepatitis in women or alcoholic cirrhosis with continued alcohol intake may lead to development of Cushing's Syndrome as a result of inadequate inactivation of corticosteroids by the liver.

VII. TREATMENT

The poor prognosis in untreated Cushing's Syndrome renders early diagnosis and treatment essential. The object of treatment is to reduce or to abolish the production of cortisol by the adrenals. In the rare cases in which a pituitary tumor is demonstrable it should be removed by formal hypophysectomy. If an adrenal tumor is present it must be removed. If the adrenals are hyperplastic or overactive an attack should be made either or the pituitary by surgery or radiotherapy or on the adrenals themselves. Surgery remains the treatment of choice.

ADRENALECTOMY

If permanent remission is to be achieved in a high proportion of cases at least 90% of the adrenal tissue must be removed. General agreement has not been reached as to whether subtotal or total adrenalectomy is the better procedure. Total adrenalectomy has the advantage of a permanent cure and guarantee against recurrence. On the

other hand, it necessitates lifelong replacement therapy. Subtotal resection attempts to overcome such difficulties by leaving a small fragment of adrenal tissue and if successful, enables the patient to regard himself as cured in a way that would not be possible if he had to take tablets for the rest of his life. If the adrenal remnant prove inadequate Replacement therapy is then unavoidable. Furthermore the adrenal tissue may regenerate and lead to recurrence of Cushing's Syndrome. However provided the whole of one gland and 95% of the other is removed, recurrence is not likely. It seems wise therefore to remove the whole of one gland, since if there is recurrence, the side to be re-explored is known.

PREOPERATIVE PREPARATION

The advent of modern imaging techniques has simplified greatly the work of the adrenal surgeon, where these facilities are available. Explorative operations are seldomly necessary. The knowledge of the site and size of the lesion makes it possible for operative technique to be adopted to the individual patient.

Generally a broad spectrum cephalosporin antibiotic is given, one dose preoperatively and continuing for 5 days post operatively. Patients are put on subcutaneous heparin 5000 units twice daily post-operatively until fully ambulant. Where necessary preoperative Potassium chloride

4-12gm daily by mouth is given. No preoperative administration of cortisol is necessary unless one is forced to give 100 mls. of cortisol acetate instramuscularly 12 and 2 hours before surgery because of an unreliable nursing situation. Endocrine and Anaesthesia Medical colleagues are usually consulted to give advise on optimum timing of surgery.

SURGICAL APPROACH

Three general surgical approaches are available: Flank (posterior) incision with retroperitoneal approached on each side, Transthoracic (lateral) approach through the diaphragm below the pleura; and a high trans-abdominal (Anterior) approach.

ANTERIOR APPROACH

This is a transabdominal approach, it affords the greatest opportunity for thorough exploration and has few complication in expert hands. It does allow inspection of both glands, as well as the para-aortic region and viscera, and is usually ideal for patients for pheochromocytomas and smaller adrenal tumors. The patient lies in supine position on the table and abdomen is opened via a single reverse Kochers, roof top or transverse abdominal incision. A full laparotomy is performed. Access to the left adrenal is gained by mobilisation of the spleen and tail of pancreas reflecting them out of the wound into the anterior

abdominal wall- this reveals the kidney and adrenal in a bed of perinephric fat. The right adrenal is exposed by mobilisation downwards of the hepatic flexure of the transverse colon, exposing the vena cava. The liver is retracted superiorly and the duodenum inferiorly to the left. A short adrenal vein, or sometimes several enters the vena cava directly just above the kidney. this was the approach used in the index case.

POST OPERATIVE MANAGEMENT

All patients should be maintained post operatively on an intravenous infusion of crystalloid, adjusted against daily serum electrolyte analysis until gut function is sufficient to sustain fluid and electrolyte balance. Antibiotic cover, Cortisone Replacement therapy and heparin should continue. For patients who had undergone ribbed exploration CXR should be performed. If significant pneumothorax is present an under water seal drainage is performed. Because post operative basal atelectasis is common, chest physiotherapy in mandatory.

COMPLICATIONS

1. Following total bilateral adrenalectomy the development of a corticotropin-secreting chromophobe adenoma may be seen in from 10 to 15% of the cases. As the tumor grows it may interface with vision. This possibility calls for careful observation for melanin pigmentation which

usually precedes the development of the tumor. Baseline lateral and posteroanterior view of the skull to estimate the size of the pituitary fossa should be done every six months. Once such a tumor has been found it can be treated with Radiotherapy 4000 rads, or a trans sphenoidal hypophysectomy.

2. Acute Adrenal crisis is a serious emergency which takes the form of acute peripheral circulatory failure. Collapse, hypertension and tachycardia may develop with dramatic suddenness after 24 hours after operation. It may follow operation on the first side. even though the other adrenal is present and hyperplastic. It should be treated with immediate infusion of 100mg of cortisol hemisuccinate. This is usually effective in 15-30 minutes. Nor-Adrenaline (4.mg in 500mls of saline) may be added to the drip at whatever speed is required to maintain the blood pressure at or above 100mmHg.
3. Acute salt deficiency may also be a manifestation of acute adrenal insufficiency, but less common than an adrenal crisis. Its danger lies in its being mistaken for pure cortisone deficiency. The symptoms of weakness apathy, nausea and vomiting are present. Daily serum electrolyte analysis is necessary. If serious depletion is present immediate correction by sodium chloride intravenously is necessary.

4. Subacute adrenal insufficiency is also known as the cortisone withdrawal syndrome and develops if the dose of cortisone is reduced too rapidly. The earliest symptoms are anorexia and nausea, first experienced in the morning and aggravated by exercise. Later they may become persistent and vomiting, weakness, lethargy, abdominal discomfort, low grade fever and tachycardia follow. A characteristic sign is a scaly desquamation of the skin of the face or even the whole body. The condition, including the skin lesion, respond rapidly to an increase in the dose of cortisone. If treatment is delayed hypotension, anuria, mental changes and death may follow.
5. Other postoperative complication maybe trouble some. These include wound infection or dehiscence, pulmonary collapse and infection, temporary psychosis, persistent tachycardia and, later rheumatic-like pains in the muscles and joints.

REMISSION OF THE DISEASE

The first sign of improvement is the scaly desquamation of the skin of the face which usually starts within two or three weeks of operation. This skin change is an index of adequate and successful surgery and it continues until remission is complete.

Within a few weeks the facial and bodily appearances start to improve and bodily configuration are regained. Cutaneous striae fade and leave pale scars. Facial hair usually diminishes, but may not be lost completely. The menstrual periods return normal, sexual function is restored and pregnancy with normal childbirth may follow. Blood pressure falls significantly in nearly all, but return to normal in only a few. All metabolic disorders are corrected. Recalcification of bone occurs slowly, but fractures heal and deformities become stabilized in time.

CONCLUSION

Cushing's Syndrome may not be a common condition, however, whenever it occurs it throws a big challenge to both the Surgeon, the Physician(endocrine) and Anaesthesiologist.

It is the aim of this paper to show that despite these challenges, adrenal surgery is possible even in developing societies like ours.. The index patient was discharged in good condition on cortisone tablets. His truncal obesity is clearing and he is loosing weight. He continues to attend out patient clinic for follow-up.

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7. A REVIEW OF 26 CASES OF CRYPTORCHIDISM

ABSTRACT:

A review of 26 patients with cryptorchidism treated in the department of Surgery from June 1988 to June 1990 is presented. The patients ages ranged from 3 to 69 years. 19 patients accounting for 73.1% of the case had left sided unilateral cryptorchidism while 6 cases (23.1%) had right sided cryptorchidism. Only one case (3.9%) had bilateral maldescent. An associated hernia was found in six patients accounting for 23.1% and in two of the six cases the hernia was the presenting problem. One case of true hermaphroditism was seen. One patient presented with a seminoma. 59.3% of the patients underwent orchiopexy while 40.7% had orchidectomy. Late presentation of patients reflect lack of public awareness of cryptorchidism as a medical problem and health education to traditional birth attendants, midwives, obstetricians and parents is advocated so that patients can be seen at an earlier age.

INTRODUCTION.

The testis develops on the posterior abdominal wall overlying the mesonephros, its lower pole is attached to a mesodermal mass called the gubernaculum. The mesonephros disappears but its ducts persist and some of the tubules gain attachment to the developing testis. The mesonephric tubules and proximal mesonephric duct develop into the

epididymis and the distal duct into the vas deferens. At about the seventh month the testis lies at the deep inguinal ring and should be in the scrotum at birth. Testicular descent is most probably achieved by relative lack of growth of the gubernaculum compared with the rest of the body.

The descent of the testis can be arrested any where in its course or the organ may take an aberrant path once it has passed through the external inguinal ring. The types of descent may be classified as follows:

1. Normal
2. Migratory (retractile)
   - low
   - High-
3. Undescended
   - External inguinal
   - Inguinal
   - Abdominal
4. Ectopic
   - Superficial Inguinal
   - perineal
   - pubic
   - femoral

The terms **"Cryptorchidism" and "Maldescent"** imply failure of descent into the scrotum. The condition may be unilateral or bilateral.

The normal testis enters the scrotum just before birth. The retractile testis completes its descent later and often at puberty. It should therefore be regarded as a variant of the normal.

Contraction of the cremaster muscle holds the testis at the top of the scrotum or in a superficial inguinal pouch which lies above the pubic tubercle lateral to the external ring and between the fascia of the external oblique and deeper layer of superficial fascia. In the low retractile type the testis can be manipulated to the bottom of the scrotum. In high retractile type, shortness of the spermatic cord as well as retraction holds the testis in the superficial inguinal pouch and manipulation will bring it down to the upper part of the scrotum but no further.

In the external inguinal position the testis is accompanied with a hernia sac along which it steps in and out of the inguinal canal either spontaneously or with manipulation. The testis is an elongated soft structure and is impalpable in the canal because it is covered by the firm apponeurosis of the external oblique muscle. If a tests is palpable therefore in the region of the canal it is almost certainly in the inguinal pouch which is superficial to the

aponeurosis. An inguinal undescended testis which is less common lies within the inguinal canal. Like the external inguinal type it may be associated with a hernia sac along which it moves freely. It does not however emerge from the external ring and is never palpable. The abdominal type which is more rare lies just inside the internal ring, however it is sometimes found on the posterior abdominal wall. It is rarely associated with a hernia sac and can be distinguished from the inguinal type only at operation.

Ectopic testes leave the external ring normally but then instead of descending into the scrotum, becomes deflected into abnormal positions and do not therefore complete their descent spontaneously. The commonest type is the superficial inguinal. The testis lies in the superficial inguinal pouch but unlike the retractile type it cannot be coaxed into the upper end of the scrotum. It may sometimes be manipulated back into inguinal canal. The other types ectopia include perineal, pubic and femoral.

CASE REPORT

N.J. IP. 580983 was a 31 year old male patient first seen in the Surgical outpatient clinic for an empty left scrotal sac since childhood. His parents had noted this problem since his birth, however they did not seek medical attention. He grew up normally to reach puberty with normal sexual function. He was married with one child.

On examination he had a normal male build, a deep male voice and a male hair distribution. He had normal cardiovascular, respiratory and abdominal findings. He had a normal sized penis with a right scrotal testis of normal size and consistency. The left scrotum was empty and there was a palpable "testis" in the left inguinal region inpossible to be manipulated into the scrotum. A diagnosis of left cryptorchidism was reached and the patient was planned for left groin exploration.

A left inguinal skin incision was made. Dissection was done down to the aponeurosis of the external oblique. The external inguinal ring was identified and the inguinal canal was opened.

An atrophic left testis was found in the inguinal canal and was associated with a small hernia sac continuous with the tunica vaginalis. Mobilisation could not bring the testis further down as the spermatic cord was short. Orchidectomy was therefore decided upon and carried out. The vascular pedicle was ligated twice with number one chromic catgut and divided. The hernial sac was transfixed at its neck with catgut number one and herniotomy was done. Reconstruction of the anterior inguinal wall was done with nylon number one. The skin was closed with nylon number three zero. The patient was discharged on the first post-

operative day. The skin incision stitches were removed on the seventh post operative day.

The histopathology report noted thickening of the tubules, basement membrane and decreased spermiogenesis associated with marked degeneration of spermatogonia and sertoli cells. The stroma was fibrotic and hyperplasia of the Leydig cells was noted. No malignant transformation was observed.

METHODOLOGY

All patients treated surgically for cryptorchidism in the Department of Surgery Muhimbili Medical Centre from June 1988 to June 1990 were retrospectively reviewed. The only criterion for inclusion in the study was surgical treatment of cryptorchid patient. Case notes were retrieved from the medical records and information on age when the patient sought treatment was recorded. The side affected and operative as well as histopathologic reports where applicable were traced. In this manner 26 patients treated for cryptorchidism were reviewed.

RESULTS

The patients age ranged from 3-69 years with a mean of 26.7 years. The majority of the cases 19/26 had left cryptorchidism accounting for 73.1% of the cases. Right sided cryptorchidism occurred in 6 cases or 23.1% of the patients. Only one case 3.9% had bilateral cryptorchidism.

In six of the patient accounting for 23.1% an associated hernia was found. In two of the six cases the presentation was primarily due to the hernia in that in one case a strangulated left inguinal hernia was noted while in the other an obstructed right inguinal hernia was the presenting feature. In these two patients orchidectomy was done.

One case of true hermaphroditism was seen in which there was a left abdominal ovotestis and right scrotal testis this patient had in addition a chordee. Two other patients had chordee one in addition having a hypospadias. One patient presented with a sigmoid volvulus and at laparotomy an incidental right intra-abdominal testis was found. A single case of seminoma was seen. This patient presented himself at 47 years of age with a short history of a rapidly enlarging right groin testis which he had been having since birth. He had no clinical evidence of metastases and orchidectomy was carried out.The patient who is a Member of Parliament was still alive and well three years after surgery. All in all 59.3% of the patients had orchiopexy while 40.7% underwent orchiectomy.

DISCUSSION

The mean age of 26.7 years at presentation is definately late reporting. It may suggest lack of community awareness that cryptorchidism is a serious problem. In a paediatric

survey reported recently more than 94% of the cases reported after the age of 1.5 years in Muhimbili and KCMC(1).

At birth the incidence of undescended testis in all males is about 10% for full term babies. The incidence is about 23% for premature babies. After the first year of life the figure drops spontaneously to about 1.7 to 3 percent (2,3). During prepuberty period this number is further reduced by the late descending testes. Most common of these is the retractile type which migrate periodically into the scrotum. Post puberally about 0.3 to o.4% of males have either unilatteral or bilateral maldescent of the testes(3). Unilateral maldescent is four to five times as frequent as bilateral maldescent. This is evident even in this series where only one case among the 26 had bilateral cryptorchidism. In the paediatric series cited above(1), 43% of the patients had bilateral cryptorchidism. Unilateral maldescent is twice as common on the right side as on the left side(2,3), however in this series 73% were left sided maldescent.

. The rate of inguinal hernia has been reported to range from 57 to 93 percent but usually such hernias are of anatomic rather than clinical importance(4). In this series inguinal hernias were noted in 23% of the patients and in two cases the hernias were the presenting problem.

One should beware of Turners syndrome, Klinefelters syndrome, Sertoli-cell-only syndrome, male pseudohermaphroditism, true hermaphroditism and hypogonadotropic eunuchoidism. In this series one case of true hermaphroditism was encountered.

The abnormally placed testis is particularly liable to suffer trauma and a hydrocele may form around it. Sterility is however the commonest complication of cryptorchidism and is probably caused by the relative warm environment of the maldescended testis. Retarded development has always been observed after the age of six. Other studies have mentioned an even earlier age of testicular degeneration. In some, cryptorchidism is the result of a failure in development (germinal aplasia/dysplasia) which also prevents the testis from developing normally even if it was in the scrotum. The leydig cells are not as dependant on temperature as the seminifereous tubules and they usually function normally unless the testis is intra-abdominal.

Malignancy especially seminoma is common, however the controversial point is its relative importance. Carroll(5). suggest that the incidence is over rated. It has been noted that 12% of all the cases of primary testicular carcinoma occur in cryptorchid man who make up less than 0.4% of the adult male population. Therefore he

concludes that maldescended testis is likely to become malignant than a scrotal testis. It should be pointed out that placing the testis into the scrotum does not guarantee against later development of cancer, for it seems that the now-scrotal-once cryptorchid testis still has an increased chance of becoming malignant[6,7]. In this Muhimbili series of 26 patients one case turned out to be a seminoma. With the low incidence (0.002% of all males) of testicular carcinoma, prophylactic orchidectomy in cryptochidism does not appear to be justified[7].

There is little agreement about the treatment of cryptorchidism, and discussions of the subject are often characterized by dogmatism. Surgical methods usually succeed in placing the testes in the scrotum but they do not necessarily result in fertile gonads. Indeed by damaging the blood supply they may cause the leydig cells to degenerate so that a patient who would otherwise have been sterile but normally developed, becomes both sterile and eunuchoid.

It is now advised that surgical correction of maldescended testis be carried out at two years. There is now ample histological evidence that the organ deteriorates during the third year of life. Interestingly, the normally descended testis may become damaged by the continuing

presence of a maldescended testis beyond this age - the mechanism of which is unknown(8).

Operation at the age of two result in histological evidence of improved spermatogenesis in the maldescended organ and prevention of deterioration in the normally developed and descended organ. If an associated hernia is present, operation is indicated in infants or neonates regardless of age.

Human Chorionic gonadotrophin in a dose of 500 units three times a week for up to eight weeks have been given to boys with bilateral impalpable testes. One or both testes may become palpable and surgery is facilated by an increase in the size of the organ. In some cases the need for surgery is obviated.

The orchiopexy operation demands patience and great delicacy in the handling of the tissues. It aims at the following:-

1. To secure sufficient length of the spermatic cord to allow the testis to reach the scrotum.
2. To place and retain the testis in the scrotum.
3. To repair any coexisting hernia
4. To improve fertility and
5. Possibly prevent malignancy

In this series as mentioned earlier 59.3% of the patients underwent orchiopexy while the rest 40.7% had orchiectomy.

RECOMMENDATIONS

The late presentation of patients suggest lack of community awareness of cryptorchidism as a medical problem. The Traditional birth attendants, the Midwives and Obstericians should be the target of health education so that patient with cryptorchidism can be presented at an earlier age. Follow up studies to determine fertility after orchidopexy would be very enlightening.

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8. A CASE OF TRUE HERMAPHRODITISM

ABSTRACT

A case of true hermaphroditism treated in the surgical department of Muhimbili Medical Centre is reviewed. About one in 2000 newborn babies has an ambiguity of the external genitalia and the extent of the ambiguity gives no clue of the underlying etiology. Despite the rapid development of modern biochemical, hormonal and karyotype studies, the final diagnosis of this condition often has to await surgical exploration and histological reports.

INTRODUCTION

About one in 2000 new born babies have an ambiguity of the external genitalia. The extent of the ambiguity gives no clue of the underlying etiology (1). Though these abnormalities should arouse the suspicion of the possibility of a true hermaphroditism, that is an individual with ovarian and testicular tissue, this is a rarity there are probably less than one hundred such cases on record (2).

Eshleman and Carneiro reported twelve cases of intersex abnormalities at K.C.M.C during 1978-1986 period. Of the 12, four were true hermaphrodites, six Adrenogenital syndrome, one male pseudo hermaphrodite and one Klein felters syndrome (3).

There is no single criterion of an individual sex. In the normal female the chromosomal sex is 46xx, the gonadal sex is ovarian and the genital sex is characterised by presence of a vulva, vagina, uterus, and fallopian tubes. In the normal male the chromosomal sex is 46xy, the gonadal sex is testicular and the genital sex is characterised by the presence of the phallus, the scrotum epidydimis, vas deference, seminal vesicle and the prostate.

Biologically there are two important inter-relationship: the first consist of the connection between the sex chromosome constitution of the zygote and the nature of the gonad; and second, the influence of the gonad on the development of the other genital apparatus. Following fertilisation, primitive gonads differentiate into testes. In the normal male there is one X and one Y chromosome, however even if these were more than one for X, the presence of the Y chromosome results in testicular development. If there is no Y chromosome but two X chromosomes the primitive gonads differentiate into ovaries. A single X chromosome in the karyotype is capable of influencing gonadal analogues to start to differentiate as ovaries but these soon atrophy and become streaks of fibrous tissues.

Cells that have only a single Y chromosome are not viable. Thus sex chromosomes are important in so far as they

determine gonadal sex but once this has been achieved they have no further function. Normally developed fetal testicular tissue produces two critical substances- androgens and mullerian inhibitor. Androgens bring about the development of the Wolffian structures into epidydimides, vasa deferentia, seminal vesicles and so on and they also mesculinise the indifferent external genitalia. The mullerian inhibitor prevents the growth of the primitive mullerian apparatus which if not inhibited develop into a vagina, uterus and fallopian tubes.

The normal development of the male genitalia is therefore brought about by a normally functioning testis. If during the first twelve weeks of embryonic life there are no testes or the testes do not function properly or the metabolic products are not utilized the genital organs develop as the female phenotype. The presence or absence of the ovaries is however immaterial to the development of the genital organs. A female can be considered as a non-masculised individual.

The formation and utilisation of the androgens are critical to the development of the male external and internal genitalia. Testosterone is formed from cholesterol through two basic path ways one via progesterone. Both of these metabolic chains require enzyme action in order to produce the required androgens especially testosterone for complete

masculinisation. Testosterone is then converted to dihydrotesterone by the enzyme 5α-reductase in the target cells of responsive tissues [4]. The receptor for testosterone is a protein located in the cytosol, that is the extra-nuclear fluid compartment of the cell. The combination of dihydrotestosterone and the receptor is stable and this complex is transferred to the cell nucleus which then allows gene activation with resultant synthesis of RNA[5,6]. Testosterone utilisation can therefore be summarised as to occur in three steps; formation; conversion to active dihydrotestosterone and finally cytosol binding to receptor and nuclear activation.

SUMMARY OF THE PATIENT K.S. REGISTRATION NUMBER 508605

K.S. was a 22 year old, referred from Kigoma and admitted first on 29/8/87 on account of childhood voice not breaking, development of feminine breast and abnormal external phallus and scrotum had been smaller than that of his agemates. At school he had to hide when passing water to conceal his abnormality. To make matters worse he developed feminine breasts which were subject to "teasing" by fellow pupils at school. His voice remained infantile and he did not develop facial hair. He had no history of cyclic abdominal pains or passage of blood urine.

He had history of mental illness- over talkativeness, aggressiveness and wondering about- treated by traditional

healers and necessitating admission once in a Kigoma Hospital.

He is a fifth born in a family of 9, the rest of the siblings are alive and well. He was born normally at term. There is positive history of consanguinity in the parents who are cousins. The father died of an abdominal "illness" was alcoholic and had frequent quarrels with the mother who is still alive. He was reared up as a male though he felt at times that he was not a "real" man. He attained standard seven primary school education.

On examination he was noted to be of rather short stature, feminine morphology, infantile voice though husky no facial hair and bilateral feminine breast. For external genitalia there was a small phallus 3-4 cm hidden between scrotal folds with chordee. The right testis was palpable in the scrotum and was small.The right testis was not histologically examined The left scrotal sac was empty.

No abnormality was detected in the cardiovascular respiratory and gastrointestinal systems. The clinical diagnosis was an intersex problem with gynaecomastia, left cryptorchidism and ambiguous external genitalia:-

Investigations done include:-

1. Barr bodies (buccal smear) 15% (normal range M=0 F= 20%-40%).
2. Serum electrolytes Na 137, K 3.8 mmol/l.

3. Seminal Fluid analysis revealed complete azoospermia
4. IVU- revealed normal radiologic findings.
5. Hemoglobin was 10.6 g/dl.

After these investigations the patient was thought to be a Klinefelter's Syndrome XXY who has been raised as a male.

SUMMARY OF TREATMENT CHRONOLOGICALLY

20.10.87 The patient had psychiatric breakdown with overtalkativeness, auditory hallucination and was preoccupied with his penile abnormalities. Psychotherapy and counselling were given.

27.10.87 Urological consultation was made and the patient and planned for correction of the external genitalia malformation.

23.11.87 At operation a tethered penile shaft was noted. Release of fibrous tissues was done however there was still shortening of the penis. Transection of the urethra and first stage urethroplasty was therefore done. Post operatively warm sitz baths and regular bouginage of the distal and proximal stomas were done.

23.6.88 The patient was discharged to Kigoma for review after one year for closure of the perineal stoma and left groin exploration.

10.8.89 The patient was readmitted. He had during this time a mild psychotic illness which was

controlled with Melleril. The patient had several sessions with a clinical psychologist and a psychiatric for psychosexual orientation. The psychotic symptoms were controlled and surgery was planned.

20.10.89 Closure of the perineal stoma and exploration of the left groin was done:-

(a) The distal and proximal urethral stomas were dilated up to number 8 bougie. A foley's size 18 urethral catheter was passed into the bladder. An appropriate skin island was developed fresh edges created and tubulation effected. Closure of the stoma was done in three layers with dexon 4/0 and monofilament nylon 4/0 to skin.

(b) At left groin exploration a well developed gubernaculum testis and underdeveloped testis and seminiferous tubules were noted in the groin. A well developed intra abdominal left "ovary" was found. No uterus or right intra abdominal gonad was found. The left "Ovary" and the rudimentary testis and seminiferous tubules were removed and the left inguinal canal was reconstructed. Ten days post-operatively the urethral catheter was removed and the surgical wound was noted to have

healed well and the patient could pass urine normally.

20.12.89 The patient underwent bilateral subcutaneous mastectomy. Histological examination revealed proliferation of fibrous tissue and few glands. Histological examination of the abdominal "testis" showed primitive and atrophic seminiferous tubules. Some of the tubes showed cystic dilatation and hyalinisation. Histological examination of the "ovary" showed fallopian tube in part and other areas showed marked cystically dilated follicles filled with hyalinised pink material. The follicles were lined by a single layer of cuboidal epithelium and were surrounded by a delicate connective tissue which was mostly hyalinised with very few stomal cells. Corpus luteum, corpus albicans, follicular cyst and premodial follicles were occasionally encountered. The final Diagnosis of true hermaphroditism was therefore reached. The patient was discharged for follow up at the referring Regional hospital.

DISCUSSION

At present several general classes of inter sex are recognized, as follows:

1. PSEUDOHERMAPHRODITISM FEMALE TYPE

In pseudohermaphroditism of the female type the individual as a genetic as well as a gonadal female but the external genitalia and some acquire male characteristics. The most common cause of the disorder is congenital adrenal hyperplasia. This condition results from an abnormality of one of the enzymes involved in cortisol formation. This in turn results in increased ACTH secretion which, in turn, leads to increased androgen production by the adrenals. Ninety percent of cases are due to deficiency of 21-hydoxylase, while the remainder are caused by lack of 11-hydroxylase or 3B dehydrogenase. Half the patients with 21-hydroxylase deficiency have severe salt loss due to inadequate mineralocorticoid secretion. Similarly patients will 11-hydroxylase deficiency may be hypertensive due to accumulation of 11-deoxycorticosterone. Androgen producing tumors in the mother is an extremely rare cause of this condition as they would usually preclude pregnancy in the first place.

MALE TYPE:

In male pseudohermaphroditism the individual is a genetic and gonadal male, but body build and external genitalia have female features. The causes of this disorder are impaired testosterone secretion and impaired utilisation by peripheral tissues.

Impaired testosterone secretion may be due to:-

(a) Partial deficiency of any of the enzymes en route to testosterone formation. Total lack of one of these enzymes leads to an XY female who presents in later life with absent puberty.

(b) A structural defect of the testis eg. hypoplasia of Leydig's cells or more severe form of dysgenesis which border on the syndrome of abnormal gonadal differentiation. In all these conditions, the testes differentiate and secrete normal quantities of antimullerian hormone. Testosterone secretion during the critical period of development is however, insufficient to virilize the external genitalia. In additions the gubernaculum function poorly and crytorchidism is common.

(c) Impaired metabolism of androgens by peripheral tissues. These cases are about 20 times as frequent as biosynthesis defects. 5α reductase deficiency= this autosomal recessive defect causes the development of male internal genital structures with ambiguous external genitalia. The wolffian structures being testosterone dependant develop normally while the external genitalia being mainly dihydrotestosterone dependant are abnormal. A male psychosexual orientation and body build develop without

G.5 CARCINOMA OF THE CERVIX STAGE Ib TREATED BY WERTHEIMS HYSTERECTOMY.

Name: M.S. Age: 30 years Reg. No: 494295

Address: Mpwapwa

Date of admission: 8/4/1987

Date of operation: 7/5/1987

Date of discharge: 24/5/1987

SUMMARY.

Mrs. M.S. 30 years old para 3 + 2 was admitted as a referral from Mpwapwa district hospital with a 12 months history of post coital bleeding. Carcinoma of the cervix clinically stage Ib was diagnosed. Examination under anaesthesia staging and biopsy was done. Histology confirmed the diagnosis. Wertheim's hysterectomy was done. The post-operative recovery was uneventful. She was discharged home two weeks later in good condition. She is being followed at the gynaecological outpatient clinic. The case is presented; treatment modality and possible complications related to it are discussed.

HISTORY.

Mrs. M.S. 30 years old para 3 + 2 was admitted at Muhimbili on 8/4/1987 as a referral from Mpwapwa district hospital where she had been attended three weeks prior to admission in Muhimbili. She was well until a year prior to admission when she started experiencing vaginal discharge. At the begining the discharge was small necessitating change of pad only once in a day. The discharge was only in certain

gynaecomastia, small phallus with little or no body hair. Androgen receptor and post-receptor defects: This is a spectrum of sex-linked conditions ranging from an apparently normal female to an infertile male. Patients with complete testicular feminisation present after puberty and have no genital ambiguity. The diagnosis may however, be suspected on the basis of bilateral inguinal hernias in a phenotypic female. A partial defect of the receptor apparatus leads to an intersex state with variable degrees of clitoromegaly and fusion of the labioscrotal folds. Virilisation may occur at puberty accompanied by gynaecomastia.

2. CHROMOSOMAL INTERSEXES

In the so called chromosomal intersexes, the nuclear chromatic and sex chromosomes are abnormal. In this group of condition, the intersex disorder results from impaired differentiation of the gonads during fetal life. Genital ambiguity occurs in patients who have testicular tissue that is dysgenetic and has not secreted sufficient concentrations of testosterone for normal male development during the critical period of embryogenesis.

(a) In ovarian dysgenesis the pattern is after XO (but XX or Xx has been reported) with the body build and genitalia of immature female and other somatic anomalies such as webbing cubitus valgus, coarctation

of the aorta etc. which can be a feature of Bonnevie-Ullrich-Turner Syndrome. The Karyotype maybe 46xy, 45X0/46xy or a more complex mosaic pattern.

(b) The XX male. Such patients often have hypospadias and undescended testes and the condition is thought to result from transposition of the gene for the HY antigen to an X chromosome. Only fifty cases have been described in world literature ‹1›. In Klinefelter's Syndrome body build, external genitalia and gonads are grossly male, but the patient usually appears enuchoid, gynaecomastia develops and the sex chromosomes are frequently abnormal with an XXY,XX, XXY/XX pattern or other variants. The pattern is a normal XY in the remaining one third.

3. TRUE HERMAPHRODITISM

True hermaphrodites have testicular tissue with seminiferous tubules and ovarian tissue with primodial follicles. These components may be found separate or combined in an ovotestis. The suspicion should be greatly heightened in any individual who gives history of abdominal pain at monthly intervals, especially if intermittent bleeding occurs either via penile urethra on rudimentary vagina. The commonest karyotype is 46XX followed by the 46XX/46Xy chimera. After removal of the testicular tissue, normal ovulation and even pregnancy may ensue.

RELATIVE FREQUENCY AND LOCATION OF TESTIS OVARY AND OVOTESTIS

Of the verified cases of true hermaphroditism whom adequate information is available about fifty percent have been characterized by presence of discrete collections of testicular and ovarian tissue. In the remainder ovotestis were present either alone or with an additional separate gonad. In those with separate ovarian and testicular tissues the ovaries generally have been located in the abdomen and the testes in the scrotum. However inguinal or scrotal ovaries and inguinal or scrotal testes have occasionally been encountered. The ovotestis when present, have been accompanied more often than not (in a ratio of about 2:1) by another discrete gonad. Four times out of five this was an ovary.

SOMATIC FEATURES OF TRUE HERMAPHRODITES

In their childhood most of the true hermaphrodites believed themselves to be males, largely because of a fair sized phallus and masculine somatic configurations. However with pubiscence and adolescence a tendency to a female body habitus with breasts and paucity of facial hair becomes evident in most of them.

In practically none of them, however could the penis be considered of average size and hypospadias, chordee and related abnormalities were frequent.

A vagina usually rudimentary in size was present in more than one half on whom definitive information on this point was given. In general the accessory sex structure associated with the gonad was appropriate i.e, tubes accompanied ovaries and an epididymis was connected to testis etc.

In the case of an ovotests, the ovarian component appears to have been dominant in this respect.

HISTOLOGICAL FEATURES OF THE GONADS

Usually in terms of gamate production, the ovaries appeared to be functioning more after than the testis. Thus the graafian follicles or evidence of recent ovulation in ovaries or ovotestes were more frequent that the incidence of mature spermatozoa in testes or ovotestes. It is however easier to detect immaturity or abnormalities of spermatozoa than of ova.

SEXUAL BEHAVIOUR

It is interesting to note that in true hermaphroditism two thirds of the individuals had been raised and considered themselves to be males. This was true even though as pointed out above that gamate maturation in the ovary was more common than in the testes. This may be a reflection of the fact that androgen secretion induces masculinisation and that testes which were not producing mature spermatozoa were none the less producing androgens. However it should

be noted that behaviour is psychologically oriented and that the individuals believed themselves to be males. This orientation should be considered when corrective surgery is undertaken [2].

Though the essential feature of true hermaphroditism is the presence of both ovarian and testicular tissue, this does not mean that the gonads of each sex are necessarily producing normal and fully developed gametocytes. Fertility in such gametes is unlikely and in addition gross anatomical abnormalities in the sex organs make reproduction impossible. Thus even though about one hundred examples of hermaphroditismus vera have been reported, pregnancy or impregnation had not occurred in any unoperated cases. However after removal of the testicular tissue, normal ovulation and even pregnancy may ensue[2].

CLINICAL INVESTIGATION, ASSESSMENT DIAGNOSIS AND TREATMENT

If the individual presents in early life diagnosis or exclusion of congenital adrenal hyperplasia is the first step; thereafter the pattern of investigation depend on chromosomal sex. In many cases however the final diagnosis depend on gonadal histology.

The only life-threatening cause of genital ambiguity is the salt-losing form of 21-hydroxylase deficiency. This diagnosis should therefore be made or refuted immediately and this is done by means of a serum 17-hydroxyprogesterone

estimation. Specific diagnosis of the deficiency enzyme depend on demonstration of an accumulating precursor hormone. Thus 21-hydroxylase deficiency is diagnosed on the basis of accumulation of 17-hydroprogesterone while 11-hydroxylase deficiency causes an increase in both this substance and 11-deoxycortisol. Measurements of these substance in blood by radioimmunoassay has taken the place of chemical tests for urinary metabolites such as pregnanetriol.

If congenital adrenal hyperplasia is not confirmed, a karyotype should be obtained in practice this is requested at the same time as the 17 hydroxyprogesterone estimation. Chromosomal analysis is carried out on cultured lymphocytes, and because a result can be obtained within three days, this method has replaced the less reliable Barr-body assessment on buccal smear.

For those with normal female karyotype once a transfer of androgens from the mother to the fetus has been excluded by a detailed history and maternal examination the differential diagnosis rests between unexplained female hermaphroditism and true hermaphrodite or XX male. Indeed, one of the latter diagnoses is certain if one or more of the gonads in palpable.

For those with a normal male karyotype either the patient is a male pseudohermaphrodite or there is some form of

abnormal gonadal differentiation (usually asymmetrical gonadal dysgenesis). Most patients in this category are male pseudohermaphrodites with impaired peripheral utilisation of testosterone.

Testosterone level especially in response to HCG 1500 units as a single dose or repeated over 3 days should be measured. An inadequate testesterone response to HCG can be caused by either a biochemical or a structural defect in the testis. As in the case with congenital adrenal hyperplasia, diagnosis of the former depends on demonstration of precursor steroid accumulating prior to the enzyme block. In addition, some of enzyme defects, such as 17α-hydroxylase deficiency, are accompanied by an inadequate cortisol response to ACTH.

If one of these biochemical defects has been excluded then a structural abnormality of the gonad may be suspected and final diagnosis is by histology. If one or no gonads are palpable then the diagnosis of asymmetrical gonadal dysgenesis is highly likely and this can be confirmed preoperatively by radiological contrast studies, which frequently demonstrate mullerian structures. However, surgery is still required for final diagnosis and removal of the gonadal streak.

A normal testosterone response to HCG strongly suggest a peripheral tissue defect in androgen metabolism. The

differentiation of 5α reductase and partial androgen receptor defect is however difficult.

In some 46xy patients with abnormal gonadal differentiation who have a reasonable amount of testicular tissue and a predominantly male phenotype there is a normal testosterone response. All such patients with asymmetrical gonadal dygenesis, and the vast majority with true hermaphroditism have one or more impalpable gonads; the correct diagnosis is therefore made when surgery is carried out to find any usually remove, the undescended gonad. If the patient is to be raised as a female then all testicular and streak tissues is removed. If the male gender is to be assigned then any streak or ovarian tissue is removed.

OBSERVATIONS AND CONCLUSION THE INDEX PATIENT

The index patient was raised as a male. His main problems were gynaecomastia, chordee and left cryptorchidism. He had a fair sized phallus. The patient had mild psychotic episodes probably related to the psychosocial stress due to his sexual ambiguity. Hormonal, biochemical and karyotype studies could not be performed for lack of facilities. As in most cases of this nature the correct diagnosis was made after surgery and histological studies.

Treatment was aimed at reinforcing and maintaining the belief that the patient was a male. Both the correction of malformation of the external genitalia and subcutaneous

mastectomy were aimed at achieving the assigned gender. In between the surgical treatment the patient had sessions with a psychologist for psychosexual orientation. A psychiatrist took care of the psychotic episodes.

The management of this patient underscores the importance of team work. The cooperation of the surgeons, clinical psychologist and psychiatrist was instrumental in the successful management of the patient.

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PART III: ORTHOPAEDIC/TRAUMA.

9. THE PATTERN OF KNEE INJURIES IN MUHMBILI MEDICAL CENTRE

ABSTRACT

A review of 21 patients with knee injuries is presented. There were 15 males (71.4%) and 6 females (28.6%). Their ages ranged from 12 to 50 years with a mean of 28 years. The leading cause of knee injuries was Road Traffic Accidents seen in 9 patients (42.9%) followed by sports accidents contributing 7 cases (33.3%). There were six patients with tibial plateau fractures (28.6%) and also six cases of distal femoral epiphyseolysis (28.6%). There were four patients with supracondylar femoral fractures (19%). There were two cases each for dislocation of the knee and ligamentous injury. One patient with proximal tibial epiphyseolysis was recorded. In general eleven patients (52.4%) were treated conservatively while 10 patients (47.6%) were treated with open reduction and internal fixation. One patient who had posterior dislocation of the knee developed gangrene of the leg necessitating an above amputation. This was the only complications seen in this series. Safer road use and curbing of sports violence is recommended to reduce incidence of knee injuries.

Keywords: Knee injuries MMC: causes, types, treatment and complications.

INTRODUCTION

No joint sustains so wide a range of injuries as does the knee. Its ligaments are both extra and intra-articular and alone amongst the major joints it contains menisci [1]. Injuries to the knee may be divided into those which affect the ligaments, the menisci, the extensor apparatus and the tibial and femoral condyles. Road traffic accidents and sports are the leading cause of injury[2].

Every fracture leads to a complex tissue injury involving bone and soft tissue . Immediately after the fracture and during the repair phase, we see local circulatory disturbances, manifestations of local inflammation, as well as pain and reflex immobilisation. These three factors, circulatory disturbances, inflammation and pain, a result of dysfunction of joints and muscles, lead to the so called "fracture disease"[3].

Fracture disease is caused by two main pathogenic factors; pain and lack of physiological challenge to the bone muscle complex by movement and changing mechanical load. In the lower limb this means lack of weight bearing and in the upper limb, lack of normal muscle work.

Fracture disease is therefore a clinical state which is manifested by chronic edema, soft tissue atrophy and patchy osteoporosis. These fibrotic processes cause muscles to develop unphysiological adhesions to bone and fascia and

therefore lead to stiffness of adjacent joints. These sequela, if fully developed, are very often not completely corrected by longterm physiotherapy. At best, they keep the patient out of work for weeks or months, but all too often they lead to partial or even total invalidity. Fracture treatment should therefore aim at first anatomic reduction of fracture fragments, particularly in joint fractures. Secondly where applicable a stable internal fixation designed to fulfil the local biomechanical demands. Thirdly, preservation of blood supply to bone fragments and soft tissue by means of atraumatic surgical technique. Fourthly, early active pain free mobilisation of muscles and joints adjacent to the fracture. In this way, the development of "fracture disease" is prevented.

CASE REPORT

G.S. IP 644656 was a 40 years old male who was admitted to hospital having fallen from a height of about two metres from the ground landing on his left leg. He immediately felt pain followed by swelling and inability to use the left knee joint. He had no other complaint. Examination at the time of admission revealed a young man in pain, fully conscious and not pale. He had a blood pressure of 120/80mmHg, a pulse of 84 per minute, regular and full volume. His heart sounds were normal. Respiratory and abdominal findings were normal. The vertebral column and pelvis were normal.

Local examination revealed a swollen tender left knee without external wound, bruises or laceration. The knee was held in slight semiflexion. Joint movement were limited by severe tenderness. The tenderness was especially severe on the lateral aspect of the joint. The dorsalis pedis and posterior tibial pulse felt strong and of full volume. Capillary filling at the toes and sensation were not impaired. X-Ray of the knee revealed a depressed fracture of the lateral tibia plateau.

The patient was planned for open reduction and internal fixation as this was an intra-articular fracture. He was admitted and the limb was elevated on a Braun splint to reduce the swelling. His hemogram revealed a hemoglobin of

13.6gm/dl. Skin preparation with antiseptic solution (savlon) was started three days prior to surgery in the ward with initial scrubbing with soap and water.

Under general anaesthesia, the patient in supine position, a tourniquet was applied. The skin was prepared with iodine and spirit and the leg was draped to leave only the exposed knee. The proximal tibia at the fracture site was approached by 15cm long "S" shaped lateral skin incision. The capsule was incised 1cm lateral to the patella and the joint was inspected. Hemoarthrosis of about 150 mls was found and evacuated. Further inspection revealed an intact lateral meniscus. There was no evidence of ligamentous injury. The capsular incision was continued distally over the lateral tibial condyle to the insertion of the iliotibial band. Using a periosteal elevator the proximal origin of the anterior tibial muscle was elevated from the tibia hinging it on its posterior attachments. Care was taken to safeguard the anterior tibial artery and deep peroneal nerve as they pass under cover of the anterior tibial muscle. The bone exposed on the anterior tibia provided a site in which to fashion a window through which the depressed segments of the joint surface could be elevated. Through the window in the tibia, a blunt instrument was inserted and used to push-up the depressed bone fragments. This was after identifying the fracture

line and fracture fragment. The fracture line was curated to create a fresh surface. By visualising the interior of the joint through the capsular incision and by manipulating the fracture fragment anatomical reduction was carried out. Articular congruency was achieved and fragments fixed with two cancellous screws. The tibia window was refilled with the bone initially removed for making the window. The capsule, periosteum and subcutaneous tissue were closed in layers using interrupted dexon 3/0. The skin was closed with nylon 3/0.

Post operatively the limb was nursed on a Braun splint and the patient was given intramuscular Pethidine injections 100mg 8 hourly for 24 hours.

He also received intravenous Cloxacillin injections 500mg six hourly for 48 hours. Control X-Rays showed adequate reduction. The drain was removed on the second post operative day. Skin stitches were removed on the seventh post operative day and the patient was started on active and passive knee mobilisation exercises. The patient was ambulant with non-weight bearing crutches and was discharged on the eleventh post operative day to attend orthopaedic and physiotherapy out patient clinics.

At first follow-up clinic two weeks after discharge from hospital the patient could fully flex the knee joint however he had a 10 degrees limitation to full extension.

He also complained of occasional mild pain in the knee joint. He was seen again at the clinic six weeks after surgery. He had less pain and almost full extension, he was allowed to start partial weight bearing. He had full extension and flexion of the knee joint at twelve weeks and control X-Ray showed satisfactory results. He was allowed to start full weight bearing.

DISCUSSION

Anatomy and function of the knee joint

The knee joint is a modified hinge which must allow limited rotation and flexion, yet provide complete stability and control under a great range of loading circumstances. The femoral condyles are cam-shaped in lateral profile, the slight relative lengthening of the ligament in flexion allowing rotation to occur on the tibia. In extension, the ligaments and capsule become taut as the femur screws home in internal rotation. The capsular envelop is judiciously reinforced medially and laterally by the collateral ligaments whose prime function is to resist varus and valgus stresses on the knee. Anteroposterior control is exerted by the cruciate ligaments which are key contributing structures to overall joint stability, as they tighten on each other to control the "screw home" mechanism in full extension. The menisci are major load bearing structures which offer an improved contour for femoral articulation, but their location between the moving joint surfaces render them vulnerable to trauma, especially when the knee is loaded in flexion and rotation. The muscle groups investing the knee are important in stabilizing the joint as well as controlling movement. The close integration of all these elements is vital to the smooth function of the knee joint. Damaged structures must be

reconstructed as closely as possible to reproduce the original anatomy, if optimal function is to be restored to the injured joint.

Incidence, etiology and classification

As previously stated injuries to the knee can be divided into those which affect ligaments, the menisci, the extensor apparatus and the tibia and femoral condyles. The following is a description of 21 patients with knee injuries studied by the author in a five months rotation in the Orthopaedic and Trauma Department,MMC.

Of the 21 patients 15 (71.4%) were males while 6 (28.6%) were females. There age ranged from 12 to 50 years with a mean of 28 years. As in previously quoted literature [2], Road traffic accidents were the leading cause of knee injury. Nine patients (42.9%) had knee injuries following Road Traffic accidents. Sports activities and especially football was the second most common cause of knee injuries in this Muhimbili series.

There were seven patients (33.3%) who were involved in sports accidents. Five patients (23.8%) were injured from various other causes. This group included patients who fell from heights, falling while climbing or going down stairs and falling into ditches while walking or hunting. (See table I below).

TABLE I CAUSES OF KNEE INJURIES

	N	%
1. ROAD TRAFFIC ACCIDENTS	9	42.9
2. SPORTS ACCIDENTS	7	33.3
3. OTHER CAUSES	5	23.8
TOTAL	21	100.0

TABLE II: TYPE OF KNEE INJURY AND TREATMENT IN 21 PTS.

	Non-Oper. treatment	Operative treatment	Total	%
1. Femoral condyle fractures	2	2	4	19.0
2. Tibial plateau fractures	2	4	6	28.6
3. Distal femoral epiphyseolysis	5	1	6	28.6
4. Proximal tibial epiphyseolysis	1	-	1	4.8
5. Post. dislocation of the knee	-	1	1	4.8
6. Ant. dislocation of the knee	1	-	1	4.8
7. Medial coll. lig. tear	-	1	1	4.8
8. Complete lig. disruption	-	1	1	4.8
TOTAL	11(52.4%)	10(47.6%)	21	100.0

The leading type of knee injury seen were tibial plateau fractures and distal femoral epiphyseolysis each contributing 28.6%. There were four femoral condyle fractures (19%). Other conditions seen were dislocations of the knee two cases and ligament injury two cases. One patient with posterior dislocation of the knee complicated by gangrene of the leg underwent amputation while one patient with complete ligament disruption underwent knee arthrodesis. Overall 11 patients (52.4%) had non-operative

treatment while 10 patients (47.6%) underwent operative treatment. (see table II).

Fracture of tibial condyles

The expanded cancellous proximal end of the adult tibia overhangs the shaft on either side and is inadequately supported from below by this cortical bone. The tibial condyles are thus more frequently the site of fracture than the opposing femoral condyles which, subjected to the same injury producing mechanisms are seldom fractured because of their sound architecture and adequate support from above by thick cortical bone [4]. This fact is exemplified in this Muhimbili series where tibial plateau fracture were commoner than fractures of the femoral condyles (see table II).

Fracture lateral tibial condyle

The mechanisms of injury are compression; force applied from above as in a fall from a height and abduction or adduction; force applied to the outer or inner side of the leg. The lateral aspect of the joint, as opposed to the medial is not protected by the other limb. Abduction or valgus strain is thus the common mechanism and fractures of the lateral condyle are the result. The gravity of the lesion lies not only in the difficulty of reduction, obstructed by the central portion of the condyle impacted into the tibial head , but in the comminution of articular

surface and damage to the ligamentous and meniscus components of the joint. In this Muhimbili study fractures of the lateral condyle were also commoner than those of the medial condyle.

Fracture both tibial condyles

Fracture both tibial condyle, the so called "Y" shaped fracture occur second in frequency to fractures of the lateral condyle [2]. In this Muhimbili series however no such fracture was seen. The injury usually takes place as a result of a fall from a height and both condyles are driven downwards.

Fracture medial tibial condyle

It was fortunate that fractures of the medial condyle are relatively uncommon, for the potential disability is much greater than on the lateral side. The medial condyle is weight bearing. Alteration in shape or surface contour interferes with the screw-home movement.

Clinical feature and diagnosis of tibial condyle fractures

The joint is usually grossly swollen, partly by reason of the hemarthrosis and partly from effusion into soft tissue overlying the upper end of tibial and femoral attachment of medial ligament. Even when the tuberosity is depressed, valgus deformity is masked by soft tissue swelling. In cases which have occurred as a result of a relatively trivial injury, such as stepping down unexpectedly from a

high kerb, the clinical signs may not be marked and the true nature of the injury only revealed following radiographic examination.

Aspiration of the joint performed as a diagnostic and therapeutic measure, reveals blood, synovial fluid and fat from concellous tissue of the tibial head.

Treatment of tibial plateau fractures

The management of tibial plateau fractures remain controversial. Apley [5] recommended skeletal traction with early motion. Scotland and Wardlaw [6] advocated the use of cast bracing. More recently Scharzker et al [7] and Waddell et al [8] following clinical reviews of large series of patients with tibial plateau fractures concluded that open reduction with anatomic reduction of articular surface generally produces best results. The results of treatment are dependent on the amount of intra-articular comminution, the presence or absence of a step deformity on the weight bearing surface and residual angulation deformity. Advocates of treatment method that allows early motion are numerous. Early motion can be achieved if the fracture is stabilized by open reduction and internal fixation with or without bone grafting, by per cutaneous fixation or by skeletal traction or cast-bracing combined with early knee motion. Open reduction and internal fixation should be performed according to the principles of

the A.O. These principles include obtaining an accurate anatomic reduction and maintaining that reduction with a rigid internal fixation, with early motion a pre-requisite of this treatment method.

Range of motion exercises are either began immediately or are delayed for as long as ten days to allow wound healing. Touch weight-bearing is began at 6-12 weeks. Of the six tibial condyle fractures in this Muhimbili series four were treated with open reduction and internal fixation with good results.

Supracondylar fractures of the femur

The supracondylar region of the femur is structurally strong and fractures occur only as a result of extreme direct violence. The fracture commonly occurs among coal-miners as a result of a fall of roof on the outstretched leg. Road traffic accidents is the commonest cause of this injury in Muhimbili. In selecting the method of treatment to be adopted in supracondylar fracture of the femur, it is important that the procedure chosen should not by itself be productive of disability. Put to such a test, methods involving internal fixation whether by plate or bone graft will fail (2). This view has however been challenged by Muller and Allgower who are pioneers of internal fixation (3). Cast bracing has also been used with good results (9). In this Muhimbili series there were four

patients with supracondylar femoral fractures two of whom were treated by closed reduction and two requiring open reduction and internal fixation. One of those operated had a failed attempt to closed reduction, internal fixation with a plate was done with good results. The second patient who was operated had an open intra-articular, intercondylar "Y" shaped fracture. The fracture was fixed with cross Kirshner wires.

Separation of the Distal epiphysis of femur

Separation of the distal epiphysis of femur differs from supracondylar fracture in that the mechanism of production is hyperextension or torsion rather than direct violence. In this Muhimbili series the commonest cause of injury was sports accidents especially football. Five of the six patients had closed treatment while one had open reduction and fixation with Kirshner wires.

Ligamentous injury of the knee

The problems of early diagnosis and treatment of injuries to the ligaments of the knee are of current interest to all orthopaedic surgeons involved in the management of trauma. Battle and Robson were the first surgeons to describe repair of the cruciates but Hey Groves suggested a substitution procedure for both the anterior and posterior cruciate ligaments[10]. Since then surgeons have become

more aggressive in their approach. Patients have become more demanding and unrealistic in their desire to restore their knees to "normal" following injury.

Examination under anaesthesia is now mandatory, wherever there is a suspicion of injury to the ligaments of the knee joint, supplemented where necessary by arthroscopy and arthrography. Early repair is necessary. In this Muhimbili series there was one patient with medial collateral ligament tear which was successfully repaired. One patient with complete ligamentous disruption and a painful unstable knee joint underwent arthrodesis. She sustained this serious injury after falling from a bridge.

Complications of knee injury

In this Muhimbili series one patient presented three days after initial posterior dislocation of the joint. The leg was already gangrenous and the patient was very toxic. He underwent an above knee amputation and he was ambulated with crutches pending fitting with a prosthesis. No other complications were seen.

Rehabilitation:

The patient with an injured knee requires continuous physiotherapy during the convalescence period to avoid joint stiffness. Some patients would further require fitting with orthopaedic prosthesis or orthotics. Rehabilitation, therefore, require a teamwork approach

involving the physiotherapist, orthopaedic technologist and coordinated by the attending orthopaedic surgeon.

Conclusions

Serious complications such as vascular damage leading to gangrene of the leg can occur especially with posterior dislocation of the knee. Careful handling of patients with knee injuries is therefore necessary.

Furthermore, the treatment of knee injuries may require specialized knowledge and theatre facilities. The best approach would be to prevent occurrence of knee injuries. This can in part be done by reducing Road Traffic accidents which are the leading cause of knee injuries by constructing and maintaining better roads. Safer road use by both drivers and pedestrians are desirable. Curbing of unnecessary sports violence by better refereeing would also go a long way to reduce the incidence of knee injuries.

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10. MANAGEMENT OF SUPRACONDYLAR HUMERAL FRACTURES, MUHIMBILI

ABSTRACT

A review of ten patients with supracondylar fracture of the humerus is presented. All the patients were males except one. Their ages ranged from 11 to 50 years with a mean of 22.9 years. The commonest cause of injury was a fall on the outstreched hand. This occurred in 8 of the 10 patients. One patient was involved in a motor traffic accident while another was shot by bandits. The left arm was involved in 7 patients while in 3 it was the right. Brachial artery injury occurred in the patient who was shot. Six patients were treated by closed manipulation and application of POP cast while open Reduction and internal fixation was performed in four patients who were not amenable to conservative treatment. Radial nerve neuropraxia in one patient which resolved spontaneously was the only post operative complication observed in this study.

INTRODUCTION

The motion at the elbow joint are flexion and extension by the hinge joint between the distal of humerus and the proximal radius and ulna, and supination and pronation of the forearm which requires motion of the proximal radio-ulna and radio-humeral joint. Significant injury to any of

these articulations may interfere with both forearm motion and elbow flexion and extension. Because of this complex articulation, the elbow is less able to withstand minor degrees of anatomic distortion. Injuries involving this articulation have a propensity for stiffness if immobilised too long and for production of new periosteal bone or myositis ossificans.

Supracondylar fracture accounts for the majority of fractures sustained about the elbow in children. Supracondylar fractures also occur in adults however this is rare as compared with the incidence in children. [1,2] In the one year period from April 1990 to April 1991, 39 patients were admitted into the orthopaedic wards MMC for elbow injuries amongst a total of 2111 admissions. Elbow injuries therefore accounted for 1.8% of the admissions. Twenty four patients out of the 39 had supracondylar fracture accounting for 61.5% of the elbow injuries.

Supracondylar fractures are usually caused by a fall upon the extended elbow; a small percentage of these fractures occur with the elbow flexed and should be recognized, so that the treatment and immobilisation can be applied in extension. In the older patients the fractures are often open and caused by motor traffic accidents or bullet injuries. As the humeral condyles are driven back into extension, the brachial artery may be stretched, contused

or lacerated over the distal end of the proximal fragment during the initial injury. An injury to the brachial artery may cause collateral vasospasm, producing ischemia to the flexor forearm muscles and resulting Volkmann's contractures. The ulna nerve may be injured but not surprisingly, it is the median nerve which is more commonly affected when there is an injury to the adjacent brachial artery.

Management of displaced fractures is fraught with problems including neurovascular compromise, difficulty in obtaining or maintaining reduction and poor late results because of stiffness of the elbow or cubitus-varus. Following is a case presentation and review of ten patients with supracondylar fractures treated in the orthopaedic and trauma department.

CASE REPORT

H.J. IP 646236 was a 12 year old boy attending primary school class six, who was brought to the casualty department by his grandmother having fallen from a coconut tree and landing on his left outstretched arm. He had severe pain followed by swelling of the left elbow area and could not use the limb though he could flex and extend the fingers. There was no history of loss of consciousness, convulsions or vomiting after the injury. There was no other injury. This was his first admission to hospital and

there was no family history of epilepsy. The child was a normal hospital delivery and had attended Maternal Child Health clinic where he had received all routine vaccinations. Normal growth milestones were reported. He was the sixth born in a family of six children.

General examination revealed an apparently healthy looking boy but in pain. His left arm was supported by an arm-sling in slight flexion and supination and the right arm stabilized the left arm at the wrist. The patient was also slightly leaning over to his left side. He was not pale. His radial pulse was 78 per minute, regular and full volume. He had a blood pressure of 110/60 mmHg and normal heart-sounds. He had normal abdominal and respiratory findings.

Local examination revealed a grossly swollen left elbow without external bleeding or bruises. The swelling extended distally to mid forearm and proximally to mid upperarm. The left elbow was held at about 20 degrees flexion and in supination. Posteriorly there was distortion of the humeral contour. The equilateral triangle could not be defined on the posterior aspect of the elbow due to swelling. He had a good volume radial pulse and normal capillary filling at the nail beds and finger tips. There was no loss of sensation. he was unable to perform movements at the elbow joint due to pain,

however he was able to perform active movements of fingers and the wrist joint.

X-Ray of the left arm in postero-anterior and lateral views revealed a transverse fracture of the left supracondylar region of the humerus with posterior displacement of the distal fragment. There was no lateral angulation. Closed manipulation of the fracture was planned. This was done under Pethidine and Diazepam, the patient in supine position.

Two assistant were deployed. The first was applying traction at the wrist joint and the second applied counter traction to the proximal fragment of the humerus. Traction was gradually increased while the operator was manipulating the distal fragment pushing it anteriorly and then the arm was flexed slowly to 90 degrees while the radial pulse was being monitored. The elbow was further flexed to about 100 degrees and radial pulse was still felt with good volume. This position was maintained with a POP cast. A post manipulative control X-Ray revealed unsatisfactory reduction of the fracture. A second manipulation procedure was attempted under general anaesthesia and muscle relaxant. Control X-Ray still revealed unsatisfactory reduction of the fracture. Closed reduction was therefore considered a failure and the patient was planned for open reduction and internal fixation. The parents and the

patient were informed of the decision and consent for the operation was obtained. The reason for failure of closed reduction was later noted (see operation notes below) to be soft tissue interposition.

Since the elbow was grossly edematous it was elevated with two pillow at the side of the patient in bed to encourage the edema to settle. Skin preparation with antiseptic was started three days before surgery. Ten days after the initial injury, the patient was taken to theatre for open reduction under general anaesthesia and tourniquet.

A posterior skin incision was made just above the fracture site and distally to the tip of the olecranon process. The ulna nerve was identified, taped and retracted to safeguard it. The triceps was split longitudinally along its fibers, the periosteum was also opened longitudinally, elevated and retracted. Both the proximal and distal fragments were identified and a hematoma which was present was evacuated. Soft tissues which had interposed between the fragment were excised. The two fragments were grasped with reduction forceps and manipulated for reduction. Reduction was successful and the fragments were temporarily maintained in position by reduction clamps. Two Kirshner wires were drilled in from the short distal fragment into the long proximal fragment. The wires ran from the lateral

epicondyle subcutaneously into the medullary cavity of the humerus.

The wire ends were cut and bent to avoid skin injury and extrusion. The wound was closed in layers with dexon 2/0 to the periosteum and subcutaneous tissue after a tube drain was left in-situ and brought out through a separate stab incision. The skin was closed with 3/0 monofilament nylon. The wound was dressed with pressure bandage and a backslab POP was applied with the elbow at 90 degrees flexion.

The patient was given 3 doses of intramuscular pethidine 50mg. 8 hourly. The limb was elevated with pillows to prevent edema. Control X-Ray post reduction and internal fixation showed satisfactory reduction. On the second post operative day the patient was noted to have developed mild wrist drop without impairment of sensation. This was attributed to possibly neuropraxia of the radial nerve secondary to edema or tourniquet. The weakness improved and had cleared on the sixth post operative day. The drain was removed on the second post operative day. The wound was inspected and stitches removed on the 8th post operative day. There was no evidence of sepsis. The patient was discharged with a POP backslab after instruction on physiotherapy and was seen in the outpatient clinic two weeks later. He was started on more vigourous

physiotherapy to prevent elbow joint stiffness. At six weeks post operative the patient could fully flex and extend the elbow joint and pronate and supinate the forearm without difficult. There was no cubitus varus deformity. The patient was allowed to continue attending school and physiotherapy and he is planned for removal of the Kirshner wires.

DISCUSSION

During my five months rotation in the Orthopaedic and trauma department, I had the opportunity to participate in the management of ten patients with supracondylar fracture of the humerus. All the patients with the exception of one were males. Their age ranged from 11 to 50 years with a mean of 22.9 years. The commonest cause of injury was a fall on the outstretched hand. Three patients fell at football, another three fell at home and two fell from trees. One patient was involved in a motor traffic accident, he had his elbow sticking out of a car window and was injured by an over-taking vehicle. One patient was shot by bandits. In seven patients it was the left elbow which was involved while in three, it was the right elbow.

Six patients were treated by closed manipulation and application of POP cast while open reduction and internal fixation was performed in four patients who were not amenable to conservation treatment. In the one patient who was shot, the brachial artery was injured and arteriogram revealed complete block of the brachial artery with patent collaterals. The limb survived on the collaterals. Radial nerve neuropraxia which resolved spontaneously was the only post-operative complication observed in this study.

Anatomy

The elbow joint consists of three articulations within the same synovial membrane. The humeral-ulna joint provides stability and allows flexion and extension movement. Pronation and supination occurs at the proximal radio-ulna and radio-humeral articulations. The medial and lateral collateral ligaments give stability to the joint. Because of this complex articulation the elbow is less able to withstand minor degrees of anatomic distortion. The trochlear has a central groove which is directed laterally in extension. This determines the carrying angle ranging from 0-15 degrees with a mean of 7.8 degrees (3)

Classification

The level of most supracondylar fractures is usually proximal to the attachment of the collateral ligaments and capsule. The distal epiphysis is not usually involved and the fracture is extra-synovial (4,5). Supracandylar fractures of the humerus are usually caused by trauma. In the younger age group the commonest cause is a fall on a outstretched hand whereas in the adults motor traffic accidents are a common cause of the fracture. These facts are exemplified even in this small study. The extension type of supracondylar fracture is the most common fracture of the elbow (2). In this series of patients all had the extension type of supracondylar fractures. The fracture

line usually extends transversely or obliquely across the metaphysis close to the epiphyseal line [6]. The shaft of the humerus is driven anteriorly while there is posterior displacement of the distal fragment. The extension type of supracondylar fractures has been classified after Mitchell as follows [5].

Type I: Fracture without displacement

Type II: Fracture with lateral displacement

Type III: Fracture with rotation, with or without lateral displacement

Type IV: Fracture with complete displacement and no contact between the fragments.

The median and radial nerves and the brachial artery are especially liable to injury in the extension type of fractures being at risk of being stretched over the jagged edge of the humeral shaft. The brachial artery and median nerves may also be injured where there is radial displacement of the distal fragment. The radial nerve is however injured mainly with medial displacement because radial displacement pulls the nerve away from the sharp edge of the supracondylar fractures in extension. The nerve is usually relaxed and carried posteriorly away from the fracture fragments.

The anteriorly (flexion type) displaced supracondylar fractures of the humerus are usually rare. They occur

after a fall upon the flexed elbow [1,7]. In this series of ten patients no such a fracture was observed.

Clinical features

The diagnosis of a supracondylar fracture of the humerus is made from history, clinical examination and radiological confirmation of the fracture. Sometimes vascular studies such as arteriograms may be needed as when injury to the brachial artery is suspected. A Dopler ultrasound probe can also be used to assess the perfusion of the hand. A history of trauma is usually present. Displaced supracondylar fractures present with marked swelling about the elbow with deformity and sometimes ecchymosis while undisplaced fractures present with considerable swelling and pain without deformity. The patient is usually unable to use the affected limb.

It may be difficult to differentiate clinically between a supracondylar fracture and a posterior dislocation of the elbow joint. Generally, however, supracondylar fractures are more mobile than posterior dislocations and anatomical relations between the olecranon and internal and external epicondyles is not disturbed in the fractures as in posterior dislocation. The arm is shortened in supracondylar fractures while it is the forearm which is shortened in posterior dislocation. Usually the patients hold their limb with the elbow semi-flexed and in

supination. If the blood supply has been compromised as in brachial artery injury with subsequent collateral supply vasospasm, there is, severe pain, numbness, cyanotic and cold fingers, loss of finger function and absence of radial pulse and capillary circulation. Nerve injury is rare but when it occurs there is loss of sensory and motor function in the affected area. In neglected patients they may present with malunion with cubitus varus deformity[8].

Treatment

The treatment of supracondylar fractures needs to be carefully undertaken in order to avoid complications. Patients should be hospitalized and observed for at least 24 hours before they can be discharged. Forceful joint motion, massage and passive stretching are best avoided. Treatment depend on the severity of the fracture in terms of displacement, neurovascular involvement, open or closed fracture and severity of edema.

Conservative treatment

Closed manipulation of supracondylar fracture of the humerus required a minimum of three persons. One should study the X-Ray carefully and rehearse the manipulation procedure. The procedure can be done under local or general anaesthesia. The surgeon does the manipulation while one assistant provides counter traction and the second assistant applies POP cast. The fragments are

initially disengaged by traction and counter traction, the sideways displacement is corrected by direct pressure. The rotational malalignement is corrected by holding the upper arm firmly while the forearm is internally rotated with the elbow at 45 degrees flexion. The posterior displacement is corrected by flexing the elbow and applying thumb pressure to the olecranon in order to engage the fragment on to the end of the humerus.

Operative treatment

Closed reduction in flexion and percutaneous fixation with cross or parallel Kirshner wires is possible under radiological control. This procedure was first described by Swenson[2].

Later series have reported fixation of the fracture by means of two Kirshner wires, introduced from the lateral aspect to minimise risk of ulna nerve damage, however the original cross-pinning technique by Swenson has continued to be used with excellent results and negligible morbidity. Open reduction and internal fixation has generally been reserved for specific indication mainly for an open fracture, a fracture requiring vascular exploration or an irreducible fracture. In four of the ten patients reported have open reduction and internal fixation was performed for irreducible fractures. In the case reported above the cause of irreducibility was soft tissue interposition.

Complications

Primary complications such as Volkmann's ischaemic contracture can occur from damage to the brachial artery. Median nerve injury can occur in which case the patient fails to flex the distal phalanx of the index finger and thumb. The median nerve is sensory to the radial three and half digits and motor to the entire thenar group of muscles, radial two lumbricals, all superficial flexions, radial half of deep flexors, pronator teres, pronator quadratus, flexor carpi radialis and palmaris longus. Secondary complications are the result of treatment manouvres or under treatment. Vascular compromise from forcible elbow flexion, tight bandaging or tight POP cast. Secondary neurovascular damage can occur with closed or open reduction. In this series one patient had injury to the brachial artery from a gun shot. The limb survived on collateral circulation. Neuropraxia in one patient was the only post operative complication seen in this series of ten patients. This resolved spontaneously.

Prognosis

The prognosis will depend on the type and site of fracture as well as the time interval from injury to treatment. The prognosis will also depend on the type of treatment offered, whether conservative or operative. The skill of

the surgeon and armamentarium available are also important prognostic factors.

Conclusion

Supracondylar fracture of the humerus is a serious injury because it often presents difficulties of diagnosis and reduction,neurological and vascular problems ,slippage in the cast ,malunion and stiffness of the elbow joint. It is noteworthy that in the Orthopaedic and Trauma department MMC all patients with displaced supracondylar fractures are admitted despite the chronic shortage of hospital beds and the majority are managed by closed reduction and application of POP cast. In a few patients however, the fracture is not amenable to conservative treatment and open reduction and internal fixation is required. The results of treatment in this MMC series are satisfactory in that in the five months period of study there was no case of amputation or Volkman's ischaemic contracture due to a supracondylar fracture. Continued vigilance against these serious complications is recommended.

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