THE PATTERN, CAUSES AND OUTCOME OF GENERALIZED PERITONITIS IN ADULT SURGICAL WARDS, AT MUHIMBILI NATIONAL HOSPITAL

By

Nicephorus Rutabasibwa: M.D

A dissertation submitted in partial fulfillment of the requirement for the Degree of Master of Medicine in surgery of Muhimbili University of Health and Allied Sciences

Muhimbili University of Health and Allied Sciences.

2007

CERTIFICATION

The undersigned certifies that, he has read and hereby recommend for acceptance by University of Dar es salaam a dissertation entitles: *The Pattern Causes and Outcome of Generalized Peritonitis in adult surgical ward, at Muhimbili National Hospital.* Submitted in partial fulfillment of the requirement for the degree of Masters of Medicine in surgery of Muhimbili University of Health and Allied Sciences.

.....

Dr. S.C.Yongolo. MD, Mmed (surg) Cert. (Urology).

Senior Lecturer and Consultant urologist at MUHAS

(Supervisor)

Date 5/12/07

COLLEGE LIBRARY
MUHIMBILI UNIVERSITY
COLLEGE OF HEALTH SIENCES

DECLARATION AND COPYRIGHT

I, NICEPHORUS RUTABASIBWA declare that, this dissertation is my own original work and that it has not been presented to any other University for a similar or any degree award.

Signature……

© Nicephorus Rutabasibwa

This dissertation, is copyright material and as such protected under the Berne Convention, the copyright Act of 1999 and other International and National enactments in that behalf on intellectual property. It may not be reproduced by any means , in full or in part expect for short extracts in fair dealing for research or private study, critical scholarly review or discourse without the written permission of the Directorate of Postgraduate Studies on behalf of both the author and the Muhimbili University of Health and Allied sciences.

ACKOWLEDGEMENTS

The completion of this study would not have been possible without the contribution of my supervisor, Dr S.Yongolo, Senior Lecturer and Consultant urology surgeon at Muhimbili University of Health and Allied Sciences. I sincerely thank him for his guidance and timely frequent corrections in the preparation of this document. He played a significant role as my mentor, my teacher, and my supervisor. His contribution is highly appreciated.

I am equally indebted to Dr M Mchembe, a specialist surgeon and lecturer, at MUHAS, for the job he did to take me through the initial development of this document as my supervisor. He is currently abroad pursuing his further studies may he excel in his ambitions

I would also like to acknowledge Prof L.E. K Lema, the then Head of Department of surgery, for his continuous encouragement and teaching.

I am equally indebted to Prof M.R Aziz, coordinator postgraduate program in the department of surgery, Prof M. Aboud, Dr N A Mbembati and all lecturers in the department of surgery, MUHAS.

I would like to extend my sincere thanks to Dr. E. Ussiri, specialist surgeon at Muhimbili National Hospital for his motivation. I am also pleased to thank all the doctors and nurses in the department of surgery for taking care of patients.

Many thanks also go to Ms Candida Moshiro and her colleagues of the department of Biostatistics and epidemiology for organizing a workshop in research methodology, which enlightened me on data processing and analysis.

Special thanks to Ms Neema Kazoka of Muhimbili Orthopaedic Institute (MOI) for her secretarial assistance.

Thanks to the Almighty God for giving me good health throughout my study period and I was filled with encouragement.

DEDICATION

This dissertation is dedicated to my beloved wife Annabella Rutabasibwa, for her love and commitment. She really encouraged me to pursue this endeavor.

Secondly, to my lovely daughter, Caroline Kemilembe. Thanks to her for being patient with me during my study period. You were very young and really needed my love and care, but you gave me strength to go on. Thanks so much.

Last but not least my special thanks to my father Boniphace K. Bernardo, and my mother Gaudentia Boniface for giving me education sufficing to make my life possible in this world.

May Almighty God bless you all.

ABSTRACT

Generalized peritonitis is a common surgical emergency. It carries a high mortality and morbidity. The causes of peritonitis vary according to geographical location, but the pattern remains the same. However, published data for Muhimbili National Hospital and Tanzania for that matter are scarce or not available at all.

This study therefore, was designed to describe the pattern, causes and outcome of generalized peritonitis in adult surgical wards, at Muhimbili National Hospital.

A prospective descriptive study of the patients who developed generalized peritonitis from 1st March, 2006 to 31st December, 2006 were included in this study and followed up for thirty days. Their socio-demographic data causes and outcome of generalized peritonitis were recorded and analyzed.

A total of 124 patients who presented with generalized peritonitis were surgically treated at Muhimbili National Hospital in Dar es salaam, Tanzania during the study period. All patients underwent thorough peritoneal lavage and drainage after the cause of peritonitis had been established and treated surgically.

Antibiotics were given to all patients, and those with gastro-duodenal perforation secondary to PUD, an acid reducing drug was added.

Most of the patients were males (80.6%) and the age group between 21 and40 years was the most affected constituting 58.1%. This is the population group that is involved in social productivity. Seventy nine percent of these patients belonged to low socio- economic strata.

Gastro- duodenal perforation was the leading cause of peritonitis accounting for 33.1% followed by small bowel perforation (31.5%). Typhoid ileal perforation was the leading cause of small bowel perforation constituting 59%. Appendicular perforation was observed in the younger age group of 11 -20 years constituting 8.1% The morbidity rate was 39.6% of which long hospital stay was the commonest outcome, with a mean hospital stay of 11.1 days.

Twenty seven patients, died during the post-operative period resulting in a mortality rate of 21.8%, septicemia being the primary cause of death in most cases.

TABLE OF CONTENTS

Certification	ii
Declaration and copyright	iii
Acknowledgement	iv
Dedications	vi
Abstract	vii
Table of contents	ix
List of tables	xi
Abbreviations	xii
1.0 Introduction	1
1.1 Definition	1
1.2 Historical background	2
2.0 Literature review	5
2.1 Anatomy of peritoneum	5
2.2 Etiology of generalized peritonitis	7
2.3 Diagnosis of peritonitis	36
2.4 Treatment of peritonitis	37
3.0 Problem statement	40
4.0 Rational of the study	41
5.0 Research questions	41
6.0 Objectives	42
7.0 Methodology	42
7.8 Ethical clearance	47

7.9 Data collection 47

8.0 Data analysis 49

10.0 Results 50

11.0 Discussion 58

12.0 Conclusions 70

13.0 Recommendation 72

References 73

Appendices 88

Questionnaire 88

Consent form 91

LIST OF TABLES

Table 1. Pattern of peritonitis according to age and sex.....50

Table 2. Causes of peritonitis.....52

Table 3 Causes of small bowel perforation.....52

Table 4. Cause of peritonitis according to age.....53

Table 5. Time interval from onset of symptoms to report to hospital.....54

Table 6. Outcome of peritonitis.....55

Table 7 Time interval between onset of symptoms to hospital presentation and Outcome of peritonitis.....56

Table 8. Occupation.....57

ABBREVIATIONS

APACHE……Acute physiology and chronic health evaluation

CT- scan…….. Computer tomography scan

CVP……..Central venous pressure

DU……Duodenal ulcer

HSV…… Highly selective vagotomy

ICU……..Intensive Care Unit

MNH……Muhimbili National Hospital

MOSF……..Multiple organ systemic failure

MUCHS……..Muhimbili University College of Health Sciences

NSAIDs…….. Non Steroid anti inflammatory drugs

PID……..Pelvic Inflammatory Disease

RTI……..Respiratory tract infection

SIRS……Systemic inflammatory response syndrome

SP……Secondary peritonitis

SPSS……Statistical package of social science

V&A……Vagotomy and antrectomy

1. INTRODUCTION

1.1 Definition

Peritonitis is defined as inflammation of the serosal membrane that lines the abdominal cavity and the organs contained therein.[1]

It may be local or have a systemic consequence generating multiple organic dysfunction. Most of the studies report a mortality of 30% in severe intra-abdominal infection.[1]

Secondary peritonitis is caused by the loss of integrity of the gastrointestinal apparatus, which contaminates the peritoneal cavity with pathogens. Invariably they are polymicrobial infections, mostly due to facultative aerobic and anaerobic Gram negative bacilli.[1]

The prognosis of peritonitis depends on the struggle between two forces: local and systemic immunity of the host and the volume, nature and length of the contamination. Microorganisms and their products stimulate cellular defenses in the host and activate numerous inflammatory mediators responsible for sepsis[1]

Peritonitis is often caused by introduction of infection into the otherwise sterile peritoneal environment through perforation of the bowel, or ruptured appendix.

The inflammation may also be caused by introduction of a chemically irritating material, such as gastric acid from a perforated ulcer or bile from a perforated gall bladder or lacerated liver, and acute pancreatitis.

In sexually active women, Pelvic Inflammatory Disease (PID) is secondary to a variety of agents; Gonococcus and Chlamydia are most common[2]. Bacteria may invade the pelvic cavity secondary to a septic abortion or presence of an intrauterine device, the pathogens then simultaneously infect the fallopian tubes which may lead to intraperitoneal abscesses. Localized peritonitis most often occurs in the pelvis from an infected fallopian tube or a ruptured ovarian cyst.[2]

1.2 Historical background

Physicians in antiquity dreaded abdominal infections. Despite the fact that peritonitis was extremely common, reports of successful surgical interventions were only anecdotal before the past century. Medicine's comprehension of the pathophysiology of the peritoneal cavity is still

evolving. The history of our understanding of the process could be considered to be as recent as the current literature.[3]

The surgical treatment of peritonitis started with the first laparotomy for an infected ovarian cyst by McDowell in the beginning of the 19th century. Thereafter the surgical approach developed parallel to the advances in abdominal surgery[4].

In the last decade of the 19th century Mikulicz felt that laparotomy was indicated in all patients with purulent peritonitis[4].

In the beginning of the 20th century Korte and Kirschner defined the principles of surgery for peritonitis that are valid up to this day, early surgical intervention, elimination of the source of infection, and peritoneal toilet. Since that time surgeons have debated on the utility of draining and irrigating the peritoneal cavity. Postoperative lavage was advocated in the beginning of the last century, but generally regarded ineffective.[4]

Thirty years ago postoperative lavage was again strongly advocated, but evidence for its benefit was still missing.[4] The history of the treatment of peritonitis exemplifies that basic concepts which have been discovered in the past, often remain valid and can serve as guidance for a long time. Such

discoveries deserve recognition and should give reason for a critical appraisal of current practice.

Thus, the statement of Trendelenburg made one hundred years ago remains true: "...in medicine, too, the today is based on the yesterday, and to follow a gradual development is of immense interest. In daily practice, especially in surgery and obstetrics, many things are much older than an ignorant can imagine."[4.]

Untreated cases of acute peritonitis may be fatal. In 1926, the fundamental role of operative therapy in the treatment of peritonitis was documented. Kirschner [5] reported that the mortality rate from intra-abdominal infections decreased from more than 90% to less than 40% during the period from 1890-1924 with the introduction of operative management as an effective therapeutic modality.

Current treatment of peritonitis and peritoneal abscesses consists of a multimodality approach directed at correction of the underlying cause, administration of systemic antibiotics, and supportive therapy to prevent or limit secondary complications due to organ system failure.[5]

2.0 LITERATURE REVIEW

2.1 Anatomy of the peritoneum

The peritoneum is the largest and most complex serous membrane in the body. It forms a closed sac (i.e., coelom) by lining the interior surfaces of the abdominal wall (anterior and lateral), by forming the boundary to the retroperitoneum (posterior), by covering the extra peritoneal structures in the pelvis (inferior), and by covering the undersurface of the diaphragm (superior).[2] This parietal layer of the peritoneum reflects onto the abdominal visceral organs to form the visceral peritoneum. It thereby creates a potential space between the 2 layers (i.e., the peritoneal cavity).[2]

The peritoneum consists of a single layer of flattened mesothelial cells over loose areolar tissue. The loose connective tissue layer contains a rich network of vascular and lymphatic capillaries, nerve endings, and immune-competent cells, particularly lymphocytes and macrophages. Junctional complexes, thus forming a dialyzing membrane that allows passage of fluid and certain small solutes join the peritoneal surface cells. Pinocytotic activity of the mesothelial cells and phagocytosis by macrophages allow for clearance of macromolecules.[2]

The peritoneal cavity is divided incompletely into compartments by the mesenteric attachments and secondary retroperitonealization of certain visceral organs.[2] A large peritoneal fold, the greater omentum, extends from the greater curvature of the stomach and the inferior aspect of the proximal duodenum downward over a variable distance to fold upon itself (with fusion of the adjacent layers) and ascends back to the taenia omentalis of the transverse colon. This peritoneal fold demonstrates a slightly different microscopic anatomy, with fenestrated surface epithelium and a large number of adipocytes, lymphocytes, and macrophages, and it functions as a fat storage location and a mobile immune organ.[2]

The compartmentalization of the peritoneal cavity, in conjunction with the greater omentum, influences the localization and spread of peritoneal inflammation and infections.

Inflammation and/or infection of the peritoneal cavity are commonly encountered problems in the practice of clinical medicine today.

In general, the term peritonitis refers to a constellation of signs and symptoms, which includes abdominal pain and tenderness on palpation, abdominal wall muscle rigidity, and systemic signs of inflammation.[2]

Patients may present with an acute or insidious onset of symptoms, limited and mild disease or systemic and severe disease with septic shock.

The peritoneum reacts to a variety of pathologic stimuli with a fairly uniform inflammatory response. Depending on the underlying pathology, the resultant peritonitis may be infectious or sterile (i.e., chemical or mechanical).[6]

Peritoneal infections are classified as primary (i.e., spontaneous), secondary (i.e., related to a pathologic process in a visceral organ), or tertiary (i.e. persistent or recurrent infection after adequate initial therapy). The intra-abdominal infection may be localized or generalized, with or without abscess formation.[2]

2.2 Etiology of generalized peritonitis.

The common etiologic entities of secondary peritonitis include gastro duodenal perforations, small bowel perforation, perforated appendix perforated colon caused by diverticulitis, volvulus, or cancer and strangulation of bowel. [2, 7]

In a study by Bohnem J *et al*, generalized peritonitis was assessed in 176 patients, 67 (38%) of whom died. Cases were divided into causative groups (a) appendicitis and perforated duodenal ulcer, (b) intraperitoneal origin

other than appendix or duodenum, and (c) postoperative peritonitis. Mortality rates were 10%, 50%, and 60%, respectively.[7] Postoperative peritonitis was characterized by lack of influence of age on outcome, late operation, and more frequent organ failure. Delayed surgery carried a worse prognosis. Organ failure was a major risk factor resulting in 76% mortality, and was usually associated with delayed surgery. More sensitive indicators of early organ dysfunction might improve survival.[7]

In a series of 239 patients with surgical infection in the abdomen and an APACHE (Acute physiology and chronic health evaluation) II score greater than 10, seventy-seven patients (32%) died.[8]

Kriger *et al*, reviewed 956 patients with generalized peritonitis who underwent surgery. The causes of peritonitis were: perforating gastro-duodenal ulcers (454), acute ruptured appendicitis (190), acute perforated cholecystitis (82), perforation of small intestine and colon (96), postoperative peritonitis (80), other causes (54). In the postoperative period, out of 691 patients with low degree of bacterial contamination, wound infection was seen in 7.9%, intra abdominal infection in 2.5%, multiple organ insufficiency in 1.0%. Mortality was 5.9%. In the postoperative period of 256 patients with high degree of bacterial contamination wound

infection decreased to 6.0%, intra abdominal infection increased to 4.2%, multiple organ insufficiency in 42.6%; mortality was 18.1%.[9]

In a retrospective analysis done in India[10], 98 cases were managed surgically. Duodenal ulcer perforations, necrotizing enteritis, acute cholecystitis with perforation and small bowel perforations were the common causes. Most of them presented late; many had associated conditions. While there was significant decrease in the number of typhoid and tubercular peritonitis, there appeared to be an increase in the incidence of necrotizing enteritis and acute cholecystitis.[10]

2.2.1 Gastro duodenal perforations.

These are due to Peptic Ulcer Disease complication.

A peptic ulcer is an ulcer of an area of the gastrointestinal tract that are usually acidic. A more general term, Peptic Ulcer Disease (PUD), is also in use. Most ulcers are associated with Helicobacter pylori, a spiral shaped bacterium that lives in the acidic environment of the stomach.[11,12]

Ulcers can also be caused or worsened by drugs such as aspirin, and other NSAIDs. Other factors like tobacco smoking, blood group, spices etc, which were suspected to cause ulcers until late in the 20th century, have been found to be of relatively minor importance in the development of PUD.[13]

A major causative factor of peptic ulcer disease is chronic inflammation due to Helicobacter pylori, a spiral but not a spirochete, rather a microaerofilic gram negative bacillus, which colonizes the antral mucosa. The immune system is unable to clear the infection, despite the appearance of antibodies. Thus the bacterium can cause a chronic active gastritis, resulting in a defect in the regulation of gastrin production by part of stomach, and gastrin secretion is increased. Gastrin in turn, stimulates the production of gastric acid by parietal cells. The acid erodes the mucosa and causes the ulcer. Reinbach *et al* [14] studied 80 patients with acute perforated peptic ulcer and found that only 47% were positive for H.pylori versus 50% in the control group.

Another major cause of PUD is the use of NSAIDs. The gastric mucosa protects itself from gastric acid with a layer of mucus, the secretion of which is stimulated by certain prostaglandins. NSAIDs block the function of cyclo-oxigenase 1 (cox-1), which is essential for the production of these prostaglandins. Newer NSAIDs (celecoxib, rofecoxib), only inhibit cox-2, which is less essential in the gastric mucosa, and roughly halve the risk of NSAID-related gastric ulceration. Horowitz *et al* [15] found that 50% of patients with perforated duodenal ulcer, had prior history of NSAIDs use, while Lanas *et al*[16] found that the use of aspirin was associated with 70% of

upper gastrointestinal perforations compared to 26.9% in controls (p=0.0001). One can predict that NSAIDS may contribute to one fifth to one third of ulcer perforations.[17]

Glucocorticoids lead to atrophy of all epithelial tissues. Their role in ulcerogenesis is relatively small[18, 19].

Other factors, which have attributed to peptic ulcer perforation, include smoking and alcohol.[13] Smoking leads to atherosclerosis and vascular spasms, causing vascular insufficiency and promoting the development of ulcers through ischemia. Smoking is a risk factor of major importance for ulcer perforation[20, 21]. A study from Norway showed a strong association between ulcer perforation and smoking with a relative risk factor of ten[22]. It has been estimated that smoking might account for 77% of all ulcer perforations in the age group younger than 75 years[22]. A major role for smoking in the etiology of ulcer perforation is also supported by studies reporting smoking prevalence of 84% and 86% among ulcer perforation patients[20,23]. Smokers with ulcer perforation have a higher mortality risk as compared to nonsmokers[21].

Anderson *et al* [24] when assessing the association between smoking, intake of alcohol and risk of peptic ulcer perforation, found that smoking more than 15 cigarettes per day increased the risk of perforation more than 3 fold.

COLLEGE LIBRARY
MUHIMBILI UNIVERSITY
COLLEGE OF HEALTH SIENCIES

Drinking alcohol more than 2 litres per week increased the risk of ulcer perforation.

Perforation is a life-threatening complication of peptic ulcer disease. The management of peptic ulcer disease has evolved over the decades, due to advances in operative techniques, bacteriology and pharmacology.

While the recognition of the role of *Helicobacter pylori (H. pylori)* in peptic ulceration has resulted in a paradigm shift in the management of uncomplicated peptic ulcers, debate continues about the appropriate management of perforated duodenal bulb and prepyloric ulcers.[25]

A new dimension has been added to this controversy by the advent of laparoscopic techniques for closing the perforation. The first clinical description of perforated peptic ulcer was made by Crisp in 1843[26]. The features of the disease and of the patients affected have changed ever since. During the nineteenth century ulcer perforation was a rare disease that occurred mainly in young women with the perforation located near the cardia of the stomach[26]. During the early twentieth century, ulcer perforation increased in incidence and these were situated in the duodenum in middle-aged men[27].

Mikulicz introduced closure of perforation by suture in1880 when he closed a gastric ulcer perforation[28]. Nonoperative management for perforated peptic ulcer disease (PUD) was first described in 1935 by Wangensteen and is still applicable in specific conditions[29]. The first two cases of primary gastric resection for ulcer procedure was described in 1919 by Von Haberer[30]. In 1937, Roscoe Graham described simple closure of duodenal perforation[31]. Recognition of the high rate of both recurrent symptoms and recurrence of ulcer with simple closure led many authors to recommend a definitive ulcer curative procedure[32, 33, 34,35,3]

The revolution in ulcer treatment that occurred with the discovery of the role of *H. pylori* has not yet led to any detectable changes in the incidence of ulcer perforation[37]. The potential for prevention thus lies in better understanding of causal factors which have not been known until lately, but apparently differ somewhat from those of uncomplicated ulcer[22,23].

The recent recognition that *H. pylori* eradication after simple closure might prevent ulcer recurrence has challenged the role of definitive surgery and will complicate the debate surrounding the management of this difficult problem[38]. About half a million people are diagnosed to have peptic ulcer

each year in the United States. Duodenal bulb and prepyloric ulcers are approximately five times as common as gastric ulcers[39].

Ulcer perforation was a rare disease in the nineteenth century; however its incidence increased greatly at the turn of the twentieth century. Since then the world has seen an epidemic of duodenal perforations among young men which now seems to be waning[40,41].

Owing to the typical and dramatic onset of ulcer perforation, the hour of the day when the ulcer perforation occurred can be assessed. A pronounced circadian variation with a greater incidence during the day and lower incidence during late night was described as early as 1903[42]. The circadian pattern seemed to differ between gastric and duodenal perforation with duodenal perforation showing peak incidence during afternoon and evening, and gastric perforation showing a primary peak around midday and secondary peak around midnight[43].

Perforated PUD typically causes severe epigastric pain that may progress to involve the entire abdomen. The degree of peritoneal findings is, however influenced by a number of factors, including the size of perforation, the amount of bacterial and gastric contents contaminating the abdominal cavity, the time between the perforation and presentation and spontaneous

sealing of the perforation[44]. These patients also demonstrate the signs and symptoms of early shock, such as tachycardia, hypotension and anuria. Abdominal and thoracic plain erect film radiographs are sensitive in the demonstration of pneumoperitoneum (60% - 75% of patients) [44]. Even though this finding is not specific for perforated peptic ulcer, free air is usually in much smaller amounts in perforations of the other abdominal viscera. An upper gastrointestinal study with water soluble contrast can demonstrate extravasation if the ulcer has not spontaneously sealed.

At present, upper endoscopy is not recommended if perforation is present or strongly suspected because it may disrupt the spontaneous sealing[44]. Ninty two percent of perforated duodenal ulcers do so through the anterior duodenal wall. Ten percent of these are also associated with a concurrent bleeding ulcer along the posterior wall, the so-called "kissing ulcer"[44].

Traditionally, perforated peptic ulcers have been treated by urgent surgical repair, with or without ulcer curative procedure. However, several studies have indicated that many patients can be managed without an operation. Wangensteen first advocated nonoperative treatment in 1935 for patients with perforation but no pneumoperitoneum on the premise that the perforation would probably have been sealed [45]. More recently, Berne and

Donovan emphasized the use of water-soluble upper gastrointestinal study to demonstrate spontaneous sealing of perforation. They noted that 40% of perforated peptic ulcers have sealed on upper gastrointestinal study performed shortly after the patient presents[46, 47].

Nonoperative management for perforated duodenal ulcers consists of resuscitation with intravenous fluids, intravenous antibiotics, nasogastric suction and acid reducing drugs. For the latter, intravenous H2 blockers have been in use for a long time, although an intravenous form of proton pump inhibitor (pantoprazole) is now available.[45]

Treatment for *H. pylori* should begin when the patient is able to take orally. The patient should undergo serial abdominal examinations with operative therapy reserved for either progression or failure of improvement of peritonitis[45].

Taylor[48] advanced arguments favoring a nonsurgical approach nearly 50 years ago, at a time when the mortality rates for surgical treatment was close to 20%. In the study by Berne and Donovan, the mortality rate for patients treated operatively was 6.2%, compared to a mortality of 3% in those treated

nonoperatively. In addition, the authors noted a very low incidence of intra abdominal abscess or reperforation in patients managed conservatively. In another study by Crofts *et al*[49] over a 13-month period, patients with a clinical suspicion of perforated peptic ulcer were randomly allocated to conservative treatment (n=40) or to emergency surgery (n=43). In the surgically managed group, 40 had duodenal ulcer, two had gastric ulcers and one had a perforated gastric carcinoma. Eleven patients (27.5%) including, three with an erroneous diagnosis (two perforated gastric carcinomas and one perforated sigmoid carcinoma), who had no improvement within 12 hours of conservative treatment and therefore underwent operation. Extravasation of contrast on upper gastrointestinal study was not an indication for operation. Morbidity was similar in the nonsurgical and surgical groups (20 vs. 17 patients); intra abdominal abscesses developed in six and two patients, respectively. Two deaths occurred in each treatment group with an overall mortality of 4.8%. The duration of hospital stay was significantly longer ($p<0.001$) in the nonsurgical group (12.0 days) than in the surgically treated group (7.8 days) Failure of nonoperative policy was significant ($p<0.05$) in patients over 70 years of age (6/9, 67%) compared to younger patients (5/31, 16%).

In a later study by Donovan *et al*[47], the authors proposed stratifying the patients based on their *H. pylori* status. They noted a high incidence of *H. pylori* in patients with perforated duodenal ulcer and demonstrated that long-term eradication of *H. pylori* could be achieved with appropriate medical therapy. In patients, known to be either *H. pylori* negative or with perforations despite previous treatment for *H. pylori* infection, definitive surgery was recommended in suitable candidates. *Helicobacter pylori* negative patients without prior treatment usually have another cause for ulceration, such as NSAID use, severe acute stress, or Zollinger-Ellison syndrome.

Despite evidence that nonoperative management may be appropriate for many patients with perforated peptic ulcer; many surgeons still practice universal operative intervention in these patients. The type and timing of operative intervention has been a matter of debate for decades. The first issue is the performance of simple closure versus closure with ulcer curative procedure. Secondly, doubt exists about both the type of ulcer curative procedure that should be performed and whether this should be performed concurrently with simple closure or as a staged procedure.

Although the method of closure described by Graham continues to be effective at providing adequate closure of the perforation, many authors have noted a high recurrence of both ulcer symptomatology and re ulceration.[32, 33] In fact 37% to 58% of patients treated with simple closure were found to have recurrence of an ulcer, whereas 48% to 85% were found to have recurrent symptoms of PUD.[42, 31, 34,36,50,51] However, some authors argue that definitive surgery prolongs what is already an emergency procedure and that recurrences can usually be managed with medical therapy[52,53]. Moreover, several of the definitive ulcer operations have been associated with significant side effects, including diarrhea, dumping, vomiting, and weight loss. It is because of these untoward sequelae, as well as the emergent nature of the situation, that some surgeons have argued against routine performance of a definitive procedure at the time of simple closure.

Simple closure of a perforated duodenal ulcer was compared to definitive surgery in three randomized trials. Patients with risk factors were not included in two of these studies. Only one postoperative death was reported among the 328 patients included in the three randomized trials[34,36,54]. Postoperative morbidity was not significantly increased by definitive surgery, except for wound sepsis in one trial[55]. With a mean follow up of 20

months, ulcer recurrence was reported in 61% (38/62) and 6% (6/92) of cases following simple closure and definitive treatment, respectively[34]. Boey *et al*[56] identified certain risk factors that increased operative mortality in patients with perforated PUD. Specifically, significant co-morbidity, preoperative shock and perforation of more than 48 hours increased mortality. Interestingly, age over 70 years and gross peritoneal soiling did not increase mortality. They concluded that simple closure alone is indicated when any of these aforementioned risk factors are present.

When selecting a patient for a definitive ulcer operation, one must decide which procedure offers an acceptably low rate of recurrence while minimizing attendant side effects. Traditionally, the most effective forms of definitive surgery have been hemigastrectomy and vagotomy or vagotomy and/with antrectomy (V&A). However, most surgeons currently confine definitive therapeutic options to vagotomy and antrectomy, vagotomy and drainage or highly selective vagotomy (HSV)[45].

Vagotomy with antrectomy has an ulcer recurrence rate of 1% at 5 to 10 years. However, the procedure carries a mortality rate of 1% to 2% and is associated with side effects, such as anastomotic leak, gastric stasis, dumping, diarrhea, and bilious vomiting[57]. Drainage procedure usually

performed in association with truncal vagotomy is pyloroplasty, although a gastrojejunostomy may be performed when local anatomy prevents the former. The operation carries a mortality of 0.7% with a 3% to 30% risk of ulcer recurrence[36, 57]. Avoiding both denervation of the viscera and attendant drainage procedure with the associated long-term sequelae are the goals of highly selective vagotomy. This has an excellent mortality rate (0% to 0.31%) with a risk of recurrent ulcerations approaching 3% to 12%, five to fifteen years after operation.[52, 57]

Most of the recurrent ulcerations after this procedure are caused by incomplete denervation of the parietal cell mass. Thus, the outcome of this operation appears to be operator dependent with the incidence of postoperative sequelae being extremely low[57]. Consequently, many surgeons have adopted HSV as a compromise between simple closure with its high rate of recurrence and either V&D or V&A and their associated postoperative side effects[57, 58].

With the advent of laparoscopic surgery another dimension has been added to the controversy. Lau et al randomized 103 patients with a clinical diagnosis of perforated peptic ulcer to laparoscopic or open repair and inside each group, between suture and sutureless repair (plug of gelatin sponge and

COLLEGE LIBRARY
MUHIMBILI UNIVERSITY
COLLEGE OF HEALTH SIENCIES

fibrin glue). Four patients in the laparoscopic group and six in the open group were excluded after randomization either because the ulcer closed spontaneously (n=5). Of 48 patients in the laparoscopic group, 11(23%) were converted to open surgery. Laparoscopic repair of perforated peptic ulcer took significantly ($p<0.001$) longer than open repair (94 vs. 54 min respectively). Patients who had laparoscopic repair required fewer analgesic doses postoperatively than those who had open surgery ($p<0.03$). No statistically significant differences were noted between the laparoscopic and open groups with regard to duration of nasogastric aspiration, intravenous drips, hospital stay, time to resume normal diet and Visual Analog Scale pain scores for the first 24 hours after surgery. There was no significant difference in morbidity, reoperation rate, or mortality between patients who underwent laparoscopic and open repair[59]. This study demonstrated the advantages of decreased analgesic requirement by laparoscopic closure without any increase in mortality or morbidity. However, this advantage was offset by the increased operative time. Increasing familiarity with laparoscopic techniques and innovative ways of repairing the perforation like sutureless closure, single suture closure and use of fibrin glue have enabled surgeons to decrease operative time.

Siu *et al*[60] in a recent trial have tried to establish the laparoscopic approach as the gold standard for the management of a perforated peptic ulcer. By using a modified technique involving a single suture closure of the perforation they demonstrated a clear advantage in terms of decreased operating time, less postoperative pain, earlier ambulation, significantly lower incidence of respiratory tract infection (RTI), lower median length of hospitalization and earlier return to normal activity. They also defined the patient population who will stand to gain the above benefits by excluding those patients with a nonpyloric gastric ulcer perforation, an ulcer perforation >10mm in diameter and technical difficulties. Since only one out of a total of 63 patients in this study had a presentation after 24 hours, and only 2 of the 63 patients presented with systolic blood pressure <90 mmHg, thus patients presenting may not sustain the advantages defined by this group.

Controversy exists regarding the relationship between *H. pylori* and perforated DU. Although the relationship between uncomplicated peptic ulcer disease and *H. pylori* is widely supported, the association with perforation is not accepted fully[23,61]. However, a recent randomized study sought to determine if eradication of *H. pylori* in patients undergoing simple closure of a perforated duodenal ulcer was effective in preventing ulcer

recurrence[12]. The authors noted that fewer surgeons are acquiring enough experience and expertise in performing definitive procedures such as highly selective vagotomy and argued that combining medical therapy for *H. pylori* with simple closure would be more desirable than undergoing definitive operation, given at least a similar rate of recurrence.

This argument does assume an association between H. pylori and ulcer perforation, but the study showed that 95% of *H. pylori* positive patients undergoing simple repair of the perforated duodenal ulcer followed by combined medical therapy and eradication were ulcer-free at one year.[12]

In the study about the management of large perforations of duodenal ulcers, Sanya Gupta *et al*[62] characterized perforations in 3 groups-Group A (<1 cm Perforation), Group B (1-3 cm perforation), Group C (>3 cm perforation), named them small, large, and giant perforations respectively. Most patients out of 162 had small perforations. 38 (23.46%) had large perforations and giant perforations were only seen in 2 patients (1.23%).

Many of the perforations were treated by omental patch method, and 5 patients (12.5%) had leak following omental patch. Kuremu [63] in Kenya found that 83.3% of patients with perforated duodenal ulcer were managed

by simple closure with omental patch. A similar study by Lawal *et al*[64] in Nigeria found that 93% of patients were treated by simple closure.

Therefore the recommended treatment for perforated duodenal ulcers, include omental patch closure, antisecretory medications and antibiotics to eradicate H. pylori infection.[17, 64,65,66,67]

2.2.2 Small bowel perforation.

Intestinal perforations are common causes of generalized peritonitis, it is often severe because of fecal contamination and overwhelming sepsis, resulting in high morbidity and mortality. In the study by Adesunkanmi *et al*[55] patients with acute generalized peritonitis from intestinal perforation were prospectively studied, it was found out that, males were affected more, male: female ratio 3:1. The overall mean age was 27.6 years. Typhoid perforation occurred in 58%, acute appendicitis in 16.3%, peptic ulcer disease in 14%, trauma in 8%, 5 were idiopathic. Wound infection occurred in 58.3%, and mortality was 18.8%, being high in traumatic and typhoid intestinal perforation.

The most significant poor prognostic factor is late presentation, which prolongs perforation-surgery interval and other complications, and mortality indices are directly influenced by it.

Small bowel perforation is a common emergency, having an extremely high mortality. However, this has been reduced over years with a better understanding and management of shock and septicemia, improved surgical technique and the use of appropriate antibiotics. In spite of all these measures, the mortality still remains between 10 and 36.5 %.[68]

Dickson *et al* [69] recorded 50% mortality, in a study of 38 cases. Mehendale *et al* [70] reported 31.5% mortality in a study of 32 cases, whereas Nadkarni *et al* [71] found a mortality of only 12.57%.

The toxemia after bowel perforation is probably due to the absorption of toxins by the lymphatics and the capillaries which form a rich network in the peritoneum. Though the peritoneal fluid has a large number of mononuclear cells providing an excellent barrier against infective processes, the latter crumbles against the onslaught of the toxic material pouring out of the perforation.

Various surgical techniques have been tried to reduce the high mortality resulting from bowel perforations, but have not significantly altered the mortality rate[72]. The cause of death in the majority of such patients was toxaemia. In our environment, the causes of small bowel perforation

include; typhoid ileal perforation; tuberculosis of the abdomen and trauma to the abdomen.

2.2.2.1 Typhoid perforations.

Typhoid fever is still a major health problem through out the developing world. One of its major surgical complications is ileal perforation.[73, 74, 75, 76]

The most lethal complications of typhoid fever are bleeding and ileal perforation, both arising from necrosis of Peye's patches in the terminal ileum. These undergo swelling and ulceration that can progress to capillary thrombosis and subsequent necrosis. These ulcerations are always located on the anti mesenteric border of the intestine and may perforate, usually in the third week of disease. An increase in titer of agglutinins against the somatic (O), and flagella (H) antigens of S.typhi occurs. (Basis for Widal test).

Perforation has been reported to occur in 0.9% to 39% of cases[73, 74] with wide variation often depending on geographical location.[75, 76] Butler reviewed 15980 cases of typhoid in world's literature and reported an overall 2.8% perforation rate.[76]

Typhoid perforation is common in the second and third decade of life. The high percentage of cases (40.5%) amongst age group of 21-30 reported by Beniwal *et al*[77] is similar to that reported by Vyas[78] and Singh and Kohli. [79]

Typhoid perforation usually occurs in the second and third week of fever. Beniwal *et al* [77] reported the maximum incidence in the second week of fever followed by those in the first week. Dickson and Cole[69], Olurin *et al*[80] and Purohit[81], reported that the majority of perforations occurred in the first week of fever, and Eggleston and Santoshil reported 33% in the second week of fever.[84]

In a review of typhoid perforations, Adesunkanmi ABK, *et al*[82] commented that generalized peritonitis from typhoid ileal perforation is a common cause of surgical emergency in developing countries, associated with high morbidity and mortality, affecting mostly young people. In the study, the mean age was 23.6, morbidity rate ranged from 8.8-71.3%, and mortality 17.5%. Edino ST[83] commented that typhoid perforation was the most important surgical complication of typhoid enteritis and was associated with high morbidity and mortality. Among the 47 patients, 12.8% perforated in the first week, 61.7% perforated in the second week and 25.5% in the third week of illness and the prevalence was found to be 87.2% in northwestern Nigeria.

The pre-operative diagnosis of perforation is based on findings of peritonitis in a patient with history of prolonged febrile illness. The intra-operative finding of perforation on the anti-mesenteric border of the terminal ileum is so characteristic that the diagnosis can be based on this alone[84, 85]

The treatment of typhoid perforation remain somewhat controversial.[86] Surgery for treatment of typhoid perforation began in the late 1800s[87]. After the work of Finney[88] and Cushing[89], surgery became the standard treatment, with mortality rate ranging from 60% to 80%[90]. The surgical management of typhoid perforations includes freshening of perforation margins, closure of the perforation in the direction of peristalitic wave to avoid sternosis of the lumen. Thorough peritoneal lavage is normally done with warm normal saline. Closed rubber suction is inserted in the paracolic gutters for drainage. Antibiotics to which Salmonella typhi is sensitive are administered, such as ciprofloxacin or a cephalosporin, for at least 14 days for complete eradication of the bacteria. Intravenous fluids are administered to enhance proper hydration.

The introduction of chloramphenicol greatly improved the outcome of non-perforated typhoid fever. A wave of enthusiasm for the treatment of typhoid

perforation with chloramphenicol and supportive care without surgery occurred in the early 1960s.[91] Despite initial reports of mortality rates of 20% to 30% with such conservative care, the weight of evidence over the last 25 years has been in favor of surgery, supplemented with chloramphenicol.[73,75,76]

Spontaneous sealing of the perforation rarely occurs[74, 84]. The resulting spillage causes gross fecal contamination thereby decreasing survival[84]. The inability of chloramphenicol to sterilize the bowel contents even when given before the perforation, leads to a continued source of sepsis unless surgical closure of the perforation is undertaken.[74, 84]

Enteric perforation is best managed surgically as it prevents further peritoneal contamination by intestinal contents. After proper peritoneal toilet, correct management of perforation should be done. A wide variety of operative procedures have been tried in enteric perforation cases but all have a high mortality and morbidity. In a review of 2245 cases of typhoid perforation reported in the world's literature, Butler *et al*[76] found the overall mortality rate to be 70% with medical treatment and 26% with surgery. Mortality rates ranging from 20% to 40% are most commonly reported for typhoid ileal perforations[73, 74].

Rates as low as 3% to 9% have been reported from areas in the developing world with better economic conditions including Mexico,[92] South Korea,[85] South Africa,[93] and Hong Kong. [94] Such mortality rates have been achieved by the addition of close electrolytes and blood gas monitoring, intensive care unit (ICU) nursing, central venous pressure (CVP) monitoring and use of total parenteral nutrition.[85, 86, 92, 93] Most of these measures are beyond the reach of the majority of hospitals in the developing world, especially in rural areas. Variables that can be manipulated to improve survival in such locations include more aggressive fluid and electrolyte resuscitation, the type of surgical procedure and the antibiotic regimen.[73, 74]

Repair of perforation should be the choice of treatment in enteric perforation, because this is a simple, quick and cost effective procedure. Ileostomy is more expensive as all patients have to undergo re-operation for the closure of ileostomy, and it further needs specialized care prior to closure. A variety of surgical procedures have been recommended for the treatment of typhoid perforations. In moribund patients, simple drainage of the peritoneum, under local anaesthesia may be all that is feasible.[85, 87] The most definitive treatment for non-moribund patients has been debridement and simple transverse closure of the perforation in either one, [86] or two layers.[74] More aggressive treatment in the form of wedge resection [95] or small bowel resection[85] has been recommended.

2.2.2.2 Traumatic bowel perforation.

Most cases of small bowel perforation after blunt trauma occur as a result of motor vehicle accidents and fall from heights, and are often associated with multiple injuries.[96] In such cases, the mechanism of injury is usually mesenteric laceration due to direct compression, or small bowel rupture due to deceleration injury. This would typically affect fixed segments such as the duodenum, duodeno-jejunal flexure,[97] proximal jejunum and terminal ileum.[96] Isolated rupture of the mid-small bowel occurs due to application of a localized blunt force to the central abdomen, which results in jejunal transection, presumably by direct compression against the lumbar spine.[96]

Small bowel rupture after a soccer injury is similarly extremely rare, and there has been only one previous report of traumatic jejunal rupture by this mechanism.[98]

Owing to the rarity of small bowel injury after blunt abdominal trauma, absence of peritoneal signs and insensitivity of radiological imaging, the diagnosis of small bowel perforation is often delayed and is associated with marked morbidity and mortality.[99] Therefore, the importance of obtaining a thorough history, paying particular attention to the nature and velocity of the projectile, the mechanism of injury and the exact anatomical site of the applied force is emphasized.[99]

Patients sustaining apparently minor injuries to the central abdomen may be at particular risk of small bowel perforation.[96] Such patients should be reviewed at frequent intervals by an experienced clinician and should undergo prompt surgical intervention at the earliest sign of clinical deterioration, such as pyrexia, tachycardia and guarding.

After elective abdominal operations for noninfectious etiologies, the incidence of secondary peritonitis (SP) caused by anastomotic disruption, breakdown of enterotomy closures, or inadvertent bowel injury, should be less than 2%.[100,101,102] Operations for inflammatory disease (i.e., appendicitis, diverticulitis, cholecystitis) without perforation carry a risk of less than 10% for the development of SP and peritoneal abscess. This risk may rise to greater than 50% in gangrenous bowel disease and visceral perforation.[101, 102]

Regarding operations for penetrating abdominal trauma, SP and abscess formation is observed in 1% (after operations for stab wounds) to 6% (after operations for gunshot wounds).[103] Duodenal and pancreatic involvement, as well as colon perforation, gross peritoneal contamination, and perioperative shock are factors that increase the risk of infection in these cases.[103] Peritoneal abscess describes the formation of an infected fluid collection encapsulated by fibrinous exudate, omentum, and/or adjacent visceral

organs. The overwhelming majority of abscesses occur subsequent to SP.[104] Approximately half of patients develop a simple abscess without localization, and the other half develop complex abscesses secondary to fibrinous septation and organization of the abscess material [104] Abscess formation occurs most frequently in the sub hepatic area, the pelvic cavity and the paracolic gutters, but it may also occur in the perisplenic area, the lesser sac, and between small bowel loops and their mesentery. [104] In general, the incidence of abscess formation after abdominal surgery is less than 1-2%, even when the operation is performed for an acute inflammatory process.[105] This incidence increases with preoperative perforation of the hollow viscus, significant fecal contamination of the peritoneal cavity, bowel ischemia, and delayed diagnosis. In these instances, the risk of abscess formation may be as high as 10-30%.[105]

2.2.2.3 Chemical peritonitis

Chemical (sterile) peritonitis may be caused by irritant substances such as bile, blood, barium and other substances or by transmural inflammatory processes of visceral organs (e.g., Crohn disease) without bacterial inoculation of the peritoneal cavity. Clinical signs and symptoms are indistinguishable from those of SP or peritoneal abscess, and the diagnostic and therapeutic approach is the same.

2.2.3 Bowel strangulation.

Intestinal obstruction requires emergency surgery, and early recognition is often life-saving since delay in treatment is an independent predictive factor of mortality. In addition, bowel strangulation is an independent predictor of complication and, even more, of mortality while the mortality rates of patients with strangulated obstruction are two to 10 times higher than those of patients with non-strangulated obstruction. [106,107,108,109,110,111,112,113,114] Moreover, accurate early recognition of intestinal strangulation in patients with mechanical bowel obstruction is important to allow safe nonoperative management of carefully selected patients. [115,116,117,118] Traditionally, such recognition is based on the presence of one or more of the classical signs: vascular compromise, continuous abdominal pain, fever, tachycardia, peritoneal signs on physical examination, leukocytosis, and metabolic acidosis.[117,118] Close and careful clinical evaluation, in conjunction with laboratory and radiologic studies, is essential for the decision of proper management of patients with acute mechanical bowel obstruction; if any uncertainty exists, prompt operative intervention is indicated.[115] It should be emphasized, though, that great caution should be taken for the management of these patients since studies have shown that preoperative diagnosis of bowel strangulation cannot be made or excluded reliably by any known

clinical, laboratory, or radiologic parameter, combinations of parameters, or by experienced clinical judgement.[119]

2.3 Diagnosis of peritonitis.

The diagnosis of peritonitis is usually clinical. Essentially, all patients present with some degree of abdominal pain. This pain may be acute or more insidious in onset. Initially, the pain is often dull and poorly localized (visceral peritoneum) and then progresses to steady, severe, and more localized pain (parietal peritoneum).

If the infectious process is not contained, the pain becomes diffuse. In certain disease entities (e.g., gastric perforation, severe acute pancreatitis, intestinal ischemia), the abdominal pain may be generalized from the beginning. Anorexia and nausea are frequently present and may precede the development of abdominal pain for some time.

Vomiting may occur because of the underlying visceral organ pathology (i.e. obstruction) or secondary to the peritoneal irritation. On physical examination, patients with peritonitis most often appear unwell and in acute distress. Fever with temperatures that can exceed 38°C is usually present, however, patients with severe sepsis may present with hypothermia. Tachycardia is caused by the release of inflammatory mediators and intravascular hypovolemia caused by anorexia and vomiting, fever, and third-space losses into the peritoneal cavity.

With progressive dehydration, patients may become hypotensive, and may demonstrate decreased urine output. Severe peritonitis may present in overt septic shock. Nearly all patients demonstrate increased abdominal wall rigidity. The increase in abdominal wall muscular tone may be voluntary in response to or in anticipation of the abdominal examination or involuntary because of the peritoneal irritation. Patients with severe peritonitis often avoid all motion and keep their hips flexed to relieve the abdominal wall tension. The abdomen is often distended, with hypoactive-to-absent bowel sounds. This finding reflects a generalized ileus and may not be present if the infection is well localized. Occasionally, the abdominal examination reveals an inflammatory mass.[2]

Rectal examination often elicits increased abdominal pain, particularly with inflammation of the pelvic organs but rarely indicates a specific diagnosis. A tender inflammatory mass toward the right may indicate appendicitis, and anterior fullness and fluctuation may indicate a *cul de sac* abscess

2.4 Treatment.

Current treatment of peritonitis and peritoneal abscesses consists of a multimodality approach directed at correction of the underlying cause, administration of systemic antibiotics and supportive therapy to prevent or limit secondary complications due to organ system failure.[5]

COLLEGE LIBRARY
MUHIMBILI UNIVERSITY
COLLEGE OF HEALTH SIENCIES

The general principles guiding the treatment of intra-abdominal infections are 4-fold:

(1) to control the infectious source,

(2) irrigation of the peritoneal cavity

(3) to maintain organ system function, and

(4) to control the inflammatory process.

Medical support includes;

(1) Systemic antibiotic therapy;

(2) Intensive care with hemodynamic and pulmonary support;

(3) Nutrition and metabolic support; and

(4) Inflammatory response modulation therapy

Treatment of peritonitis and intra-abdominal sepsis always begins with volume resuscitation, correction of potential electrolyte and coagulation abnormalities, and empiric broad-spectrum parenteral antibiotic coverage.

Surgery remains an important therapeutic modality for all cases of peritoneal infection. Any operation should address the first 2 principles of the treatment of intra-peritoneal infections: early and definitive source

control and purging of bacteria and toxins from the abdominal cavity. The issue of timing and adequacy of surgical source control is paramount because an improper, untimely, or incorrect operation may have an overwhelmingly negative effect on outcome.

The technique to repair the tear/perforations is different in different pathologies and includes;

a) PUD use of 3-4 sutures preferably vicryl or silk, interrupted with a vascularised omental patch.
b) bowel tear, use of 3-4 vicryl or silk, interrupted, sutured in a way to avoid stenosis, or narrowing of the lumen.
c) Appendectomy.
d) Drainage of abscess and use of closed suction rubber drainage.

In all operations, thorough peritoneal lavage using warm saline is recommended.

Broad-spectrum antibiotics (e.g. cephalosporin and metronidazole) are advised up to five days post operatively to control infection. Antisecretory medications are advocated in perforated PUD. Intravenous infusion therapy is used to restore hydration.

3.0 PROBLEM STATEMENT.

Despite all efforts made in improving intensive care and surgery, Generalized Peritonitis remains a challenging condition that is associated with a high mortality rate.[120]

Severe abdominal infections are accompanied with a high level of endotoxin production, resulting in the so-called systemic inflammatory response syndrome (SIRS), which is often complicated by multiple organ failure [120] The surgical eradication of the infectious focus is the most important prerequisite for a successful treatment. According to the severity of the local inflammation, different forms of abdominal lavage can be applied. [120]

Severe morbidity and high mortality is especially experienced in developing countries.[82] Some of the risk factors are attributed to low socio-economic status, [55, 82] poor health facilities, disorganized social infrastructure and unavailable health Insurance programs.

4.0 RATIONALE OF THE STUDY.

Up to one third of patients still die of generalized peritonitis, [121] despite improved diagnostic modalities, potent antibiotics, modern intensive care, and aggressive surgical treatment,

Peritonitis in African communities is mainly in the young age group[82,83] This is a group that is actively involved in social productivity. Also this condition afflicts communities who live in slums where sanitary conditions and housing infrastructures are poor.

The pattern causes and outcome of generalized peritonitis are not well documented in our region. Therefore this study provides a database on the profile of this fatal condition. The knowledge acquired will aid setting up strategies necessary for prevention and early intervention.

Also this study was done as a partial fulfillment of the requirement for the degree of Masters of Medicine in Surgery, University of Dar es salaam.

5.0 RESEARCH QUESTIONS.

1. What is the pattern of generalized peritonitis at MNH?
2. What are the causes of generalized peritonitis at MNH?
3. What is the treatment outcome of generalized peritonitis at MNH?

6.0 OBJECTIVES.

6.1 Main objective

To describe the pattern, causes and outcome of generalized peritonitis in adult surgical wards at MNH.

6.2 Specific objectives:

1. To describe the pattern of peritonitis according to age and sex in adult surgical wards at MNH
2. To describe the causes of peritonitis in adult surgical wards at MNH
3. To describe the time interval between onset of symptoms to hospital presentation and undergoing surgery at MNH
4. To describe the morbidity and mortality of peritonitis in adult surgical wards at MNH
5. To describe the socio-economic status of patients with peritonitis in adult surgical wards at MNH

7.0 METHODOLOGY:

7.1 Study area:

This was a hospital-based study, which was conducted in the adult surgical wards at Muhimbili National Hospital. MNH is one of the referral and teaching hospital in the country. It is located in the city of Dar es salaam which has a population of about 3.5 million. Apart from serving city residents as a referral centre, the hospital also serves as a National referral hospital. MNH consist among other departments, the department of surgery,

which encompasses three firms, which deal with a specific specialty. Furthermore, all firms also perform general surgical procedures.

All surgical patients are received at the casualty department where they are sorted and sent to admitting surgical wards. The patients are then clerked by an intern and then reviewed by a registrar or a resident, followed by a consultant. Patients from non-surgical wards that need surgical attention are referred accordingly.

Most of the adult surgical emergencies are performed by Residents and Registrar, who have been supervised by consultants until they are capable of working independently. They are however, required to get advise from their consultants wherever necessary.

Both public and private patients are currently treated in the hospital. However there is a Social Welfare Unit, which helps those patients who fail to pay hospital bills and those who are not capable of paying for expensive investigations such as CT-scan and ultrasound.

7.2 Study design:

This was a prospective descriptive study of patients who presented with generalized peritonitis at MNH.

7.3 Study population:

All patients who were admitted in the surgical wards, with clinical features of generalized peritonitis necessitating exploratory laparotomy.

7.4 Sample size:

All patients who presented to hospital with clinical features of peritonitis during the period of the study were taken to constitute the sample size.

7.5 Patients recruitment:

Once the patient was suspected to have generalized peritonitis, a thorough history was taken and clinical evaluation of the patient was done

Basic investigation were taken including, blood for hemoglobin level. Blood grouping, and cross matching, and request of at least two units of blood for transfusion if required. Serum electrolytes were also measured,

Intravenous fluids were initiated immediately; crystalloids (lactated ringers and normal saline) were used to resuscitate the patients as most of them were severely dehydrated and clinically in shock.

Urethral catheterization was done in every patient, so as to monitor urine output, and thus the input and output chart was commenced immediately. No patient was taken to the operation theatre with inadequate urine output i.e. less than 30mls per hour. The minimum resuscitation time was within two hours. Nasal-gastric tube was inserted for decompression of the stomach, this helped to reduce the risk of aspiration during intubations. Following adequate resuscitation, the patient was taken to theatre for emergency exploratory laparotomy.

Intra-operatively, the cause of peritonitis and site of perforation if present was identified, suction of the peritoneal spillage most of which was purulent material was done, and the volume aspirated was noted. The cause of peritonitis was then taken care of surgically. Incase of a perforated peptic ulcer, simple closure with vascularized omental patch was performed. Different surgical techniques were used in ileal perforation including refreshening and closure, wedge resection and end to end anastomosis, or resection and ileo-transverse anastomosis depending on the size and number of perforations and the surgeon's preference. Appendectomy was done for perforated appendix, and bowel resection performed if it was strangulated.

Thorough peritoneal lavage using 3 to 5 litres of warm normal saline was done to neutralize the toxins and/or to purge the bacteria from the

peritoneum. Closed drainage was done in all patients to drain the paracolic, sub hepatic and pelvic spaces accordingly to avoid collection and post-operative abscess formation.

All patients received the first dose of parenteral cefriaxone and metronidazole immediately after establishing the diagnosis. The antibiotics were also continued during the post-operative period for a minimum of five days to take care of the sepsis. Ceftriaxone was given in a dosage of 1g daily and metronidazole 500mg, at eight hours interval. For patients with PUD, omeprazole, an anti-secretory medication was added. Adequate analgesics e.g., pethedine was given during the first twenty four hours post-operatively at a dosage of 100mg at a minimum of six hourly intervals if required. Vital signs i.e. blood pressure, pulse rate, temperature, and pain were evaluated.

7.6 Inclusion criteria:

All patients with an intra-operative diagnosis of purulent peritonitis, either due to peptic ulcer perforation or bowel perforation or strangulation, were included in the study.

7.7 Exclusion criteria:

Patients with acute abdominal symptoms and signs but no pus was found at laparotomy.

Children who were ten years and below, who were admitted in pediatric surgery unit.

7.8 Ethical clearance:

The study was ethically cleared by MUHAS research and publication committee.

The patients were explained about the study and were requested to participate. All consenting patients were recruited into the study. However no patient was denied of appropriate and adequate treatment upon not consenting.

The information gathered about the patient was kept confidential and could only be available to interested parties only with permission of the investigator and the patient.

7.9 Data collection:

Data were filled in a structured questionnaire by the investigator from the patients who had consented for the study. Information on socio-demographic characteristics that included age, sex, marital status, employment status was obtained by interviewing the patients, as well as

obtained from the patients' files. The average duration of illness i.e. from onset of symptoms to admission to hospital was inquired. The cause of generalized peritonitis and type of surgical repair performed was obtained from the surgical notes in the file, and incase of inadequate information, efforts were made to consult the surgeon who performed the operation for further clarification.

Follow up.

All patients were followed up during the post-operative period for development of any complication such as wound sepsis, burst abdomen, fistula formation, length of hospitalization, and these data were recorded. Post operative hospital deaths were recorded and possible cause of death established.

Patients who were discharged within seven days post operatively were considered to have recovered uneventfully. They were followed up for thirty days in the outpatient clinic for any late complications. Patients who discharged following uneventful recovery and were not seen in the clinic within the one month follow-up period were regarded completely cured.

8.0 Data analysis:

Data collected were entered into statistical package of social sciences (SPSS) version 11.0. It was then analyzed to determine the distribution of peritonitis according to age and sex, causes, and the outcome of peritonitis.

10.0 RESULTS

A total of 124 patients presented with generalized peritonitis during the study period of 10 months, from March 2006-December 2006.

All of these patients were recruited to the study and were operated on an emergency basis.

The mean age was 32.3, range 11-82 years

Males constituted the majority (80.6%), male to female ratio was 5:1. The age group between 21 and 40 years was the most affected constituting 58.1 %. Table 1, illustrates these results.

Table 1;

Pattern of peritonitis according to age and sex

		sex		
		male	**female**	**Total**
Age group in years	**11-20**	20 (16.1%)	3 (2.4%)	23 (18.5%)
	21-30	36 (29.0%)	12 (9.7%)	48 (38.7%)
	31-40	19 (15.3%)	5 (4.0%)	24 (19.4%)
	>40	25 (20.2%)	4 (3.2%)	29 (23.4%)
	Total	100 (80.6%	24 (19.4%)	124 (100.0%)

Gastro duodenal perforation was the most common cause of generalized peritonitis (33.1%), peptic acid ulcer disease being the absolute cause of these perforations.

Small bowel perforation constituted 31.5% in which typhoid ileal perforation was the commonest cause, comprising 59% of all small bowel perforation, followed by blunt trauma to the abdomen due to motor traffic accidents and kicks to the abdomen by blunt objects (28.2%) Tuberculosis of the abdomen comprised 12.8%.

However typhoid ileal perforation constituted 18.5% overall causes of peritonitis, trauma 8.9% and tuberculosis 4%. Tables 2& 3 summarize this information.

Appendicular perforation accounted for 19.4%, strangulation and/or perforation of bowel, 11.3%, and others 4.8%. (Table 2)

Table 2;

Causes of peritonitis

causes	Frequency(n=124)	Percent (%)
Gastro duodenal perforations	41	33.1
Typhoid perforation	23	18.5
Abdominal trauma	11	8.9
TB abdomen	5	4.0
Perforated appendix	24	19.4
large bowel perforation/strangulation	14	11.3
others	6	4.8
Total	124	100.0

Table 3

Causes of small bowel perforation

Cause	Frequency(n=39)	Percent (%)
Typhoid perforation	23	59.0
Abdominal trauma	11	28.2
TB abdomen	5	12.8
Total	39	100.0

Appendicular perforation was much more common in age group 11- 20 years (8.1%), while gastro duodenal and small bowel perforation were in the upper age groups (32.3% and 24.2% respectively) as seen in Table 4.

Table 4:

Causes of peritonitis according to age (years)

		Cause of peritonitis					
		Gastro-duodenal perforation	Small bowel perforation	Perforated appendix	large bowel perforation	others	Total
Age group in years	11-20	1 (.8%)	9 (7.3%)	10 (8.1%)	3 (2.4%)	-	23 (18.5%)
	21-30	17 (13.7%)	18 (14.5%)	8 (6.5%)	3 (2.4%)	2 (1.6%)	48 (38.7%)
	31-40	10 (8.1%)	7 (5.6%)	1 (.8%)	4 (3.2%)	2 (1.6%)	24 (19.4%)
	>40	13 (10.5%)	5 (4.0%)	5 (4.0%)	4 (3.2%)	2 (1.6%)	29 (23.4%)
	Total	41 (33.1%	39 (31.5%)	24 (19.4%)	14 (11.3%)	6 (4.8%)	124 (100.0%)

The time taken by the patients between onset of symptoms and presentation to hospital was less than 72 hours in 37 (29.8%) cases, and 72 hours or more in 87 (70.2%) cases. (Table 5).

Table 5:

Time interval (hrs) from onset of symptoms to report to hospital

		Frequency(n=124	Percent (%)
Time interval	**<72 hrs**	37	29.8
	72+ hrs	87	70.2
	Total	124	100.0

Forty nine out of 124 patients developed post operative complications, giving a morbidity of 39.6%. Prolonged hospitalization being the leading cause of morbidity. (Table 6). The mean hospital stay was 11.1 days. In the majority of patients especially with peritonitis due to perforation,

contamination was either purulent or fecal, and this could be contributory to high morbidity and mortality.

The overall mortality rate was 21.8% (Table 6) with septicemia associated with Multiple Organ Systemic Failure (MOSF) being the most common cause.

Table 6:

Outcome of peritonitis

	Frequency(n=124)	**Percent (%)**
recovered well	48	38.7
wound sepsis	8	6.5
long hospitalization	31	25.0
Anastomotic leakage	10	8.1
Death	27	21.8
Total	124	100.0

The outcome of peritonitis in this study was greatly influenced by late presentation to hospital. Table 7 summarizes this information.

Table 7:

Time interval (hrs) between onset of symptoms to hospital presentation and outcome of peritonitis

		Outcome of peritonitis					
		recovered well	**wound sepsis**	**long hospital stay**	**anastomotic leakage**	**died**	**total**
time interval (hrs)	**<72 hrs**	18 (14.5%)	4 (3.2%)	10 (8.1%)	1 (0.8%)	4 (3.2%)	37 (29.8%)
	72+ hrs	30 (24.2%)	4 (3.2%)	21 (16.9%)	9 (7.3%)	23 (18.5%)	87 (70.2%)
	Total	48 (38.7%)	8 (6.5%)	31 (25.0%)	10 (8.1%)	27 (21.8%)	124 (100.0%)

Thirty eight (30.6%) out of 124 cases were unemployed while 48.4% had temporally jobs (table 8). Thus majority of our patients belonged to lower socio-economic status class, who probably are unable to pay for their medical expenses.

Table 8:

Occupation

Employment status	Frequency(n=124)	Percent (%)
Employed	26	21.0
unemployed	38	30.6
temporary jobs	60	48.4
Total	124	100.0

11.0 Discussion:

Generalized peritonitis is a frequently encountered surgical emergency in tropical countries like Tanzania, mostly affecting young people in their prime of life, a fact that has been seen in this study.

Lawal OO *et al* [64] found that three quarters of the affected were working class young men. The high morbidity and 20% mortality observed were attributed to late presentation with presence of advanced bacterial peritonitis at admission.

In the majority of our cases, the presentation to hospital was late with well established generalized peritonitis with fecal or purulent contamination, and varying degree of septicemia. 70.2% of the patients presented to the hospital more than 72 hours after onset of symptoms (table 5). The most possible explanation could be due to poor socio-economic status of our patients (table 8), and inefficient referral system in which the patient present first to the health centre then district hospital, and finally to the referral hospital, usually in a moribund situation due to the delay. Also these patients are not able to pay for the cost sharing hospital expenses, which may delay appropriate treatment.

The time taken from resuscitation to preparation of the patient for emergency operation was between 2 hours to 72 hours. Some delay may

have been due to major renovation taking place at the hospital. However, considerable delay occurred getting to the hospital either from home or referred from a peripheral hospital. Sometime resuscitation took long as some of the patient arrived in hypovolemic or septicemia shock with impending acute renal failure

The decision for a laparotomy was mainly clinical sometimes supplemented by investigations. Late presentation, delay in operation (>48 hours), multiple perforations and drainage of copious quantities of pus and fecal material from the peritoneal cavity, contributed to increased incidence of fecal fistula and subsequent mortality[7].

The etiological factors show a wide geographical variation. Khana *et al* [122] from India studied 204 consecutive cases of gastrointestinal perforation and found that, over half (108) cases were due to typhoid. They also had perforations due to duodenal ulcer (58), appendix (9), amoebiasis (8), and tuberculosis (4). These figures show the importance of infection and infestation in the third world countries, where sanitary measures and housing constructions are very poor. This is in accordance with this study in which gastro-duodenal perforation accounted for 33.1% followed closely by small bowel perforation 31.5%. Typhoid accounted for 59% of small bowel perforations and 18.5% in terms of overall causes of peritonitis.

Tuberculosis was found in five cases constituting 12.8% of small bowel perforation and 4.0% as overall cause of peritonitis.

Adesunkanmi AR *et al*[123] in reflecting the importance of infection, found out that ileal perforation constituted 38%, perforated appendix 17.3%, and peptic ulcer perforation 9%. Traumatic bowel perforation accounted for 5.6% and the wound infection rate was 42.6%. This trend is slightly different from this study, however, the importance of infection is nevertheless emphasized.

On the other end of the spectrum, Noon *et all* [124] from Texas studied 430 patients with gastrointestinal perforation and found 210 cases to be due to penetrating trauma, 92 to perforated appendix and 68 due to peptic ulcer disease. The study reveals the importance of trauma as a cause of bowel perforation in the developed world. However the increased incidence of road traffic accidents in our environment mainly due to high speed as well as blunt abdominal trauma due to social violence, accounted for the traumatic perforation cases seen in this study. Trauma accounted for 28.2% of small bowel perforation and 8.9% as overall cause of peritonitis.

The high mortality (21.8%) seen in this study is comparable to other studies. Mehendale *et al* [70] reported a mortality rate of 31.5%, Rajender S.J *et al* [125]

found mortality to be 10% and attributed it to delay in seeking medical treatment.

Most of the patients affected were young people in the working class age group, and many belonged to the low socio-economic status group as 48.4% were having non specific temporal jobs and 30.6% were unemployed. Only 21% were employed. City congestion associated with poverty with limited or poor toilet systems contributing to poor sanitation and hygiene may be factors leading to ill-health and perforation. This is comparable to a study by Ugwu *et al*[126] in which 78% of patients with peritonitis belonged to low socio-economic strata.

The most significant poor prognostic factor was late presentation and other complications and mortality were directly influenced by it. In this study 79% of patients belonged to low socio-economic group, and morbidity was 39.6% and mean hospital stay of 11.1 days.

Gastro duodenal perforations:

Perforation of peptic ulcers formed the major group among the gastro duodenal perforations. These perforations are usually encountered along the first part of the duodenum anteriorly and in the pylorus of the stomach. The advances in the medical treatment for peptic ulcer disease have led to a dramatic decrease in the number of elective surgeries performed. However,

the number of patients undergoing surgical intervention for complications such as perforations remains relatively unchanged or probably has increased[33, 64].

Although the surgical options are many from simple closure to definitive acid reducing procedures, it has been experienced that simple closure of the perforation using a pedicled omental patch, gives good results even in large perforations up to 3 centimeters in diameter. [62] This was the modality of treatment which was used in this study for all the gastro-duodenal perforations, and it was found to be an appropriate and easy technique for registrars and residents who usually perform the emergency surgery.

The mortality rate for these perforations varies from 4-11% [60, 62] and the prognoses is poorer in the elderly, those with concomitant disease, were in peri operative shock, have larger size perforation, delayed presentation and delayed operation. [127]

Ersumo T *et al*[128] in his study found out that perforated peptic ulcer accounted for 3.4% of the adult emergency surgical procedures. In nearly 22% of patients, no previous history of peptic ulcer disease was present. Delay in diagnosis was noted in 95% of the cases, with 78% of patients were found to have purulent peritonitis at laparotomy. The mortality rate was

18.9%, equivalent to this study in which a tendency to late presentation was also seen.

Small bowel perforations:

The next commonest type of perforations encountered in this study, were those arising in the small intestines. Typhoid was the commonest cause (59%).

Typhoid fever is a predominant cause of non traumatic perforation in developing counties.[72] Typhoid fever, a severe febrile infectious disease caused primarily by Salmonella typhi, occurs in areas where poor socio-economic levels and unsanitary environmental conditions prevail. This was evident in this study where most of the patients (79%) belonged to low socio-economic strata. After ingesting contaminated water or food, the infection leads to bacteremia, high grade fever, and signs of systemic sepsis with characteristic normal or low blood counts and anemia. [129]

The gut in typhoid fever is edematous and friable especially in the terminal 60 centimeters of the ileum as was seen in our series of patients.

Aggressive resuscitation, antibiotics and early surgery has reduced the mortality rate and complications in this subset of small bowel perforations to less than 10%. [130] One of the most severe complications of typhoid enteritis is perforation of ileal ulcerations. The typically high mortality rates from these perforations are in part due to extremely limited supportive care in hospitals in typhoid endemic areas. In the setting of rural African hospitals, Mock C *et al* [130] demonstrated a decrease in overall mortality rate from 40% using one layer closure with chloramphenicol alone to 19% using two layer closure with a combination of chloramphenicol, gentamicin and metronidazole. There was also a decrease in overall mortality rate from 43% using less than 10 ml/kg of intra operative fluid administration to 14% when using more than 10 ml/kg. Therefore even with constraints of the rural developing world, more aggressive initial fluid resuscitation and use of broad spectrum antibiotics can decrease mortality. [130]

Surgical management of intestinal perforation remains a challenge. It is the teaching that since these patients have bowel edema that precludes any suturing, exteriorization of the perforation as a loop ileostomy is the safest and fastest procedure to be done. Closure of this loop ileostomy is performed electively after 6-8 weeks. A primary anastomosis (simple

closure) is to be considered only when the patient presents early and the bowel is healthy. Unfortunately no matter what procedure is used, post operative mortality and morbidity remains high, the most catastrophic complications being fecal fistulae and wound dehiscence

This is contrary to what is experienced at MNH, whereby the commonly performed operation is simple closure, resection and anastomosis, or end to side ileo-transverse anastomosis. However, these procedures have been complicated by high failure rate in which re-perforation and high out put fecal fistulas predominate. Probably an ileostomy would have been a better procedure, but its maintenance in our developing country and the need for second operation is a dilemma.

Typhoid enteritis with perforation is still a major problem in many hospital centres in the tropics and the post operative intestinal fistula accounts for much of the morbidity and mortality among those who survive the septicemic phase of the disease.[130] In poorly equipped hospitals with limited resources, early aggressive re-exploration to close the intestinal fistula when the patient is still clinically stable, is advocated in preference to conservative treatment which is prolonged, of uncertain outcome and liable to be complicated by the patient's financial constraints.

Small bowel perforations due to intestinal tuberculosis was another cause. Most of these (50-80%) occur in the ileum usually proximal to stricture of the bowel, [131] as it was in this study in which all of the tuberculous perforations were at the terminal ileum. The mortality rate reported in these tubercular perforations is very high, up to 70%. [132]

The diagnosis of perforated tubercular enteritis is usually not one that is made pre-operatively, due to the non specific signs and symptoms and absence of radiological evidence of tuberculosis in the chest. Even in the presence of tuberculous lesion in the chest, the diagnosis of TB enteritis is not entertained until the histology and culture of the biopsied tissue turns out to be positive.[133]

Mycobacterium tuberculosis is responsible for most of abdominal tuberculosis. Primary abdominal tuberculosis due to ingestion of contaminated food is very rare. [133] Secondary intestinal disease arises from swallowed sputum leading to the direct penetration of the mucosa by the organism, or during a bacteremic phase in patients with pulmonary tuberculosis, [133] in which case the peritoneum, mesenteric nodes, and the intestines may become infected along with other abdominal and pelvic organs.

The most common site of intestinal tuberculosis is the terminal ileum and ileocaecal junction. Other sites in order of incidence are the ascending colon, jejunum, appendix, duodenum, stomach and esophagus.[133, 134] This is in accordance with the observation done in the current series in which all the perforations were in the terminal ileum.

Patients may also present with abdominal pain, diarrhea, and blood in stool along with symptoms of pulmonary tuberculosis. The most common symptoms are anorexia, abdominal pain and weight loss. [134] A mass may be palpable in the right iliac fossa. Patients are usually anemic with normal white cell count. Complications include obstruction, perforation, hemorrhage and fistula formation.[131, 133] Diagnosis is aided by keeping such possibilities in mind, given the appropriate risk factors and symptoms. A definite diagnosis is made by identification of acid fast bacilli in tissue culture.

All patients in this study were treated with surgical resection, and multi-drug anti tubercular medications in this case, isoniazid, pyrazinamide, ethambutol and rifampicin were initiated on clinical basis, according to the National TB guidelines.

The other cause of small bowel perforation is blunt abdominal trauma mostly from motor traffic accidents, and they go undiagnosed until generalized peritonitis sets in.

Appendicular and colorectal perforations

Perforated appendix and large bowel perforations were among the causes of generalized peritonitis in this study. Appendicular perforation was found in higher frequency in the young age group. This compares with other studies in which peritonitis due to perforated appendix have been found mainly in the young age group.[135, 136]

Early surgical intervention with appendectomy before perforation is the ideal treatment. Even if the detailed symptoms and signs of acute appendicitis make it possible to operate before perforation, it is known that young children still have disproportionately high perforation rate due to, (i) low incidence, (ii) thin appendiceal wall, and (iii) vague or non specific clinical history. Since perforation of the appendix in children is not uncommon, a high index of suspicion is helpful. If a child has persistent spiking fever, vomiting and diarrhea, the probability of perforated appendix must always be kept in mind, particularly when the abdomen becomes tender and gradually distended.[136]

Current evidence suggests that spontaneous resolution of untreated, non-perforated appendicitis is common and if perforation had to occur, it could rarely be prevented. It is also associated with less or decrease in mortality than it was previously thought. This motivates a shift in focus from the prevention of perforation to detection and treatment of appendicitis.

12.0 Conclusions:

Generalized peritonitis remains a common and significant surgical problem at MNH, and in the developing world at large.

Majority of patients belonged to poor socio-economic strata in which 79.4% of them were either unemployed, or were casual laborers. They also live in unsanitary environment, with poor housing system which also explains the importance of the burden of infection and infestation in the third world communities.

The most affected group was the young individuals in the prime of their life. This is the working class group who is mostly involved in social productivity, and thus has an impact on the economy of the society.

Peritonitis is usually associated with high morbidity and mortality. In this study morbidity and mortality was 39.6% and 21.8% respectively. This situation was attributed to late presentation to hospital 70.2%, there by prolonging onset of illness to surgery interval, resulting into moribund patients with purulent or fecal contamination and severe septicemia.

Advanced management such as close electrolyte and blood gas monitoring, central venous pressure monitoring and use of total parenteral nutrition are not available in most hospitals in the developing world.

Parameters such as aggressive fluid and electrolyte resuscitation, type of surgical procedures and use of appropriate antibiotics should be emphasized in order to improve survival.

Limitations of the study:

The amount of funding T sh 600,000/= allocated for research is not sufficient to cover high laboratory costs. It would have also been appropriate to do bacteriological studies, but funds were inadequate. Also antibiotics like ceftriaxone, metronidazole, ciprofloxacin and omeprazole as an antisecretory could have been given to patients in the study preferably free of charge to ensure adequate recovery and minimize ulcer recurrences.

13.0 Recommendations:

1. Decreasing the burden of diseases is paramount to our survival. Health education on sanitary measures and environmental preservation should be emphasized to all patients, and population at large. This will help minimize risk to infections and infestations such as worms

2. The habit of seeking medical attention and advice as early as possible should be encouraged so that adverse complications of peritonitis can be reduced.

3. Hospitals should be equipped to try and improve patient care, acquire modern diagnostic and resuscitation techniques have enough trained personnel including nursing staff; the pharmacy services should be available in hospital and have adequate stock of drugs instead o relatives buying medications from outside the hospital.

REFERENCES:

1. Gnocchi C A. Intraabdominal infection and new quinolones. Medicina (B Aires) 1999; 59 suppl. 1: 47-54
2. Thomas Genuit, Lena Napolitano: Peritonitis and abdominal sepsis www.eMedicine.com.inc September 2004.
3. Steeb G, Wang YZ, Siegrist B, O'Leary JP: Infections within the peritoneal cavity: a historical perspective. Am Surg. 2000 Feb; 66(2): 98-104
4. Wolff H: On the history of surgical treatments of peritonitis. Zentralbl Chir.2002 Jan; 62-7
5. Kirschner M. Die Bahandlund der akuten eitrigen freien Bauch fellentzundung Langenb. Arch. Chir. 1926; 142: 253-267
6. Finlay-Jones J J, Kenny P A, Nulsen M F, et al. Pathogenesis of Intraabdominal abscess formation; abscess-potentiating agents and inhibition of complement-dependent opsonization of abscess inducing bacteria. J Infect Dis. 1991Dec; 164(6): 1173-9
7. Bohnem J, Boulanger M, Meakins JL, Mclean AP. Prognosis in generalized peritonitis. Relation to cause and risk factors. Arch. Surg. 1993; March 118(3)
8. Chistou NV, Barie PS, Dellinger EP, Waymack JP, Stone HH. Surgical Infection society intraabdominal infection study. Prospective evaluation of management techniques and outcome. Arch. Surg. 1993 Dec 128(2)

9. Kriger A G, Shurkalin B K, Gorskii V A, Faller A P, Andreitsev I ,Glushkov PS, et al. Results and prospects of treatment of general forms of peritonitis Khirurgii (MOSK). 2001; 8: 8-12

10. Subramanyam SG, Sunder N, Saleem KM, Kilpadi AB. Peritonitis in patients over the age of 50 years; 98 cases managed surgically. Trop. Doct. 2005 Oct; 35(4): 247-50

11. Svanes C, Soreide O. Gastric ulcer bleeding and perforation. NSAIDs or Helicobacter pylori? Scand J Gastroenterol. 1996; 31(suppl 220):128-31

12. Enders KW, Lam YH, Sung JJY. et al. Eradication of Helicobacter pylori prevents recurrence of ulcer after simple closure of duodenal ulcer perforation: a randomized controlled study. Ann Surg 2000; 231:153-8

13. Svanes C, Ovrebo K, Soreide O. Ulcer bleeding and perforation: non-steroidal anti-inflammatory drugs or H.pylori. Scand J Gastroenterol suppl. 1996:220:128-31

14. Reinback DH, Cruickshank G, McColl KE. Acute perforated duodenal ulcer is not associated with H.pylori infection. Gut .1993; 34: 1344-7

15. Horowitz J, Kukora JS, Ritchie WP. Jr. All perforated ulcers are not alike. Ann. Surg. 1989; 209: 293-6

16. Lanas A, Serrano P, Bajodar E, et al, Evidence of aspirin use in both upper and lower gastrointestinal perforation. Gastroenterology. 1997; 112: 683-9

17. Sawyers JL, Herrington JL, Mulherin JL, et al. Acute perforated duodenal

ulcer. An evaluation of surgical management. Arch. Surg. 1975:110: 527-30

18. Rodriguez LAG, Jick H. Risk of upper gastro intestinal bleeding and perforation in individuals on non steroidal anti inflammatory drugs. Lancet. 1994; 343:769-72

19. Henry D, Dobson A, Turner C. Variability in the major risk of gastrointestinal complications from non steroidal anti-inflammatory drugs. Gastroenterology. 1993; 105:1078-88

20. Smedley F, Hickish T, Taube M, Yale C, Leach R, Wastell C. Perforated duodenal ulcer and cigarette smoking. J R Soc Med. 1988; 81:92-4

21. Doll R, Peto R, Wheathly K, Gray R, Sutherland I. Mortality in relation to smoking: 40 years observation in male British doctors. BMJ. 1984; 309:901-11

22. Svanes C, Soreide JA, Skarstein A, Svanes BP, Soreide O. Smoking and ulcer perforation. Gut. 1997; 41:177-80.

23. Reinbach DH, Cruickshank G, Mc Coll KEL. Acute perforated duodenal ulcer is not associated with Helicobacter pylori infection. Gut. 1993; 34:1344-7.

24. Andersen IB, Jorgensen T, Bonnevie O, Gronback M, Sorensen TI. Smoking and alcohol intake as risk factors for bleeding and perforated peptic ulcers: a population based cohort study. Epidemiology. 2000; 11: 434-9

25. Sawyers JL, Herrington JL Jr. Perforated duodenal ulcer managed by proximal gastric vagotomy and suture placation. Ann. Surg. 1977; 185:656-60

26. Crisp E. Cases of perforation of the stomach. Lancet 1843; 1:639

27. De Bakey ME. Acute perforated gastro-duodenal ulceration. Surgery. 1940; 8:1028-30

28. Miculicz J. Ueber laparotomy bei magen und Darm perforation. Samml Klin Vort Leipzig. 1885; 262:2307-15

29. Wangensteen OH. Nonoperative treatment of localized perforations of the duodenum. Proc Minn Acad Med. 1935; 18:477-80

30. Von Haberer H. Zur therapic acuter Geshwursperforationen des Magens and duodenums in die frece Banchhohle. Weinklin Wochnsahr. 1919; 32:413-5

31. Graham RR, The treatment of perforated duodenal ulcers. Surg Gynaecol Obstet. 1937; 64:235-8

32. Jordan PH Jr, Korompai FL. Evolvement of a new treatment for perforated duodenal ulcer. Surg Gynaecol Obstet. 1976; 142:391-5

33. Jordan GL, De Bakey ME, Duncan JM. Surgical management of perforated peptic ulcer. Ann Surg. 1974; 179:628-83

34. Boey J, Branick FJ, Alaganatham TT, *et al.* Proximal gastric vagotomy: the preferred option for perforations in perforated duodenal ulcer. Ann Surg. 1988; 208:168-74.

35 Boey J, Lee NW, Koo J *et al.* Immediate definitive surgery for perforated

duodenal ulcers. Ann Surg. 1982; 196:338-44.

36. Tanphiphat C, Tanprayoon T, Na Thalang A. Surgical treatment of perforated duodenal ulcer: a prospective trial between simple closure and definitive surgery. Br J Surg. 1985; 72:370-2

37. Rauws EAJ, Tytget GNJ. Cure of duodenal ulcer associated with eradication of Helicobacter pylori. Lancet. 1990; 335:1233-5.

38. Enders KW, Lam YH, Sung JJY, *et al.* Eradication of helicobacter pylori prevents recurrence of ulcer after simple closure of duodenal ulcer perforation: randomized controlled trial. Ann Surg. 2000; 231:153-8.

39. Ashley SW, Evoy D, Daly JM. Stomach. In: Schwartz SI, Shires GT, Spencer FC *et al* editors. Principles of Surgery(7^{th} ed). New York. Mc Graw Hill. 1999:1191-7.

40. Svanes C, Lie RT, Kvale G, Svanes K, Soreide O. perforated peptic ulcer over 56 years time trends in patient characteristics. Gut. 1993; 34:1666-71.

41. Coggon D, Lambert P, Langman MJS. 20 years of hospital admissions for peptic ulcer in England and Wales. Lancet. 1981; 1:1302-4.

42. Brnner F. Das acrut in die freie Bauchhohle perforimde Magen und Duodenal Dtsch Zchir. 1903; 69:101-5

43. Hannessey E. Perforated peptic ulcer: mortality and morbidity in 603 cases. Aust NZ J Surg. 1969; 38:243-8

44. Jordan PH, Marrow C. Perforated peptic ulcer. Surg Clin North Am. 1988; 68:315-29

45. Menakuru SR. Current Management of Peptic Ulcer perforations. Pak J Med Sci. 2004; 20 (2); 157-163.

46. Berne TV, Donovan AJ. Non operative treatment of perforated duodenal ulcer. Arch Surg. 1989; 124:830-2

47. Donovan AJ, Berne TV, Donovan JA. Perforated duodenal ulcer. Arch Surg. 1998; 133:1166-71

48. Tylor H. Perforated peptic ulcer treated without operation. Lancet. 1946; 2:441-3

49. Crofts TJ, Park KGM, Steele RJC, Chung SSC, Li AKC. A randomized controlled trial of non operative treatment for perforated peptic ulcer. N Engl J. Med. 1989; 320:970-3

50. Drury JK, Mc Kay AJ, Huchison JSF, Joffe SN. Natural history of perforated duodenal ulcers treated by suture closure. Lancet. 1978; 2:749-50

51. Griffen GE, Organ CH. The natural history of perforated duodenal ulcer treated by suture placation. Ann Surg. 1976; 183:382-5

52. Bomman PC, Theodorou NA, Jeffrey PC et al. Simple closure of perforated duodenal ulcer: a prospective evaluation of a conservative management policy. Br J Surg. 1990; 77:73-5

53. Kaushik SP, Sikora SS. Perforated duodenal ulcer. Is definitive management warranted? (editorial). Indian J Gastroenterol. 1993; 12:75-6

54. Hay JM, Lacaine F, Kohlman G, Fingerhut A. immediate definitive surgery for perforated duodenal ulcer does not increase operative mortality. A prospective controlled trial. World J Surg. 1988; 12:705-8

55. Adesunkanmi ARK, Ajao OG. The prognostic factors in typhoid ileal perforation. A prospective study of 50 patients. JR Coll Surg Edinb. 1997; 42:395-399

56. Boey J, Wong J, Ong GB. A prospective study of operative risk factors in perforated duodenal ulcers. Ann Surg. 1982; 195:265-9

57. Johnston D, Martin J. Duodenal ulcer and peptic ulceration. In: Zinner MJ, Schwartz S, Ellis A. eds Maingot's abdominal operations Stanford CT: Appleton and Lange (10^{th} ed) 1997; 941-53

58. Donahue PE. Extended highly selective vagotomy. In: Wastel C, Nyhus LM, Donahue PE eds. Surgery of esophagus, stomach and small intestine. Boston Little Brown. 1995; 505-17

59. Lau WY, Leung KL, Kwong KH et al. A randomized study comparing laparoscopic versus open repair of perforated peptic ulcer using suture or sutureless technique. Ann Surg. 1996; 224:131-8

60. Siu WT, Leong HT, Lau BKB et al. laparoscopic repair for perforated duodenal ulcer: a randomized controlled trial. Ann Surg. 2002; 235:313-9

61. Sebastian M, Chandran VPP, Elasheal YM, Sim AJ. Helicobacter pylori infection in perforated peptic ulcer disease. Br J Surg. 1995; 82:360-2

62. Sanya Gupta, Robin Kaushik, Rajeev Sharma, Ashok Attri. The

management of large perforations of duodenal ulcers. BMC surgery. 2005, 5:15 doi: 10. 1186/1471-2482-5-15

63. Kuremu RT. Surgical management of peptic ulcer disease. East Afr Med J. 2002; 29: 545-6

64.. Lawal OO, Fadiran OA, Oluwole SF, Campbell B. Clinical pattern of perforated prepyloric and duodenal ulcers. Ife-Ife, Nigeria. Trop Doct. 1998; 28: 152-5

65. Minmanli M, Isgor A, Kabukcuogln F et al. The effect of H. pylori in perforation of duodenal ulcers. Hepatogastroenterology. 1998; 45: 1610-2

66. Kumar D, Sinha AN. Helicobacter pylori infection delays ulcer healing in patients operated for duodenal ulcer. Indian J Gastroenterol. 2002; 21: 19-20

67. Kauffman GL, Jr. Duodenal ulcer disease: treatment by surgery, antibiotics or both. ADV SURG 2000; 34: 121-35

68. Bapat RD, Supe AN, Sathe MJ. Management of small bowel perforation with intra- and post operative lavages with povidon iodine. Postgr Med J. 1983; 29(1):29-33

69. Dickson JA, Cole GJ. Perforations of terminal ileum-a review of 38 cases. Br J Surg. 1964; 51:893-897

70. Mahendale VG, Samsi AB. Jejunal-ileal perforations. J Postgrad Med. 1979; 25:41-47

71. Nadkarni KM, Shetly SD, Kagzi RS, Pinto AC, Bhalerao RA. Small bowel

Perforations-a study of 32 cases. Arch Surg. 1981; 116:53-57

72. Swadia ND, Trived PM, Thakkar AM. The problem of enteric ileal perforations. Ind J Surg. 1979; 41:643-651

73. Archampong E.Q. Typhoid ileal perforations: why such mortalities? Br J Surg. 1976; 63:317-115

74. Archampong E.Q. Operative treatment of typhoid perforation of the bowel. Br Med J. 1969; 3:273-276

75. Archampong E.Q. Tropical diseases of the small bowel. World J Surg. 1985; 9:887-896

76. Butler T, Knight J, Nath SK, et al. Typhoid fever complicated by intestinal perforation. Rev Infect Dis. 1985; 7:244-255

77. Beniwal US, Jindal D, Sharma J, Jain S, Shyan G. Comparative study of operative procedures in typhoid perforation. Indian J Surg. 2003; 65(2):172-177.

78. Vyas PN. Study of 15 cases of intestinal perforation in enteric fever. Indian J Surg. 1964; 26:1-8

79. Singh KP, Singh K, Kohli JS. Choice of surgical procedure in typhoid perforation: experience in 42 cases. J Indian Med Assoc. 1991; 89:255-6

80 Olurin EO, Ajari OO, Bohrer SP. Typhoid perforations. J Roy Coll Surg. Edinb. 1972; 17:353-63

81. Purohit PG. Surgical treatment of typhoid perforations: experience of 1976 Sangli epidemic. Indian J Surg. 1978; 40:227-38

82. Adesunkanmi A.R.K, Badmus Tjudeen A, Fadiora FO, Agbakwuru EA. Generalized peritonitis secondary to typhoid ileal perforation: Assessment of severity using modified APACHE II score. Indian J Surg. 2005; 67(1):29-33

83. Edino ST. Surgical abdominal emergency in northwestern Nigeria. Nigerian J Surg. 2002; 8:13-17

84. Eggleston FC, Santoshi B, Singh CM. Typhoid perforation of the bowel. Ann Surg, 1979; 190:31-35

85. Kim JP, Oh SK, Jarrett F. Management of ileal perforations due to typhoid fever. Ann Surg. 1975; 181:88-91

86. Gibney EJ. Typhoid perforation. Br J Surg. 1989; 76:887-889

87. Bifar J, Tarpley J. Intestinal perforation in typhoid fever: a historical and state of art review. Rev Infect Dis. 1985; 7:257-271

88. Finney JMT. On the surgical treatment of perforating typhoid ulcer. Johns Hopkins Hospital Reports. 1900; 8:155-208

89. Cushing HW. Laparotomy for intestinal perforation in typhoid fever: a report of four cases occurring in 1898. Johns Hopkins Hospital Reports. 1900; 8:209-240

90. Patterson FD. The surgical treatment of perforation of the intestines in typhoid fever: a review of literature. Am J Med Sci. 1909; 137:660-666

91. Woodward TE, Smadel JE. Management of typhoid fever and its complications. Ann Intern Med. 1964; 60:144-157

92. Vargas M, Pena A. Perforated viscera in typhoid: a better prognosis for children. J Pediatr Surg. 1975; 10:531-532

93. Keenan JP, Hadley GP. The surgical management of typhoid perforation in children. Br J Surg. 1984; 71:928-929

94. Li FWP. Surgical treatment of typhoid perforation of the intestine. Br J Surg. 1963; 50:976-979

95. Welch TP, Martin NC. Surgical treatment of typhoid perforation. Lancet. 1975; 1:1078-1080

96. Neugebauer H, Wallenboeck E, Hungerford M. Seventy cases of injuries of the small intestine caused by blunt abdominal trauma: a retrospective study. J Trauma. 1999; 46:116-21

97. Stevens SL, Maull KI. Small bowel injuries. Surg Clin North Am. 1990; 70:541-6090.

98. Baker B.E. Jejunal perforation occurring in contact sports. Am J Sports Med. 1978; 6:403-4

99. Fakhry SM, Brownstein M, Watts DD et al. Relative short diagnostic delays (>8 hours) produce morbidity and mortality in blunt small bowel injury: an analysis of time to operative intervention in 198 patients from a multi centre experience. J Trauma. 2000; 48:408-14

100. McQuaid KR; Diseases of peritoneum. In: Tierney LM, Mc Phee SJ, Papadakis MA, eds. Current Medical Diagnosis and Treatment. New York, NY: McGraw Hill Professional Publishing 1999: 558-563

101. Bufalari A, Giustozzi G, Moggi L: Post operative intraabdominal abscess; percutaneous versus surgical treatment. Acta Chir Belg. 1996 Sept-Oct, 96(5); 197-200

102. Paik PS, Towson JA, Anthone GJ, et al: Intraabdominal abscess following laparoscopic and open appendectomies. J. Gastrointest. Surg. 1997 Mar, 1(2); 188-193

103. Ivatury RR, Zobowski R, Psarras P, et al. Intraabdominal abscess after penetrating abdominal trauma. J Trauma. 1988 Aug; 28(8); 1238-43

104. L, Di Palo S, Castrucci M, et al; [Abdominal abscess; their treatment and the study of prognostic factors]. Minerva Chir. 1997 Apr; 52(4): 369-76

105 Reid RI, Dobbs BR, Frizelle FA. Risk factors for post appendecectomy intraabdominal abscess. Aust NZ J Surg. 1999 may, 69(5): 373-4

106. Ihedioha U, Alani A, Modak P, Chong P, O'dwyer PJ. Hernias are the most common cause of strangulation in patients presenting with small bowel obstruction. Hernia. 2006; 10:338-340

107. Cheadle WG, Garr EE, Richardson JD. The importance of early diagnosis of small bowel obstruction. Am Surg. 1988; 54: 565-569

108. Lopez-Kostner F, Hool GR, Lavery IC. Management and causes of acute large-bowel obstruction. Surg Clin North Am. 1997; 77: 1265-1290

109. Chiedozi LC, Aboh IO, Piserchia NE. Mechanical bowel obstruction. Review of 316 cases in Benin City. Am J Surg. 1980; 139: 389-393

110. Kuremu RT, Jumbi G. Adhesive intestinal obstruction. East Afr Med J. 2006; 83: 333-336

111. Lawal OO, Olayinka OS, Bankole JO. Spectrum of causes of intestinal

obstruction in adult Nigerian patients. S Afr J Surg. 2005; 43: 34-36

112. Tamijmarane A, Chandra S, Smile SR. Clinical aspects of adhesive intestinal obstruction. Trop Gastroenterol. 2000; 21: 141-143

113. Bizer LS, Liebling RW, Delany HM, Gliedman ML. Small bowel obstruction: the role of nonoperative treatment in simple intestinal obstruction and predictive criteria for strangulation obstruction. Surgery. 1981; 89: 407-413

114. Uludag M, Akgun I, Yetkin G, Kebudi A, Isgor A, Sener A. Factors affecting morbidity and mortality in mechanical intestinal obstruction. Ulus Travma Derg. 2004; 10: 177-184

115. Mucha P Jr. Small intestinal obstruction. Surg Clin North Am. 1987; 67: 597-620

116. Miller G, Boman J, Shrier I, Gordon PH. Natural history of patients with adhesive small bowel obstruction. Br J Surg. 2000; 87: 1240-1247

117 Richards WO, Williams LF Jr. Obstruction of the large and small intestine. Surg Clin North Am. 1988; 68: 355-376

118. Sarr MG, Bulkley GB, Zuidema GD. Preoperative recognition of intestinal strangulation obstruction. Prospective evaluation of diagnostic capability. Am J Surg. 1983; 145:176-182

119. Renzulli P, Krahenbuhl L, Sadowski C, al-Adili F, Maurer CA, Buchler MW. Modern diagnostic strategy in ileus. Zentralbl Chir. 1998; 123: 1334-1339

120. Giessling U, Petersen S, Freitag M, Kleine-Kraneburg H, Ludwing K. Surgical Management of severe peritonitis. Zentralbl Chir. 2002 Jul; 127(7): 594-7

121. Wittman DH, Schein M, Condon RE. Management of secondary peritonitis. Ann Surg. 1996 Jul; 224(1): 10-18

122. Kanna AK, Mishra MK. Typhoid perforation of the gut. Postgr Med J. 1984; 60:523-524

123. Adesunkanmi AR, Oseni SA, Adejuygbe O, Agbakwuru EA. Acute generalized peritonitis in African children: Assessment of severity using Modified APACHE II Score. ANZ J Surg. 2003 May; 73(5): 275-9

124. Noon GP, Beal AC, Jorden GL. Clinical evaluation of peritoneal irrigation with antibiotic solution. Surgery. 1967; 67-73

125. Rajender SJ, Ashock KA, Robin K et al. Spectrum of perforation peritonitis in India. World J Emerg Surg. 2006; 1:1-26

126. Ugwu BT, Yiltok SJ, Kidmas AT, Opaluwa AS. Typhoid intestinal perforation in North Central Nigeria. West Afr J Med 2005 Jan-Mar; 24(1):1-6

127. Rajesh V, Chandra SS, Smile SR. Risk factors predicting operative mortality in perforated peptic ulcer disease. Trop Gastroenterol. 2003; 24:148-50

128. Ersumo T, W/Meskel Y, Kotisso B. Perforated peptic ulcer in Tikur Anbessa Hospital: a review of 74 cases. Ethiop Med J. 2005 Jan; 43(1):9-13

129. Santillana M. Surgical complications of typhoid fever enteric perforation. World J Surg. 1991; 15:170-175

130. Mock C, Visser L, Denno D, Maier R. Aggressive fluid resuscitation and broad spectrum antibiotics decrease mortality from typhoid ileal perforation. Trop Doct. 1995 Jul; 25(3):115-17

131. Sleisenger MH, Fordtran JS eds. Gastrointestinal disease. Vol 2, 5th edn. London. WB Saunders. 1993; 1158-60

132. Ja Kubowski A, Elwood RK, Enarson BA. Clinical features of abdominal tuberculosis. J Infect Dis. 1989; 344-4

133. Moris PJ, Wood WE eds Oxford Textbook of Surgery 2nd edn. London: Oxford Press. 2000:1844-6

134. Burnand K, Young AE, Lucas AE, eds. The new Aid's companion in surgical studies. 2nd edn. London: Churchill Livingstone. 1998:702-4

135. Roland EA. The natural history and traditional management of appendicitis. World J Surg. 2007 Jan ;(1):31

136. Yu Tang, Jan You Lin, Yu-Shung Huang. Appendicitis in children. Kaohsiung J Med Sci. 2006 Sept; 22(9):452-6