Causes of Upper Gastrointestinal
Bleeding in Sudanese Patients

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Dedication

To the soul of my parents…

To Tyseer …

To Ahmed …

And all my friends …

Tariq
## LIST OF CONTENT

<table>
<thead>
<tr>
<th>Subject</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acknowledgement</td>
<td>i</td>
</tr>
<tr>
<td>Abstract English</td>
<td>ii</td>
</tr>
<tr>
<td>Abstract Arabic</td>
<td>iv</td>
</tr>
<tr>
<td>Abbreviation</td>
<td>v</td>
</tr>
<tr>
<td>List of tables</td>
<td>vi</td>
</tr>
<tr>
<td>List of figures</td>
<td>vii</td>
</tr>
</tbody>
</table>

**Chapter One**

Introduction & Literature Review 1

Objectives 29

**Chapter Two**

Patients and Methods 30

**Chapter Three**

Result 34

Discussion 60

Conclusion 64

Recommendation 65

References 66

Appendix (Questionnaire)
ACKNOWLEDGMENT

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I am greatly indebted to a lot of my colleagues who filled the questionnaire and helped me in preparation of this work.

My utmost thanks to the dear friend Mr. Adil Amin for his patience, co-operation and statistical guiding.
ABSTRACT

Upper gastrointestinal bleeding in Sudan is a common problem with significant mortality and morbidity and consumption of already scarce medical resources in our country.

This study was conducted in Khartoum province, in the main three Tertiary hospitals; Khartoum teaching hospital; Ibn Sina specialized hospital and Soba university hospital. One hundred cases were studied in each hospital in the period from October 2002 to August 2003.

The aim of the study was to know the potential causes of upper gastrointestinal tract bleeding in Sudanese patients. Important investigations were done to the patients. These include: oesophagastroduod-endoscopy, abdominal ultrasonography, liver function tests prothrombin time, complete blood count with peripheral blood picture.

The study showed the dominance of the variceal causes, mainly due to bleeding oesophageal varices, resulting from schistosomal periportal fibrosis which contributed to 76% of the causes. Most of the patients were from Gezira area which is endemic for schistosomiasis.

Peptic ulcer disease accounted for 9.7% of the cases. Other causes found were gastric erosion (2%), carcinoma of the stomach (2.7%), and Mallory-Weiss (0.7%). Other rare causes found in this study include: Dieulafoy lesions (0.7%), watermelon stomach (1%), duodenal tumors, and A-V malformations (0.3%).

There is a rise in the percentage of patients with bleeding oesophageal varices compared to a previous study done almost twenty years ago. Also, this study shows a decrease in the percentage of patients with bleeding peptic ulcers.
خلاصة الأطروحة

النزيف الحاد من الجهاز الهضمي العلوي من المشاكل الشائعة في السودان بما له من تأثير مباشر على الحياة ونوعيتها واستنزاف الموارد الطبية المتاحة على قلتها، هذه دراسة مستقبيلة مقطعية وصفية أجريت بالمستشفيات التالية: مستشفى الخرطوم ومستشفى ابن سينا التخصصي ومستشفى سوبا الجامعي، حيث درست حالة ثلاثمئة مريض، مائة مريض من كل مستشفى من هذه المستشفيات الثلاثة.

أجريت هذه الدراسة في الفترة من أكتوبر 2002م وحتى أغسطس 2003م، الهدف من الدراسة هو معرفة أسباب النزيف من الجهاز الهضمي العلوي في المرضى السودانيين، أهم هذه الفحوصات تشخيص هذه الحالات كان منظار المرئي والمعدة والأثني عشر والموجات فوق الصوتية للبنط ووظائف الكبد وزمن البروتوربين وفحص الدم العام.

هذه الدراسة أوضحت أن أكثر الأسباب شيوعا كان النزيف من دوالي المرئي حيث كان السبب في إحدى وسبعين في المائة من الحالات بسبب التليف حول الوريد الباليبي بسبب داء المنشقات، غالبية الحالات من المنطقة المبوءة بهذا الداء.

القرح المعدة كانت السبب في 9.7% من الحالات وأسباب الأخرى كانت على النحو التالي: سرطان المعدة 2.7% تعرية مخاطية المعدة 2% وهناك أسباب نادرة مثل المعدة البطيخية 1% داء ملري فايز 2% أورام المرئ 0.7% والتشوهات الوريدية 0.3%.

هذه الدراسة أوضحت أن نسبة المرضى بالنزيف بسبب الدوالي المرئية في ازدياد مقارنة بالدراسات السابقة وأيضا أوضحت النزيف من القرح قد تنافصت.
# ABBREVIATIONS

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tr>
<td>ASGE</td>
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<tr>
<td>CRS</td>
<td>Cherry Red Spots</td>
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<tr>
<td>DIC</td>
<td>Disseminated Intravascular Co-agulopathy</td>
</tr>
<tr>
<td>DR</td>
<td>Diffuse Redness</td>
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<tr>
<td>DU</td>
<td>Duodenal Ulcer</td>
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<td>EUS</td>
<td>Endoscopic Ultra Sonography</td>
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<td>GI</td>
<td>Gastrointestinal</td>
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<td>GV</td>
<td>Gastric Varices</td>
</tr>
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<td>HCS</td>
<td>Haematocystic Spots</td>
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<td>HVPG</td>
<td>Hepatic Venous Pressure Gradient</td>
</tr>
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<td>NIEC</td>
<td>North Italian Endoscopic Club</td>
</tr>
<tr>
<td>NSAIDs</td>
<td>Non Steroidal Anti Inflammatory Drugs</td>
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<tr>
<td>OV</td>
<td>Oesophageal Bleeding</td>
</tr>
<tr>
<td>PPI</td>
<td>Proton Pump Inhibitor</td>
</tr>
<tr>
<td>PT</td>
<td>Prothrombin Time</td>
</tr>
<tr>
<td>PVT</td>
<td>Portal Vein Thrombosis</td>
</tr>
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<td>RWM</td>
<td>Red Wale Markings</td>
</tr>
<tr>
<td><strong>UGIB</strong></td>
<td>Upper Gastrointestinal Bleeding</td>
</tr>
<tr>
<td>WHO</td>
<td>World Health Organization</td>
</tr>
</tbody>
</table>
LIST OF TABLES

Table 1: Distribution of patients according to age group
Table 2: Distribution of patients according to presenting complain and past medical history
Table 3: Distribution of patients according to physical examination findings
Table 4: Distribution of patients according to haematological findings
Table 5: Distribution of patients according to liver function test and prothrombin time
Table 6: Distribution of patients according to their ultrasonographic findings and endoscopy
LIST OF FIGURES

Figure 1: Distribution of patients according to gender
Figure 2: Distribution of patients according to residence
Figure 3: Distribution of patients according to occupation
Figure 4: Distribution of patients according to past medical history of schistosomiasis in PPF
Figure 5: Relationship between age group and gender in the study population
Figure 6: Relationship between diagnosis and vomiting blood among the study population
Figure 7: Distribution of patients according to serology for both hepatitis B and C viruses in patients with pure schistosomal periportal fibrosis
Figure 8: Distribution of patients with liver involvement according to ultrasonography
Figure 9: Distribution of patients according to endoscopic finding
Figure 10: Diagnosis of variceal causes according to the underlining pathology
Figure 11: Distribution of the variceal causes UGIB according to type of varices
Figure 12: Distribution of non-variceal causes UGIB according to endoscopic findings
INTRODUCTION & LITERATURE REVIEW

Upper gastrointestinal bleeding (UGIB) was firstly described *as ground coffee and melena by Galen Claudius in 200 A.D.\(^{(1)}\) Since that time it remains a common medical presentation for patients seen by gastroenterologist. It is associated with significant morbidity, mortality and use of health resources.

It is estimated that more than 300,000 hospital admissions for UGIB occur annually in USA. The annual rate of hospitalisation for upper gastrointestinal bleeding has been estimated at 36 to 102 patient per 100,000 of the general population and is twice as common in men compared to women. It causes an overall mortality rate of approximately 10\%(2,3), and it costs about 15-80,000 dollar per patient per year.\(^{(4)}\)

Mortality ranges from 1-7\% for bleeding duodenal ulcer, 7-16\% for bleeding gastric ulcer and 22-63\% for bleeding oesophageal varices, and it is usually high among advanced age group.\(^{(5)}\)

Upper gastrointestinal bleeding may present with hematemesis, melena, and hematochezia, or in form of signs and symptoms of iron deficiency anaemia due to occult blood loss.

Definitions:

- Haematemesis is vomiting fresh red blood.\(^{(6)}\)
- Coffee ground vomiting is vomiting of altered black blood.\(^{(6)}\)
- Melena is the passage of tarry stools.\(^{(6)}\)
- Haematochezia is the passage of red blood per rectum; this is usually due to bleeding from the lower gastrointestinal tract but occasionally can be due to massive upper gastrointestinal bleeding. It indicates at least 1000ml of blood\textsuperscript{(7)} general, patients who present with haematemesis and melena have more severe bleeding than those who present with melena alone.

**Pathophysiology & Clinical features of upper gastrointestinal bleeding (UGIB):**

- The colour of vomited blood depends on the concentration of hydrochloric acid in the stomach in addition to the duration of its contact with the blood.\textsuperscript{(6)} When vomiting takes place shortly after the onset of bleeding it will appear dark red, and when it stays longer it will appear brown or black. The coffee ground appearance is due to the action of hydrochloric acid on hemoglobin which will change to hematin giving it this characteristic appearance.

- Hematemesis usually indicates UGIB as a cause, because bleeding distal to the duodenum rarely enters the stomach.

- Almost all patients with hematemesis have melena, but only half of patient with melena have hematemesis\textsuperscript{(6)}. This is because melena usually occurs in both UGIB and bleeding down to the ascending colon specially when the transit time is sufficiently prolonged\textsuperscript{(6)}. The black colour of melena results from contact of the blood with hydrochloric acid to produce hematin.\textsuperscript{(6)} About 50-60 ml of blood may produce
melena for about seven days and a positive test for occult blood for seven days after the stool colour change back to normal.\textsuperscript{(6)}

Positive results for occult blood loss may indicate a serious disease and should be thoroughly investigated.\textsuperscript{(6)}

- Black stool that is negative for occult blood may result from ingestion of iron, bismuth, or various formulae and should not be mistaken for melena.\textsuperscript{(7)}

- The manifestations of UGIB depend on the source, rate of bleeding, and underlying or coexistent disease; e.g. a patient with underlying Ischemic heart disease may present with angina or MI after brisk UGIB. Coexistent heart failure, hypertension, pulmonary disease, renal failure and diabetes mellitus may be aggravated by severe GI bleeding, which may present as shock. Lesser degrees of bleeding may manifest as orthostatic changes in pulse (a change $> 10$ beat/min) or BP (a drop of $\geq 10$mmHg). Orthostatic changes should be interpreted with caution in patients with underlying heart disease or peripheral vascular disease or in those taking drugs known to influence peripheral vascular resistance\textsuperscript{(6)}. UGIB of 60 ml gives only melena. Bleeding of 500 ml is rarely associated with systemic signs; exception are mentioned (elderly, coexistent heart disease or anaemia). Rapid haemorrhage of greater volumes results in decreased venous return to the heart, decreased cardiac output, and increased peripheral resistance due to reflex
vasoconstriction. Orthostatic hypotension greater than a change of 10mmHg usually indicate a 20 percent or greater of blood loss.

**Causes of upper gastrointestinal bleeding**

The causes of UGIB are many and are different according to the geographical area. The commonest cause of UGIB in the United States is peptic ulcer disease which accounts for more than 50 percent of the causes, this is followed by gastric erosion, and variceal bleeding.(7) In the United Kingdom the commonest cause of again is peptic ulcer causing 30 to 50 percent of all causes of UGIB, but interestingly no cause was identified no in 24%. (8) of the cases.

Here in Sudan, which is a tropical country, however, bleeding due to oesophageal varices is the commonest cause. (9) The causes of UGIB can be divided into two groups:

A. Variceal causes

B. Non-variceal causes

**Variceal Causes of UGIB**

Anatomical & physiological and pathological consideration:
The portal vein is formed by the confluence of the superior mesenteric and splenic veins and it is about 6-8cm in length and has a mean diameter of 12 mm.

The superior mesenteric vein receives blood from the small intestine, colon and the head of the pancreas. The splenic vein is formed from several tributaries originating at the splenic hilum, so receiving blood from pancreas and left gastroepipoic and inferior mesenteric vein and the short gastric veins from the fundus of the stomach.(10)

The portal blood flow is approximately 1 liter per minute, the mean pressure is about 7 mm Hg, and 70% of the hepatic oxygen is supplied by the portal vein.(11)

The level of portal pressure is determined by flow rate and vascular resistance. The fundamental abnormality in almost all forms of portal hypertension is increased resistance.(11) In portal hypertension there is a rise in portal pressure and a collateral circulation develops, diverting the portal flow into systemic veins. Thus while in normal circumstances 100% of portal blood flows through the liver, in cirrhosis with a severe intrahepatic block to portal venous flow, only approximately 10% of the blood flow reaches the hepatic veins, and the rest circumvents the liver via the collateral circulation.(11)

It is contravertial whether the opening of these collaterals decrease the level of hypertension. One Study shows that do not decrease portal hypertension.(11) Others believe that the development of these collaterals decrease the level of portal
hypertension. The liver is depends mainly on the hepatic artery for oxygen and nutrient so, the liver is shrunken with impaired ability to regenerate.\textsuperscript{(14)} Many of these collateral systems are clinically benign, but the most important and dangerous are gastroesophageal collaterals, so-called varices. A part from gastroesophageal varices, similar collateral develop between the inferior mesenteric vein and the haemorrhoidal vein, and the umbilical veins and the cutaneous vein of the abdominal wall. Similarly, blood from the portal system drains in a retrograde fashion via the left renal vein, the gonadal veins and many retroperitoneal veins drain into the azygos system and the vena cavae. Collaterals also develop at the sites of previous surgery or inflammation and frequently around an ileostomy or colostomy stoma.

Longstanding portal hypertension is not only the cause of discrete collaterals with dilated and tortuous varices, but also causes mucosal changes due to abnormalities in the microcirculation. Thus portal gastropathy is well recognized with vascular ectasia of the gastricmucosa.\textsuperscript{(12)}

Similar changes have been described in the small and large intestines of patients with portal hypertension. Such changes contribute to occult gastrointestinal bleeding in these patients.

As indicated, gastroesophageal varices are clinically the most important collateral to develop. There are two main inflows, from the left gastric vein and from the splenic hilum through the short gastric veins. Interestingly, the finding of isolated varices in the
gastric fundus raises the question of a block in the splenic vein with the formation of collateral only via the short gastric vessels. Oesophageal varices are fed principally by the reversed flow in the left gastric vein.

Regarding the formation of the oesophageal varices, normally there are four layers of veins in the lower esophagus. The intraepithelial veins and superficial venous plexus, the deep intrinsic venous plexus, perforating vein and the adventitial veins. The veins within the epithelium form "Cherry red spots" under condition of portal hypertension; this predict hemorrhage. Typically, large varices occur when portal hypertension causes dilatation and tortuosity of the deep intrinsic veins in this region where portal and systemic circulation connect. The venous system in the gastroesophageal region has been classified into zones: The truncal zone, the perforating zone the palisade zone, and the gastric zone. The palisade zone is believed to be the water shed between the portal and systemic system. Turbulent flow in the veins of the perforating zone with thining of the muscularis mucosa lead to rupture of varices in this region frequently.

Recurrence of varices after endoscopic sclerotherapy may be related to the communications between various venous channels or perhaps to the enlargement of veins in the superficial venous plexus. Failure of sclerotherapy may also be due to failure to thrombose the perforating veins.

Although gastric varices are mainly confined to the cardia, subtle intramucosal vascular abnormalities have been described
throughout the stomach in patients with portal hypertension. Ectatic capillaries and venules are seen in the lamina propria and they communicate with deep vessels. Microthrombi may be present and there may be increased numbers of vertically orientated smooth muscle fibres in the mucosa. This is referred to as portal hypertensive gastropathy. Gross lesions in the antrum have been described in which the striking endoscopic appearances led to the term 'water melon stomach'; longitudinal rugal folds which contain dilated vessels that converge on the pylorus.

Varix: Is a diated and tortuous vein usually on intrinsic vein.

Pathophysiology of gastroesophageal varices:
Cirrhosis accounts for up to 90 percent of the causes of portal hypertension in North America and Europe. In the Sudan, prevalence of infection with schistosoma mansoni and periportal fibrosis in areas not covered by control programs reaches 70% and 18%, respectively; more than half of those with periportal fibrosis have oesophageal varices and 3-4% have hematemesis. Two major factors contribute to the development of portal hypertension. Firstly it is the increased resistance to portal blood flow from an architectural distortion of the hepatic parenchyma and secondly a hyperdynamic circulation secondary to both systemic vasodilation and an increase in plasma volume due to salt and water retention.

Collateral circulation develops in response to portal hypertension by opening of pre-existing collateral vessels and
possibly by active angiogenesis. From a clinical prospective, collateral vessel under pressure become a problem when they occur in the oesophagus and stomach, in the anorectal area, around surgical stomas, and occasionally in other parts of the gastrointestinal tract, which is in continuity with the lumen. Variceal haemorrhage is more likely in patients with hepatic venous pressure gradient (HVPG) above 12 mm Hg. HVPG is an independent and significant indicator for variceal bleeding and death.\(^{(21)}\)

**Natural history of varices in cirrhosis**

1. **Development of varices:**

   The rise in portal pressure is associated with the development of collateral circulation which allows the portal blood to be diverted into the systemic circulation, these spontaneous shunts occur at the cardia through the intrinsic and extrinsic gastro-oesophageal veins and the remaining other portal-systemic junctions.

   Numerous lines of evidence suggest that, varices develop and enlarge with time.

   A study of 532 patients with cirrhosis, showed that the cumulative incidence of patients with varices increased from 12% to 90% over 12 years.\(^{(23)}\)

   In a study involving 80 patients followed for 16 months, Cales and Pascal,\(^{(24)}\) showed that 20% of patients who did not have
varices, developed new varices and 42% of patients with small varices, showed definite enlargement.

The two factors that appear to determine the development of varices are continued hepatic injury and the degree of portosystemic shunting. Evidence for the former is derived from studies in which varices were shown to regress with time. Baker and colleagues\(^{(25)}\) followed a cohort of 112 patients, with oesophageal varices and showed that varices had disappeared in nine patients, regressed in seven, and remained unchanged in six. They concluded that the disappearance and regression of varices may be related to abstinence from alcohol. This observation was confirmed in a study by Dagradi and colleagues\(^{(26)}\) who followed a cohort of patients with alcoholic cirrhosis over three years and showed a reduction in variceal size in 12 of the 15 patients with alcoholic cirrhosis who stopped drinking and an enlargement in variceal size in 17 patients who continued to drink. On the other hand, Cales and Pascal\(^{(24)}\) showed that regression of varices occurred in 16% of patient with alcoholic cirrhosis who continued to imbibe alcohol. This may be related to the development of large portosystemic collaterals which decompress the portal system.

Concerning natural history of varices secondary to schistosomal periportal fibrosis, no published literature is available.

**Risk factor for first variceal bleeding:** These factors are still not clear. The most important factors that have been responsible include: (i) pressure within the varix, (ii) variceal size (iii) tension
on the variceal wall, and (iv) severity of the liver disease in cirrhotic patients.

i. Portal vein pressure In most cases, portal pressure reflects intra variceal pressure\(^{21,27}\) and a hepatic venous pressure gradient more than 12 mm Hg is necessary for the development of bleeding from oesophageal varices but there is no linear relationship between the severity of portal hypertension and the risk of variceal haemorrhage.\(^{28,29}\) However, the hepatic venous pressure gradient (HVPG) tends to be higher in bleeders as well as in patients with larger varices. In a prospective study comparing porpranolol with placebo for the prevention of first variceal haemorrhage, Groszman and colleagues\(^{30}\) showed that bleeding from varices did not occur if the portal pressure gradient could be reduced to less than 12 mm Hg. This pressure has since been accepted as the aim of pharmacological therapy of portal hypertension.

ii. Variceal size The theoretical background of this correlation can be provided by La-place's law which state that the tension on the wall of a rigid container is proportional to the radius, thus the increase in the size of the varices increases the tension of the wall and liability to bleed. This is best assessed endoscopically. Variable results in the literature occur because of the lack of a definition regarding the distinction between large and small varices. Numerous studies\(^{28,31,32,33}\) have shown that the risk of variceal haemorrhage increases with the size of varices.\(^{36,37}\) The grades of the varix is as follows: Grade I (F1): The varices can be depressed by the endoscope. Grade II (F2): The varices cannot be depressed
by the endoscope, and Grade III (F3): The varices are confluent around the circumference of the oesophagus.\(^{(34)}\)

**iii. Variceal wall and tension** Again La-place low play a role here. Increasing the size of the varix and decreasing the thickness of the variceal wall causes variceal rupture. This was confirmed by Polio and Grossman using an in vitro model.\(^{(35)}\)

The colour of the varices is extremely important. Varices usually appear white and opaque. Endoscopic features such as "red spots" and "wale" markings were first described by Dagradi. These features represent changes in variceal wall structure and tension associated with the development of microtelangiectasias.

Beppu and colleague\(^{(36)}\) showed that 80% of patients who had blue varices or cherry red spots bled from varices, suggesting that this was an important predictor of variceal haemorrhage.

**iv. Severity of liver disease and bleeding indices** Both the North\(^{(34)}\) Italian Endoscopic Club (NIEC) and the Japanese, Prada\(^{(37)}\) showed that the risk of bleeding was based on three factors: severity of liver disease as measured by Child Class, variceal size, and red wale markings.

**v. Oesophagitis** The suggestion that oesophagitis may precipitate variceal haemorrhage has been discarded.\(^{(38)}\)

The common types of varices are oesophageal varices and gastric varices, but varices can develop elsewhere in gastrointestinal tract due to portal hypertension where they are called ectopic varices.\(^{(39)}\)
The examples of ectopic varices are duodenal varices and jejunal varices which can cause UGIB.\(^{(39)}\)

Common causes of portal hypertension in Sudan are:

- Schistosomal periportal fibrosis
- Liver cirrhosis

**Schistosomiasis**

Schistosomiasis is the second most prevalent disease, after malaria, in the world. It affects an estimated 200 million people in 76 countries and additional 600 million people are at risk of acquiring this infection.\(^{(40)}\)

According to the WHO, the global distribution of schistosomiasis has changed in recent years. It has been eradicated from Japan and the Lesser Antilles Islands; transmission has been stopped in Tunisia; and transmission is very low in Morocco, Saudi Arabia, Venezuela, and Puerto Rico.\(^{(40)}\)

Here in Sudan, the infection rate varying from 5 to 23\(^{(41)}\). Elgadal\(^{(42)}\) conducted a study which showed *schistosoma mansoni* had become the dominant infection in the Gezira irrigated areas with prevalence rates up to 80\% in local population.

Human infections occur in the course of bathing in or wading through contaminated streams, ponds, or irrigation canals. After infection by *schistosoma cercariae*, there is a latent period before the worms settle in the veins of their preferred visceral site. For *S.
mansonii, S. Japonicum and S. mekongi these are the mesenteric and portal veins.

About 4 weeks after infection, female worms commence egg-laying by a rate of 300 egg/day. For S. mansonii. Eggs not retained in the intestinal wall or excreted in the faeces are carried to the liver; about 50% of all eggs laid are retained in the body. An acute infection syndrome called 'Katayama fever' may occur at this time, with fever, systemic upset and eosinophilia.

Young adults are the main age group affected. Liver metabolic function is generally well preserved and encephalopathy is rare after variceal haemorrhage.

Ascites is found in up to one-third of patients presenting with haemorrhage\(^{(43)}\), but is more frequent in advanced decompensated cases.\(^{(44)}\)

Clinically decompensated hepatosplenic schistosomiasis is characterized by wasting, low serum albumin, prolonged prothrombin time, ascites and hepatic encephalopathy. It is pathogenesis is disputed but predisposing factors include malnutrition, severe deposition of collagen in the space of Disse, Ischaemic damage from repeated variceal bleeding, and severely distorted, arteriovenous relationships in the portal tracts. Alcohol abuse and hepatitis type B and C will produce additional effects.\(^{(44)}\)

Cross-sectional studies have also indicated that the chronic sequelae of HBV infection – chronic hepatitis and cirrhosis – are more likely to develop in patients with hepatosplenic schistosomiasis.\(^{(45)}\)
Macroscopic features of advanced hepatosplenic schistosomiasis are a firm, enlarged liver with a bosselated thickened capsule, and a characteristic portal fibrosis termed 'symmers' clay-pipe stem fibrosis.\textsuperscript{(45)} On cut section, there are thick, white tracts of collagen around major portal tracts, in rounded or stellate shapes. Liver architecture is preserved and there is no cirrhosis. Microscopically, ova penetrate and obstruct the portal branches and are deposited either in the larger radicals, producing the coarse type of billarzial hepatic fibrosis, or in the small portal tracts, producing the fine diffuse form. In early lesions the live eggs are surrounded by eosinophils and an eosinophil abscess may form with fibrinoid materials surrounding the eggs. Then an epithelioid granulomas with or without giant cells develop. The eggs live for 3 weeks then gradually degenerate into empty shells. Within the thick clay-pipe stem fibrous lesions, are seen variable numbers of dead eggs, healing granulomas, fibroblasts, dense collagen, hypertrophied elastic fibres, tortuous arterioles and venules, and entrapped bile ducts.\textsuperscript{(46)}

The portal hypertension of hepatosplenic schistosomiasis is presinusoidal. It result from fibrosis due to host's cell-mediated granulomatous reaction to secreted egg antigen.\textsuperscript{(48)} It is delayed type hypersensitivity reaction.

TH\textsubscript{0} and TH\textsubscript{2}-type helper lymphocytes play an important role in granuloma formation.\textsuperscript{(50)} Autopsy studies of S. mansoni show that clinically significant hepatic fibrosis does not usually develop until there has been chronic infection of more than 160 worm.\textsuperscript{(51)}
The clinical feature shows three stages. Itching follows the entry of the cercariae through the skin. This is followed by a stage of fever, urticaria and eosinophilia. Finally, the third stage of deposition of ova results in intestinal, urinary and hepatic involvement. Initially, the liver (as mentioned), and the spleen are firm, smooth and easily palpable. This is followed by hepatic fibrosis and eventually portal hypertension which may appear years after the original infection.\(^{(13)}\)

Oesophageal varices develop. Bleeding episodes are recurrent but rarely fatal.\(^{(13)}\) The liver shrinks in size and the spleen becomes much larger. Dilated abdominal wall veins and a venous hum are indication of the portal venous obstruction. Ascites and oedema may develop. The blood shows leucopenia and anaemia. The faeces at this stage contain few, if any, parasites.\(^{(13)}\) Patients tolerate blood loss well and hepatic encephalopathy is unusual.\(^{(13)}\)

Diagnostic test: Detection of ova in urine, stool or rectal mucosal biopsy are still the accepted method of diagnosing active infection. Bleeding may be a complication of rectal biopsy in those with portal hypertension. Serological test indicate past exposure without specifying the time. Detection of circulating schistosomal antigens indicates active disease. An ELISA for detecting circulating soluble egg antigens in serum correlates with egg output.\(^{(13)}\)

Ultrasound provides a non-invasive means of assessing hepatic fibrosis and can be correlated with morphological studies\(^{(52)}\) and it is an effective as wedge biopsy in diagnosing symmer's fibrosis.
Ultrasound studies in Egypt also suggest that a mild degree of fine portal fibrosis accompanies mild hepatosplenomegaly in children infected only with S. haematobium.\textsuperscript{(53)}

The degree of the periportal fibrosis is graded as follows\textsuperscript{(54)}:

Grade I: Minimal echogenic thickening of the wall in two or more portal radicles with little change in the diameter of the main portal vein.

Grade II: Mild echogenic thickening of the walls of two or more portal radicles, mainly peripherally, with little or no thickening of the wall of the main portal vein. The gall bladder wall is thickened.

Grade III: Moderate to severe periportal thickening of most portal vein radicles with marked narrowing of the central lucency. The walls of the veins are irregular in thickness. The thickening is marked at the bifurcation of the portal vein and extends to the surface of the liver. The wall of the main vein is thickened from 2-10 mm. The gall bladder wall is thickened.

Grade IV: Marked thickening of the wall of the portal vein radicles with obliteration of the central lucency in the peripheral branches forming thick echogenic bands ranging in thickness from 10-20 mm, reaching the periphery of the liver. The liver is small and shrunken with an irregular surface. The gall bladder greatly thickened.

Upper GIT endoscopy is superior over barium examination in the diagnosis of UGIB. Here, in schistosomiasis upper GIT endoscopy should be done to diagnose varices, bleeding site of varices that
occurs during the first 8 hours, because known patient with oesophageal bleeding has approximately 50% chance of bleeding from a source other than varices.\(^{(55)}\) Endoscopy may predict bleeding from varices. This by presence of the red colour sign (RCS), which is a risk factor for bleeding. The RCS is subdivided into four categories according to Japanese Research Society for Portal Hypertension:\(^{(56)}\)

- **Red wale marking (RWM):** these are longitudinal dilated venules.
- **Cherry red spots (CRS):** These are small red spots usually multiple about 2 mm in diameter.
- **Haematocystic spots (HCS):** These are larger and solitary spots found on tortuous varices and indicate a high risk of haemorrhage.
- **Diffuse redness (DR):** The surface of the varices is diffusely red in a given area. No surface elevation or depression.

**Therapy:**

- The fibrosis of advanced hepatosplenic schistosomiasis is not reversible on antischistosomal therapy with praziquantel. Biochemical studies of patient treated before occurrence of variceal haemorrhage show a reduction in fibrogenesis and a diminution of the obstruction to portal vein blood flow.\(^{(57)}\)
- Ultrasound evaluation of patients with S. Japonicum has indicated that moderate but not severe hepatic fibrosis diminishes on praziquantel with a reduction in spleen
size, and that serum total albumin improve with chemotherapy.\(^{(58)}\)

- Praziquantel is the safest of all anti-helminthics even during pregnancy and lactation.\(^{(59)}\)

- No vaccine is available against schistosomiasis, but Sm-P80 is a potentially excellent candidate for a schistosomiasis vaccination.\(^{(60)}\)
Portal Vein Thrombosis

(PVT)

*Portal vein thrombosis usually is due to one of three main causes:

1. Hyper-coagulable states\(^{(61)}\). This may be due to the use of contraceptives, protein C or S deficiency, antithrombin deficiency, dysfibrinogenaemia, heparin cofactor II deficiency and myeloproliferative disorders which may be subclinical.

2. Stasis or mass lesion which includes:
   - Liver cirrhosis (0.6-40% of cirrhotic will develop PVT) which will increase the risk of bleeding\(^{(62)}\).
   - 20-60%\(^{(63)}\) of patient with hepato-cellular carcinoma will develop PVT with increasing risk of encephalopathy and bleeding.
   - 28-48% of cases with myeloproliferative disorders will develop PVT\(^{(64)}\).

3. Inflammation of the portal vein:
   - This occurs with appendicitis, diverticulitis, omphalitis, chemical injury due to pancreatitis and primary sclerosing cholangitis with bile leak.
   - Diagnosis of portal vein thrombosis is by ultrasound or Doppler imaging.
   - Treatment is usually repeated endoscopic therapy or non-selective B-block if no contraindication.
Splenic vein thrombosis\textsuperscript{(10)}:-

Causes:-

1) Acute and chronic pancreatitis (65%).
2) Pancreatic carcinoma (18%).
3) Lymphoma.
4) Trauma.
5) Hyper coagulable states.

- It causes sinistral (left sided) portal hypertension. Because splenic vein drains the spleen and the stomach through the short gastric veins, it causes isolated gastric varices.

- Gastric varices here are diagnosed endoscopically. But when not activity bleeding, gastric varices may be difficult to distinguish from benign prominent gastric folds. Endoscopic ultrasonography (EUS) identifies hypoechoic, tortuous dilated blood vessels in the submucosa that are characteristic for gastric varices.

- Treatment usually by splenectomy. But acute bleeding can be treated endoscopically; however, rebleeding is the rule, and the mortality rate is as high as 55%.
Non-variceal causes of UGIB

Peptic ulcer disease (PUD):

Peptic ulcer disease is a common cause of acute UGIB, accounting for about 50% of all causes in the western countries. Bleeding from duodenal ulcers is more common than from gastric ulcers – a two-fold or more difference in most series\(^{(65,66)}\). Approximately 150,000 patients are hospitalized for bleeding ulcers in the United States each year\(^{(67)}\). Rates of hospitalization and surgery for bleeding ulcers have not decreased since the 1970s, and the rate of mortality from bleeding ulcers has remained at about 6% to 12% over the same period\(^{(68,69,70,71)}\). The incidence of bleeding ulcer, appear to rise in the winter and decline in the summer\(^{(72)}\).

There are several factors for developing peptic ulcer disease. The main three are acid, H. pylori, and NSAIDs. Of these factors only NSAIDs appear to be an important risk factor for the development of bleeding ulcer.

**Acid**: The role of acid concentration is established only in Zollinger – Ellision syndrome. Nonetheless, reducing acid concentration in patients with bleeding peptic ulcer remains an important issue.

The prevalence of H. pyloric infection in patients presenting with complicated ulcer is decreased. Hosking and coworkers reported that 71% of patients presenting with bleeding duodenal ulcers yielded culture positive for H.pylori, whereas 93% of patient presenting with non-bleeding duodenal ulcers during the same period had H. pylori infection \(p<.01\)\(^{(73)}\). Sixty percent of patients
with bleeding gastric ulcer, had H. pylori – positive cultures, whereas 75% of patients with non-bleeding gastric ulcers had H. pylori infection. Jensen and colleagues\(^{(74)}\) reported an H. pylori prevalence rate of only 72% in patients with bleeding gastric ulcers; 10% of patients neither had H. pylori – positive cultures nor were using NSAIDs.

**NSAID Ingestion:** NSAIDs ingestion is considered as the most important risk factor identified for the development of bleeding in patients with peptic ulcer disease. A number of epidemiologic studies have demonstrated an increased risk of complicated ulcer (e.g., bleeding, perforation, hospitalization and death) and overall GI complications in patients taking NSAIDs. Shorr and associates found that the relative risk of bleeding ulcers in elderly patients taking NSAIDs in Tennessee is 4.0\(^{(75)}\). In the United Kingdom, Langman and Colleagues\(^{(76)}\) reported a relative risk of 4.5 for peptic ulcer bleeding with NSAID use; the risk of bleeding from gastric ulcers and duodenal ulcers were similar.

Experimental studies provide a much more reliable estimate of the risk of NSAID-associated GI complication. The best estimate of the risk of regular NSAID are probably comes from a prospective study of patients with rheumatoid arthritis. Silverstein\(^{(77)}\) and coworkers observed 4439 patients taking NSAIDs for rheumatoid arthritis and a placebo for up to 6 months. Of importance, 42% of these patients were taking steroid, which potentially increase the risk of complication, of the 4439 patients 052% developed bleeding caused by ulcers or erosions.
Four clinical characteristics were identified as independent predictors of NSAID-associated GI complications: Age 75 years or older, history of peptic ulcer, history of GI bleeding, and history of heart disease.

Aspirin now is commonly prescribed for vascular prophylaxis, which requires assessment of the risk of complications. Serrano and coworkers studied 903 consecutive patients discharged at low-dose aspirin and they found that: Cardiovascular patients on long-term low-dose aspirin have a stable risk for major UGIB, which is higher than published controlled clinical trials. Again they found that anti-secretory and nitrovasodilator drugs protect from UGIB, whereas previous peptic ulcer or UGIB and higher doses of aspirin increase the risk. The risk of UGIB with NSAIDs varies twenty-fold depending on the drug, and by three to seven-fold depending on the dose chosen. Risk is maximal during the first week and decreases thereafter. Paracetamol is not associated with UGIB at any dose and should be the first-line analgesic wherever possible. (Non-aspirin, non-steroidal anti-inflammatory are types, when put in order according to their ulcerogenicity were found in this order): Ibuprofen, diclofenac, indomethacin, naproxen, piroxicam, and ketoprofen. Striking dose response relationships were seen with four to eight-fold increases in risk within conventionally used dose ranges for all except ketoprofen. Shorrock and associates, in a study of patients receiving prophylaxis for transient ischaemic attacks, found that aspirin, 300mg q.d., was associated with a 7.7-fold increased risk of UGIB in comparison with placebo, and
600mg b.i.d increased the risk 14.4-fold. Cryer and colleagues\textsuperscript{(81)} showed that doses of aspirin as low as 10mg/day still significantly decrease gastric prostaglandin production – to levels that are similar to the inhibition seen with 81mg and 325mg of aspirin.

\textit{Anti coagulant therapy} : Shorr\textsuperscript{(75)} and associates reported that the relative risk for a bleeding ulcer in patients taking oral anticoagulants was 3.3. The relative risk when patients were taking both oral anticoagulants and NSAID was 12.7. Ulcers that are located high on the lesser curve of the stomach or on the posterio-inferior wall of the duodenal bulb are more likely to rebleed. Bleeding tends to occur when an ulcer loops up to the floor of the crater and commonly protrudes with an aneurysmal dilation.

\textbf{Oesophagitis and Hiatal Hernias}

Major hemorrhage from oesophagitis is rare, accounting for 2\% of patients who presented with clinically significant UGIB in one study\textsuperscript{(82)}. Other series have reported oesophagitis as a cause of UGIB in 0\% to 8\% of cases, and hiatal hernia in 0\% to 12\%\textsuperscript{(83)}. The most common findings with oesophagitis are anaemia and occult blood in the stool; and Brisk hemorrhage suggests an oesophageal ulcer.
Mallory – Weiss Tears:

There are lacerations in the region of the gastroesophageal junction that account for about 5% to 15% of cases of UGIB\(^{(82,84,85,86)}\). The classic history is of one of vomiting, retching, or coughing preceding hematemesis in an alcoholic patient. These are typical antecedent symptoms have been reported in 29% to 86% of patients and a history of heavy alcohol use in 30% to 60%\(^{(84,85,86)}\). Most Mallory tears affect gastroesophageal junction, although 10% to 20% may involve the oesophagus\(^{(84,85,86)}\). Haemorrhage from the tear stops spontaneously in 80-90% of cases. Rebleeding occurs in 0% to 5%\(^{(84,87,88)}\). Most patients require only supportive care.

Gastritis:

Gastritis is a histologic diagnosis that indicates inflammation in the gastric mucosa. There is an evidence that histologic gastritis causes upper GI hemorrhage exists. Gastritis is frequently diagnosed as a cause of upper GI hemorrhage\(^{(89)}\). In this author’s experience, gastritis is a relatively unusual cause of serious upper GI hemorrhage. Because subepithelial hemorrhages and erosions are strictly mucosal lesions, and because all blood vessels of significant size are in the submucosa or below, subepithelial hemorrhages and erosions cannot cause major bleeding, in contrast to ulcers, which may induce serious bleeding when erode into arteries below the mucosa. Subepithelial hemorrhages and erosions develop in
various clinical settings, the most important of which are drug ingestion (e.g. NSAIDs), stress, and alcohol ingestion\(^{(90)}\).

A meta-analysis of prophylaxis for stress induced ulcers\(^{(93)}\) indicated that H\(_2\) receptor antagonists, antacids, or sucralfate led to a significant reduction in clinically significant bleeding (defined as hemodynamic changes or a drop in hemoglobin plus transfusion). However, no evidence indicates that prophylactic therapy decreases mortality rates. Bleeding in control patients in some trials has been low\(^{(91)}\).

**Duodenitis:**

In contrast to gastritis, duodenitis is uncommonly reported as a cause of upper GI hemorrhage. Although erosive duodenitis was said to be the cause of upper GI bleeding in 6\% of patients in the ASGE survey, 6 other series have made no mention of duodenitis as a source of bleeding\(^{(65)}\).

**Neoplasms:**

Neoplasms of the GI tract often produce chronic, occult bleeding but are unusual causes of profuse, acute GI bleeding\(^{(92)}\). They may be either primary (e.g., adenocarcinomas, stromal tumors, neuroendocrine tumors, lymphoma, or polyps) or metastatic from non-GI sources (e.g. breast, melanoma). Treatment of bleeding neoplasms is usually aimed at treatment of the neoplasm itself, but palliative therapy to stop the bleeding may also be necessary. Surgical resection is generally the first choice, if such a procedure is feasible and the patient is a candidate for surgery, if not, endoscopic therapy (injection), angiographic therapy, or radiation therapy may be tried\(^{(93)}\).
Dieulafoy’s lesion:

Dieulafoy’s lesion consists of an abnormally large artery that unlike all other vessels that penetrate the gut wall, retains the large caliber of its feeding vessel as it approaches the mucosa(94). This large vessel apparently compress causing a tiny erosion and rupture of the vessel into the lumen. Dieulafoy’s lesions are difficult to identify unless actively bleeding or covered by a stigma of recent hemorrhage, such as a clot. Bleeding can be massive and recurrent. Formerly, surgical wedge resection was required. However, more recent reports indicate that endoscopic thermal therapy or injection of sclerosant is effective. Unusually found in the proximal stomach, Dieulafoy’s lesions have now been reported throughout the GI tract(94).

Gastric antral vascular ectasia (watermelon stomach):

Gastric antral vascular ectasia may be isolated or may be characterized by linear red streaks running longitudinally in the gastric antrum. Because of the appearance of alternating stripes the latter has been called watermelon stomach(95). Found primarily in older women, the lesion consists of collections of dilated venules, often with focal thrombosis, and fibromuscular hyperplasia in the propria. Therapy is required if there is anemia refractory to iron supplementation, and it consists of either endoscopic thermal therapy or surgical antrectomy(95).
Objective of the study

- To know the causes of upper GIT bleeding in Sudanese patients.
PATIENT AND METHODS

Study design:

Prospective cross-sectional descriptive study.

Area of study:

Khartoum Teaching Hospital, Ibn Sina and Soba University Hospital.

Period of the study:


Sample size:

300 patients, 100 patients from each hospital.

Patients:

Patients were included in the study if they fulfilled the following criteria:-

(1) Have upper gastrointestinal bleeding as evident by haematemesis, and/or melena.
(2) Bleeding should be witnessed or documented by aspiration through nasogastric tube in difficult cases.
Patients were excluded if they were:

(i) Very ill or moribund.
(ii) Below 18 years of age.

Methods:-

Data collected in this study were obtained from the following sources (A questionnaire was designed):

(1) Medical history.
(2) Physical examination.
(3) Investigation.

(1) Medical history:

This includes full medical history of each individual patient with reference to his/her age, sex, residence and occupation. This includes:

a- Information about the bleeding (melena or haematemesis) whether once or recurrent, blood replacement, hospital admission, and symptoms of haemodynamic unstaibility (dizziness).

b- Past history of hospital admission, peptic symptoms, symptoms of liver disease (Jaundice, lower limb swelling, abdominal distension), and risk factors to develop liver disease (alcohol, family history of liver disease). Again it includes drug history.
(2) Physical examination:

All patients were subjected to through physical examination. General examination includes measurement of pulse rate, blood pressure, presence or absence of anaemia, stigmata of chronic liver disease, signs of hepatic decompensation, signs of portal hypertension, epigastric tenderness, and skin for bleeding.

(3) Investigations:-

The following haematological investigations were done:

1- Haemoglobin estimation by cyanmethaemoglobin method using a photo electric calorimeter.
2- Total white cell count and platelet counts by visual methods using number chamber.
3- Prothrombin time by quick are stage method (PT).

Chemical investigation include:

1- Estimation of serum albumin level by the dye binding method using bromocresol green.
2- Serum bilirubin was measured using Van Den – Berg reaction.

Oesophagogastroscopy:

This was performed in all patients using Olympus GIF-p3 fibo-optic endoscope. It was done within 72 hours maximally from
time of bleeding. It was carried out by trained personnel with sub-specialty in gastroenterology.

**Ultra-sonographic examination:**

This was performed in these patients to look for periportal fibrosis, liver cirrhosis, and extra-hepatic causes of varices this was done using ALOK 500 SD with a convex 3.5 MHz transducer.
RESULTS

A total number of three hundred patients who presented with upper gastrointestinal bleeding (UGIB) were studied. One hundred from each hospital: Khartoum Teaching Hospital, Ibn Sina Hospital, and Soba University Hospital. Patients were studied to know the causes of UGIB. Various clinical, endoscopic, ultrasonographic, and laboratory data obtained were statistically analyzed using uni-and-multivariate tests:

Table (1) through table (6) show the main clinical features, laboratory, endoscopic, ultrasonographic, biochemical, and serological data of all patients studied. Males outnumbered females by 2.45 to one. Most of the patients were coming from El-Gazira area which is known by the wide spread canals of the greater Gezira scheme.

Age groups lied between 20 to 40 years (P. value .006) which is statistically significant. The mean age was (41.8 ± 10) years. Twenty eight percent of patients were farmers. 22% of the patients, all males were unemployed due to their illness. Recurrent hematemesis was reported in 53.7% of patients, while 39.3% of cases had one episode of haematemesis. Symptoms of postural hypotension were elicited in 23.7% of cases. History of alcohol consumption was positive in 16% of cases. Other common symptoms were dyspepsia and abdominal distension which were elicited in 34.7% and 28.97% of cases respectively. Past medical history of schistosomasis and history of non steroidal anti-
inflammatory drugs (NSAIDS) were reported in 15.7% and 4.7% of all cases respectively. The physical examination was carried-out in these patients. Thirty percent of the patients showed rapid pulse more than 100 beat per minute while 13.7% of the patients showed a systolic blood pressure less than 100mmHg on presentation. Seventy four percent of the patients were pale, while only 8.7% of them were jaundiced. Only one patient was cyanosed.

Signs of chronic liver disease were found in 23.3% of the patients. Signs of portal hypertension were found in 11% of the cases (mainly venous hum and dilated veins), and 64% of the patients had splenomegaly. Epigastric tenderness was elicited in 13.7% of the patients. Ascites was found in 21.3% of the patients.

**Investigation:**

Eighty one percent of the patients have hemoglobin value below 10gram/dl, while patients with pancytopenia were about 30% of all cases. Those showing features of iron deficiency anaemia were 55%. Prothrombin time was prolonged in 28.7% of the cases. Liver function test showed low serum albumin in 27% while 4.7% of the cases have high billirubin with low serum albumin.

Upper gastro intestinal tract endoscopy was performed in all of these patients. It was normal in 5 patients (1.7% of all cases). The commonest lesion found was isolated esophageal varices 76% (Fig. 9). Combination between esophageal and gastric varices was found in 6.3% of cases, while isolated gastric varices was found in
only 3 cases (1%), two of these patients their abdominal ultrasound revealed splenic vein thrombosis and the third one who was on sclerotherapy for esophageal varices which was successfully treated. Non variceal bleeding from patients esophageal varices was reported in this study four patients (1.4%) bled from duodenal ulcer while already they were have esophageal varices, two patients were having periportal fibrosis and two patients were having liver cirrhosis. Esophagitis was reported in this study in 1.7% of patients with esophageal varices, almost all of them have had periportal fibrosis. Peptic ulcer disease accounted for 9.7% of all cases, with a ratio of 3.14 between duodenal and gastric ulcer. Neoplasm accounted for 3.6% of cases (these include carcinoma of the stomach 2.7%, esophageal cancer 0.3%, gastric leiomyoma 0.3% and duodenal cancer 0.3%). Esophagitis was reported in 4.3% of all cases. Other lesions encountered include: watermelon stomach 1%, gastric erosions 2%, dieulafoy's lesion 0.7% Mallory Weiss 0.7% and arteriovenous malformation in 0.3% of the patients.

Ultrasonography was reported as normal in 18.7% of cases, it showed periportal fibrosis in 51.7% of patients. It showed combination between liver cirrhosis periportal fibrosis in 14% of cases (Table 6). Ultrasonography revealed extrahepatic lesions in 2% of patients with portal vein thrombosis, and 0.7% of cases with splenic vein thrombosis. Again, it showed 5.3% of cases with pure periportal fibrosis who were having ascites (Table 6).
Serology for hepatitis B and C was performed in patients with past medical history of jaundice, and those with features of stigmata of chronic liver disease.

It was positive to one of the two viruses in 8% of cases.
Table (1) Distribution of patients according to age group

<table>
<thead>
<tr>
<th>Age group</th>
<th>Frequency</th>
<th>Percent</th>
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<tr>
<td>&lt;20</td>
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<tr>
<td>20–40</td>
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<td>41–60</td>
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<td>&gt;60</td>
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<td>Total</td>
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</tr>
<tr>
<td>Complain &amp; past medical history</td>
<td>Frequency</td>
<td>Percent</td>
</tr>
<tr>
<td>---------------------------------</td>
<td>-----------</td>
<td>---------</td>
</tr>
<tr>
<td>Pallor</td>
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<td>Past medical history of schistosomiasis</td>
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<td>Jaundice</td>
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<td>Melena</td>
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Table (2b) Distribution of patients according to presenting complain and past medical history

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<tr>
<th>Haematemesis</th>
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<td>Once</td>
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<tr>
<td>Recurrent</td>
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Table (3) Distribution of patients according to physical examination findings

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<tr>
<th>Physical examination</th>
<th>Frequency</th>
<th>Percent</th>
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<tbody>
<tr>
<td>Signs of chronic liver disease</td>
<td>62</td>
<td>20.7</td>
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<tr>
<td>Signs of liver decompensation</td>
<td>30</td>
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<tr>
<td>Signs of portal hypertension</td>
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<tr>
<td>Other than splenomegaly</td>
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<td>Splenomegaly</td>
<td>192</td>
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Table (4a) Distribution of patients according to haematological findings

<table>
<thead>
<tr>
<th>Hb level (g/dL)</th>
<th>Frequency</th>
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<tr>
<td>&lt;10</td>
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Table (4b) Distribution of patients according to haematological findings

<table>
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<tr>
<th>Total WBCs count (cell/dL)</th>
<th>Frequency</th>
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Table (4c) Distribution of patients according to haematological findings

<table>
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<th>Platelet count/dL</th>
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Table (5a) Distribution of patients according to liver function test and prothrombin time

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<th>Prothrombin</th>
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<td>Prolonged</td>
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Table (5b) Distribution of patients according to liver function test and prothrombin time

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<th>Liver function test</th>
<th>Frequency</th>
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<td>High Billirubin</td>
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<tr>
<td>Low Albumin</td>
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<td>High billirubin + low albumin</td>
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Table (6) distribution of patients according to their ultrasonographic findings and endoscopy

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<tr>
<th>Ultrasound</th>
<th>Normal</th>
<th>Liver cirrhosis</th>
<th>PPF</th>
<th>Liver cirrhosis + Ascites</th>
<th>PPF+Ascites</th>
<th>Portal vein thrombosis</th>
<th>Splenic vein thrombosis</th>
<th>Liver cirrhosis+PPF</th>
<th>Liver cirrhosis+PPF+Ascites</th>
<th>PPF+Portal vein thrombosis</th>
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<tbody>
<tr>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
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<tr>
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P. value: 0.000
Figure (1): Distribution of patients according to gender

- Male: 71%
- Female: 29%
Figure (2): Distribution of patients according to residence
Figure (3): Distribution of patients according to occupation

- Unemployed: 22%
- Farmer: 28%
- Others: 50%
Figure (4): Distribution of patients according to past medical history of schistosomiasis in PPF
Figure (5): Relationship between age group and gender in the study population

P. value: .006
Figure (6): Relationship between diagnosis and vomiting of blood among the study population.

P value: .001
Figure (7): Distribution of patients according to serology for both hepatitis B or C viruses in patients with pure Schistosomal periportal cirrhosis.
Figure (8): Distribution of patients with liver involvement according to ultrasonography
Figure (9): Distribution of patients according to endoscopic findings.
Figure (10): Diagnosis of variceal causes according to the underlying pathology
Figure (11): Distribution of the variceal causes of UGIB according to type of varices

- OV: 90.6%
- OV+GV: 8.1%
- GV: 1.3%
Figure (12): Distribution of non-variceal causes of UGIB according to endoscopic findings
DISCUSSION

This study included 300 cases of upper gastrointestinal bleeding (UGIB). The most important investigation is oesophago gastro duodenoscopy (OGD) done by trained personnel in the subspecialty of gastroenterology. This is to reduces the diagnostic error.

Age groups lied between 20 to 40 years (P. value .006) which is statistically significant. The mean age was (41.8 ± 10) years. Twenty eight percent of patients were farmers. Twenty two of the patients, were unemployed males due to their illness. This important category of our young community is the reproductive group which further hinders the already jeopardized gross domestic income in basically agricultural country like Sudan.

The orphan study done concerned with causes of UGIB in Sudan was done by A.M.A Saad in Khartoum T.H. in 1985 (4 year period study)(96). He studied 256 cases presenting with recent attack of UGIB (1-30 days). The commonest cause was found to be bleeding esophageal varices resulting from schistosomal periportal fibrosis. It was reported in 41.8% of cases. Peptic ulcer disease accounted for 30.5%. ? hernia and oesophagitis occurred in 7.8% of cases. Duodenitis represented 3.1% and 4.7% were miscellaneous causes. In our study which is bigger in size than that of Saad we found that oesophageal varices accounted for 71%; almost double that of Saad. This increase in the percentage of oesophageal varices as a cause UGIB can be explained by the higher number of patients
from Gezira included in this study. This high number of patients from this area probably is due to occupational factors and migration. Poor medical services in an area of growing population with over 4 millions and only one hospital in Wad Medani is a major contributor to the vast number of patients from Gezira in our study.

The explanation for the lower number of patients with bleeding peptic ulcer in this study compared with that of Saad is the recently introduced highly effective proton pump inhibitor (PPI) used by patients; prescribed and non prescribed. Peptic ulcer disease usually causes self-limiting bleeding which may manifest as melena, which may be over looked by the patient.

We also observed in Saad study higher percentage of patients without obvious lesion during endoscopy(96). This occurred in only 1.7% in our study. This could be explained by the time lapse between bleeding and endoscoping, patients being endoscoped up to 30 days later in Saad's study. Possible explanations were the fact that newer endoscopies are of higher diagnostic yield plus the simple fact that minor pathology like erosions, small tears would have healed over few days. This stresses the importance of doing endoscopy soonest following an episode of upper GI bleed. In the recent years push enteroscopy has become the most important method in the examination of patients with obscure gastrointestinal bleeding. The source of bleeding could be identified in 57.8% of patients. The most common lesions were small bowel tumor and vascular malformation. Several patients (22.86%)were referred for enteroscopy had lesions in the esophagus, stomach and proximal duodenum that had been missed at upper GI endoscopy(54).
Lule from Keynya showed that prevalence of OV was 34% whereas DU was 17%. Superficial inflammatory lesions represented 17.5%. Multiple lesions had been shown in 17% (97).

Fadali (98), Maduar (99), Zakaria (100) from Egypt reported an incidence of OV 68%, 79%, 52.4% respectively. Whereas that of DU was 15.5% - 31.6%. In all these studies there was a higher prevalence of DU as compared to our study in the face of the vast prevalence of bleeding from OV. This could be accounted for by the different ethnicity and geographical distribution of patients and the lesser use of NSAIDS in our community which is less sophisticated than that of Egypt and even Kenya. Obviously the similarity in prevalence of bleeding from oesophageal varices in the Egyptian studies and our study; the many things in common between the Sudanese and Egyptians couldn't be over emphasized.

The difference was even greater in the developed western countries. It had been shown that bleeding oesophageal varices as a cause of upper GI haemorrhage was only 10% in UK (8). Infections as a cause of UGIB are scarce in the west, population is older the use of NSAIDS is widely prevalent.

The incidence of mixed lesions in this study is about 3.3%. This includes oesophagitis on top of varices in 4% of patients with periportal fibrosis. Duodenal ulcer was a cause of bleeding in 1.7% of patients with liver cirrhosis.
The commonest cause of UGIB is esophageal varices secondary to schistosomal periportal fibrosis which showed increased number compared with that in the study carried by Prof. A.M.F Saad done in 1985.

Peptic ulcer disease comes second to esophageal varices as a cause of UGIB. Generally this goes with Prof. A.M.A Saad's, but it showed decreased number.

Esophageal cancer, dieulafoy lesions, benign gastric tumor. DIC, gastric and duodenal tumors are reported as rare causes of UGIB in this study.

Esophageal, gastric erosion and watermeton stomach accounted for significant figures, of 2.3%, 2%, and 1% respectively.

70% of the patients were pale, 30% showed rapid pulse, more than 100 beat per minute, 13.7% of patients showed systolic blood pressure less than 100mmHg. This may contribute to subsequent mortality.

Age group and malignancy.

RECOMMENDATION
• Schistosomal periportal fibrosis is still the dominant cause of UGIB, so primary care health provider should intensify their activities against this disease.

• Chemotherapy is effective in early disease and in patients with mild to moderate schistosomal periportal fibrosis and it reverses the pathology so mass chemotherapy should cover the endemic areas.

• Specialized centre for the management of UGIB should be set especially in endemic areas.

• A protocol for management of UGIB should be set with clear guidelines to improve the outcome of the management of these patients and to make the best use of the scarce medical resources in our country.

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