"ETIOLOGICAL PROFILE OF PATIENTS PRESENTING WITH UPPER GASTROINTESTINAL BLEEDING - ONE YEAR CROSS SECTIONAL STUDY"

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ENDORSEMENT

This is to certify that the dissertation entitled **"ETIOLOGICAL PROFILE OF PATIENTS PRESENTING WITH UPPER GASTROINTESTINAL BLEEDING-ONE YEAR CROSS SECTIONAL STUDY "** is a bonafide research work done by **REG NO. BG0112011.**

Dr. Rekha S Patil MD
Professor and Head,
Department of Medicine,
J. N. Medical College,
Nehru Nagar, Belgaum – 10

Date:
Place: Belgaum

Dr (Mrs) N.S.Mahantshetti MD (Paed)
Principal,
J. N. Medical College,
Nehru Nagar, Belgaum – 10

Date:
Place: Belgaum

LIST OF ABBREVIATIONS

ACR	-	American College of Radiology
ASGE	-	American Society for Gastrointestinal Endoscopy
BMP	-	Basic metabolic profile
bpm	-	Beats per minute
BUN	-	Blood urea nitrogen
CBC	-	Complete blood count
CE	-	Capsule endoscopy
cm	-	Centimeter
CT	-	Computed tomography
DU	-	Duodenum
e.g.	-	For example
ECG	-	Electrocardiogram
EGD	-	Esophagogastroduodenoscopy
GAVE	-	Gastric antral vascular ectasia
GERD	-	Gastroesophageal reflux disease
GI	-	Gastrointestinal
h	-	Hour
ICU	-	Intensive care unit
INR	-	International Normalized Ratio
mg	-	Milligram
min	-	Minute
mL	-	Milliliter
mm Hg	-	Millimeters of mercury
n	-	Total number

NSAIDs	-	Nonsteroidal antiinflammatory drugs
PPI	-	Proton pump inhibitor
PT	-	Prothrombin time
PUD	-	Peptic ulcer disease
SBP	-	Systolic blood pressure
SIGN	-	Scottish Intercollegiate Guidelines Network
TAE	-	Transcatheter arterial embolization
UGI	-	Upper Gastrointestinal Endoscopy
UGIB	-	Upper gastrointestinal bleeding
UK	-	United Kingdom
US	-	United States
USA	-	United States of America
y	-	Years

ABSTRACT

Background and objectives

Acute upper gastrointestinal bleeding is one of the common medical emergencies. Geographically there is wide variation in the etiology. The present study was an attempt to find out the etiological profile of patients presenting with upper gastrointestinal bleeding so as to choose the optimal endoscopic therapy and further management.

Methodology

The present one year cross-sectional study was carried out in the Department of Medicine and Gastro-enterology, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. A total of 50 patients presenting with upper gastro-intestinal bleeding during the study period that is January 2013 to December 2013 were included. The selected patients underwent upper GI endoscopy.

Results

Majority of the patients were males (94%) and male to female ratio was 15.66:1. The commonest age groups were 40 to 50 years and 61 to 70 years comprised of 24% of patients each and mean age was 52.78 ± 13.52 years. Most of the patients reported past history of liver disease (28%) and alcohol consumption was noted in 44%. 76% of the patients presented with hematemesis. On clinical examination most of the patients (68%) had pallor followed by hepatosplenomegaly(50%). On UGI endoscopy, 54% of patients had esophageal

varices as the source of bleed. The commonest diagnosis was cirrhosis of liver-alcohol induced with portal hypertension noted among 26% of the patients.

Conclusion

Upper GI bleeding is widely prevalent among males and patient is likely to present with hematemesis and melena with history of liver disease. Esophageal varices is the commonest cause of upper gastro-intestinal bleeding.

Keywords

Upper GI scopy; Upper gastro-intestinal bleeding; Varices;

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Chapter 1

Introduction

INTRODUCTION

Upper gastrointestinal bleeding refers to blood loss within the intraluminal gastrointestinal tract from any location between the upper oesophagus to the duodenum at the level of the ligament of treitz.[1] It is a common medical emergency associated with significant morbidity and mortality. Bleeding from the upper gastrointestinal tract is approximately five times more common than lower gastrointestinal tract.[2]

The overall incidence of acute upper gastrointestinal hemorrhage has been estimated at 50 to 100 per 100,000 patients per year, with an annual hospitalization rate of approximately 100 per 100,000 hospital admissions.[3,4] The incidence of upper gastrointestinal bleeding is increasing in elderly people; The incidence of upper gastrointestinal bleeding is 2-fold greater in males than in females, in all age groups, however, the death rate is similar in both sexes. Mortality increases with older age (>60 y) in males and females.[2]

The common causes include duodenal ulcer, gastric ulcer, erosive mucosal disease, varices of portal hypertension and Mallory Weiss syndrome. Less common causes include esophagitis, neoplasm and angiodysplasia[5] Bleeding from the gastrointestinal tract may present as hematemesis, melena, hematochezia, occult gastrointestinal bleeding, and anaemia.[2]

Further ,age more than sixty years, concurrent diseases, hemodynamic status disorder, active bleeding, hypertension, coagulation problems, NSAIDs usage and helicobacter pylori infection are the main risk factors for upper gastrointestinal bleeding.[7]

Bleeding from the GI tract may present in five ways. Hematemesis is vomitus of red blood or coffee ground material. Melena is black, tarry, foul smelling stool. Hematochezia is the passage of bright red or maroon blood from rectum. Occult GI bleeding may be identified in the absence of overt bleeding by special examination of the stool . Finally, patients may present only with symptoms of blood loss or anemia such as light- headedness, syncope, angina, or dyspnea.[8]

If vomiting occurs shortly after the onset of bleeding, the vomitus appears red and later the appearance is dark red, brown, or black precipitated blood clots in the vomitus and produces a coffee ground appearance. Melena develops after as little as 50- 100 ml of blood loss in upper gastrointestinal tract. Acute blood loss greater than 50-100 ml may produce melena for as long as a week. After stool color returns to normal, the test for occult blood test may remain positive for a week. Stool is black tarry and foul smelling. The black, tarry character of melena is due to degradation of blood to hematin of other hemochromes by bacteria and should not be confused with greenish character of ingested iron or the black, non foul- smelling stool caused by ingestion of bismuth.. Hematemesis results from a combination of large amount of blood filling the stomach together with the urge to vomit. So hematemesis generally indicates a more severe bleeding episode than melena. Clinical presentation depends on the site, extent and rate of hemorrhage and presence of coincidental disease.[8]

In the recent years the number of studies exclusively examining epidemiologic patterns of UGIB has been quite limited. However, most epidemiologic studies have shown a decrease in the incidence of all causes of

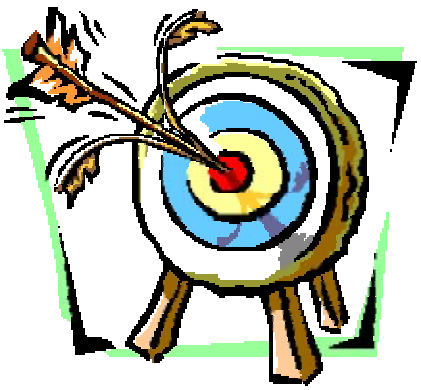
upper gastrointestinal bleeding. Although the incidence of peptic ulcers has remained unchanged.[9]

Therefore, information about etiology is helpful for physicians in order to choose the best treatment techniques and set the ground to control and manage the disease and its consequences.[10,11]

Hence the present study was designed to study the etiological profile of patients presenting with upper gastrointestinal bleeding.

Chapter 2

Objectives



OBJECTIVES

The objective of the present study was to assess the etiological profile of patients presenting with upper gastrointestinal bleeding.

Chapter 3

Review of Literature

REVIEW OF LITERATURE

Upper gastrointestinal bleeding (UGIB) is one of the most common GI medical emergencies, which all GI units deal with everyday. Tremendous advancement has been made in pharmacological and endoscopic interventions in management of upper GI bleeding over the past decade.

However, it still carries considerable morbidity, mortality and health economic burden. Peptic ulcer bleeding is the predominant cause of nonvariceal bleeding in the US and Europe. Recent data indicate a decline in the incidence of peptic ulcer disease and peptic ulcer bleeding in both the United States and Europe, which can be attributed to a decrease in H. pylori infection. Numerous studies from US, Europe and some Asian countries have reported peptic ulcer bleeding mortality 5-12%.[13,14] Existing literature indicates that the mortality rate with each variceal bleeding episode is 30-50%.[14]

Epidemiology

Gastrointestinal bleeding is a very common emergency, accounting for 7-8% of acute medical admissions. Upper GI bleeding (UGIB) is 4-5 times more common than the lower GI hemorrhage. The incidence varies greatly from country to country: from 144/100,000 in Sweden, 111 in Aberdeen and 100 in the USA, to 47 in the UK.[15]

Annually, approximately 100,000 patients are admitted to US hospitals for therapy for UGIB. UGIB is a common occurrence throughout the world. A report from France concludes that the mortality from UGIB has decreased from

about 11% to 7%; however, a similar report from Greece finds no decrease in mortality. In a nationwide study from Spain, UGIB was 6 times more common than lower GI bleeding.[16]

The incidence of UGIB is 2-fold greater in males than in females, in all age groups; however, the death rate is similar in both sexes.[17] Upper GI bleed is more common in men than women (ratio 3:2) and the frequency increases with age – a 20-30 fold increase has been witnessed from the 3rd to the 9th decade. The elderly, thus, appear to be at particular risk, with persons above 60 years accounting for more than 40% of cases.[15]

The population with UGIB has become progressively older, with a concurrent increase in significant comorbidities that increase mortality. Increase in mortality with older age (>60 y) in males and females.[18]

Etiology

Peptic ulcer disease (PUD) accounts for about half of all UGIB. Major risk factors for PUD include Helicobacter pylori infection, use of nonsteroidal antiinflammatory drugs (NSAIDs) or aspirin, smoking, alcoholism, and prior history of PUD. Patients who bleed after admission for another problem usually have PUD.[19]

A recent moderate decrease in PUD as a cause of UGIB has been suggested by a study, despite a marked increase in the proportion of elderly patients who have UGIB from PUD related to NSAIDs.[20] PUD surgery is performed less than previously, but increasingly bariatric surgery causes postoperative bleeding

ulcers. Variceal hemorrhage accounts for 10% to 25% of UGIB, depending on the catchment area. Other relatively common causes of UGIB are inflammatory lesions of the upper gastrointestinal tract,Mallory-Weiss tears, angiodysplasia, and Dieulafoy lesions. Postprocedural bleeding is usually related to endoscopic biopsy or therapy.[20]

Causes of upper gastrointestinal hemorrhage[20]

Major causes

- Peptic ulcer disease
- Esophageal and gastric varices
- Hemorrhagic gastritis
- Esophagitis
- Duodenitis
- Mallory-Weiss tear
- Angiodysplasia
- Upper gastrointestinal malignancy
- Anastomotic ulcers (after PUD surgery or bariatric surgery)
- Dieulafoy lesion

Minor causes

- Cameron lesion
- Gastric antral vascular ectasia (watermelon stomach)
- Portal hypertensive gastropathy
- Post chemotherapy or radiation sequelae

- Gastric polyps
- Aortoenteric fistula
- Submucosal lesion/mass (eg, leiomyoma)
- Connective tissue disease
- Hemobilia
- Hemosuccus pancreaticus
- Kaposi sarcoma
- Foreign bodies
- Postprocedural: nasogastric tube erosions, endoscopic biopsy, endoscopic polypectomy, EMR, endoscopic sphincterotomy

Varices related UGIB

Variceal hemorrhage occurs at a yearly rate of 5–15%, and the size of varices is the most important predictor of hemorrhage with the highest risk of first hemorrhage (15% per year) occurring in patients with large varices. Although bleeding from esophageal varices ceases spontaneously in up to 40% of patients, and despite improvements in therapy over the last decade, it is associated with a mortality of at least 20% at 6 weeks. Gastric varices are less prevalent than esophageal varices and are present in 5–33%of patients with portal hypertension with a reported incidence of bleeding of about 25% in 2 years, with a higher bleeding incidence for fundal varices. Gastric varices are commonly classified based on their relationship with esophageal varices as well as their location in the stomach.[21]

Ulcer-related UGIB

As previously mentioned, peptic ulcer disease is strongly associated with H pylori infection. The organism causes disruption of the mucous barrier and has a direct inflammatory effect on gastric and duodenal mucosa. As the ulcer burrows deeper into the gastroduodenal mucosa, the process causes weakening and necrosis of the arterial wall, leading to the development of a pseudoaneurysm. The weakened wall ruptures, producing hemorrhage.[22]

The flow through the vessel varies with the fourth power of the radius; thus, small increases in vessel size can mean much larger amounts of blood flow and bleeding, with more severe hypotension and more complications, especially in older patients. Visible vessels usually range from 0.3-1.8 mm. Exsanguinating hemorrhage has been reported from larger vessels. The larger vessels are located deeper in the gastric and duodenal submucosa and serosa. The pancreatoduodenal artery and its major branches are located posteroinferiorly in the duodenal bulb, larger branches of the left gastric artery are found high on the lesser curvature[22]

Vomiting-related UGIB

During vomiting, the lower esophagus and upper stomach are forcibly inverted. Vomiting attributable to any cause can lead to a mucosal tear of the lower esophagus or upper stomach. The depth of the tear determines the severity of the bleeding. Rarely, vomiting can result in esophageal rupture (Boerhaave syndrome), leading to bleeding, mediastinal air *entry*, left pleural effusion (salivary amylase can be present) or left pulmonary infiltrate, and subcutaneous emphysema.[22]

Mallory-Weiss tears in UGIB

Mallory-Weiss tears account for 15% of acute upper GI hemorrhage.[23] Kenneth Mallory and Soma Weiss first described the syndrome in 1929.[24] The massive UGIB 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 intragastric and intrathoracic pressures, leading to a gastric mucosal tear from the forceful distention of the gastroesophageal junction.[25]

In 80-90% 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 gastroesophageal junction.[24]

Acute stress gastritis in UGIB

The predisposing clinical conditions that have the potential to alter local mucosal protective barriers, such as mucus, bicarbonate, blood flow, and prostaglandin synthesis results in acute stress gastritis. Any disease process that disrupts the balance of these factors results in diffuse gastric mucosal erosions. This is most commonly observed in patients who have undergone episodes of shock, multiple trauma, acute respiratory distress syndrome, systemic respiratory distress syndrome, acute renal failure, and sepsis. The principal mechanisms involved are decreased splanchnic mucosal blood flow and altered gastric luminal acidity.[22]

Dieulafoy lesions in UGIB

The Dieulafoy lesion, first described in 1896, is a vascular malformation of the proximal stomach, usually within 6 cm of the gastroesophageal junction along the lesser curvature of the stomach. However, it can occur anywhere along the GI tract. This lesion accounts for 2-5% of acute UGIB episodes.[26]

Endoscopically, the lesion appears as a large submucosal vessel that has become ulcerated. Bleeding can be massive and brisk because of the large size of the vessel. The vessel rupture usually occurs in the setting of chronic gastritis, which may induce necrosis of the vessel wall. Alcohol consumption is reportedly associated with the Dieulafoy lesion.[22]

In a review of 149 cases, the Dieulafoy lesion mostly occurred in men and mostly in those in their third to tenth decade.[27]

NSAIDs in UGIB

NSAIDs cause gastric and duodenal ulcers by inhibiting cyclooxygenase, which causes decreased mucosal prostaglandin synthesis which results in impaired mucosal defenses. Daily NSAID use causes an estimated 40-fold increase in gastric ulcer creation and an 8-fold increase in duodenal ulcer creation.[28]

Long-term NSAID use is associated with a 20% incidence in the development of mucosal ulceration.[29] Medical therapy includes avoiding the ulcerogenic drug and beginning a histamine-2 (H2)–receptor antagonist or a proton pump inhibitor that provides mucosal protection.[22]

Clinical assessment

The history and physical examination of the patient provide crucial information for the initial evaluation of persons presenting with a GI tract hemorrhage.[23]

History findings include weakness, dizziness, syncope associated with hematemesis (coffee ground vomitus), and melena (black stools with a rotten odor).[22]

Occasionally, a brisk UGIB manifests as hematochezia (red or maroon stools); the redder the stool, the more rapid the transit, which suggests a large upper tract hemorrhage. Laine and Shah found that 15% of patients presenting with hematochezia had an upper gastrointestinal source of bleeding identified at urgent esophagogastroduodenoscopy.[30]

Patients may have a history of dyspepsia (especially nocturnal symptoms), ulcer disease, early satiety, and NSAID or aspirin use. A history of recent aspirin ingestion suggests that the patient may have NSAID gastropathy with an enhanced bleeding diathesis from poor platelet adhesiveness.[23]

Many patients with UGIB who are taking NSAIDs present without dyspepsia but with hematemesis or melena as their first symptom, owing to the analgesic effect of the NSAID. Low-dose aspirin (81 mg) has been associated with UGIB with or without the addition of NSAID therapy. Using the lowest effective dose for both short-term and long-term users is recommended.[31]

Continuous acid suppression with a proton pump inhibitor (PPI) should be administered to patients with a history of ulcers as they are at an increased risk for UGIB when placed on aspirin or NSAID therapy . The patient's ulcer history is also important because recurrence of ulcer disease is common, especially if he or she has not been treated for *H pylori* gastritis or antibiotic therapy has failed.[22]

Patients may present in a more subacute phase, with a history of dyspepsia and occult intestinal bleeding manifesting as a positive fecal occult blood test result or as iron deficiency anemia. A history of chronic alcohol use of more than 50 g/d or chronic hepatitis (B or C) increases the risk of variceal hemorrhage, gastric antral vascular ectasia (GAVE), or portal gastropathy.[22]

The finding of subcutaneous emphysema with a history of vomiting is suggestive of Boerhaave syndrome (esophageal perforation) and requires prompt consideration of surgical therapy. The presence of postural hypotension indicates more rapid and severe blood loss.[22]

A meta-analysis documented the incidence of acute UGIB symptoms as follows:[32]

- Hematemesis - 40-50%
- Melena - 70-80%
- Hematochezia - 15-20%
- Either hematochezia or melena - 90-98%
- Syncope - 14.4%
- Presyncope - 43.2%
- Symptoms 30 days prior to admission - No percentage available

- Dyspepsia - 18%
- Epigastric pain - 41%
- Heartburn - 21%
- Diffuse abdominal pain - 10%
- Dysphagia - 5%
- Weight loss - 12%
- Jaundice - 5.2%

The importance of the above clinical signs in determining the source of GI bleeding is demonstrated in the table below.[1]

Probable Source of GI Bleeding Within the Gut

Clinical Indicator	Probability of Upper GI Source	Probability of Lower GI Source
Hematemesis	Almost certain	Rare
Melena	Probable	Possible
Hematochezia	Possible	Probable
Blood-streaked stool	Rare	Almost certain
Occult blood in stool	Possible	Possible

Medical history

Although a complete medical history is obtained, it should be focused on the gastrointestinal tract, other highly relevant history, and significant comorbid conditions. In a patient in hypovolemic shock or otherwise in extremis, the

medical history is initially obtained rapidly in summary form because of the need for emergency resuscitation, but a complete history is obtained after patient stabilization. The medical history includes past episodes of gastrointestinal bleeding and their causes, because up to 60% of UGIB is from the same gastrointestinal lesion that previously bled; prior use of gastrotoxic drugs, such as NSAIDs or aspirin; and prior use of drugs that promote bleeding, such as antiplatelet agents or anticoagulants. The risk of bleeding from PUD is increased up to fivefold with administration of nonselective NSAIDs or higher doses (R325 mg/d) of aspirin. Administration of anticoagulants in the therapeutic range, low-dose aspirin (100 mg/d), or the antiplatelet drugs clopidogrel and ticlopidine increase the risk of UGIB by threefold, whereas the selective COX-II (cyclooxygenase-II) inhibitor celecoxib only modestly increases the risk.[33,34]

A history of alcoholism increases the risk for cirrhosis, portal hypertension, and bleeding from esophageal varices. In cirrhotics, about 60% of an initial UGIB is from esophageal varices. Alcoholism also increases the incidence of PUD. A history of smoking cigarettes is relevant.[19]

Duodenal ulcers heal more slowly and recur more frequently with therapy in smokers than in nonsmokers. Smoking and alcoholism are also associated with gastrointestinal malignancy. The patient should be asked about prior H pylori infection and therapy.Hpylori most commonly produces PUD when contracted at a young age, a phenomenon more frequent in immigrants to America. Aortoenteric fistula is strongly associated with prior aortic surgery, aortic aneurysms, and severe atherosclerosis.[19]

Gastrointestinal symptoms are highly relevant. Patients who have PUD often have chronic epigastric pain. A duodenal ulcer typically causes abdominal pain that is initially relieved by eating, but recurs 1 to 2 hours postprandially. Mesenteric ischemia often presents with self-limited gastrointestinal bleeding associated with severe abdominal pain. Reflux esophagitis typically causes pyrosis. Other forms of gastrointestinal bleeding are typically painless. Vomiting, coughing, or retching before bleeding suggests a Mallory-Weiss tear.[35]

Involuntary weight loss suggests chronic disease, particularly gastrointestinal malignancy. Regurgitation, water brash, or dysphagia suggests possible gastroesophageal reflux disease (GERD). The differential diagnosis of dysphagia with UGIB includes reflux esophagitis, esophageal infections, esophageal malignancy, benign peptic stricture, pill esophagitis, and esophageal ulcers.[19]

The presentation and appearance of the blood helps to localize the site of bleeding and to evaluate its acuity and severity. Melena is recognized as black and tarry stools. It should be differentiated from black stools secondary to iron or bismuth ingestion. Patients who have gross UGIB present with melena in about 75% of cases and with hematemesis in about 50%.[17]

Hematemesis indicates bleeding proximal to the ligament of Treitz. Moderate amounts of ‘‘coffee-ground’’ or altered blood emesis suggest more limited bleeding than does hematemesis. About 90% of melena arises from bleeding proximal to the ligament of Treitz because of degradation of blood during gastrointestinal transit, whereas 10% of melena arises from the small

bowel or right colon. Bright red blood per rectum usually arises from a lower gastrointestinal source, most commonly from hemorrhoids. Hemorrhoidal bleeding classically presents with a clinical triad of bright red blood per rectum attributable to the presence of arterialized blood in the hemorrhoidal plexus; blood coating the stools attributable to insufficient time for admixture; and post defecatory bleeding attributable to hemorrhoidal trauma during stool evacuation. Bright red blood per rectum occasionally arises from a massive UGIB.[36]

A UGIB must be excluded when fresh rectal bleeding is accompanied by signs of hypovolemia or hypoperfusion.[19]

The patient's evaluation of the severity of gastrointestinal bleeding is generally not quantitative and often inaccurate.[37] Orthostatic dizziness, mental confusion, cold clammy extremities, angina, or severe palpitations suggest hemodynamic compromise from massive bleeding. Symptoms of excessive bleeding when brushing the teeth, hematuria, or easy bruisability suggest a coagulopathy that can contribute to the bleeding. Jaundice, weakness, fatigue, anorexia, and abdominal distention from ascites are consistent with chronic liver disease.[19]

Physical Examination

The goal of the patient's physical examination is to evaluate for shock and blood loss. Patients present with an ulcer that has bled or is actively bleeding (although approximately 80% of ulcers stop bleeding). Hematemesis and melena are the most common presentations of acute UGIB, and patients may present with both symptoms.[22]

Assessing the patient for hemodynamic instability and clinical signs of poor perfusion is important early in the initial evaluation to properly triage patients with massive hemorrhage to ICU settings. Worrisome clinical signs and symptoms of hemodynamic compromise include tachycardia of more than 100 beats per minute (bpm), systolic blood pressure of less than 90 mm Hg, cool extremities, syncope, and other obvious signs of shock, such as ongoing brisk hematemesis or the occurrence of maroon or bright-red stools, which requires rapid blood transfusion.[38]

Pulse and blood pressure should be checked with the patient in supine and upright positions to note the effect of blood loss. Significant changes in vital signs with postural changes indicate an acute blood loss of approximately 20% or more. Signs of chronic liver disease should be noted, including spider angiomata, gynecomastia, splenomegaly, ascites, pedal edema, and asterixis.[22]

Signs of tumor are uncommon but portend a poor prognosis. Signs include a nodular liver, an abdominal mass, and enlarged and firm lymph nodes. The finding of telangiectasias may indicate the rare case of Osler-Weber-Rendu syndrome.[22]

The medical history, physical examination, and initial laboratory values are important in assessing resuscitation requirements, triage, endoscopy timing, consultation requirements, and prognostication.[39]

The physical examination, while complete, is directed at findings relevant to gastrointestinal bleeding. The severity of blood loss is roughly estimated by the

hemodynamic status and other key signs. Resting tachycardia, in the absence of another cause, suggests mild to moderate hypovolemia.[19]

Orthostatic hypotension is defined as a decrease in the systolic blood pressure of more than 20 mm Hg or an increase in the pulse of more than 20 beats/min from recumbency to standing. Orthostatic hypotension suggests loss of 15% or more of the blood volume. Hypotension is associated with a 40% loss of blood volume.[40]

Patients in shock typically have a thready, weak pulse and cold, clammy extremities. Stigmata of chronic liver disease, such as jaundice, spider angiomata, palmar erythema, hepatomegaly, ascites, and caput medusa suggest UGIB from esophageal varices. Ecchymoses or petechiae are signs of a coagulopathy. The abdomen is carefully examined. Hyperactive bowel sounds are consistent with a UGIB because blood in the proximal gut is an irritant that stimulates peristalsis, whereas normoactive bowel sounds are more consistent with lower gastrointestinal bleeding. Hypoactive bowel sounds suggest bowel ischemia, an ileus, or mechanical obstruction. Abdominal tenderness is uncommon with uncomplicated UGIB, except occasionally for PUD. Severe abdominal tenderness suggests gastrointestinal bleeding associated with bowel ischemia, gastrointestinal obstruction, or gastrointestinal perforation.[19]

Severe direct abdominal tenderness, rebound tenderness, or involuntary guarding suggests a possible acute abdomen that requires exclusion of gastrointestinal perforation before performing EGD. A careful rectal examination should be performed, including determination of the type of bleeding, whether

hematochezia, maroon stools, or melena; testing for fecal occult blood; and inspection for external hemorrhoids or anal fissures.[19]

Critical aspects of the physical examination in patients who have acute gastrointestinal bleeding[19]

- Hemodynamic stability
    - Tachycardia, thready pulse
    - Hypotension
    - Orthostatic hypotension
    - Hypoxia
- Careful abdominal examination
    - Bowel sounds
    - Abdominal tenderness
    - Ascites shifting dullness
- Signs of chronic liver disease or portal hypertension
    - Hepatomegaly
    - Splenomegaly
    - Palmar erythema
    - Caput medusa
    - Spider angiomata
    - Peripheral edema
- Signs of shock
    - Cold clammy extremities
    - Poor mentation

- Rectal examination
    - Occult blood
    - Gross blood
        - Bright red blood per rectum
        - Melena
        - Burgundy stools
        - Blood coating stools versus within stools
        - Bloody diarrhea

Laboratory data

The decline in hematocrit reflects the degree of blood loss after a delay of 24 hours or more from an acute UGIB. The hematocrit does not immediately decline during bleeding because whole blood, containing a proportionate amount of plasma and erythrocytes, is initially lost. The hematocrit subsequently declines because of dilution from influx of extravascular fluid into the vascular space. This dilution is augmented by intravenous hydration. The initial hematocrit on admission is best interpreted when a recent prior baseline hematocrit is available for comparison. Serial hematocrits are helpful to assess the severity of a UGIB but should be integrated with the hemodynamic assessment because overhydration falsely depresses the hematocrit.[19]

A central venous pressure or Swann-Ganz catheter more accurately reflects the volume status than the physical examination or serial hematocrit levels.[41,42] These catheters are indicated when such information is essential for proper fluid management. Other important laboratory parameters include the

coagulation profile; routine serum chemistries, especially the blood urea nitrogen (BUN) and creatinine levels; and serum biochemical parameters of liver function. Patients who have UGIB typically have an elevated BUN level because of absorption of degraded blood during intestinal transit and prerenal azotemia from hypovolemia,[43] and have a BUN/creatinine ratio greater than 20:1.[44] The erythrocytes are typically normocytic with an acute UGIB and are typically microcytic with a chronic UGIB. Iron deficiency anemia is consistent with chronic blood loss. Although leukocytosis may be secondary to the stress of acute bleeding, leukocytosis requires exclusion of underlying infection by appropriate cultures and analyses of blood, sputum, urine, or ascitic fluid as necessary.[19]

Myocardial infarction should be excluded by serial electrocardiography and serum cardiac enzymes in elderly patients who have hypotension and in all patients who have massive bleeding because of a significant risk for myocardial infarction from coronary artery hypoperfusion from hypovolemia. For example, in a study of 113 patients who had severe gastrointestinal bleeding undergoing serial serum cardiac enzyme determinations, 16 (12.3%) patients had myocardial infarction.[45]

Patients who have myocardial infarction consequent to massive bleeding often do not experience chest pain, or the chest pain may be misinterpreted as epigastric pain. For example, in a study of 36 patients who had gastrointestinal bleeding complicated by simultaneous myocardial infarction only half of the patients had chest pain.[46]

An electrocardiogram (ECG) should be ordered to exclude arrhythmia and cardiac disease, especially acute myocardial infarction due to hypotension. Esophagogastroduodenoscopy may increase the risk of arrhythmias.Performing a troponin test may be useful in identifying patients with severe coronary ischemia or atypical myocardial infarction.[22]

Assessment of hemorrhagic shock

As previously mentioned, patients who present in hemorrhagic shock have a mortality rate of up to 30%. Hemorrhage may be classified based on the amount of blood loss, as noted in the following table.[47]

Estimated Fluid and Blood Losses in Shock

	Class 1	**Class 2**	**Class 3**	**Class 4**
Blood Loss, mL	Up to 750	750-1500	1500-2000	>2000
Blood Loss,% blood volume	Up to 15%	15-30%	30-40%	>40%
Pulse Rate, bpm	< 100	>100	>120	>140
Blood Pressure	Normal	Normal	Decreased	Decreased
Respiratory Rate	Normal or Increased	Decreased	Decreased	Decreased
Urine Output, mL/h	>35	30-40	20-30	14-20
CNS/Mental Status	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic
Fluid Replacement, 3-for-1 rule	Crystalloid	Crystalloid	Crystalloid and blood	Crystalloid and blood

This classification scheme aids in understanding the clinical manifestations of hemorrhagic shock. In early class 1 shock, the patient may have normal vital signs, even with a 15% loss of total blood volume. As the percentage

of blood volume loss increases, pertinent clinical signs, symptoms, and findings become more apparent.

Although early cardiovascular changes occur as blood loss continues, urine output, as a sign of end organ renal perfusion, is only mildly affected until class 3 hemorrhage has occurred.[22]

Bornman et al[47] correlated the presence of shock (defined as a pulse rate >100 bpm or SBP < 100 mm Hg) with the incidence of rebleeding rates after initial nonsurgical intervention. They found that rebleeding (a marker for increased mortality and need for surgery) occurred in 2% of patients without shock, in 18% with isolated tachycardia, and in 48% with shock.

Unless the patient has evidence of shock, orthostatic testing should be performed to assess and document a hypovolemic state. A positive tilt test finding is defined as an SBP decrease of 10 mm Hg and a pulse rate increase of 20 bpm with standing compared to the supine position. The American Society for Gastrointestinal Endoscopy (ASGE) survey was able to correlate orthostatic changes with the incidence of mortality.[48] The mortality rate when orthostatic changes are present is 13.6%, compared to 8.7% when they are absent.

A positive tilt test result consistently correlated with a blood loss of 1000 mL. This becomes extremely useful when evaluating patients with class 1 hemorrhagic shock.[15]

CBC With Platelet Count and Differential

A complete blood count (CBC) is necessary to assess the level of blood loss in a patient with upper gastrointestinal bleeding. Where possible, having the patient's previous results is useful to gauge this loss. CBC should be checked frequently (q4-6h) during the first day.[22]

Hemoglobin Value and Type and Crossmatch Blood

Based on the patient's initial hemoglobin level and clinical assessment of shock, a type and screen or type and crossmatch should be ordered. The patient should be crossmatched for 2-6 units, based on the rate of active bleeding. The hemoglobin level should be monitored serially in order to follow the trend. An unstable hemoglobin level may signify ongoing hemorrhage requiring further intervention.[22]

Patients generally require blood transfusions because of hypoperfusion and hypovolemia. Patients with significant comorbid conditions (eg, advanced cardiovascular disease) should receive blood transfusions to maintain myocardial oxygen delivery to avoid myocardial ischemia.[49,50]

According to the 2008 Scottish Intercollegiate Guidelines Network (SIGN) guideline, patients in shock should receive prompt volume replacement.[51]

One of the criteria used to determine the need for surgical intervention is the number of units of transfused blood required to resuscitate the patient. The more units required, the higher the mortality rate.[26] Operative intervention is

indicated once the blood transfusion number reaches more than 5 units, as noted in the following table.[26]

Effect of Number of Packed Erythrocyte Transfusions on Need for Surgery and Mortality from UGIB

Number of Units Transfused	Need for Surgery, %	Mortality Rate, %
0	4	4
1-3	6	14
4-5	17	28
>5	57	43

BMP, BUN, and Coagulation

The basic metabolic profile (BMP) is useful in evaluating for renal comorbidity; however, blood in the upper intestine can elevate the BUN (blood urea nitrogen) level as well. Measurement of coagulation parameters is necessary to assess for continued bleeding. Abnormalities should be corrected rapidly. The BUN-to-creatinine ratio increases with upper gastrointestinal bleeding (UGIB). A ratio of greater than 36 in a patient without renal insufficiency is suggestive of UGIB.[22]

Coagulation Profile

The patient's prothrombin time (PT), activated partial thromboplastin time, and International Normalized Ratio (INR) should be checked to document the presence of a coagulopathy. The coagulopathy may be consumptive and

associated with a thrombocytopenia. A platelet count of less than 50 with active acute hemorrhage requires a platelet transfusion and fresh frozen plasma in an attempt to replete lost clotting factors. The coagulopathy could be a marker for advanced liver disease.[22]

The PT is used in calculating the Child-Pugh score.[28] Elevated aminotransferase levels are a result of hepatocellular injury. Increased levels of alkaline phosphatase and gamma–glutamyl transpeptidase are indicative of cholestatic liver disease.[22]

Prolongation of the PT based on an INR of more than 1.5 may indicate moderate liver impairment. A fibrinogen level of less than 100 mg/dL also indicates advanced liver disease with extremely poor synthetic function.[22]

Calcium Level

Assessing patients' calcium levels is useful in identifying individuals with hyperparathyroidism as well as in monitoring calcium in patients receiving multiple transfusions of citrated blood. Hypercalcemia increases acid secretion.[22]

Gastrin level

A gastrin level can identify the rare patient with gastrinoma as the cause of upper gastrointestinal bleeding and multiple ulcers.[22]

Endoscopy

The development of endoscopy has provided clinicians with the ability for diagnostic and therapeutic approaches to bleeding from the gastrointestinal (GI)

tract. Endoscopic examination of the upper GI tract provides useful information regarding the source and site of bleeding.[48] Endoscopic findings and their incidence rate in patients with upper GI bleeding (UGIB) include the following:

- Duodenal ulcer - 24.3%
- Gastric erosion - 23.4%
- Gastric ulcer - 21.3%
- Esophageal varices - 10.3%
- Mallory-Weiss tear - 7.2%
- Esophagitis - 6.3%
- Duodenitis - 5.8%
- Neoplasm - 2.9%
- Stomal (marginal) ulcer - 1.8%
- Esophageal ulcer - 1.7%
- Other/miscellaneous - 6.8%

Endoscopy should be performed immediately after endotracheal intubation (if indicated), hemodynamic stabilization, and adequate monitoring in an intensive care unit (ICU) setting have been achieved. The 2010 American College of Radiology (ACR) appropriateness criteria for UGIB recommend upper endoscopy as the initial diagnostic examination for all patients presumed to have UGIB.[52]

Capsule endoscopy (CE) may identify low-risk lesions in UGIB, potentially allowing a subset of patients to be safely treated as outpatients. In a study of CE in patients with UGIB, of those with duodenal visualization on CE,

23 of 25 (92%) CE findings were concordant with esophagogastroduodenoscopy (EGD) for low-risk lesions that would have been candidates for outpatient management.[53]

A cause for bleeding was identified in 62 (75%) patients. CE and EGD findings were concordant in 34 (55%) patients. Among the patients with positive EGD findings, 21 (38%) had negative CE results. Of these, 7 were a result of a lack of duodenal visualization. Of 28 patients with normal EGD results, 7 (25%) had positive CE results.[53]

Chest Radiography

Chest radiographs should be ordered to exclude aspiration pneumonia, effusion, and esophageal perforation; abdominal scout and upright films should be ordered to exclude perforated viscus and ileus.[22]

Barium Contrast Studies

Barium contrast studies are not usually helpful and can make endoscopic procedures more difficult (ie, white barium obscuring the view) and dangerous (ie, risk of aspiration).[22]

CT Scanning

Computed tomography (CT) scanning and ultrasonography may be indicated for the evaluation of liver disease with cirrhosis, cholecystitis with hemorrhage, pancreatitis with pseudocyst and hemorrhage, aortoenteric fistula, and other unusual causes of upper GI hemorrhage.[54]

The 2010 ACR criteria state that CT is particularly useful for localizing obscure UGIB and for evaluating a patient with UGIB and a history of aortic reconstruction or pancreaticobiliary procedure.[52]

CT scanning is useful in the diagnosis of aortoenteric fistula because images may reveal thickened bowel, perigraft fluid collection, extraluminal gas, or inflammatory changes in the area of the duodenum and aortic graft.[22]

Nuclear Medicine Scanning

Nuclear medicine scans may be useful in determining the area of active hemorrhage. However, the 2010 ACR criteria state that Tc-99m-labeled erythrocyte scans are of limited value in diagnosing UGIB, but continue to be useful in certain cases of obscure UGIB.[52]

Angiography

Angiography may be useful if bleeding persists and endoscopy fails to identify a bleeding site. According to the 2010 ACR guidelines, angiography along with transcatheter arterial embolization (TAE) should be considered for all patients with a known source of arterial UGIB that does not respond to endoscopic management, with active bleeding and a negative endoscopy.[52]

In cases of aortoenteric fistula, angiography requires active bleeding (1 mL/min) to be diagnostic.[22]

Nasogastric Lavage

This procedure may confirm recent bleeding (coffee ground appearance), possible active bleeding (red blood in the aspirate that does not clear), or a lack of blood in the stomach (active bleeding less likely but does not exclude an upper GI lesion). A nasogastric tube is an important diagnostic tool, and tube placement can reduce the patient's need to vomit. Placement for diagnostic purposes is not contraindicated in patients with possible esophageal varices.[55]

The characteristics of the nasogastric lavage fluid (eg, red, coffee grounds, clear) and the stool (eg, red, black, brown) can indicate the severity of the hemorrhage. Red blood with red stool is associated with an increased mortality rate from more active bleeding compared with negative aspirate findings with brown stool.[22]

Histologic Findings

The bleeding vessel lies in the deepest layer of the ulcer. Fibrinoid necrosis is observed at the site of perforation of the vessel. Pseudoaneurysmal dilation of the vessel may be present at the site of perforation. Biopsy samples should be taken from the edge of a gastric ulcer to rule out carcinoma. The characteristic lesion of *H pylori* is chronic active gastritis with the organisms observed after routine staining. The lesion of gastric antral vascular ectasia is capillary dilation with fibrin clots and fibromuscular hyperplasia.[22]

Literature on etiological profile of upper GI bleeding

In a study by Elghuel A at Tripoli Medical Centre the most common cause of UGIB was peptic ulcer, which represents 37% of all cases, followed by bleeding due to varices in 26.7%, reflux oesophagitis(9.8%),erosions(11.4%) as the most common causes.[56]

Rathod JB et al published in 2011 showed hemetemsis as most common presenting symptom and acute erosive gastritis(34%) as most common cause followed by esophageal varices(24%),Peptic ulcer (22%),Reflux oesophagitis(18%).[57]

In a study by Kaviani MJ et al in October 2010 shows Gastric ulcer as most common etiology followed by duodenal ulcer and oesophageal varices and also compares the mode of presentation.[58]

In a study by Sugawa C et al showed acute gastric mucosal lesion(26%) as the most common cause followed by gastric ulcer(26%),duodenal ulcer(23%) and oesophageal tear(7%).[59]

Pruthi HS et al showed incidence was most common among males and duodenal ulcer(31.5%),Erosive mucosal disease (30.8%), oesophageal varices (16.4%), gastric ulcer (6.2%), Miscellaneous(3.3%) were the most common causes.[60]

Lakhwani MN et al published in Med J Malaysia in December 2010 showed duodenal ulcer(32%) followed by gastric ulcer(29.7%), erosion(10.9%) and esophageal varices(10.9%) as the most common causes.[61]

Chapter 4

Methodology

METHODOLOGY

This one year cross-sectional study was conducted in the Department of Medicine and Gastro-enterology, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2013 to December 2013.

Study design and duration

The study design was a cross-sectional study.

Study period

This study was carried out from January 2013 to December 2013.

Source of Data

Patients with upper gastro-intestinal bleeding presenting at Department of Medicine and Department of Gastro-enterology, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum were studied.

Sample size

A total of 50 patients with upper gastro-intestinal bleeding were included in the study.

Sampling procedure

The sample size was determined considering 80% of the average three year hospital statistics on patients presenting with upper GI bleed as below.

Year	Number of patients
2010	58
2011	62
2012	66
Total	186
Average	62
80% of average	49.6

Hence the sample size of 50 was considered.

Selection criteria

Inclusion Criteria

- Patients presenting with upper gastrointestinal bleeding
    - Hematemesis
    - Melena
- Patients aged 18 years and above

Exclusion Criteria

- Patients unfit for upper Gastrointestinal video endoscopy.

Ethical clearance

The study was approved by the Ethical and Research Committee of Jawaharlal Nehru Medical College, Belgaum.

Informed consent

The patients presenting with upper gastro-intestinal bleeding were screened for eligibility. Those who fulfilled the selection criteria were informed about the nature of study and included after obtaining a written informed consent (Annexure–I).

Data collection

Patients were interviewed and demographic data, history of present illness, other comorbid conditions, personal history and diet pattern were obtained. Significant alcoholism was considered as alcohol consumption of more than 160gram/day for atleast 8 years. Further these patients underwent clinical examination and UGI endoscopy. The findings were noted on a predesigned and pretested proforma (Annexure-II).

Procedure

Patients were subjected to upper gastrointestinal video endoscopy. Endoscopy was performed in all patients within 24 h of admission after hemodynamic stabilization by using an Olympus forward viewing flexible video endoscope. Endoscopy was performed by placing the patients in a left lateral position by standard technique.

Statistical methods

The data obtained was coded and entered into the Microsoft Excel Spreadsheet (Annexure III). The categorical data was expressed in terms of rates, ratios and percentages and continuous data was expressed as mean ± standard deviation.

Chapter 5

Results

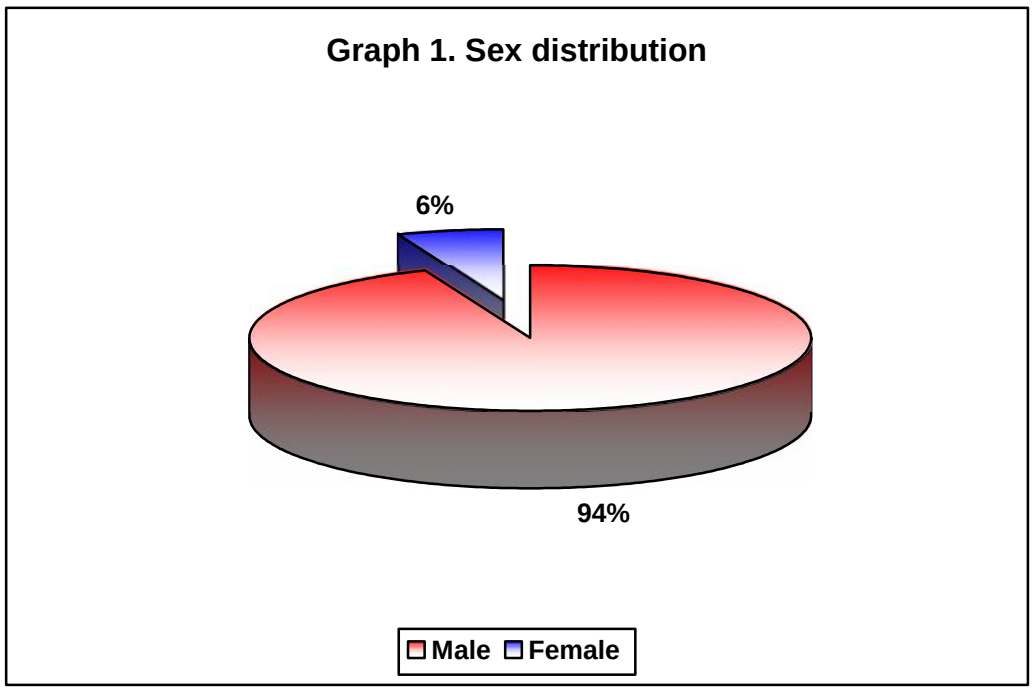
RESULTS

The present one year cross-sectional study was conducted from January 2013 to December 2013. A total of 50 patients presenting with upper gastro-intestinal bleeding at the Department of Medicine and Gastro-enterology, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum were studied.

Data obtained was analysed and the observations and interpretation were tabulated as below.

Table 1. Sex distribution

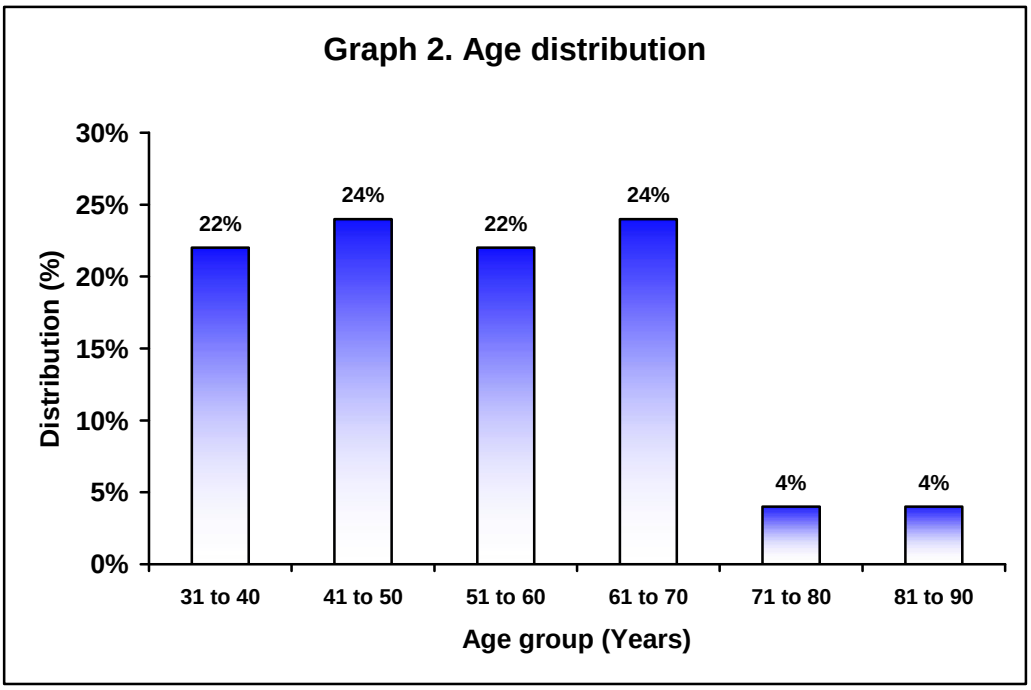
Sex	Distribution (n=50)	
	Number	**Percentage**
Male	47	94.00
Female	3	6.00
Total	**50**	**100.00**



In the present study majority of the patients were males (94%). The male to female ratio was 15.66:1.

Table 2. Age distribution

Age group (Years)	Distribution (n=50)	
	Number	**Percentage**
31 to 40	11	22.00
41 to 50	12	24.00
51 to 60	11	22.00
61 to 70	12	24.00
71 to 80	2	4.00
81 to 90	2	4.00
Total	**50**	**100.00**

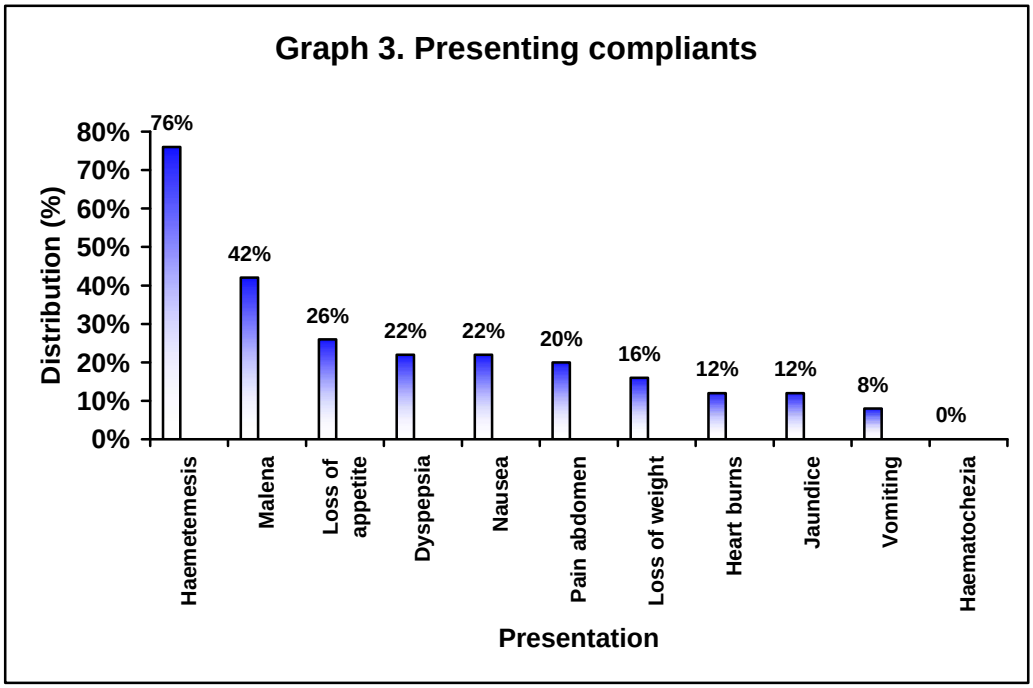


In this study, 24% of the patients each presented with age between 40 to 50 years and 61 to 70 years. The mean age was 52.78 ± 13.52 years.

Table 3. Presenting complaints

Presentation	Distribution (n=50)	
	Number	Percentage
Hematemesis	38	76.00
Melena	21	42.00
Loss of appetite	13	26.00
Dyspepsia	11	22.00
Nausea	11	22.00
Pain abdomen	10	20.00
Loss of weight	8	16.00
Heart burns	6	12.00
Jaundice	6	12.00
Vomiting	4	8.00
Haematochezia	0	0.00

Multiple presentations hence total not shown

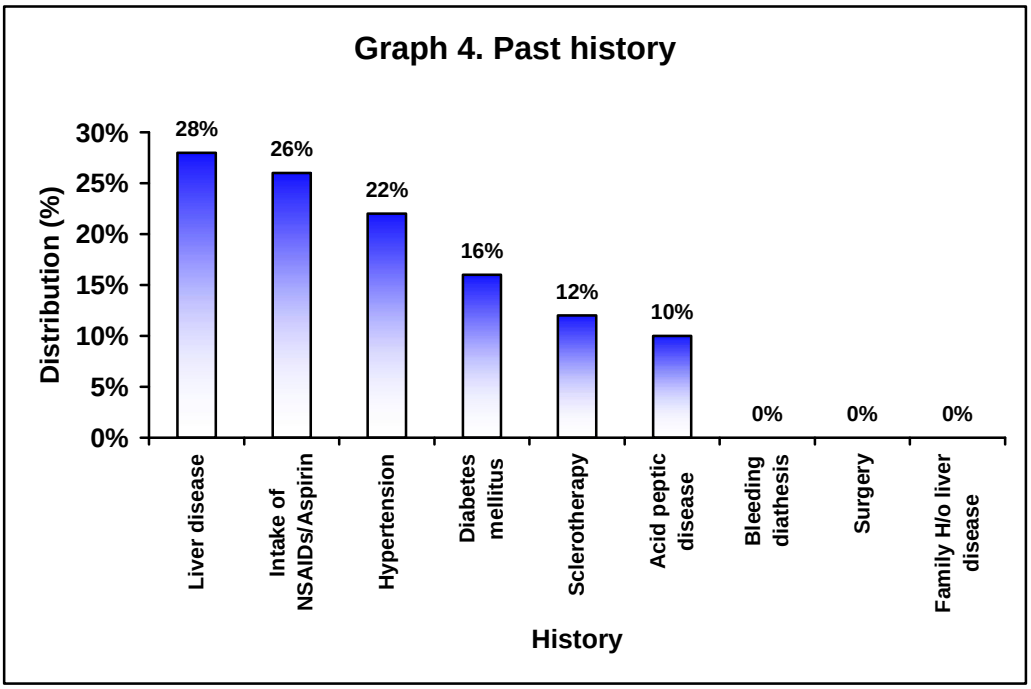


In this study majority of the patients presented with hemetemesis (76%)

Table 4. Past history

History	Distribution (n=50)	
	Number	Percentage
Liver disease	14	28.00
Intake of NSAIDs / Aspirin	13	26.00
Hypertension	11	22.00
Diabetes mellitus	8	16.00
Sclerotherapy	6	12.00
Acid peptic disease	5	10.00
Bleeding diathesis	0	0.00
Surgery	0	0.00
Family H/o liver disease	0	0.00

Multiple presentations hence total not shown

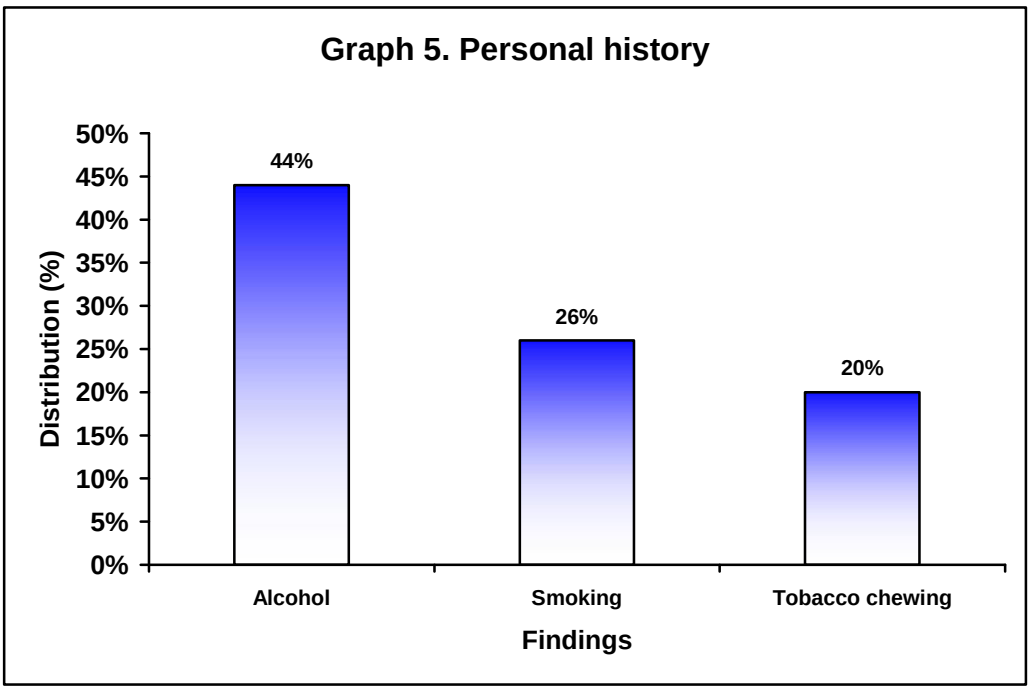


In the present study most of the patients reported past history of liver disease (28%) and intake of NSAIDs and aspirins (26%).

Table 5. Personal history

Findings	Distribution (n=50)	
	Number	Percentage
Alcohol	22	44.00
Smoking	13	26.00
Tobacco chewing	10	20.00

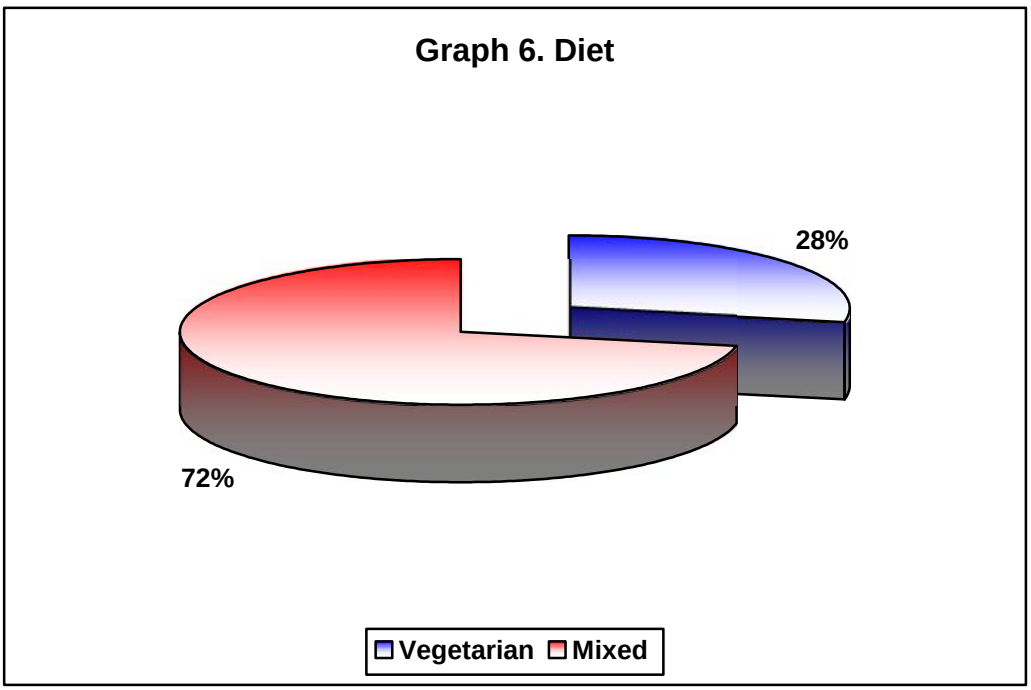
Multiple presentations hence total not shown



In this study history of alcohol consumption, smoking and tobacco chewing was noted in 44%, 26% and 20% respectively.

Table 6. Diet

Diet	Distribution (n=50)	
	Number	**Percentage**
Vegetarian	14	28.00
Mixed	36	72.00
Total	**50**	**100.00**



In the present study 72% of the patients followed mixed diet that is, vegetarian and non vegetarian diet.

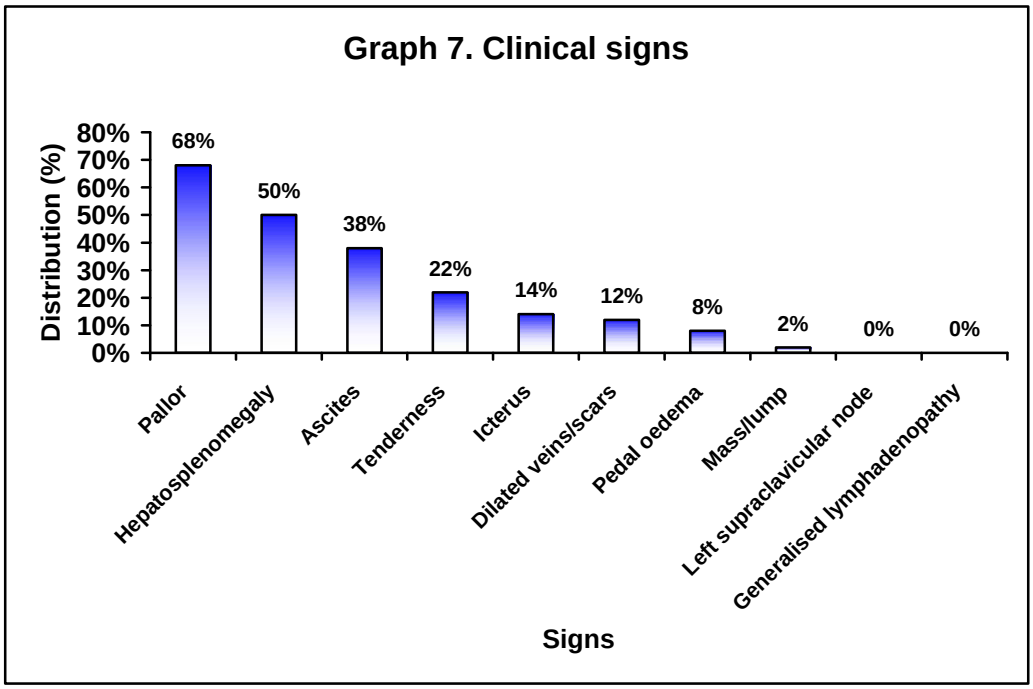
Table 7. Vitals

Variables	Distribution (n=50)	
	Mean	**SD**
Pulse rate (/Min)	81.12	9.85
Systolic blood pressure (mm Hg)	128.94	16.09
Diastolic blood pressure (mm Hg)	76.88	8.16

The mean pulse rate, systolic and diastolic blood pressure of the study population is as depicted in Table 7.

Table 8. Clinical signs

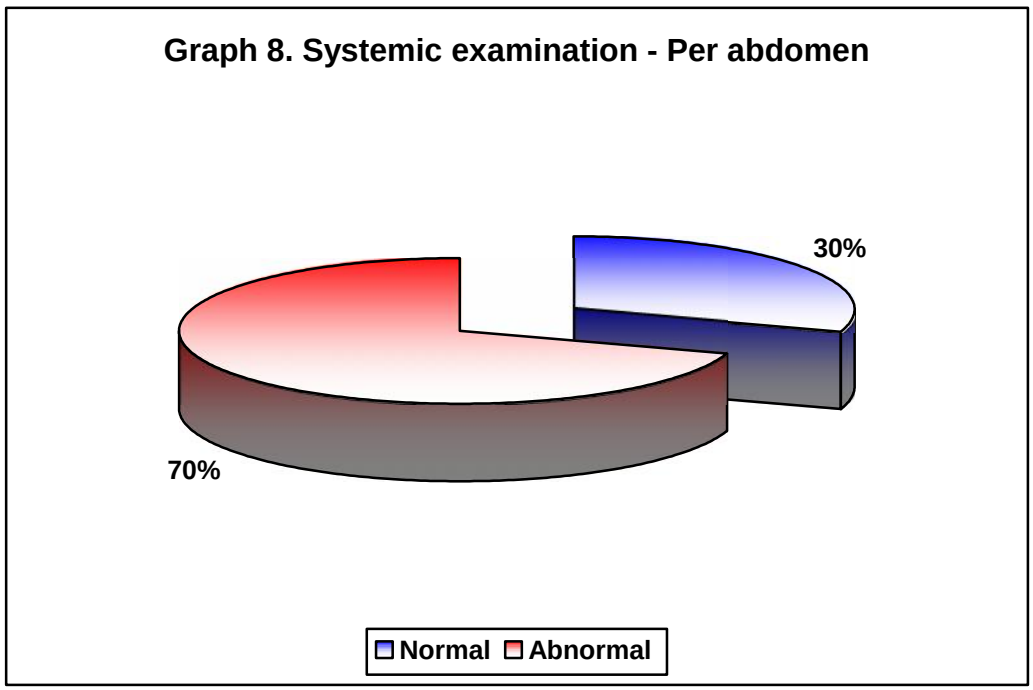
Signs	Distribution (n=50)	
	Number	Percentage
Pallor	34	68.00
Hepatosplenomegaly	25	50.00
Ascites	19	38.00
Tenderness	11	22.00
Icterus	7	14.00
Dilated veins/scars	6	12.00
Pedal oedema	4	8.00
Mass / lump	1	2.00
Left supraclavicular node	0	0.00
Generalised lymphadenopathy	0	0.00



In the present study, on clinical examination most of the patients (68%) had pallor followed by tenderness (50%). The other signs are as shown in table 8 and graph 9.

Table 9. Systemic examination - Per abdomen

Findings	Distribution (n=50)	
	Number	**Percentage**
Normal	15	30.00
Abnormal	35	70.00
Total	**50**	**100.00**

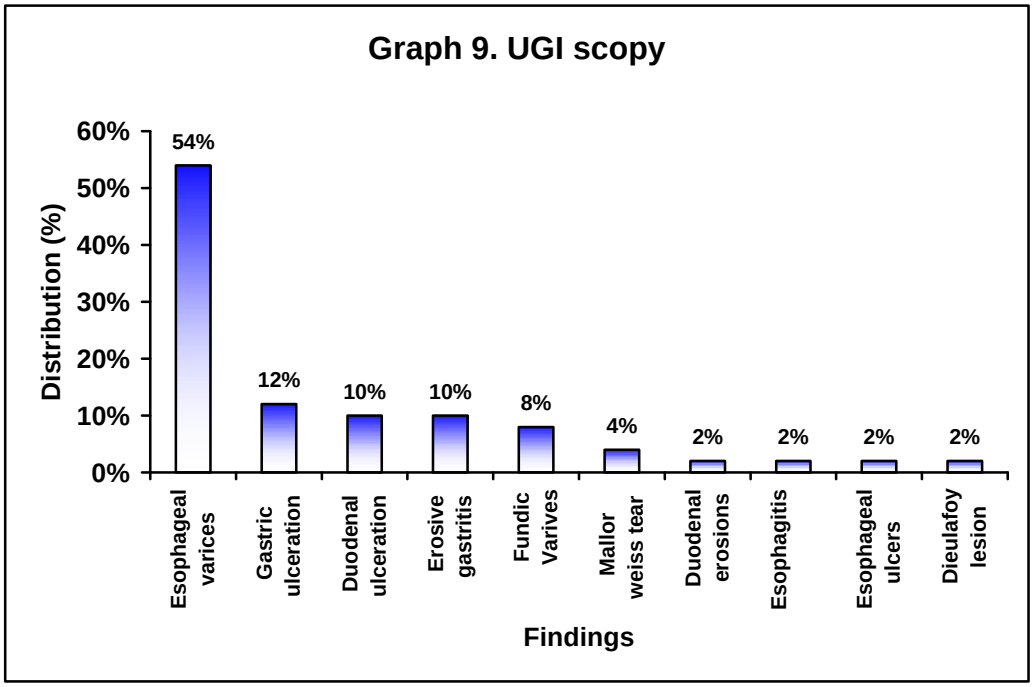


In this study, on systemic examination majority (70%) of the patients had abnormal findings on per abdomen examination.

Table 10. UGI scopy

Findings	Distribution (n=50)	
	Number	Percentage
Esophageal varices	27	54.00
Gastric ulceration	6	12.00
Duodenal ulceration	5	10.00
Erosive gastritis	5	10.00
Fundic varices	4	8.00
Mallory weiss tear	2	4.00
Duodenal erosions	1	2.00
Esophagitis	1	2.00
Esophageal ulcers	1	2.00
Dieulafoy lesion	1	2.00

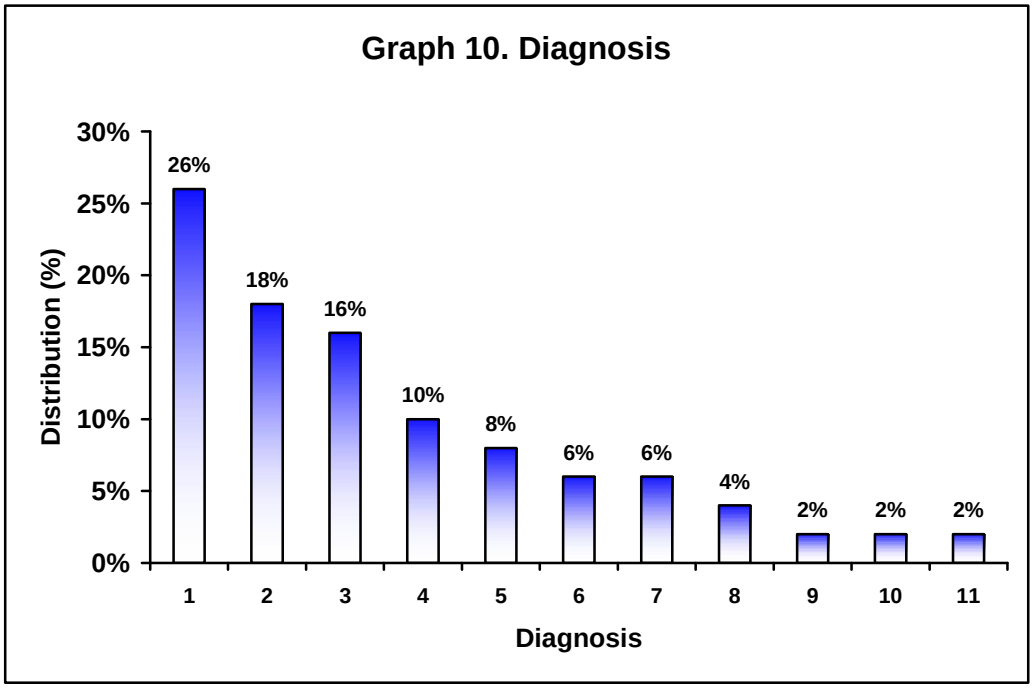
Multiple presentations hence total not shown



In the present study, on UGI, most of the patients had esophageal varices (54%).

Table 11. Diagnosis

Diagnosis	Distribution (n=50)	
	Number	Percentage
1	13	26.00
2	9	18.00
3	8	16.00
4	5	10.00
5	4	8.00
6	3	6.00
7	3	6.00
8	2	4.00
9	1	2.00
10	1	2.00
11	1	2.00
Total	**50**	**100.00**



Diagnosis

1. Cirrhosis of liver – alcohol induced with portal hypertension .
2. Cryptogenic cirrhosis of liver with portal hypertension..
3. Drug induced gastropathy
4. Gastric ulcerations
5. Erosive gastritis
6. Cirrhosis of liver secondary to hepatitis B infection with portal hypertension
7. Non cirrhotic portal hypertension
8. Non alcoholic fatty liver disease with portal hypertension
9. Mallory weiss tear
10. Dieulafoy's lesions
11. Duodenal ulcerations

In the present study the most common diagnosis was Cirrhosis of liver – alcohol induced with portal hypertension noted among 26% of the patients. The next common diagnosis was cryptogenic cirrhosis of liver with portal hypertension (18%) followed by gastric ulcerations (10%), erosive gastritis (8%), Cirrhosis of liver secondary to hepatitis B infection with portal hypertension and non cirrhotic portal hypertension (6% each).

Chapter 6

Discussion

DISCUSSION

Acute upper gastrointestinal bleeding is one of the common medical emergencies that have a hospital mortality of approximately 7% to 10%. The incidence of UGI bleeding is estimated to range from 50 to 150 cases per 100,000 population in developed countries. Despite the advances in therapeutic management, mortality has remained unchanged which may be due to increased longevity, comorbid conditions in the elderly, liver disease, frequent use of non-steroidal anti-inflammatory drugs (NSAID) and anticoagulants.[6]

Non variceal UGI bleeding is the most common cause where peptic ulcer disease accounts for 50% to 70%. Oesophageal varices account for less than 10% of all causes of GI haemorrhages but have a very high mortality rate of at least 30% during their initial hospitalization, with a one year mortality rate approaching 60%. Other less common causes are inflammatory lesions, Mallory Weiss tears, angiodysplasia, and Dieulafoy's lesion.[6] The present study was aimed to study the etiological profile of patients presenting with upper gastrointestinal bleeding.

This one year cross-sectional study included a total of 50 patients presenting with upper gastro-intestinal bleeding from January 2013 to December 2013 to the Department of Medicine and Gastro-enterology, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum.

In this study, males (94%) outnumbered females (6%) with male to female ratio of 15.66:1. A similar male preponderance of 98% is described in a study,[62] although the incidence among men is approximately double that of

women in other studies.[63] A similar study[8] from Rajshahi in 2008 also reported male preponderance that is, 88% males and 12% females with male to female ratio of 7.3:1. Another study[64] from Shimla in 2005 also reported with 78.4% of the males. Recently a similar study[2] from Sikkim, India reported 72.9% males and 27.1% females. In contrast, a study[65] from Tata Main Hospital, Jamshedpur, of the 500 patients, there were 257 males in comparison to 243 female patients. However, The findings of the present study were consistent with the studies from Rajshahi,[8] Shimla[64] and Sikkim[2] which reported male preponderance. The male preponderance observed in this study could be explained by the presence of various risk factors like smoking and alcohol consumption which are common in male population leading to UGI bleeding.

In the present study, nearly one fourth that is, 24% of the patients each presented with age between 40 to 50 years and 61 to 70 years. In a study from Shimla, maximum patients (47.7%) with UGIB were in the age group of 41 to 60 years. Similarly study from Sikkim[2] reported maximum patient with age group of 51-60 years and with mean age of 53.70 years. The mean age in this study was found to be 52.78 ± 13.52 years. The mean age is variably reported in different series. A study from Nepal[6] reported mean age as 48.76±17.19 years. Other studies from India by Anand et al[66] reported mean age of 41 years and Rao et al[67] reported a mean age of 43 years, from West Indies, Kaliamurthy et al[68] reported higher mean age of 55 years. Recently published UK audit showed even higher mean age of 64.4.[69] The mean age of the present study was comparable with a study from Sikkim[2] and Kaliamurthy et al[68] from West Indies.

In this study 76% of the patients presented with hematemesis. The next common complaint was melena (42%), followed by loss of appetite (26%), dyspepsia (22%) and nausea (22%) and pain abdomen (20%). However, less common presentations were loss of weight (16%), heart burns (12%), jaundice (12%) and vomiting (8%). A study[8] from Rajshahi also reported that, most of the patients with upper gastrointestinal haemorrhage presented with both haematemesis and melena (42%). Rathod JB et al[70] in 2011 reported hemetemsis as most common presenting symptom. In a study[2] from Sikkim, India the commonest presentation was hematemesis and melena in over half of the patients like other studies,[71,72] which predict massive bleeding. However, a bleeding duodenal ulcer is likely to be presented with melena more frequently than hematemesis while a bleeding gastric ulcer patient may present with hematemesis more frequently than melena. Variations in presentation among the cases of upper GI haemorrhage in the present study compared to other studies may be explained by the fact that hematemesis and melena are dependent upon the rate, amount and site of bleeding.

In the present study, 28% of the patients reported history of liver disease while 26% reported intake of NSAIDs and aspirins. The other comorbid conditions included hypertension (22%), diabetes mellitus (16%) and acid peptic disease (10%). Personal history revealed alcohol consumption in 44%, smoking in 26% and tobacco chewing in 20% of the patients. It has been estimated that more than 30 million people all over the world consume NSAIDs daily[14] and numerous studies have documented the toxic effect of NSAIDs on the gastrointestinal mucosa. In a study, 37.5% patients gave history of NSAIDs,

aspirin, and other drugs intake within 48 hours, the probable precipitating factors. NSAIDs, aspirin, can cause bleeding ulcer and also increase the chances of bleeding from pre-existing ulcers.[73] A study[64] reported that, alcohol was a probable precipitating factor in 12.5% of DU patients. In other studies, an overall 18% of patients of upper GI bleed had alcohol as a precipitating factor.[74] In 78.4% patients, ulcer-like symptoms were present before onset of UGI bleeding. Laszlo et al[75] found frequent absence of preceding symptoms in upper GI bleeding patients regardless of NSAIDs use.

In the present study based on UGI endoscopy the most common etiology was esophageal varices noted in more than half of the study population (54%). The next common etiology was gastric ulceration (12%) followed by duodenal ulceration (10%), erosive gastritis (10%) , fundic varices (8%) and Mallory Weiss tear (4%). However, duodenal erosions, esophagitis, esophageal ulcers, dieulafoy lesion were noted in 2% of the patients each. Few studies showed esophageal varices as a leading cause of bleeding. A study[6] from Nepal also reported that, the commonest endoscopic finding was bleeding from esophageal and gastric varices (47.7%). Gurung et al[76] from Nepal reported esophageal varices in 15.6% and Bhattarai et al[77] in 33.3%. A study done in Pakistan by Adam et al[78] found that esophageal varices were responsible for bleeding in 44.4% cases. In contrast, another study done in Pakistan by Bhutta et al[79] reported esophageal varices only in 21%. The discrepant result was explained due to various factors like hospital reporting of all the major and minor cases and doing endoscopy in time. In India, Anand et al[66] investigated 408 patients of UGI bleed and found that 45.5% had esophageal varices and another study done by Rao et al[67] showed esophageal

varices as the most common cause (51%). This shows that the trend of UGI bleeding in south East Asian countries is different from the developed countries as UK Audit 2007 has reported only 11% bleeding varices[69] and Snaders et al[80] have reported only 4.4%. In US, variceal bleeding accounts for around 6%.[81] Various risk factors may be responsible such as heavy alcohol consumption, ignorance, low socio-economical and poor nutritional status in the developing countries.

In a study by Elghuel A[56] at Tripoli Medical Centre the most common cause of UGIB was peptic ulcer, which represents 37% of all cases, followed by bleeding due to varices in 26.7%, reflux oesophagitis (9.8%), erosions (11.4%) as the most common causes. Rathod JB et al[57] in 2011 showed acute erosive gastritis (34%) as most common cause followed by esophageal varices (24%), Peptic ulcer (22%), Reflux oesophagitis (18%). In a study by Kaviani MJ et al[58] in 2010, Gastric ulcer was the most common etiology followed by duodenal ulcer and oesophageal varices. Sugawa C et al[59] reported acute gastric mucosal lesion (26%) as the most common cause followed by gastric ulcer (26%), duodenal ulcer (23%) and oesophageal tear (7%). Lakhwani MN et al[61] in 2010 reported duodenal ulcer (32%) as the most common causes of UGI followed by gastric ulcer (29.7%), erosion (10.9%) and esophageal varices (10.9%).

In the present study, the most common diagnosis was cirrhosis of liver-alcohol induced with portal hypertension noted among 26% of the patients. The next common diagnosis was cryptogenic cirrhosis of liver with portal hypertension (18%) followed by gastric ulcerations (10%), erosive gastritis (8%), cirrhosis of liver secondary to hepatitis B infection with portal

hypertension and non cirrhotic portal hypertension (6% each). Portal hypertension is responsible for several sources of upper and lower gastrointestinal bleeding, including esophageal varices, gastric varices, ectopic varices in the small and large intestine, congestive gastropathy, and a higher incidence of peptic ulcer.

Chapter 7

Conclusion

CONCLUSION

Esophageal varices is the commonest cause of upper gastro-intestinal bleeding in this region. The other etiologies are gastric ulceration, duodenal ulceration and erosive gastritis. Upper GI bleeding is common among males, likely to present with hematemesis and melena as common symptoms .

Chapter 8

Summary

SUMMARY

Acute upper gastrointestinal bleeding is one of the common medical emergencies. Geographically there is wide variation in the incidence of etiology. The present study was an attempt to find out the etiological profile of patients presenting with upper gastrointestinal bleeding to enable the optimal endoscopic therapy and further management.

The present one year cross-sectional study was carried out in the Department of Medicine and Gastro-enterology, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. A total of 50 patients presenting with upper gastro-intestinal bleeding during the study period that is January 2013 to December 2013 were included. The selected patients underwent upper GI endoscopy.

Majority of the patients were males (94%) and male to female ratio was 15.66:1. The commonest age groups were 40 to 50 years and 61 to 70 years comprised of 24% of patients each and mean age was 52.78 ± 13.52 years. Nearly one third of the patients reported past history of liver disease (28%) and alcohol consumption was noted in 44%. Hematemesis (76%) was the most common presenting symptom. On clinical examination 68% of the patients had pallor followed by hepatosplenomegaly(50%). On UGI endoscopy , 54% of the patients had esophageal varices as the source of bleed followed by gastric ulcerations in 12 % of patients. The commonest diagnosis was alcohol induced cirrhosis of liver with portal hypertension noted among 26% of the patients.

The etiology of varices that is portal hypertension was secondary to alcohol induced cirrhosis of liver in 26% of patients ,cryptogenic cirrhosis of liver in 18% of patients, hepatitis B induced cirrhosis of liver in 6% of patients, non cirrhotic portal hypertension in 6 % of patients and non alcoholic fatty liver disease in 4 % of patients.

Chapter 9

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Annexures

Annexure II

ANNEXURE II – PROFORMA

ETIOLOGICAL PROFILE OF PATIENTS PRESENTING WITH UPPER GASTRO INTESTINAL BLEEDING- ONE YEAR CROSS SECTIONAL STUDY

PROFORMA

NAME:

AGE:

SEX:

SERIAL NO.:

OP/IP NO.:

OCCUPATION:

1) PRESENTING COMPLAINTS DURATION:

1. Hematemesis- quality, quantity
2. Melena- present or absent
3. Jaundice
4. Dyspepsia
5. Pain abdomen
6. Heart burns
7. Haematochezia
8. Regurgitation- postural, non postural
9. Loss of appetite
10. Loss of weight- sudden, slow
11. Nausea

12. Vomiting

PAST HISTORY

1. DM/HT
2. H/O acid peptic disease
3. H/O intake of NSAIDS, Aspirin
4. Any H/O surgery
5. H/O Sclerotherapy
6. H/O Bleeding diathesis
7. H/O Liver disease

3) PERSONAL HISTORY

Smoking- Duration

Consumption of alcohol Duration

Diet- Veg/ Non veg

Tobacco chewing Duration

4) FAMILY HISTORY

Any H/O Liver disease in the family

5) MARITAL STATUS

6) GENERAL EXAMINATION

Built/ nourishment

Pallor / icterus/ pedal edema/generalized lymphadenopathy/left supraclavicular node

PULSE RATE

BLOOD PRESSURE

ABDOMEN

Tenderness

Dilated veins /scars Mass/ lump

Hepatomegaly

Ascitis /free fluid

CARDIOVASCULAR SYSTEM

RESPIRATORY SYSTEM

CENTERAL NERVOUS SYSTEM

2) UGI Endoscopy-

DIAGNOSIS

Annexures

Annexure III

ANNEXURE III – KEY TO MASTER CHART

AB - Abnormal

BP - Blood pressure

Diagnosis

1. Cirrhosis of liver – alcohol induced with portal hypertension.
2. Cryptogenic cirrhosis of liver with portal hypertension.
3. Drug induced gastropathy
4. Gastric ulcerations
5. Erosive gastritis
6. Cirrhosis of liver secondary to hepatitis B infection with portal hypertension .
7. Non cirrhotic portal hypertension
8. Non alcoholic liver disease with hypertension
9. Mallory weiss tear
10. Dieulafoy's lesions
11. Duodenal ulcerations

F - Female

M - Male

m - Mixed

Min - Minute

mm Hg - Millimeters of mercury

N - Normal

NSAID - Non steroidal anti-inflammatory drug

UGI - Upper gastrointestinal Endoscopy

Upper gastrointestinal endoscopy

1	-	Esophageal varices
2	-	Gastric ulceration
3	-	Duodenal ulceration
4	-	Erosive gastritis
5	-	Fundic varices
6	-	Mallory weiss tear
7	-	Duodenal erosions
8	-	Esophagitis
9	-	Esophageal ulcers
10	-	Dieulafoy lesion

V	-	Vegetarian

Annexures

Annexure I

ANNEXURE I – CONSENT FORM

"ETIOLOGICAL PROFILE OF PATIENTS PRESENTING WITH UPPER GASTRO INTESTINAL BLEEDING" A ONE YEAR CROSS SECTIONAL STUDY

Objective and purpose of the study:

To study the etiological profile of patients presenting with upper gastrointestinal bleeding. The principal investigator of the study **M. D. in GENERAL MEDICINE,**

Risks and Benefits:

Upper gastrointestinal bleeding is a common condition encountered during practice. The presentation, treatment and the prognosis depends on the site and amount of bleeding. Hence it is beneficial to find out the etiological profile for the effective management.

Procedure:

If you agree to be part of the research study you will be asked the relevant history and have to undergo upper gastrointestinal video endoscopy.

Alternatives

Taking part in this study is voluntary. You may choose not to take part in this study, or if you decide to take part now, you can later change your mind and withdraw from the study. Your decision will not change the present or future health care or other services that you receive. The study doctor or sponsor may you're your participation in this study any time. If you choose not to take part in the study you will receive the standard treatment for patients with your condition.

Privacy and Confidentiality

All information collected about you during the course of this study will be kept confidential to the extent permitted by law. The code numbers will identify you in this research record. Information from this study may be published but your identity will be confidential in any publication.

Institution / Sponsor's policy

Does not apply to this research

Financial incentives for participation You will not be paid / offered any gifts /incentives for participating in the study

Authorization to publish the results

The results of the study would be forwarded to the KLE University, Belgaum as part of requirement towards the completion of MD degree, review and publishing.

Queries and contact details

If you have any questions about the research you may please contact:

1. Investigator, _____ Post Graduate student, Department of Ophthalmology, JNMC, Belgaum.
2. Guide, _____ Professor, Department of Ophthalmology, JNMC, Belgaum
3. _____ Principal, JNMC, Belgaum and Chairman, Institutional Ethics Committee.

Consent Statement

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicates that I have read, or it has been read to me, this entire consent form, and have had all my questions answered.

In case of the queries during study or in future you may contact following person

Principal investigator : ----------

Guide : ----------

Participant: _____ Signature / Thumb print _____

Name of the Witness _____ Signature _____

Name of the investigator _____ Signature _____

Date: Place:

ANNEXURE III - MASTER CHART

Serial Number	In/Out patient number	Age (Years)	Sex	Complaints											History												General physical examination findings																			Systemic examination					
																							Personal					BP (mm Hg)		Signs																						
				Haemetemesis	Malena	Jaundice	Dyspepsia	Pain abdomen	Haematochezia	Heart burns	Loss of weight	Loss of appetite	Nausea	Vomiting	Diabetes mellitus	Hypertension	Acid peptic disease	Intake of NSAIDs / Aspirin	Surgery	Sclerotherapy	Bleeding diathesis	Liver disease	Family history of liver disease	Smoking	Alcohol	Tobacco chewing	Diet	Pulse rate (/Min)	Systolic	Diastolic	Pallor	Icterus	Pedal oedema	Generalised lymphaden	Left supraclavicular nod	Tenderness	Dilated veins/scars	Mass / lump	Hepatosplenomegaly	Ascites	Free fluid	Cardiovascular system	Respiratory system	Per abdomen	Central nervous system	UGI scopy	Diagnosis
1	510689	48	M	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	+	-	m	72	136	80	+	+	+	-	-	-	-	-	+	+	-	N	N	AB	N	1	1
2	512466	82	M	+	+	-	-	+	-	-	-	-	-	-	+	+	+	+	-	-	-	-	-	+	-	-	m	80	160	80	+	-	-	-	-	-	-	-	-	-	-	N	N	N	N	3	3
3	512508	38	M	+	-	-	+	-	-	-	+	+	-	-	-	-	-	-	-	-	-	+	-	-	+	+	m	72	126	80	+	-	-	-	-	-	-	-	+	-	-	N	N	AB	N	5	2
4	512903	80	M	+	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	-	-	-	-	-	-	m	84	150	90	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	3,6	3
5	513710	42	M	-	+	-	-	-	-	+	+	+	-	-	-	-	-	-	-	-	-	+	-	-	+	-	m	86	126	80	+	-	-	-	-	-	-	-	+	+	-	N	N	AB	N	1	1
6	514931	50	M	+	-	-	+	-	-	+	+	+	-	-	+	+	+	+	-	-	-	-	-	-	+	+	m	102	120	70	+	-	-	-	-	+	-	-	-	-	-	N	N	AB	N	7	3
7	515927	87	M	-	+	-	-	-	-	-	-	+	+	-	-	+	-	+	-	+	-	+	-	-	-	-	m	76	150	80	+	-	-	-	-	-	-	-	-	-	-	N	N	N	N	5	2
8	515127	58	M	+	-	-	+	-	-	-	-	+	-	-	+	-	-	-	-	+	-	+	-	-	+	-	m	108	100	60	+	-	-	-	-	-	-	-	+	+	-	N	N	AB	N	1	1
9	519536	63	M	-	+	-	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	+	-	-	-	-	V	88	142	90	+	-	-	-	-	+	+	-	-	+	-	N	N	AB	N	1	2
10	523740	68	M	+	-	-	-	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	V	70	130	70	-	-	-	-	-	-	-	-	-	-	-	N	N	AB	N	2	4
11	527087	39	M	+	-	+	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	+	-	-	+	-	m	102	90	60	+	+	+	-	-	-	+	-	+	+	-	N	N	AB	N	1	1
12	530780	32	M	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	m	82	118	70	+	-	-	-	-	-	-	-	+	-	-	N	N	AB	N	1	1
13	530594	43	M	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	m	80	120	70	-	-	-	-	-	-	-	-	+	+	-	N	N	AB	N	2	4
14	532375	48	M	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	m	82	118	80	+	-	-	-	-	-	-	-	+	+	-	N	N	AB	N	1	2
15	529902	62	M	-	+	-	-	+	-	-	-	-	-	-	-	+	-	+	-	-	-	-	-	-	+	-	m	82	160	90	+	-	-	-	-	-	-	-	-	-	-	N	N	N	N	3,7	3
16	528598	65	M	+	-	-	+	-	-	-	-	-	+	+	-	-	-	+	-	-	-	-	-	-	-	+	m	66	136	80	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	8,9	3
17	534376	42	M	+	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	V	90	142	80	+	-	-	-	-	-	-	-	+	+	-	N	N	AB	N	1	6
18	524187	35	M	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	m	70	130	70	+	-	-	-	-	+	-	-	-	-	-	N	N	AB	N	6	9
19	536504	35	M	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	m	82	126	80	+	-	-	-	-	+	-	-	-	+	-	N	N	AB	N	1	2
20	536353	53	M	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	+	-	-	-	+	V	82	130	80	+	-	-	-	-	-	-	-	+	-	-	N	N	AB	N	1	2

ANNEXURE III - MASTER CHART

Serial Number	In/Out patient number	Age (Years)	Sex	Complaints											History														General physical examination findings																Systemic examination					
																								Personal					BP (mm Hg)		Signs																				
				Haemetemesis	Malena	Jaundice	Dyspepsia	Pain abdomen	Haematochezia	Heart burns	Loss of weight	Loss of appetite	Nausea	Vomiting	Diabetes mellitus	Hypertension	Acid peptic disease	Intake of NSAIDs / Aspirin	Surgery	Sclerotherapy	Bleeding diathesis	Liver disease	Family history of liver disease	Smoking	Alcohol	Tobacco chewing	Diet	Pulse rate (/Min)	Systolic	Diastolic	Pallor	Icterus	Pedal oedema	Generalised lymphaden	Left supraclavicular nod	Tenderness	Dilated veins/scars	Mass / lump	Hepatosplenomegaly	Ascites	Free fluid	Cardiovascular system	Respiratory system	Per abdomen	Central nervous system	UGI scopy	Diagnosis
21	539280	60	F	+	-	-	-	-	-	-	+	-	-	-	+	+	-	-	-	-	-	-	-	-	-	+	V	70	140	80	+	-	-	-	-	-	-	-	-	-	-	N	N	N	N	1	2
22	539254	45	F	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	-	-	m	88	136	80	+	-	-	-	-	-	-	-	+	-	-	N	N	AB	N	1,5	6
23	538181	62	M	+	-	+	+	-	-	-	-	+	-	-	-	-	-	+	-	+	-	+	-	+	-	-	m	78	130	80	-	-	-	-	-	-	-	-	+	+	-	N	N	AB	N	1	1
24	545058	60	M	+	+	+	-	-	-	-	-	-	+	+	-	-	-	-	-	-	-	-	-	-	-	-	m	82	136	80	-	+	-	-	-	-	-	-	+	+	-	N	N	AB	N	1	7
25	540498	45	M	+	-	-	-	-	-	-	-	-	+	+	-	-	-	-	-	-	-	-	-	-	+	-	V	80	140	90	+	-	-	-	-	-	-	-	+	-	-	N	N	N	N	1	6
26	540486	31	F	+	-	-	-	+	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	V	80	120	70	-	-	-	-	-	-	-	-	-	-	-	N	N	AB	N	4	5
27	543800	45	M	-	+	-	+	+	-	+	+	-	-	-	-	-	+	+	-	-	-	-	-	-	-	-	m	92	138	96	+	-	-	-	-	+	-	-	-	-	-	N	N	AB	N	4	5
28	545420	52	M	+	-	+	-	-	-	-	+	+	+	-	-	-	-	-	-	-	-	-	-	-	+	+	m	90	100	60	-	+	+	-	-	-	+	-	+	+	-	N	N	AB	N	1	1
29	504821	55	M	+	-	-	-	-	-	-	+	+	+	-	-	-	-	-	-	-	-	+	-	+	+	-	V	70	132	80	+	+	-	-	-	-	-	-	+	+	-	N	N	AB	N	1	1
30	556616	40	M	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	V	82	140	90	+	+	+	-	-	-	-	-	+	-	-	N	N	N	N	1	7
31	561023	64	M	-	+	-	-	-	-	-	-	-	-	-	+	+	-	-	-	-	-	-	-	-	-	+	m	72	132	80	+	-	-	-	-	-	-	+	+	-	-	N	N	AB	N	1	7
32	558098	50	M	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	+	+	m	76	160	70	+	-	-	-	-	-	-	-	+	-	-	N	N	N	N	5	1
33	561622	35	M	+	-	-	+	+	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	m	106	100	60	+	-	-	-	-	-	-	-	-	-	-	N	N	N	N	4	5
34	564552	35	M	+	-	+	+	-	-	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	m	86	120	70	-	-	-	-	-	+	-	-	-	-	-	N	N	AB	N	4	5
35	565197	50	M	+	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	m	78	120	70	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	2	3
36	562714	42	M	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	m	74	150	82	+	-	-	-	-	-	-	-	+	-	-	N	N	N	N	1	1
37	567523	62	M	-	+	-	-	-	-	-	-	-	-	-	+	+	-	+	-	-	-	-	-	-	-	-	m	72	126	70	-	-	-	-	-	-	-	-	+	-	-	N	N	N	N	1	8
38	511527	66	M	-	+	-	-	+	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-	-	-	-	V	70	116	80	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	10	10
39	522055	65	M	+	-	-	+	+	-	+	-	-	+	-	-	-	+	-	-	-	-	-	-	-	-	-	V	70	126	80	+	-	-	-	-	-	-	-	-	-	-	N	N	AB	N	3	11
40	520478	65	M	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	V	70	136	80	-	-	-	-	-	+	-	-	-	-	-	N	N	AB	N	4	3

ANNEXURE III - MASTER CHART

Serial Number	In/Out patient number	Age (Years)	Sex	Complaints											History												General physical examination findings																		Systemic examination					
																							Personal					BP (mm Hg)		Signs																					
				Haemetemesis	Malena	Jaundice	Dyspepsia	Pain abdomen	Haematochezia	Heart burns	Loss of weight	Loss of appetite	Nausea	Vomiting	Diabetes mellitus	Hypertension	Acid peptic disease	Intake of NSAIDs / Aspirin	Surgery	Sclerotherapy	Bleeding diathesis	Liver disease	Family history of liver disease	Smoking	Alcohol	Tobacco chewing	Diet	Pulse rate (/Min)	Systolic	Diastolic	Pallor	Icterus	Pedal oedema	Generalised lymphaden	Left supraclavicular nod	Tenderness	Dilated veins/scars	Mass / lump	Hepatosplenomegaly	Ascites	Free fluid	Cardiovascular system	Respiratory system	Per abdomen	Central nervous system	UGI scopy	Diagnosis
41	519869	31	M	+	-	-	-	-	-	-	-	-	+	-	-	-	+	-	-	-	-	-	-	-	-	-	m	72	130	80	+	-	-	-	-	+	-	-	-	-	-	N	N	AB	N	2	4
42	517703	71	M	+	+	-	-	+	-	-	-	-	+	-	-	+	-	-	-	-	-	-	-	-	+	-	m	70	150	80	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	2	4
43	516375	54	M	-	+	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	+	-	m	80	150	80	+	-	-	-	-	-	-	-	+	+	-	N	N	AB	N	1	1
44	522926	39	M	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	+	-	m	90	125	80	+	-	-	-	-	-	-	-	+	+	-	N	N	AB	N	1	1
45	540933	52	M	+	-	-	-	-	-	-	-	-	+	+	-	-	-	-	-	-	-	-	-	-	+	-	m	88	108	72	+	-	-	-	-	+	-	-	-	-	-	N	N	AB	N	2	4
46	514740	62	M	-	+	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	-	-	-	-	-	-	m	90	108	72	+	-	-	-	-	-	+	-	+	+	-	N	N	AB	N	1	8
47	545470	52	M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	m	72	110	70	-	+	-	-	-	+	-	-	-	+	-	N	N	AB	N	1	2
48	512881	59	M	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	+	V	84	112	80	-	-	-	-	-	-	+	-	+	+	-	N	N	AB	N	1	1
49	551790	55	M	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	m	80	126	70	+	-	-	-	-	-	+	-	-	+	-	N	N	AB	N	1	2
50	5517719	65	M	+	+	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	V	88	120	72	+	-	-	-	-	+	-	-	-	-	-	N	N	AB	N	3	3