THE PROFILE OF PATIENTS WITH ACUTE UPPER GASTROINTESTINAL TRACT BLEEDING AS SEEN AT MUHIMBILI NATIONAL HOSPITAL

By

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CERTIFICATION

The undersigned certify that he has read and hereby recommend for acceptance by the Muhimbili University of Health and Allied Sciences a dissertation entitled, ***The Profile of Patients with Acute Upper Gastrointestinal Bleeding as seen at Muhimbili National Hospital***, in partial fulfillment of the requirements for the degree of Master of Medicine in General Surgery.

.....

Prof. Mohamed R. Aziz

(Supervisor)

Date: 26/11/2007

DECLARATION

AND

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

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DEDICATION

To my wife and my daughter

ABSTRACT

Background

The use of various endoscopic techniques has progressively reduced the role of surgery in the emergent management of UGIB, under certain circumstances the surgeon, however still plays an important role in the management and in a specific group of patients. The problem poses a greater challenge to the surgeons in developing countries, Tanzania being among them, where endoscopy may not be widely available.

Objective

To study the profile of patients with acute upper gastrointestinal bleeding as seen at Muhimbili National Hospital.

Results

A total of 77 patients were studied. Males were approximately five times more frequently involved as compared to females. Upper gastrointestinal endoscopy was done in 67 patients. Oesophageal varices were the leading cause of acute upper gastrointestinal bleeding (48.1%) followed by duodenal ulcer (22.1%). Varices had a peak at the age of 31 to 50 years. Duodenal ulcer was commonest in patients who were above 50 years of age. A palpable spleen positively predicted varices as a source of bleeding in 91.9% of patients, while impalpable spleen predicted a source of bleeding other than varices in 96.7% of patients. The association between splenomegaly and varices was statistically significant (P value < 0.001). The mean number of units of blood transfused in these patients with acute upper gastrointestinal bleeding was 2.9 +/- 2.5. Patients with varices needed more blood transfusion than other causes. Mean hospital stay was 12.9 +/- 12.2. Hospital stay was prolonged in patients with varices. Overall mortality due to acute upper

gastrointestinal bleeding was 15.6%. Most deaths occurred in patients who had not undergone upper GI Endoscopy due to haemodynamic instability.

Conclusion

In Tanzania, at MNH, upper gastrointestinal bleeding was found to be five times more common in males than females among patients admitted to hospital. Haematemesis and/or melaena were the most common symptoms presenting in more than 90% of patients. The majority of patients admitted with acute UGI bleeding had oesophageal varices, most of whom were below 50 years. Splenomegaly is an important indicator for varices. Patients with oesophageal varices had longer hospital stay and needed greater number of units of blood transfusion.

TABLE OF CONTENTS

	PAGE:
CERTIFICATION	ii
DECLARATION AND COPYRIGHT	iii
ACKNOWLEDGEMENT	iv
DEDICATION	v
ABSTRACT	vi
LIST OF ABBREVIATION	ix
INTRODUCTION	1
LITERATURE REVIEW	5
STATEMENT OF THE PROBLEM	41
RATIONALE OF THE STUDY	43
STUDY OBJECTIVES	44
METHODOLOGY	45
ETHICAL ISSUES	46
RESULTS	47
ILLUSTRATIONS	60
DISCUSSION	63
CONCLUSION	69
RECOMMENDATIONS	70
REFERENCES	71
APPENDICES	85

LIST OF ABBREVIATIONS

AV	-	Arteriovenous
B-RT	-	Balloon occluded retrograde transvenous obliteration
CMV	-	Cytomegalovirus
COX-1	-	Cyclooxygenase-1
DU	-	Duodenal ulcer
DSRS	-	Distal splenorenal shunt
GI	-	Gastrointestinal tract
GISTs	-	Gastrointestinal stromal tumors
GIT	-	Gastrointestinal tract
GOV	-	Gastroesophageal varices
GU	-	Gastric ulcer
HBV	-	Hepatitis B virus
IGV	-	Isolated gastric varices
MNH	-	Muhimbili National Hospital
NSAID	-	Nonsteroidal anti-inflammatory drug
OGD	-	Oesophagogastroduodenoscopy
PPIs	-	Proton pump inhibitors
SPSS	-	Statistical Package for Social Sciences
TIPS	-	Transjugular Intrahepatic Portosystemic Shunt
UGIB	-	Upper gastrointestinal bleeding

INTRODUCTION

Upper gastrointestinal bleeding is defined as bleeding derived from a source proximal to the ligament of Treitz. Haematemesis and melaena are the most common presentations of acute upper gastrointestinal bleeding and patients may present with both symptoms. Occasionally, a brisk upper gastrointestinal bleeding may present as haematochezia.[1,2]

Upper gastrointestinal bleeding is a digestive disorder with both high morbidity and mortality, being among the most important digestive emergencies. Bleeding from upper gastrointestinal tract is approximately four times as common as bleeding from the lower gastrointestinal tract [3,4].

The incidence of acute upper gastrointestinal bleeding is slowly decreasing over the past three decades, but still presents a major problem in treatment. [5].

An aging patient population with an increased prevalence of associated medical co-morbidities has kept the mortality figures largely unchanged for the past thirty years, despite technological advances in endoscopy and other minimally invasive procedures. The hospital mortality rate has remained at about 10% in the past thirty years. [2,6]

All patients who develop acute gastrointestinal bleeding need urgent assessment. Almost all should be admitted as an emergency to hospital. Only a small minority of young fit patients who have self limiting bleeding can be managed as outpatients, but even these need urgent investigation. Patients who present with haematemesis tend to have more severe bleeding than those who present with melaena alone. [6].

The use of various endoscopic techniques, medical therapies and visceral angiography has progressively diminished the role of surgery in the emergent management of upper gastrointestinal bleeding. Nevertheless operative intervention still represents the most definitive intervention and remains the final therapeutic option for many bleeding lesions of the upper gastrointestinal tract. Of patients who develop acute upper gastrointestinal tract bleeding, ten to twenty percent require surgical procedures [2, 7]

At the initial assessment it is important to define factors that have prognostic importance. The main factors predicting death include increasing age, co-morbidity and endoscopic findings. Mortality is extremely low in patients under forty years old but thereafter increases steeply with advancing age. Patients who have severe co-morbidity, particularly renal insufficiency, hepatic failure or disseminated malignancy have a poor prognosis. Hospital admissions may be needed for gastrointestinal bleeding in many of these, and death is often due to the disease progression rather than bleeding. [2,6]

The history and physical examination provide crucial information for the initial evaluation of a patient presenting with gastrointestinal tract haemorrhage. The history and findings can be extremely helpful in determining the location of gastrointestinal bleeding. Alcohol abuse and cirrhosis should suggest portal hypertension and oesophageal varices as a source for the bleeding. A history of recent nonsteroidal anti-inflammatory drug (NSAID) abuse should elicit concern about bleeding from gastric ulcer. [8]

Differential diagnoses for upper gastrointestinal bleeding includes: [2,9]

- Gastric ulcer
- Duodenal ulcer
- Oesophageal varices

- Gastric varices
- Mallory-Weiss tear
- Oesophagitis
- Neoplasms of the upper gastrointestinal tract
- Haemorrhagic gastritis
- Dieulafoy lesion
- Angiodysplasia
- Haemobilia
- Pancreatic pseudocyst
- Pancreatic pseudoaneurysm
- Aortoenteric fistula

In the management of a patient with acute upper gastrointestinal bleeding the priority is to support the circulation rather than to identify the source of bleeding. Endoscopy is undertaken once resuscitation has been achieved; at least one large bore cannula is inserted in a substantial vein. The rate of infusion depends on the severity of shock. Crystalloids should be used rather than colloids (such as dextrans).

If blood transfusion is required the aim is to maintain a haemoglobin concentration of ten grammes per deciliter (10gm/dl). In patients with suspected liver disease the use of normal saline should be avoided because of the risk of precipitating ascites. [6,10]

After resuscitation endoscopy is undertaken. In most cases this is done electively on the next available routine list but within 24 hours of admission. Only a minority of profusely bleeding patients need emergency endoscopy. [6,11]

Surgery is the best way of stopping active ulcer bleeding and preventing rebleeding, but it carries a high morbidity and mortality. It is now reserved for cases in which endoscopic treatment has failed. Surgical treatment for acute variceal bleeding including oesophageal transection with devascularization and portocaval shunt is rarely done because of unacceptable mortality. It has been replaced by endoscopic therapy and transjugular intrahepatic portosystemic shunt (TIPS). However shunts may precipitate encephalopathy.[6]

LITERATURE REVIEW

Aetiology of acute upper gastrointestinal bleeding.

In Europe the commonest cause of upper gastrointestinal tract haemorrhage is peptic ulcer. A history or proved ulcer or ulcer like dyspepsia is absent in about 20% of cases. In these patients consumption of aspirin or nonsteroidal anti-inflammatory drugs is common. Infection with *Helicobacter pylori* is less prevalent in bleeding ulcers than in uncomplicated ulcers. Severe ulcer bleeding due to erosion of an artery by ulcer and severity of bleeding depends on the size of the ulcer and the size of the arterial defect. Bleeding from a defect greater than one millimeter in diameter is unlikely to stop spontaneously and does not respond to endoscopic treatment. Large ulcers arising from the posterior part of the duodenal cap erode the gastroduodenal artery and provoke brisk bleeding [6]

Bleeding from gastric erosions, oesophagitis or vascular malformations usually stops spontaneously and is not usually life threatening. Mallory-Weiss tears are a consequence of retching, and most patients have a history of alcohol misuse, have features of other gastrointestinal disease such as peptic ulcer or gastroenteritis, or have non-gastrointestinal cause of vomiting. Bleeding usually stops spontaneously although endoscopic haemostatic treatment is sometimes needed. [6]

Bleeding from upper gastrointestinal malignancy is not usually severe, and prognosis is dictated by the stage of the disease. Patients with extensive upper gastrointestinal cancer have dismal prognosis, but death is not usually a consequence of gastrointestinal haemorrhage but of disease progression. [6]

In any patient with acute gastrointestinal bleeding, liver disease should be considered because it requires specific management. Oesophageal varices account for a small proportion of cases but have a disproportionate impact on medical resources. Bleeding is often severe and other features of liver failure such as fluid retention, hepatic encephalopathy, renal failure and sepsis often develops after the bleed. About a third of patients will die, and the prognosis is related to the severity of the underlying liver disease rather than the size of variceal haemorrhage. [6]

Aortoduodenal fistula must be considered in patients who develop profuse bleeding and have undergone aortic aneurysm surgery. [6,12.]

A study from Tanzania, about 25 years ago to evaluate the diagnostic yield of endoscopy in upper gastrointestinal tract bleeding; 122 patients presented with symptoms of upper gastrointestinal bleeding of less than two weeks duration. The most frequent findings were peptic ulcer (40.9%), oesophageal varices (16.4%) and mucosal lesions (7.4% as an isolated finding and a further 16.4% associated with peptic ulcer or varices). Negative endoscopies comprised 24.6% of all 122 patients, but 19 in whom the bleeding episode preceded endoscopy by not more than 48 hours, there were none. In 105 patients whose history of haematemesis and/or melaena was not recent, endoscopy was negative in 30%. The earlier the endoscopy is done, the greater is the diagnostic yield. [13]

In a study from Kenya 15 years ago, a total of 97 consecutive patients were admitted with upper gastrointestinal bleeding. All patients underwent upper gastrointestinal endoscopy within 24 hours of presentation and a positive endoscopic diagnosis was possible in 90 (93%) of the patients.

The leading causes of bleeding were oesophageal varices (35%), duodenal ulceration (17.5%), superficial inflammatory lesions (17.5%) and multiple lesions with superficial inflammation and oesophageal varices as the commonest combination. [14]

In a study from Malawi in adults who presented with acute upper gastrointestinal bleeding, the principal causes of haemorrhage were oesophageal varices (45%), duodenal ulcer (16%), gastritis/erosion (9%) and gastric ulcer (7%). [15]

In Sudan, bleeding due to oesophageal varices is the commonest cause of upper gastrointestinal haemorrhage. The cause of portal hypertension was schistosomal periportal fibrosis in 93% of patients. [16]

In Europe the incidence of acute UGIB varies from 50 to 150 cases per 100,000 per year and is highest in areas of social deprivation. [11,17,18,19,20.] The incidence rises from 23 per 100,000 in those aged under 30 years of age to 485 per 100,000 in those aged over 75 years of age. [17] UGIB is seen predominantly among the elderly; 68% of patients are over 60 years of age and 27% are over 80 years of age. [11,20.] Mortality of patients admitted to hospital because of acute gastrointestinal bleeding is approximately 10%-14%, and have not changed over half a century. [11,19.] Almost all deaths occur in the elderly and related to decompensation of medical co-morbidity. [11,19]

In Europe the causes of acute UGIB are as shown in table below; [19]

Cause of UGIH	Frequency %
Peptic ulcer	35-50
Varices	5-12
Mallory-Weiss tear	2-5
Oesophagitis	20-30
Duodenitis/gastritis/erosions	10-20
Vascular	2-3
Tumours	2-5
Aortoduodenal fistula	< 1

In USA the annual incidence of hospitalization due to acute upper gastrointestinal bleeding was between 47.7 and 102 per 100,000. [21,22] Incidence increased markedly with age, more than 70% of patients were more than 60 years old. Males were two times more affected than females. [21] Peptic ulcer bleeding was the cause of bleeding in about one half of all cases. [21,23]

A study done in California about ten years ago revealed the causes of acute UGI bleeding as; peptic ulcer 61.6%, mucosal erosive disease 14.3%, varices 6.2%, miscellaneous causes 9.7% and unknown causes 8.1%. [22]

Bleeding from peptic ulcers.

While upper gastrointestinal bleeding is caused by a wide variety of medical conditions, most studies in Europe and America have shown that about 50% of cases are caused by peptic ulcers. Approximately 80% of all upper gastrointestinal bleeding episodes stop spontaneously. [8,10,19,22,24,25]

High risk patient population for peptic ulcer disease includes those with a history of alcohol abuse, chronic renal failure and/or nonsteroidal anti-inflammatory use. [8]

Recurrence of gastrointestinal haemorrhage is associated with an increased mortality rate, a greater need for surgery, blood transfusions, a prolonged hospital stay and increased overall health care costs. [10]

Since the publication of the historical paper in the Lancet in 1983, the role of H. pylori in the aetiology of peptic ulcer disease became well accepted and it has become clear that eradication of *H. pylori* infection prevents ulcer recurrence and reduces recurrent peptic ulcer bleeding. [21, 26.]

With the introduction of proton pump inhibitors (PPIs) in 1988, a strong acid suppression became available that is capable of healing gastritis, duodenitis, erosions and almost all ulcers. PPIs have also been found to prevent ulcer recurrence and complications when concomitantly used with nonsteroidal anti-inflammatory drugs or aspirin. Counteracting this is the fact that the population in the West is living longer, a fact that has resulted in more comorbidity and increasing use of medications including NSAIDs. [21,27,28,29.]

Peptic ulcers are commonly duodenal or gastric ulcer. Duodenal ulcers have been considered more common than gastric ulcer, but studies in the West have shown almost

even rates for gastric and duodenal ulcers. Higher incidence of gastric ulcers is due to more elderly and diseased population and more NSAID intake and lower rate of duodenal ulcers is due to effective medical therapy. [30] In most cases, the bleeding is caused by the erosion of an artery by the ulcer. Bleeding vessels larger than 1.5 millimeters in diameter are associated with an increased mortality. [8]

The appearance of the ulcer at the time of endoscopy provides important information regarding the risk of rebleeding. Forrest et al were the first to classify the stigmata of haemorrhage from peptic ulcers as follows: [31]

Class	Activity	Rebleeding %
Forrest Ia	Spurting bleeding	80
Forrest Ib	Oozing bleeding	10
Forrest IIa	Nonbleeding visible vessel	50
Forrest IIb	Adherent clot	33
Forrest IIc	Haematin on ulcer base	7
Forrest III	Clean ulcer base	3

Rockall scoring system for nonvariceal upper GI bleeding

Rockall scoring system is the best risk assessment for rebleeding of nonvariceal haemorrhage basing on both the severity of haemorrhage and general health condition of the patient. Multivariant analysis identified age, shock, medical co-morbidity and specific endoscopic findings as independent variables which predict rebleeding and death. [17]

Rockall scoring system

Variable	Score 0	Score 1	Score 2	Score 3
Age	<60	60-79	>80	-
Shock	None	Pulse>100, normal BP	Pulse>100beats/min, systolic BP < 100 mmHg	-
Co-morbidity	None	-	Cardiac, GIT cancer, other major co-morbidity	Renal failure, liver failure, disseminated malignancy
Diagnosis	Mallory-Weiss tear no lesion, no SRH	All other diagnoses	Malignancy of the upper gastrointestinal tract	-
Major SRH	None or dark spots	-	Blood in the upper GIT, adhering clot, visible or spurting vessel	-

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Rockall score, rebleeding and mortality

Risk score	% rebleed	% mortality
0	5	0
1	3	0
2	5	0.2
3	11	3
4	14	5
5	24	11
6	33	17
7	44	27
>8	42	41

Management of peptic ulcer bleeding.

Peptic ulcer bleeding is a common and potentially fatal condition. It is best managed using a multidisplinary approach by a team with medical endoscopist and surgical expertise. A prompt initial clinical and endoscopic assessment should allow patient triaged effectively into those who require active therapy versus those who require monitoring and preventive therapy. [11]

Early endoscopy and endoscopic therapy is associated with reductions in the length of hospital stay, rate of recurrent bleeding and need for emergent surgical intervention. [32,33,34.]

The endoscopic techniques used to achieve haemostasis have continued to evolve over the last 15 years as newer, more effective therapies have been developed. Various techniques currently available for achieving haemostasis: [8]

- Injection of vasoactive agents
- Bipolar electrocoagulation
- Heater probe coagulation
- Argon plasma coagulator
- Laser photocoagulation
- Application of haemostatic materials including biological glue.

The three most popular methods of haemostasis are injection therapy, coaptive coagulation and laser phototherapy.

Injection therapy involves the use of several different solutions injected into and around the bleeding lesion. The different solutions available for injection are epinephrine, sclerosants and clot producing materials such as fibrin glue. Epinephrine used for

injection is diluted (1:10,000) and injected 0.5-1 ml aliquots. It is a vasoactive agent that works by vasoconstriction and decreasing blood flow to the areas. This allows for platelet function and clot formation to attain haemostasis. Debate continues over whether the haemostatic effect of epinephrine is due to the induced vasoconstriction and subsequent platelet aggregation or to the tamponade effect produced by injecting the volume of drug into the tissue surrounding the bleeding lesion. Injecting a volume of sterile isotonic sodium chloride solution and provide tamponade effect also leads to haemostasis, although not as effectively as epinephrine. [4]

Coaptive coagulation uses direct pressure and thermal therapy to achieve haemostasis. Thermal therapy includes monopolar and bipolar electrocoagulation and heater probe application. The bleeding vessel is isolated, compressed and tamponaded prior to coagulation therapy. By using both maneuvers, the depth of tissue injury is minimized. Coaptive coagulation is as effective as injection therapy in achieving haemostasis. [4]

Laser phototherapy creates haemostasis by generating heat and direct vessel coagulation. This is a non-contact thermal method. It is not as effective as coaptive coagulation because it lacks the use of compression to create tamponade effect. It is also expensive. [4]

The endoscopic appearance of the bleeding lesion has been used to identify patients at high risk for recurrent bleeding. [35]

The indication for endoscopic therapy is based on size, site and stigmata of recent bleeding. An actively bleeding vessel is treated because it is a high risk lesion with 55% risk for recurrent bleeding. A non bleeding visible vessel is also a high risk lesion that is treated because of its 43% risk for rebleeding. Recurrent bleeding occurs most commonly within the first 72 hours. Treatment of clots adherent to a lesion is based on the patient's

risk factors to withstand and survive another bleeding episode. The risk of rebleeding varies from 14 to 36%. Low risk lesions are those that appear as flat pigmented spots and those that involve a clean ulcer base with no visible vessel. The risk of rebleeding in these is 10% and 5% respectively. Endoscopic treatment of these low risk lesions is not usually performed. [35]

Primary haemostasis can be achieved in the great majority of ulcer bleeding patients using endoscopic therapy but rebleeding occurs in 15% to 20% of cases, usually within the first 24 hours. [19]

In one study a comparison of two haemostatic procedures was done: it compared endoscopic injection of 1:10,000 epinephrine solution alone and epinephrine plus application of haemoclips. The conclusion was that both procedures are equivalent therapies in treating bleeding and rebleeding from peptic ulcers. [24]

Proton pump inhibitors (PPIs) decrease rebleeding rates in patients with bleeding ulcers associated with overlying clot or visible non bleeding vessel in the ulcer base. [36]

Surgery in patients with bleeding peptic ulcers

Epidemiological studies have demonstrated that the incidence of emergency surgery for bleeding peptic ulcer has not changed despite major improvement with endoscopic treatment. There are no proven alternatives to emergency operation for massive bleeding uncontrolled by endoscopic procedures. [28]

Operations for peptic ulcer bleeding has remained constant, such operations are performed on 10% to 20% of all patients hospitalized for upper gastrointestinal tract haemorrhage secondary to peptic ulcer. [7]

Importantly surgery on bleeding peptic ulcer was necessary on an emergent or urgent basis in 70% and 100% of patients respectively. [7]

Indications for surgical operations for peptic ulcer bleeding include:

- Active haemorrhage that is refractory or inaccessible to endoscopic control. [28]
- Endoscopic proved rebleeding despite technically successful endoscopic treatment. A second endoscopic treatment can be attempted in patients who rebleed. Permanent haemostasis can be achieved in about a half of these cases and emergency surgery is required in the rest. [28,37]
- Shock in (blood pressure less than 100 mmHg) within 12 hours of admission in patients over 50 years of age but responding to resuscitation (operation being performed as soon as shock is brought under control. [37]
- Aggressive surgical approach, developed in United Kingdom based on uncontrollable initial bleeding, extensive need for blood replacement and rebleeding. Criteria for early operation are 4 or more units of blood transfused within 24 hours, one rebleed or endoscopic stigmata; and for delayed surgery are 8 or more units of blood transfused per 24 hours, two rebleeds and persisting rebleeding. [28]
- A coexisting reason for surgery as perforation, obstruction or malignancy. [6]

The three common operations performed for a bleeding duodenal ulcer are as follows [8]

- Suture ligation of the bleeding ulcer, truncal vagotomy and pyloroplasty.
- Suture ligation of the bleeding ulcer, truncal vagotomy and antrectomy
- Duodenotomy and suture ligation of bleeding ulcer with proximal (highly selective) vagotomy

A multicentre randomized prospective trial compared minimal surgery (under-running the vessel or ulcer excision and adjuvant ranitidine) with conventional ulcer surgery (vagotomy and pyloroplasty or partial gastrectomy) for the treatment of bleeding peptic ulcer. There was no significant difference in mortality after conservative surgery as compared to that after conventional surgery. [38]

Another study reviewed patients who underwent emergency surgery for bleeding peptic ulcer. Surgical treatment was either conservative (oversewing or ulcer excision alone or associated with vagotomy) or was radical (antrectomy and vagotomy or partial gastric resection). The type of surgery did not influence postoperative mortality, whereas bleeding recurrence was more frequent in case of conservative surgery. [39]

In a study to compare the rates of postoperative bleeding recurrence, duodenal leakage and mortality in patients undergoing emergency surgery for massive persistent or recurrent bleeding from bulbar peptic ulcer, the rate of recurrent bleeding was 17% after oversewing plus vagotomy and 3% after gastric resection. The duodenal leak rate was higher after gastric resection than after oversewing and vagotomy (13% versus 3%). Overall postoperative mortality was similar, 22% (oversewing and vagotomy) versus 23% (gastric resection). [40]

The diagnosis of H. pylori infection is important in the management of patients with complicated bleeding peptic ulcer. If a patient with a bleeding ulcer requires surgery, then knowledge of the patient's H. pylori status becomes pertinent because it may help guide the decision to choose a particular definitive surgical procedure, for instance simply over sewing the ulcer as opposed to performing anti-ulcer operation.

Many studies support the decision to manage the bleeding ulcer in conjunction with eradication of H. pylori. In patients with H. pylori infection, the rate of recurrent bleeding is extremely low. Patients who are not infected with H. pylori may require a subsequent acid lowering surgical procedure or long term medical therapy for recurrent ulcer disease and bleeding. Rates of H. pylori infection are reported to be lower in patients with complicated ulcer disease compared with patients with uncomplicated ulcers. In one study incidence of H. pylori was found to be 71% in bleeding duodenal ulcers and 93% in non-bleeding ulcers. [17,41,42,43,44]

Some case series and cohort studies suggest that acute gastrointestinal bleeding decrease the sensitivity of Helicobacter pylori diagnostic testing. A prospective cohort study using patients as their own controls was done to assess H. pylori biopsy testing in patients with acute upper gastrointestinal bleeding and one month later. The results showed that acute gastrointestinal bleeding did not decrease the sensitivity of urease testing. Furthermore, bleeding did not produce falsely negative histological examinations for H. pylori density or the inflammatory cell infiltration. [45,46]

Bleeding gastric ulcer.

The choice of operation depends on the location of the ulcer and the haemodynamic stability of the patient to withstand the operation. Five types of gastric ulcers occur basing on their location and acid secretory status.

Type 1 gastric ulcers are located on the lesser curvature of the stomach at or near the incisura angularis. These ulcers are not associated with hypersecretory acid state.

Type 2 ulcers, combination of two ulcers that are associated with hypersecretory acid state. The ulcer location occurs in the body of the stomach in the region of the incisura and the second ulcer occurs in the duodenum.

Type 3 ulcers are associated with high acid output and usually within 3cm of the pylorus.

Type 4 ulcers are located high in the lesser curvature of the stomach and are not associated with high acid output.

Type 5 ulcers are related to ingestion of nonsteroidal anti-inflammatory drugs or aspirin. These ulcers can occur anywhere in the stomach.

Vagotomy is added to manage type 2 or type 3 gastric ulcers. Patients who are haemodynamically stable with intermittent bleeding requiring blood transfusions should undergo a truncal vagotomy and distal gastric resection to include the ulcer for type 1, 2 and 3 ulcers.

In patients who present with life threatening haemorrhage (poor risk patients) and a type 1,2 or 3 ulcer, a lesser procedure like biopsy and oversew or excision of the ulcer should be considered. [47,48]

There is evidence indicating that there is no difference between local and radical surgery concerning mortality, although the rebleeding rate is higher in the local group. Patients may be served by a relatively aggressive approach. [38,39,40]

Stress gastritis

Acute stress gastritis is a disease process characterized by diffuse superficial mucosal erosions that appear as discrete areas of erythema. The bleeding is usually mild and self limiting and rarely progresses to life threatening haemorrhage. Commonly observed in patients who have undergone episodes of shock, multiple trauma, acute respiratory distress syndrome, acute renal failure and sepsis. Histamine receptor antagonists are proven to be more effective at controlling stomach pH. Stress related bleeding usually occurs 7 to ten days after the initial insult, but may manifest sooner. [49,50]

Endoscopic haemostasis is attempted using electrocoagulation, laser or injection therapy. Surgical intervention becomes necessary if non operative therapy fails and blood loss continues. Simply oversewing actively bleeding erosion is sometimes effective enough to control the bleeding. Gastric resection with or without vagotomy with reconstruction may be necessary. The type of resection depends on the location of the erosions. [50]

Upper gastrointestinal bleeding secondary to oesophageal varices

Variceal bleeding is one of the most alarming complications of liver cirrhosis. Sixty percent of patients with liver cirrhosis develop oesophageal varices. Thirty percent of these patients bleed from their varices within two years of their diagnosis.

The mortality rate for variceal bleeding is 30 to 50%, which is higher than any other common cause of upper gastrointestinal bleeding. [21,33,51]

Child-Pugh classification [52]

Parameter	1 point	2 points	3 points
Bilirubin (μmol/L)	< 20	20-30	>30
Albumin (gm/L)	>35	28-35	<28
Increase in PT (seconds)	1-3	4-6	>6
Ascites	none	slight	moderate
Encephalopathy	none	1-2	3-4

Grade A score of 5 and 6

Grade B score of 7 to 9

Grade C score of 10 to 15.

The risk of bleeding within one year is less than 10% for a patient with a Child-Pugh class A cirrhotic liver with small varices, but it is 76% for a patient with Child-Pugh class C cirrhotic liver with large varices and red signs. [53]

After an acute variceal haemorrhage, bleeding resolves in 50% of patients. Bleeding is least likely to stop in patients with large varices and a Child-Pugh class C. Once bleeding stops, patients remain at increased risk of rebleeding for about six weeks; such risk is greatest during the first 48 hours after haemorrhage. Factors associated with early rebleeding include age over 60 years, renal failure and severe initial bleeding defined by haemoglobin less than 8 g/dl at presentation.

Risks for late rebleeding are severe liver failure, continued alcohol abuse, large variceal size, renal failure and hepatocellular carcinoma. [53]

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The risk of first variceal bleed may be predicted basing on variceal size, the presence of red wales and a child-Pugh score greater than or equal to 8 points. [33,54]

A score based on the appearance of oesophageal varices correlates with the risk of variceal bleeding in cirrhotics who have never bled. [55,56]

Parameters

- Form and size
- Location
- Colour
- Red colour sign

Form and size

Parameter	Finding	Points
F1	Small straight varices not disappearing with insufflation	1
F2	Large varices occupying less than less than one third of the lumen	2
F2	Large , coil shaped varices more than one third of the lumen	3

Location

Parameter	Finding	Points
Li	varices located within the lower third (distal 6 cm)	0
Lm	varices located within the lower two thirds	1
Ls	varices extending above the level of tracheal bifurcation	2

Colour

Parameter	Finding	Points
Cw	white varices appearing like large folds of mucosa	0
Cbw	varices that cannot be clearly assigned as Cw or Cb	1
Cb	dark blue varices, appearing cyanotic	2

Red colour sign

Parameter	Finding	Points
Absent		0
+	Present but not more than 10 lesions	1
++	More than 10 lesions are easily visible but not extensive	2
+++	Extensive, covering all varices in the entire oesophagus	3

Almost all patients with high grade varices are expected to bleed within two years. [55,56]

Score	Grade	% Bleeding
1-3	low	0
4-7	medium	11
8-10	high	73

Causes of portal hypertension can be pre-sinusoidal, sinusoidal or post-sinusoidal. Pre-sinusoidal can either be extra-hepatic or intrahepatic. Extrahepatic presinusoidal causes of portal hypertension are congenital umbilical sepsis, trauma, hypercoagulable state and malignant occlusion. Presinusoidal intrahepatic causes of portal hypertension are schistosomiasis, congenital hepatic fibrosis and primary biliary cirrhosis. Cirrhosis is the sinusoidal cause of portal hypertension. Postsinusoidal causes of portal hypertension include Budd- Chiari syndrome and venoocclusive disease. [52,54]

A study done in Sweden showed that variceal bleeding is still a strong risk factor for recurrent bleeding. There was no significant difference in survival between groups with varices diagnosed after a bleed to the one with varices diagnosed before a bleed. Prophylactic sclerotherapy does not reduce the incidence of bleeding from varices in patients with liver cirrhosis and low to moderate bleeding risk. Although sclerotherapy lowers the mortality attributable to variceal bleeding, overall mortality was not affected. The effect of prophylactic sclerotherapy seems dependent on the underlying bleeding risk. Beneficial effect can only be expected for patients with a high risk for bleeding. [57]

In studies from some African countries, bleeding oesophageal varices was the leading cause of upper gastrointestinal bleeding. In Kenya, a study revealed that oesophageal varices were a leading cause, contributing 35% of all cases of acute upper gastrointestinal

bleeding. In a study from Malawi, it was a leading cause of upper gastrointestinal bleeding (45%).[14,15]

In patients with upper gastrointestinal bleeding due to varices, those with portal hypertension but normal liver function (portal vein thrombosis, idiopathic portal hypertension, and schistosomiasis) have a better outcome and survival rates compared to those with liver dysfunction and other organ failure. [33,58]

In a study from Malawi, 69% of patients with oesophageal varices who were investigated, had *S. mansoni* infection. A palpable spleen positively predicted oesophageal varices in 91% of cases, while nonpalpable spleen predicted a source of haemorrhage other than varices in 97% of cases. [15]

In a study from Zimbabwe, portal hypertension was caused by alcoholic liver cirrhosis in 44%, hepatitis B induced in 14%, cryptogenic liver cirrhosis in 6%, bilharzial portal fibrosis in 34% and extrahepatic portal obstruction in 2%. [47]

Some authors have found that thrombocytopaenia (platelets less than 118,000/mm^3), splenomegaly and ascites are useful predictors of large oesophageal varices in cirrhotic patients. However a study done in Singapore showed that only 55% of patients had thrombocytopaenia, 45% had splenomegaly and 27 had ascites. So splenomegaly, thrombocytopaenia or ascites is an unreliable predictor of oesophageal varices. Urgent or emergent endoscopy is still advocated to accurately diagnose bleeding oesophageal varices. [59]

Gastric varices develop in patients with portal hypertension including liver cirrhosis, idiopathic portal hypertension as well as left sided local portal hypertension such as splenic vein thrombosis or splenic AV malformation. The inflow vein is the left gastric

vein, posterior vein or short gastric vein, while the outflow vein is the gastro-renal shunt in most of the patients with gastric varices. The form of gastric varices is classified into three types of venous dilatations according to size and shape of varices; [58]

- Tortuous type
- Notched type
- Tumour type

Gastric varices are seen in nearly one out of every five patients with portal hypertension and oesophageal varices. The commonest presentations of patients with gastric varices include bleeding and hepatic encephalopathy. [60]

Gastric varices could be classified as follows basing on their location: [58,60,61.]

- Gastro-oesophageal varices type 1 (GOV 1) – continuing as extension of oesophageal varices and present on the lesser curve.
- Ectopic gastric varices type 2 (GOV 2) – gastric varices on the greater curve, as an extension of oesophageal varices
- Isolated gastric varices type1 (IGV 1) – isolated varices in the fundus of the stomach
- Isolated gastric varices type 2 (IGV 2)- isolated varices at ectopic sites in the stomach.

Gastric variceal bleeding has a higher rate of rebleeding than oesophageal variceal bleeding, and is associated with decreased survival. The cumulative mortality of fundal varices reaches as high as 52% at the end of one year. [58]

Isolated gastric varices (IGV 2) are predominantly (84.9%) secondary in origin, developing after oesophageal variceal obliteration. [60]

Management of bleeding varices

The initial management of a patient with acute variceal bleed involves aggressive efforts at resuscitation as previously outlined. Treatment strategies can be classified into

- Pharmacologic therapy
- Endoscopic therapy
- Tamponade
- Decompression therapy
- Liver transplantation

Pharmacologic therapy

The patient with acute variceal bleeding may initially be treated with intravenous vasopressin and nitroglycerine, somatostatin or its analogs (eg octreotide). Vasopressin is a potent splanchnic and systemic vasoconstrictor including coronary vasoconstriction. Nitroglycerin should be concomitantly administered to titrate and maintain systolic blood pressure in the range of 90 to 100mmHg.

Nitroglycerin should be initiated at 40mcg/min to protect the coronary arteries from the profound adverse cardiovascular effects of vasopressin.

The intravenous infusion of vasopressin is started at 0.2-0.4U/min. A meta-analysis revealed that vasopressin was clearly superior in arresting bleeding as compared to untreated controls but showed no survival benefit. [62]

A newly developed prodrug called terlipressin has been used that has advantages over vasopressin. [63] Terlipressin has a longer half life with a biphasic vasoconstriction profile. The drug first has systemic vascular effects that are then steadily converted into a more

effective vasoconstriction of the splanchnic bed. Randomized control trials comparing terlipressin with placebo have shown clear benefit in terms of bleeding control and survival. [64]

Recombinant coagulation factor VII is a synthetic coagulation factor that is currently used to treat bleeding episodes in patients with haemophilia. It has recently been trialed in randomized studies as an adjuvant to standard methods of controlling variceal haemorrhage. Patients in end stage liver disease lose synthetic capacity to produce these coagulation factors, most notably factor VII. [65]

A recent double blind trial has shown increased success at controlling bleeding endpoints in more severe cirrhotics (classes B and C) treated with factor VII compared to placebo. Although results are promising, cost effective dosing and timing of therapy are yet to be determined. [65,66]

Endoscopic therapy in variceal bleeding

The two main endoscopic techniques available to control variceal bleeding are endoscopic sclerotherapy and endoscopic variceal band ligation. Endoscopic sclerotherapy involves injecting a sclerosing agent such as ethanolamine oleate or polidocanal into the varix lumen (intravariceal) or immediately adjacent to the vessel (paravariceal) to create fibrosis in the mucosa overlying the varix, which leads to haemostasis. Endoscopic variceal band ligation consists of placement of a rubber band around the varix. This technique is performed by first sucking the varix into a sheath attached to the distal end of the endoscope. Once the varix is suctioned into the sheath, a trigger device allows the deployment of a rubber band around the varix, a procedure that strangulates the varix. The rubber bands slough off in the following 24 to 72 hours, leaving a shallow ulcer behind. Ulceration is less severe than with sclerotherapy [67,68]

The single potential downside to endoscopic banding is poor visualization of the varices when massive bleeding is present. This is due to the 30% reduction in the field of vision through endoscope caused by the plastic sheath used to deploy the rubber band. [62]

The drawback to sclerotherapy is its potential life threatening complications such as perforation, ulceration and stricture of the oesophagus. The initial success in arresting haemorrhage comparing the two methods is comparable, as high as 95% in some studies. Variceal rebleeding rates are lower with band ligation compared to sclerotherapy which has rebleeding rates as high as 50% . [62,63]

The other available endoscopic option is the use of tissue adhesives which are useful for both oesophageal and gastric varices.

Native cyanoacrylate (N-butyl-2-cyano-acrylate) or histoacryl is a liquid tissue adhesive which has a consistency similar to water, which makes it easy to manipulate down the endoscope. After injecting the substance into the varix, the blood mixes with the adhesive agent and rapidly polymerizes into hard glue. The cyanoacrylate then plugs the lumen of the varix and creates haemostasis. Several weeks after the injection the overlying mucosa sloughs off and the adhesive cast is passed through the gastrointestinal tract. [69]

Cyanoacrylate is 90% successful in achieving haemostasis in patients with acute bleeding from either gastric or oesophageal varices. The early rebleeding rate is 0-28%. The risks of using these adhesive agents are the potential systemic effects and permanent damage to the endoscope from expose to the adhesives at the lens of the scope. The potential systemic effects are related to the risk of thrombotic complications. Cerebral stroke from anomalous right to left shunts, fatal pulmonary embolization, splenic infarction and retrogastric abscess have all been reported in literature. [69]

Balloon tamponade

Since the 1990s, the use of balloon tamponade for the emergent control of oesophageal variceal bleeding has steadily decreased and is now usually indicated for only the 10% of patients whose acute bleeding episode is not controlled endoscopically. The use of these tubes can be life saving maneuver when medical and endoscopic efforts fail to control the bleeding. Although temporary control of bleeding is effective in 85% of cases, recurrent bleeding with release of the tamponade occurs in most patients. [70]

In addition to their limited effectiveness, the tubes are associated with a 20% complication rate that includes airway obstruction, aspiration and oesophageal necrosis

with rupture. Because of severe life threatening complications and limited use, the tubes are used only as a temporary measure while the patient is resuscitated until when the patient is ready for a repeat endoscopy or a portal pressure decompression through radiological or surgical method. [70]

The two most commonly used tubes are the Sengstaken-Blakemore tube and the Minnesota tube. These tubes have an oesophageal balloon and a gastric balloon that are inflated to produce a tamponade effect after confirming appropriate anatomical placement. The physician must be familiar with the steps involved in placement of the tubes to avoid the unnecessary complications associated with balloon tamponade are as follows [58,70]

Major complications:

- Oesophageal rupture
- Tracheal rupture
- Duodenal rupture
- Respiratory tract obstruction
- Aspiration
- Haemoptysis
- Tracheooesophageal fistula
- Jejunal rupture
- Thoracic lymph duct obstruction
- Oesophageal necrosis
- Oesophageal ulcer

Minor complications

- Nasopharyngeal bleeding
- Chest pain
- Balloon impaction and/or migration (nausea and vomiting)
- Alar necrosis

Transjugular intrahepatic portosystemic shunt (TIPS)

The TIPS procedure has become the most frequently employed method, because of its effectiveness; it is considered the standard of therapy for bleeding oesophageal varices that are unresponsive to endoscopic and pharmacologic first line treatment. The placement of TIPS reduces the outflow hepatic resistance, lowers portal pressure and diverts portal blood flow from gastrooesophageal collaterals through the stent. TIPS procedure controls variceal bleeding in more than 90% of patients. The rebleeding rate is 16 to 30% at one year follow up, and this is most commonly related to stenosis of the intrahepatic shunt or obstruction of the stent. Shunt dysfunction occurs in approximately 50 to 60% of patients at six months. [58,71,72.]

Balloon-occluded retrograde transvenous obliteration of gastric varices (B-RTO)

B-RTO is a promising new transvenous sclerotherapy technique with 5% ethanolamine oleate. The criteria for B-RTO are the presence of gastro-renal shunt; 20 to 30ml of 5% ethanolamine oleate with a contrast medium is injected into the gastric varices through a balloon catheter via the right femoral vein. The eradication rate is 97%. The cumulative rebleeding rate was 14% at 8 years after treatment. [58]

Surgical shunts

Surgery for bleeding oesophagogastric varices is the most reliable method to control acute haemorrhage and is associated with recurrent bleeding rates of less than 10%. The goal of a surgical shunt is to effectively decrease the portal venous hypertension and its adverse effects without compromising liver functions. [8] The operative approaches for bleeding oesophagogastric varices consist of two general concepts;

- Decompression of the high pressure portal venous system into a low pressure systemic venous system
- Devascularization of the distal oesophagus and proximal stomach.

The following options are currently available:

- Portacaval shunt (end to side)
- Portocaval shunt (side to side)
- Small diameter interposition graft
- Mesocaval shunt
- Large diameter interposition graft
- Distal splenorenal (Warren) shunt
- Oesophagogastric devascularization, oesophageal transaction and reanastomosis
- Orthotopic liver transplantation
- Splenectomy for splenic vein thrombosis

Portosystemic shunts can be classified as non-selective, selective or partial shunts. Selective or partial shunts, in addition to decreasing portal venous hypertension preserve portal blood flow to the liver in order to avoid compromising liver perfusion and function.

Encephalopathy occurs in 10-15% of patients after a selective shunt (Distal splenorenal shunt-DSRS), in 10-20% after a partial shunt and 30-40% after a total shunt. [33]

There is no significant difference in survival between the different shunts; for elective cases, operative mortality rate is approximately 5%. [33,73,74]

Oesophagogastric devascularization, oesophageal transaction and reanastomosis

This can be done by the technique of modified Sugiura procedure, which involves a one stage transabdominal approach. A splenectomy is first performed. Next the stomach is devascularized to include the upper two thirds of the lesser and greater curvatures. The devascularization of the oesophagus is carried 6 to 7 cm through the diaphragmatic hiatus. The left gastric vein is preserved. Next a circular stapler is introduced 4-6 cm into the oesophagus through an anterior gastrotomy. The circular stapler is fired allowing concomitant transaction and reanastomosis of the oesophagus. Pyloroplasty is then performed after reinforcing the oesophageal anastomosis. [75]

Patients with preserved liver function (Child A or B cirrhosis without ascites) should first be considered for a distal splenorenal shunt (DSRS). Modified Sugiura procedure should be considered when DSRS can not be done because of a previous splenectomy, small splenic vein or splenic vein thrombosis. [75] Mortality after modified Sugiura procedure is about 9% and rebleeding rate was 7%. [75]

In a study done in China postoperative mortality after elective modified Sugiura procedure was 0% and absolute rebleeding rate was 4% and incidence of hepatic coma was 2.5%. [76]

A study done in India compared modified devascularization procedures with or without oesophageal stapler transection combined with endoscopic surgery and pharmacotherapy

for control of variceal bleeding. Postoperative mortalities were 7.1% and 10.5%, rates of rebleeding were 7.1% and 7.8%; residual varices were 30.7% and 32.3%; recurrent varices were 7.6% and 5.8% following devasularization with oesophageal transection or without oesophageal transection respectively. [77]

Distal splenorenal (Warren) shunt (DSRS)

The distal splenorenal shunt (DSRS) was conceptualized and developed at the University of Miami by Warren and Zeppa with the first patient series reported in 1967. [71]

Candidates for DSRS are those with life threatening upper gastrointestinal bleeding but with minimal or no ascites and intact prograde portal flow to the liver (hepatopetal flow). DSRS properly performed in appropriately selected patients results in a marked decrease in both oesophagogastric variceal size and episodes of upper gastrointestinal bleeding. Maintenance of portal pressure and hepatopetal flow remains the central theoretical advantage of DSRS versus less selective shunts. [71,73,78]

DSRS (Warren) is performed by anastomosing the distal splenic vein to the side of the left renal vein. The portal venous system is thereby divided into a decompressed gastrosplenic venous circulation and a high pressure superior mesenteric venous circulation that maintains hepatoportal blood flow to the liver. The liver maintains portal perfusion and the incidence of encephalopathy is lower (10-15%). [33]

Worsening of ascites can occur because of dissecting and dividing the lymphatics near and around the left renal vein. DSRS should be avoided in patients with medically intractable ascites. The splenic vein should be 6-7 mm in diameter to avoid a high risk of shunt thrombosis. [33,73,74]

Patients with presinosoidal portal hypertension, preserved or stable hepatocellular function and UGIB may be best served by a DSRS as 20-year survivals exceeding 85% are observed. [33,71]

The outcomes are poor in patients who undergo DSRS and suffer ongoing hepatic injury, particularly who are actively consuming alcohol postoperatively. No late survivors after 10 years if alcohol use was ongoing. Similarly reduced survival is observed in patients with viral induced cirrhosis, likely due to ongoing liver destruction from viral infection. In these patients , the long term benefits of DSRS appears reduced and suggests a limited role for DSRS in these patients versus other modalities, especially liver transplantation. [71]

Non-steroidal anti-inflammatory drugs (NSAIDs)

Nonsteroidal anti-inflammatory drugs (including aspirin) are inextricably linked to peptic ulcer disease. Patients with rheumatoid arthritis and osteoarthritis who take NSAIDs have a 15 to 20% annual incidence of peptic ulcer, and the prevalence of peptic ulcer in chronic NSAID user is about 25% (15% gastric and 10% duodenal).

Complications of peptic ulcer disease (specifically haemorrhage and perforation) are much more common in patients taking NSAIDs. Many of these patients remain asymptomatic until they develop these life threatening complications. NSAIDs damage the gastrointestinal mucosa by multiple mechanisms. Topical injury occurs because NSAIDs are weak acids that remain in a non-ionized lipophilic form in the acidic gastric lumen. In this non-ionized state, they migrate through the gastric mucus into the surface epithelial cells, where they dissociate into the ionized form, causing epithelial acidification and injury. NSAIDs also decrease the hydrophobidity of the gastric mucus.

The greater toxicity from NSAIDs, however, is likely a result of inhibition of mucosal prostaglandin synthesis caused by systemic absorption of the agent resulting in suppression of gastric mucosal cyclooxygenase-1 (COX-1) activity. Prostaglandins are essential for mucosal bicarbonate and mucus secretion and are important agents of mucosal protection. NSAIDs have an inherent anti-platelet effect through inhibition of thromboxane synthesis and platelet aggregation, both COX-1 activities. [79,80]

The effect of four NSAIDs, aspirin, piroxicam, ibuprofen and indomethacin on the gastric mucosa was studied. Aspirin caused the worst lesions while piroxicam caused least lesions. [81]

Helicobacter pylori and NSAIDs consumption are independent and unrelated risk factors for upper gastrointestinal bleeding. Eradication of *H. pylori* is recommended before initiation of a long term aspirin administration in elderly patients. Low aspirin dosages are associated with high risk of ulcer bleeding.

The risk of upper gastrointestinal bleeding in elderly patients is significantly higher in the cases of acute abuse of NSAIDs relative to its chronic use. The simultaneous use of NSAIDs or aspirin and selective serotonin reuptake inhibitor antidepressants increase the risk of upper gastrointestinal bleeding. [82,83,84,85,86.]

Use of nonsteroidal anti-inflammatory drugs and low dose aspirin is accompanied by risk of upper and lower gastrointestinal tract complications. Management strategies to reduce this risk include gastro-protective pharmacotherapy, use of safer NSAIDs and eradication of *H. pylori* infection. [87]

In gastritis, duodenitis and gastroduodenal erosions linked to NAIDs use, circulatory support and stopping NSAIDs are required. Also most NSAID induced peptic ulcers heal after administration of a proton inhibitor or misoprostol. [19,88]

Endoscopic therapy is indicated when there is peptic ulcer with major stigmata of recent bleeding (active spurting bleeding, non bleeding visible vessel or non adhering blood clot). Endoscopic therapy can either be injection therapy (1:10,000 adrenaline), heater probe or a combination of the two. Rebleeding after endoscopic therapy occurs in 15% to 20% of cases. [19]

Mallory-Weiss tear

Kenneth Mallory and Soma Weiss first described the syndrome in 1929. The massive upper gastrointestinal bleeding results from a tear in the mucosa of the gastric cardia. This linear mucosal laceration is the result of forceful vomiting, retching, coughing or straining.

These actions create a rapid increase in the gradient between the intragastric and intrathoracic pressures, leading to a gastric mucosal tear from the forceful distention of the gastrooesophageal junction. [89]

In 80 to 90% of cases this is a single 1.75 to 2.5 cm mucosal tear along the lesser curve of the stomach just distal to the gastrooesophageal junction. [89]

Mallory-Weiss tears account for 0.5 to 17% of acute upper gastrointestinal haemorrhage. [8,90]

The bleeding from a Mallory-Weiss tear spontaneously ceases in 50 to 80% of patients by the time endoscopy is performed. [89,91]

For other patient whom bleeding is visualized at endoscopy, the endoscopic treatment options are electrocoagulation, heater probe application or sclerotherapy. [89,92]

Surgical intervention in Mallory-Weiss tear is required to achieve haemostasis in only 10% of cases. Bleeding from gastrooesophageal is visualized through an anterior gastrotomy. Once the tear is localized, the bleeding is controlled by oversewing the lesion. The overall mortality rates for patients who require emergent surgery is 15 to 25%. [89]

Dieulafoy lesion

Dieulafoy lesion, first described in 1896 by a French surgeon Paul Georges Dieulafoy, is a vascular malformation in the proximal stomach, usually within 6 cm of the gastrooesophageal junction along the lesser curvature of the stomach. Dieulafoy lesion is an uncommon aetiology of gastrointestinal haemorrhage recently estimated to represent 4% of the cases of upper gastrointestinal haemorrhage. [93]

Endoscopically the lesion appears as a large submucosal vessel that has become ulcerated. Because of the large size of the vessel, bleeding can be massive and brisk. The initial endoscopic management of Dieulafoy lesion can be highly successful; contact thermal ablation is the most effective technique. Endoscopic injection therapy is also an effective and safe therapeutic method this condition. [93,94]

Carcinoma of the stomach

Carcinoma of the stomach can present with upper gastrointestinal bleeding. Emergency surgery was associated with a higher mortality rate (42.9%) than elective procedures (13.3%) factors associated with a poor prognosis were active haemorrhage at initial endoscopy, an admission blood urea nitrogen greater than 16 mmol/L, preoperative

transfusion greater than 4 units, total transfusion greater than or equal to 9 units and a lowest recorded haemoglobin during hospitalization of less than 7.5g/dl. [95]

Rare causes of upper gastrointestinal bleeding

Primary aortoduodenal fistulas are among the rare causes of acute upper gastrointestinal haemorrhage and are defined as communications between the native abdominal aorta and the duodenum. The mortality rate is very high in undiagnosed and untreated. [96]

Gastric tuberculosis is very rare compared with other sites in the gastrointestinal tract and upper gastrointestinal bleeding is an extremely rare manifestation of gastric tuberculosis. Also pseudoaneurysm of the splenic artery secondary to gastric tuberculosis has been reported. [97]

Neurogenic tumours of the duodenum are extremely rare and represent an unusual cause of gastrointestinal haemorrhage.

The treatment is endoscopic or surgical excision. In case of ulcerated large tumours situated periampullary, pancreaticoduodenectomy (Whipple's) represent an efficient solution. [98]

Gastrointestinal stromal tumours (GISTs) are uncommon neoplasia arising from stromal tissue of the intestinal wall. Gastrointestinal stromal tumours are a rare cause of acute upper gastrointestinal bleeding. [99]

Massive upper gastrointestinal haemorrhage is a rare manifestation of gastrointestinal cytomegalovirus (CMV) infection in patients with immunodeficiency. [100,101,102]

Hemosuccus pancreaticus, a rare form of upper gastrointestinal bleeding, may complicate chronic pancreatitis and pose a significant diagnostic and therapeutic dilemma. Selective digestive angiography can provide the diagnosis. [103]

STATEMENT OF THE PROBLEM

Upper gastrointestinal tract bleeding has a high morbidity and mortality, being among the most important digestive emergencies. [3]

Literature in many countries shows that gastroduodenal peptic ulceration, severe lesions of gastric or duodenal mucosa and oesophageal variceal rupture were the most frequent reported causes of upper gastrointestinal haemorrhage. [5,14,15,19,21,22 ,23]

The annual incidence of hospitalization for acute upper gastrointestinal haemorrhage is around 50 to 150 per 100,000; increase with age and twice as high in males as in females. [11,17,18,19,22]

Three out of 1000 individuals have peptic ulcer every year and 20% of the ulcer episodes are associated with bleeding. [22]

Epidemiologic studies have demonstrated that the incidence of emergency surgery for upper GI bleeding has not changed despite major improvement with endoscopic treatment. There are no proven alternatives to emergency operation for massive bleeding uncontrolled by endoscopic procedures. [7,28]

Operative intervention still represents the most definitive treatment and remains the final therapeutic option for many bleeding lesions of the upper gastrointestinal tract. [2]

In Tanzania and many other developing countries endoscopic therapy is not widely available. This makes a surgeon play even a major role in the management of patients with severe upper gastrointestinal tract bleeding.

Acute upper gastrointestinal bleeding was found to be common in patients admitted in Muhimbili National Hospital.

In a pilot study done retrospectively by the investigator it was found that 101 patients were admitted over a year with upper gastrointestinal bleeding. In this pilot study mortality due to this problem was about 20% which is unacceptably high.

STUDY RATIONALE

In a comparison of fibreoptic endoscopy in acute upper gastrointestinal haemorrhage in Africans and Europeans, the incidence of gastric and duodenal ulceration was similar in the two races. Peptic ulcers were the main source of bleeding in both groups and were surprisingly more common than varices in the Africans. [104] However several studies in East African countries show ruptured oesophageal varices as the leading cause of upper gastrointestinal bleeding. [14,15]

This study will reveal the burden of upper gastrointestinal bleeding to the hospital and the relative frequency of various causes in Tanzania. It would hence justify more equipment to the endoscopy unit and training of doctors in gastroenterology and endoscopy, not only at MNH but also in other regional hospitals in the country.

In addition patients will eventually benefit by acquiring improved quality care.

This study was also done as a partial fulfillment for the award of Masters of Medicine in General Surgery at the Muhimbili University of Health and Allied Sciences.

STUDY OBJECTIVES

Broad objective

To study the profile of patients with acute upper gastrointestinal bleeding as seen at the Muhimbili National Hospital (MNH)

Specific Objectives

1. To document the causes of acute upper gastrointestinal bleeding as seen at the Muhimbili National Hospital.

2. To determine the correlation between splenomegaly and gastro-oesophageal varices as a cause of acute upper GI bleeding in patients seen at the MNH

3 To determine the mean number of units of blood transfused in patients with acute upper gastrointestinal bleeding at the MNH

4 To determine the mean hospital stay in patients with acute upper gastrointestinal bleeding at the MNH.

5 To determine the mortality in patients with acute upper gastrointestinal bleeding at the MNH.

METHODOLOGY

STUDY DESIGN

The study was a prospective descriptive study among patients who presented with acute upper gastrointestinal bleeding.

STUDY PERIOD

The study covered a period of eleven (11) months from February 2006 to December 2006.

STUDY AREA

The study was conducted in MNH in the departments of surgery and internal medicine, from both the male and female wards. Aga Khan Hospital was also included.

STUDY POPULATION

This study included all patients aged 12 years and above who presented with haematemesis and melaena or haematemesis with haematochezia

EXCLUSION CRITERIA

Patient who were younger than 12 years presenting with upper GI bleeding were excluded from the study. Patients with lesion(s) in the oral cavity or hypopharynx as a cause of bleeding were also not included in the study. Unconscious patients were also excluded from the study.

SAMPLE SIZE

Since this was a descriptive study the formula for sample size calculation could not be applied. The study included all consecutive patients with acute upper gastrointestinal bleeding admitted in both surgical and medical wards from February 2006 to December 2006. A total of seventy seven patients were included in the study.

DATA COLLECTION.

Data was collected using a structured questionnaire. All patients were followed up by the author until when discharged or death occurred.

DATA ANALYSIS AND DISSEMINATION

All data obtained was analyzed using SPSS (Statistical Package for the Social Sciences) 10 for Windows program.

ETHICAL CONSIDERATION

Both informed and written consent for the study were obtained from all patients to be included in the study. Ethical clearance was obtained from the ethical committee of Muhimbili University College of Health Sciences.

RESULTS

The study population

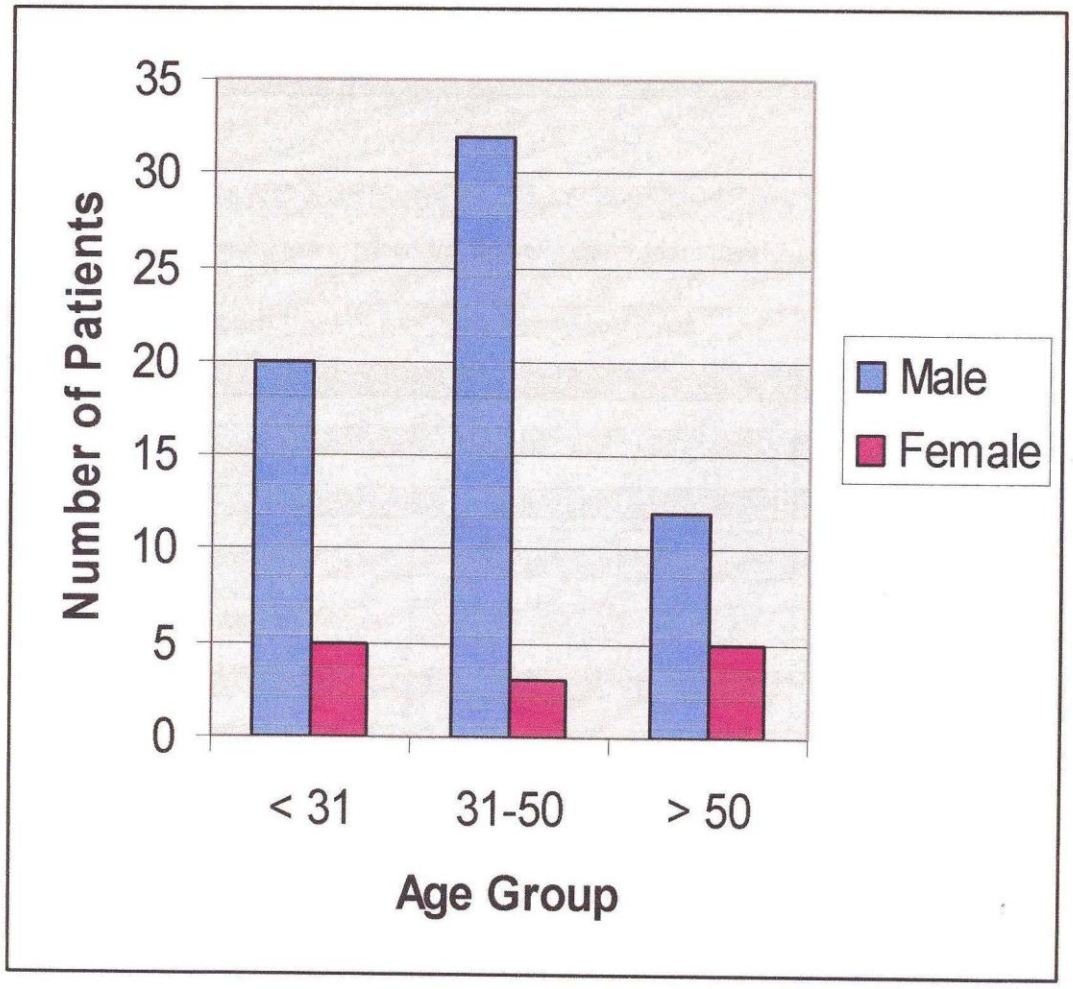
During the study period, between February 2006 and December 2006 a total of 77 patients who presented with acute upper gastrointestinal bleeding and admitted at MNH were enrolled into the study. Some of these patients were admitted in general surgical wards and others were admitted in medical wards.

Age and sex distribution

The patients' mean age was 40.48 years with a standard deviation of 17.04. The oldest patient was 89 years old and the youngest was 13 years old. The peak age groups were 31-50 years which included 35 patients (45.5%). Age group less than 31 years had 22 patients (32.5%). The age group more than 50 years accounted for the minimum number of patients (22.1%). There were a total of 64 males and 13 females (16.9%). The male to female ratio was 4.9:1.

The Bar chart below graphically summarizes the male preponderance and the peak ages.

Bar chart showing distribution of patients by age and sex.



Symptomatology

Most of the patients presented with both haematemesis and melaena, (63.6%), haematemesis only presented in 27.3% of patients and melaena only presented in minority of patients (2.6%). Haematemesis and/or melaena presented in 93.5% of all patients with acute upper GI bleeding.

Table1. The symptoms of acute upper gastrointestinal bleeding

Upper GI Bleeding symptom	**Number of patients**	**Percentages**
Haematemesis only	21	27.3
Melaena only	2	2.6
Haematemesis and melaena	49	63.6
Haematochezia	5	6.5
Total	77	100

Oesophagogastroduodenoscopy (OGD) was used as a standard diagnostic tool. Out of 77 patients who presented with upper GIT bleeding OGD was done in 67 of them. Oesophageal varices were found to be the leading cause of upper GI bleeding. Thirty seven patients had varices (48.1%). Duodenal ulcers were found in 17 patients (22.1%), gastric ulcers were found in 2 cases (2.6%) and gastric erosion in 2 cases (2.6%). OGD was normal in 5 patients (6.5%). Other diagnoses were reported in 4 cases, (5.2 %) of upper GI bleeding, these included 2 cases of gastric malignancies (confirmed by histology), a case of Mallory-Weiss tear and a case of oesophagitis. OGD was not done in 10 cases (13 %). The pie chart below summarizes these findings, highlighting varices as a major cause of UGIB.

Pie Chart showing causes of upper GI bleeding (Percentages)

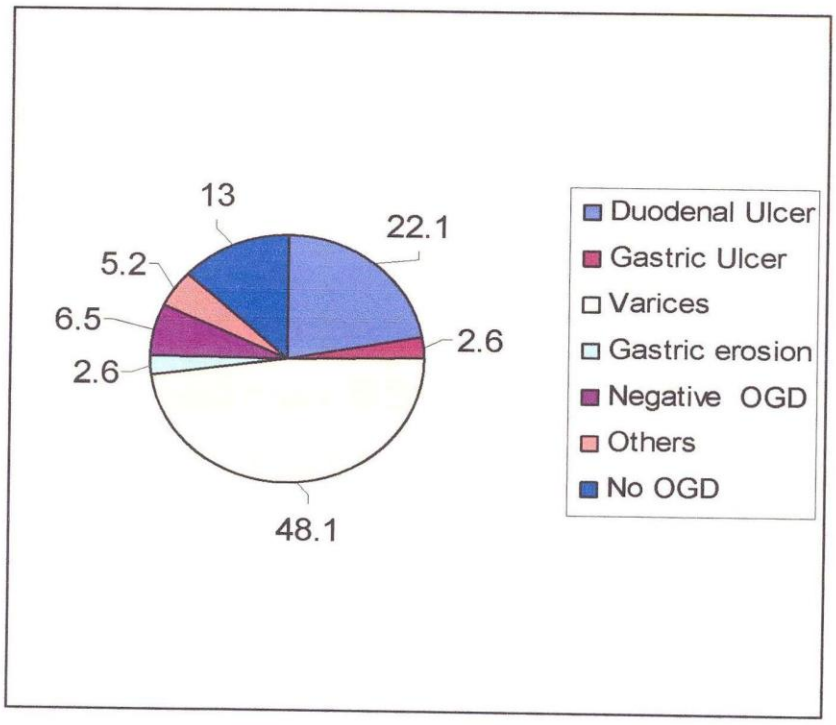


Table 2. Causes of upper GI bleeding with age

Age (Years)								Total
	DU	GU	Varices	Erosion	Normal	Others	No OGD	
<31	5 (29.4%)	0 (0%)	15 (40.5%)	1 (50%)	1 (20%)	0 (0%)	3 (30%)	25 (32.5%)
31-50	5 (29.4%)	1 (50%)	19 (51.4%)	1 (50%)	4 (80%)	1 (25%)	4 (40%)	35 (45.5%)
>50	7 (41.18%)	1 (50%)	3 (8.1%)	0 (0%)	0 (0%)	3 (75%))	3 (30%)	17 (22.1%)
Total	17 (100%)	2 (100%)	37 (100%)	2 (100%)	5 (100%)	4 (100%)	10 (100%)	77 (100%)

Duodenal ulcers were commonest in patients who were above 50 years of age, this age group contributed 7 cases (41.2%) no patient was found to be having gastric ulcer in the age group below 31 years, one case of gastric ulcer was found in age group31-50 years and more than 50 years as well. Oesophageal varices had a peak age of 31-40 years which contributed 51.4% of cases of varices followed by age group less 31 years (40.5%).

Table 3. Causes of upper GI bleeding with sex

Sex	Causes 0f UGIB							Total
	DU	GU	Varices	Erosion	Normal	Others	No OGD	
Male	15 (23.4%)	1 (1.6%)	32 (50%)	2 (3.1%)	5 (7.8%)	2 (3.1%)	7 (10.9%)	64 (100%)
Female	2 (15.4%)	1 (7.7%)	5 (38.5%)	0 (0%)	0 (0%)	2 (15.4%)	3 (23.1%)	13 (100%)
Total	17 (22.1%)	2 (2.6%)	37 (48.1%)	2 (2.6%)	5 (6.5%)	4 (5.2%)	10 (13%)	77 (100%)

Oesophageal varices were the leading cause of upper GI bleeding in both sexes. In males it accounted for 50% of all males with upper GI bleeding, and in females it accounted for 38.5% of all females with upper GI bleeding. Varices were followed by duodenal ulcer as a cause of acute upper GIT bleeding in both sexes, in males it accounted for 23.4% of all males and in females it accounted for 15.4% of all females.

Table 4: Correlation between splenomegaly and oesophageal varices

Spleen	Causes of UGI Bleeding		Total
	Varices	Other causes	
Palpable	34 (91.9%)	1 (3.3%)	35 (52.2%)
Not palpable	3 (8.1%)	29 (96.7%)	32 (47.8%)
Total	37 (100%)	30 (100%)	67 (100%)

Chi-square = 52.1

Degree of freedom = 1

P = 0.0000

Of all patients who had acute upper GIT bleeding and underwent OGD 52.2% had a palpable spleen and 47.8% had impalpable spleen. Those with palpable spleen 91.9% were confirmed to be having varices on endoscopy and those with impalpable spleen, 96.67% were found not having varices. The only patient who had palpable spleen and no oesophageal varices had upper GI bleeding due to a duodenal ulcer. The finding of splenomegaly in patients with oesophageal varices is statistically significant ($P < 0.001$).

Blood transfusion in patients with acute upper GI bleeding

On admission the mean haemoglobin level of patients with acute upper GI bleeding was 6.1gm/dl, minimum haemoglobin level was 2.5gm/dl and maximum haemoglobin was 15.7 gm/dl. The mean number of units of blood transfused was 2.9, the range being between no blood transfusion and maximum of 12 units, and standard deviation was 2.48. Following resuscitation or on discharge, haemoglobin levels were taken (control haemoglobin levels). The mean control haemoglobin level was 8.1 gm/dl, minimum control haemoglobin was 4.0 gm/dl and maximum control haemoglobin was 15.7 gm/dl

Table 5. Blood transfusion and cause of UGIB:

Units trans	Cause of upper GI bleeding							Total
	DU	GU	Varices	Erosion	Others	Normal OGD	No OGD	
0-3	14 (82.4%)	0	16 (43.2%)	2 (100%)	1 (25%)	5 (100%)	10 (100%)	48 (62.3%)
4-5	2 (11.8%)	2 (100%)	19 (51.4%)	0	3 (75%)	0		26 (33.8%)
≥ 6	1 (5.9%)	0	2 (5.4%)	0	0	0		3 (3.9%)
Total	17 (100%)	2 (100%)	37 (100%)	2 (100%)	4 (100%)	5 (100%)	10 (100%)	77 (100%)

Table 5 above shows that, patients with gastrooesophageal varices needed more blood transfusion as compared to other causes of upper GI bleeding. Most of the patients with varices (56.8%) needed 4 units of blood or more. Most patients with duodenal ulcer were transfused 0-3 units of blood (82.4%), only 5.9% of patients with duodenal ulcer were transfused more than 4 units of blood. In all patients who did not have OGD, blood transfused was 0-3 units.

Hospital stays in patients with acute upper GI bleeding

In patients admitted with acute upper GI bleeding the mean hospital stay was 12.9 days, the minimum hospital stay was less than a day and the maximum was 48 days, standard deviation was 12.2.

Table 6. Hospital stay and age

Age group (years)	Hospital stay (days)			Total
	<14	14-27	>27	
<31	15 (30%)	7 (38.9%)	3 (33.3%)	25 (32.5%)
31-50	23 (46%)	7 (38.9%)	5 (55.6%)	35 (45.5%)
>50	12 (24%)	4 (22.2%)	1 (11.1%)	17(22.1%)
Total	50 (100%)	18 (100%)	9 (100%)	77 (100%)

Fifty patients had a hospital stay of less than fourteen days, out of these, 23 patients (46%) were in age group 35-50 years, 15 patients (30%) were in age group less than 31 years and 12 patients (24%) were above 50 years of age. Of the patients who had a hospital stay between 14 and 27 days, majority were in age groups less than 31 years and 31-50 years each composed of 38.9%. Majority of patients who had a hospital stay of more than 27 days came from the age group 31-50 which consisted of 5 patients (55,6%), followed by age group less than 31 years (33.3%).

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Table7. Hospital stay and cause of upper GI bleeding.

Stay (days)	Cause of upper GI bleeding						Total
	DU	GU	Varices	Erosion	Normal	Others	
<14	13 (76.5%)	2 (100%)	15 (40.5%)	2 (100%)	5 (100%)	3 (75%)	40 (59.7%)
14-27	3 (17.6%)	0	14 (37.8%)	(0%)	0 (0%)	1 (25%)	18 (26.9%)
> 27	1 (5.9%)	0 (0%)	8 (21.6%)	0 (0%)	0 (0%)	(0%)	9 (13.4%)
Total	17 (100%)	2 (100%)	37 (100%)	2 (100%)	5 (100%)	4 (100%)	67 (100%)

The hospital stay was prolonged in patients who had gastrooesophageal varices. Out of 37 patients with varices, 15 (40.5%) had a hospital stay of less than 14 days and 14 patients (37.8%) had a hospital stay between 14 and 27 days. Out of 17 patients who had duodenal ulcers 13 (76.5%) had a hospital stay of less than 14 days.

Mortality in patients with acute upper GI bleeding

Table 8. Outcome of patients admitted with acute upper GI bleeding

Outcome	Frequency	Percentage
Discharged	63	81.8
Absconded	2	2.6
Died	12	15.6
Total	77	100

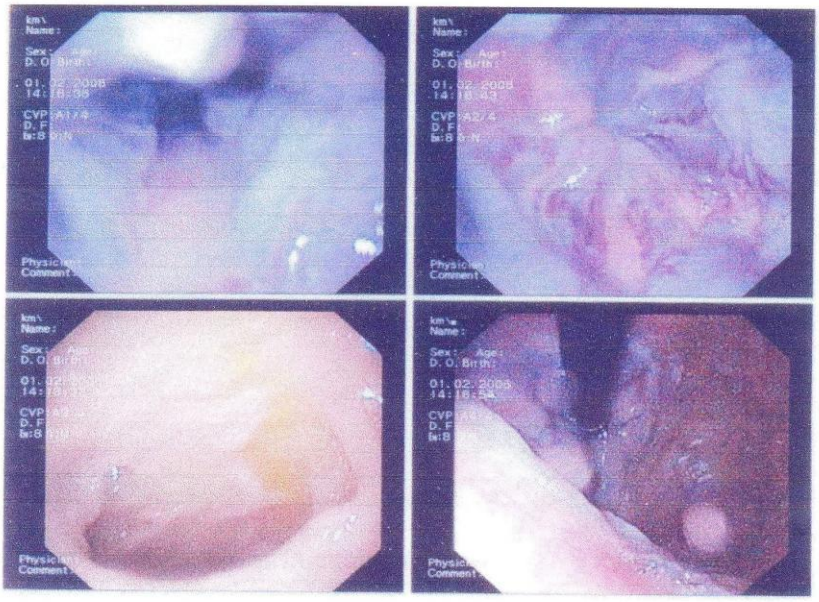
A total of 77 patients with acute upper GI bleeding were admitted. Sixty three patients (81.8%) were discharged home and 2.6% went home against medical advice. Twelve patients died giving a mortality rate of 15.6%.

Table 9. Outcome of acute upper GI bleeding and cause of upper GIB

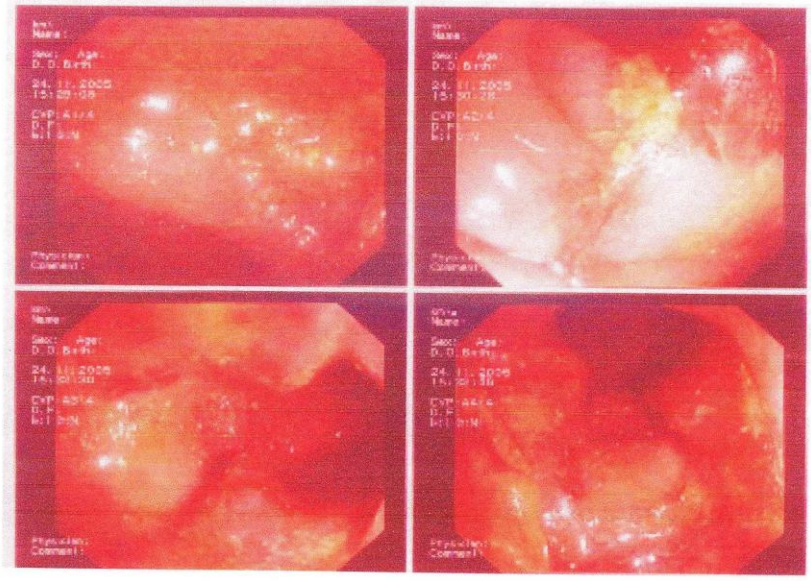
Cause	Discharged	Absconded	Died	Total
Duodenal ulcer	16 (25.4%)	0 (0%)	1 (8.3)	17 (22.1%)
Gastric ulcer	2 (3.2%)	0 (0%)	0 (0%)	2 (2.6%)
Varices	34 (54%)	0 (0%)	3 (25%)	37 (48.1%)
Gastric erosions	2 (3.2%)	0 (0%)	0 (0%)	2 (2.6%)
Normal OGD	4 (6.3%)	0 (0%)	1 (8.3%)	5 (6.5%)
Others	2 (3.2%)	2 (100%)	0 (0%)	4 (5.2%)
OGD not done	3 (4.8%)	0 (0%)	7 (58.3%)	10 (13%)
Total	63 (100%)	2 (100%)	12 (100%)	77 (100%)

Majority of patients died before endoscopy was done, these were 7 out of 12 deaths (58.3%). Most of these patients were too sick for endoscopy to be done. One patient out of the 12 (8.3%) who died had duodenal ulcer. Three patients (25%) with varices died.

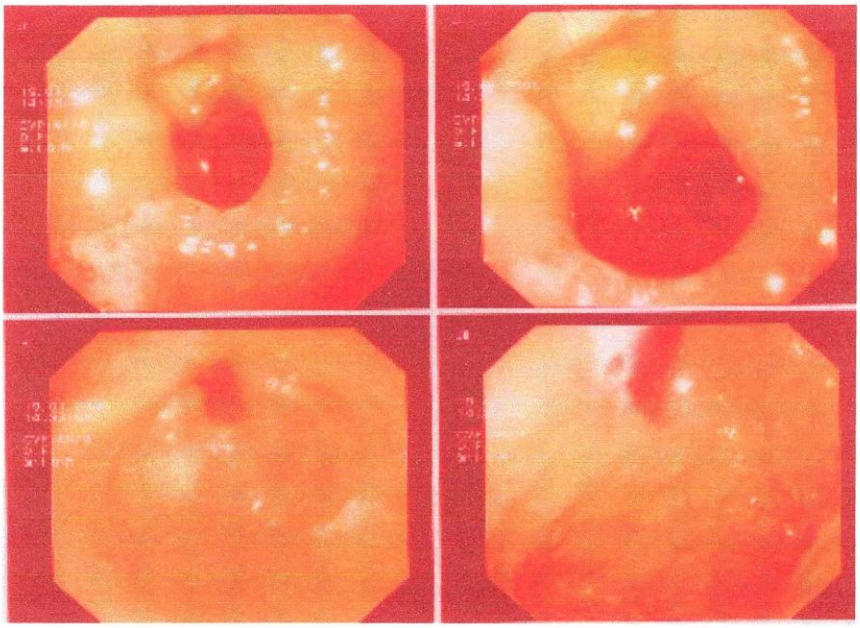
Illustrations



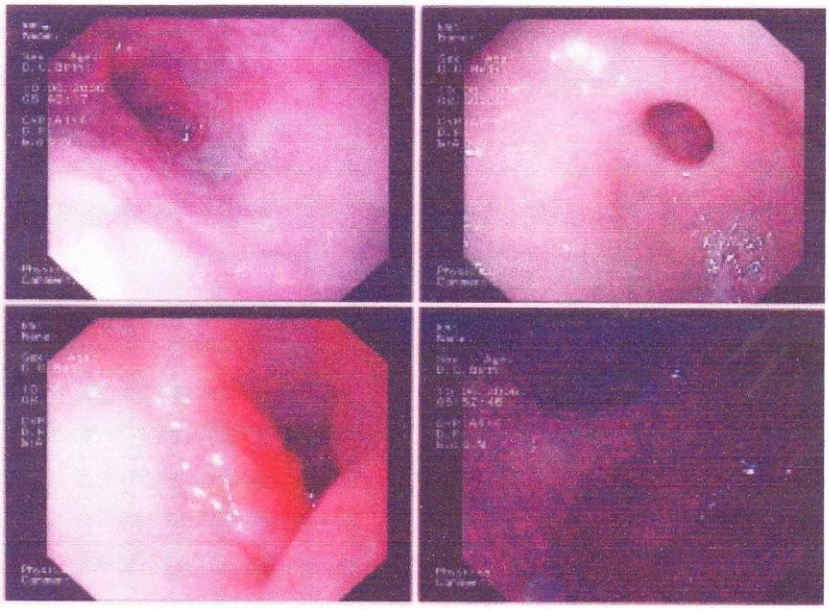
Endoscopy picture showing large blue varices almost filling the whole lumen



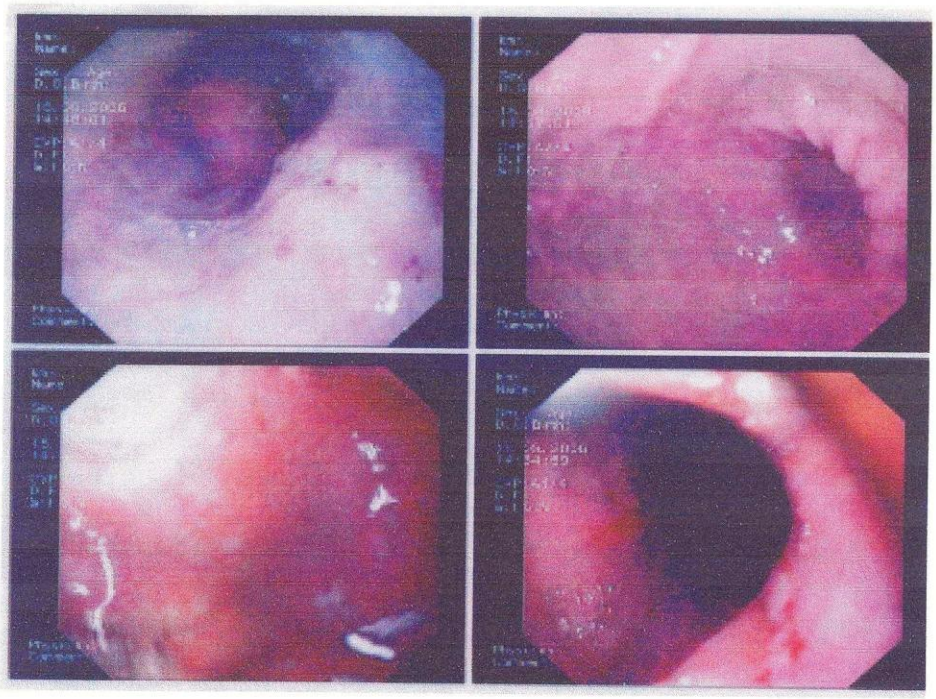
Endoscopy showing duodenal ulcer



Endoscopy showing duodenal ulcer with a clean base



OGD done a week after haematemesis showing a healing ulcer with duodenitis



Patient had haematemesis preceded with vomiting, OGD showing Mallory-Weiss tear.

DISCUSSION

The commonest causes of acute upper gastrointestinal tract bleeding include peptic ulcer disease, ruptured oesophageal varices and gastroduodenal erosive lesions. There is limited data on the profile of acute UGI bleeding in Tanzania and many patients are managed conservatively due to lack of diagnostic and therapeutic endoscopic services. This study focused on the profile of patients with acute UGI bleeding as seen at Muhimbili National Hospital, Dar es Salaam, Tanzania. MNH, apart from being Muhimbili University of Health and Allied Sciences (MUHAS) teaching hospital, was selected as a study area because it is the largest referral and national hospital in Tanzania attending to referral patients from Dar es Salaam city and upcountry districts.

A total of 77 patients were included in the study, males were 83.1% and females were 16.9% making a male to female ratio of 4.9:1. These results concur with the ones which were found in Sudan where the male: female was about 5:1[16]

However these results differ from the ones which were found in United States of America where the male to female ratio was 2:1.[21] the explanation for these is that, in both Tanzania and Sudan, the leading cause of acute UGIB was oesophageal varices, in Sudan schistosomiasis was confirmed as the cause of portal hypertension in most patients, the same may apply in Tanzania because schistosomiasis is endemic in many regions of Tanzania.

Young boys are more likely to get infested by schistosoma because they often are more exposed when swimming or fishing in dams which are habitats for snails which are intermediate host of schistosoma worms.

The majority of patients who had acute upper gastrointestinal bleeding presented with haematemesis, melaena or both (93.5%). Haematemesis alone was a presenting symptom in 63.6% of patients. These results are almost consistent with the results found in literature, whereby haematemesis was a presentation in 40% to 50%, melaena was a presentation in 70-80% of patients with acute upper GI bleeding. [2,9]

In all 67 patients who had upper GI endoscopy, the procedure was done after more than 24 hours of admission except in only one patient. This is contrary to the standard teaching or protocols; endoscopy within the first 24 hours of admission is a standard therapy for the management of the initial haemorrhage.[10] Early endoscopy and endoscopic treatment is associated with reductions in the length of hospital stay, rate of recurrent bleeding and need for emergent surgical intervention. [6,10,34,35]

The reasons for delay in having endoscopy are;

- Absence of endoscopic services at MNH in the first 6 months of the study, although some had OGD in private hospitals many needed longer time to raise funds for that.
- Inadequate equipment and staff in the new endoscopy unit which was established 5 months before the end of data collection.

  The new unit has only one upper GI endoscope and one endoscopist who is also a full time surgeon, hence endoscopies are done once a week on an elective basis. In addition no accessories for endoscopic therapy are available.
- Haemodynamically unstable patients. Some of the patients were admitted in shock and it took a long time to stabilize them for endoscopy. The delay and even

mortality was also contributed by unavailability of adequate amount of blood to resuscitate these patients.

Out of 77 patients who had acute upper GI bleeding, 48.1% had oesophageal varices as the source of bleeding, followed by duodenal ulcer which accounted for 22.1% of all cases of upper GI bleeding. These results are closer to results of several studies which were done in neighboring African countries. In a study done in Malawi, oesophageal varices was the leading cause of acute upper GI bleeding and accounted for 45% of cases, duodenal ulcer accounted for 16% of all cases of upper GI bleeding. In a study done in Kenya oesophageal varices was a leading cause of upper GI bleeding comprising 35% of all cases. [14,15,] However these results are different from the results of studies done in Europe and America where peptic ulcer disease contributes the majority of cases of acute upper GI bleeding (about 35% to 50%), oesophagitis (20% to 30%) duodenitis/gastritis/erosions (10% to 20%). [19,21,22,23]

The difference in causes of upper GIB between Tanzania and those in developed countries can be explained by the fact that many regions in Tanzania are endemic for schistosomiasis; however it is more likely that some of the patients in the study could have cirrhosis secondary to viral hepatitis. In developed countries with a more aging population, patients use NSAID for different problems like arthritis and aspirin for cardiac diseases, predisposing them to ulcers and erosions. Hence most patients in this study had varices while in developed countries most cases of upper GI bleeding are due to peptic ulcer and gastroduodenal erosive lesions.

A palpable spleen was found in 91.9% of patients of patients who had oesophageal varices. The spleen was impalpable in 96.7% of patients who did not have varices. The

association between splenomegaly and varices was found to be highly statistically significant ($P > 0.001$). This is consistent with the study from Malawi, which revealed that, a palpable spleen positively predicted oesophageal varices in 91% of cases, while no palpable spleen predicted a source of bleeding other than varices in 97% of cases. [15] This is an important finding even in a part of the world where malaria is endemic and can be useful clinical indicator of varices in UGIB.

On admission the mean haemoglobin level of patients with acute upper GI bleeding was 6.1gm/dl, with a minimum haemoglobin level of 2.5gm/dl. The mean number of units of blood transfused was about 3 (2.9). The mean control haemoglobin level was 8.1gm/dl, with a minimum control haemoglobin level of 4.0gm/dl. However the mean control haemoglobin level was still lower than the expected control found in the literature, of 10gm/dl.[6,10]

This is explained by constraints the blood bank at MNH is facing. In most cases blood was not available when needed emergently. Most of the patients (83.7%) were admitted with a haemoglobin level of 7gm/dl or less. With a mean blood transfusion of 3 units most of these patients had ended up with control haemoglobin of less than the recommended 10gm/dl. Actually some of these patients were discharged with severe anaemia.

The study revealed that patients with oesophageal varices needed more blood transfusion as compared to other causes of acute upper GI bleeding. This concurs with findings from other studies. Bleeding in oesophageal varices is often more severe than bleeding from other sources. [6]

In this study the mean hospital stay in patients with AUGIB was found to be 12.9 days with a standard deviation of 12.2. Hospital stay was prolonged in patients with oesophageal varices as compared to other causes of UGIB. In patients with duodenal ulcer, majority (76.5%) had a hospital stay of less than 14 days. The hospital stay was prolonged in patients with oesophageal varices because it is likely these had more severe bleeding as compared to other causes. [6]

Unfortunately the high demands of blood transfusion were not met by the blood bank at MNH, hence the prolonged hospital stay.

The outcome of patients with upper gastrointestinal haemorrhage is greatly influenced by recurrence of bleeding. Other independent risk factors for poor outcome include age older than 60 years, ongoing bleeding and hypotension (systolic blood pressure less than 100 mmHg). But it may be possible to identify patients who has a low risk for bleeding and can be discharged after a short hospitalization. In many studies the mean hospital stay of patients with upper GI bleeding was about 7 to 8 days.

With early discharge protocol length of hospital stay was reduced to about 3 days leading to important cost saving. [105,106,107]

Overall mortality due to acute upper gastrointestinal bleeding was found to be 15.6%. This is slightly higher compared to mortality in other studies which is around 10%.The finding of a higher mortality in this study can be explained by the fact that there is no therapeutic endoscopy at the MNH, the endoscopy unit was established recently with only a single diagnostic upper GI endoscope. About 20% of patients with upper GI bleeding rebleed and most do so within the first 24 hours after the first bleed. Endoscopic therapy within the first 24 hours can control bleeding in more than 80% of cases, hence

reduce the rate of rebleeding, hospital stay, the need for emergent surgery and mortality. [17,34,35]

The mortality rate in patients with oesophageal varices admitted at MNH was 8.1%, which is lower than what is reported in the literature. The explanation for a lower mortality due to variceal bleeding found in this study as compared to other studies where mortality was about 30% to 50% is that most of patients at the MNH with varices could have been having portal hypertension due to schistosomal periportal fibrosis. These patients usually have a good prognosis because liver function is usually normal in most patients. [21,33,51,57,58.]

CONCLUSIONS

- Acute upper GI bleeding is more prevalent in males than in females, with a male to female ratio of 4.9:1.
- Haematemesis and/or melaena are the most common symptoms and presented in 93.5% of patients with acute upper gastrointestinal bleeding.
- Oesophageal varices are an important cause of upper GI bleeding in Tanzania, comprising of 48.1% of all cases followed by duodenal ulcers (22.1%).
- Splenomegaly is found highly to correlate with the finding of oesophageal varices as the cause of upper GI bleeding. The correlation is statistically significant with a P value of less than 0.001.
- Blood transfusion is in high demand in patients with acute upper GI bleeding. Blood bank at MNH failed to meet this high demand of blood transfusion in patients with upper GI bleeding.
- The mean number of units of blood transfused in patients with acute upper GI bleeding admitted at MNH was 2.9. Blood bank at MNH failed to meet high demand of blood transfusion in patients with upper GI bleeding.
- The mean hospital stay in patients admitted with upper GI bleeding at the MNH was 12.9 days, which was prolonged compared to other studies. The hospital stay was more prolonged in patients with varices compared to other causes.
- Overall mortality in patients with upper GI bleeding at the MNH is higher as compared to other studies. At MNH the mortality is 15.6% while it is around 10% in most other studies.

LIMITATIONS OF THE STUDY

- Absence of endoscopy services at MNH in the initial course of the study, this lead to some patients failing to have endoscopy while others had it done much later.
- Absence of emergency endoscopy at MNH, this led to a long waiting times, some patients died while waiting for endoscopy while others were started on empirical treatment and later on endoscopy revealed normal findings.

RECOMMENDATIONS

- Since varices are such a common cause of acute UGIB, the actual causes of liver pathology in Tanzania need to be studied thoroughly and highlighted, since both HBV disease and schistosomiasis although difficult, are potentially preventable diseases.
- Peptic ulcers should be diagnosed early by OGD/H. pylori studies and treated to prevent complications.
- NSAIDs should be used with caution, and when necessary should be used with misoprotol or PPIs
- Public education should be given on the different causes of upper GI bleeding to avoid the preventable causes and to seek medical attention early before complication of upper GI bleeding occurs.

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