Dissertation on

VALIDATION OF HIGH RISK CLINICAL SCORING - AS A SENSITIVE METHOD TO PREDICT ENDOSCOPIC SEVERITY AND ADVERSE OUTCOME IN PATIENTS WITH UGI BLEED

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CERTIFICATE

This is to certify that this dissertation in **"VALIDATION OF HIGH RISK CLINICAL SCORING - AS A SENSITIVE METHOD TO PREDICT ENDOSCOPIC SEVERITY AND ADVERSE OUTCOME IN PATIENTS WITH UGI BLEED"** is a work done by **Dr.S.SAKTHI VELAYUTHAM,** under my guidance during the period 2004 - 2007. This has been submitted in partial fulfillment of the award of M.D. Degree in General Medicine (Branch - I) by the Tamil Nadu Dr.M.G.R. Medical University, Chennai - 600 032.

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DECLARATION

I solemnly declare that this dissertation entitled **"VALIDATION OF HIGH RISK CLINICAL SCORING - AS A SENSITIVE METHOD TO PREDICT ENDOSCOPIC SEVERITY AND ADVERSE OUTCOME IN PATIENTS WITH UGI BLEED"** is done by me at Madras Medical College and Government General Hospital during 2004-2007 under the guidance and supervision of **Prof.Dr.D.B.SELVARAJ, M.D.** This dissertation is submitted to Tamil Nadu Dr.M.G.R. Medical University, towards the partial fulfillment of requirement for the award of M.D. Degree General Medicine (Branch - I).

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CONTENTS

INTRODUCTION

Upper Gastrointestinal (UGI) bleeding is a common clinical problem with an incidence of 103-172 / 1,00,000 persons per year[1,2]. Forty four percent of hospitalisations for upper GI bleeding are for patients older than 60 yrs of age[3]. Approximately 80% upper GI bleeding are self limited and require only supportive therapy[4]. Patients with continued or recurrent bleeding have mortality rates of 25% to 30%[4].

Definition

By definition UGI bleed is bleeding from the GI tract proximal to the `ligament of Treitz'.

Gastrointestinal bleeding encompasses a broad array of clinical scenarios. The spectrum is diverse because of the multiple types of lesions that can cause bleeding, and because bleeding can occur from virtually any where in the gastrointestinal tract. Additionally, gastrointestinal bleeding varies greatly in its volume and as such may be massive or trivial, and may be clinically apparent or altogether hidden. Gastrointestinal bleeding is manifest in one or more of the following clinical scenarios (1) bleeding is from the upper gastrointestinal tract; (2) bleeding is from the lower gastrointestinal tract; (3) bleeding is occult (ie, unknown to the patient); or (4) bleeding is clinically obvious but the site (ie, whether it is from the upper or lower gastrointestinal tract) is obscure. Patients with occult bleeding are challenging because the

patient is unaware of the bleeding and clinical clues to its cause are typically lacking. Patients with obscure bleeding are particularly challenging because their bleeding is typically recurrent and the site of bleeding is difficult accurately to identify.

Bleeding from the upper gastrointestinal tract is approximately five times more common than from the lower gastrointestinal tract[5] and seems to be more common in men and the elderly[6].

The clinical presentation of patients with gastrointestinal bleeding typically reflects the site, etiology, and rate of bleeding. Gastrointestinal tract bleeding is manifest in one or more ways. Hematemesis, melena, or hematochezia are the most common manifestations of gastrointestinal bleeding. Hematemesis is defined as vomiting of blood and is caused by upper gastrointestinal bleeding from the esophagus, stomach, or proximal small bowel. Blood may be bright red or it may be old and take on the appearance of coffee grounds. Melena is defined as passage of black, tarry, and foul-smelling stools. The black, tarry character of melena is caused by degradation of blood in the more proximal colon (and is typical of bleeding from the upper gastrointestinal tract). Melena should not be confused with the greenish character of ingested iron or the black, non-foul-smelling stool caused by ingestion of bismuth (ie, in compounds, such as bismuth subsalicylate). Hematochezia refers to bright red blood from the rectum that may or may not be mixed with stool. Occult gastrointestinal bleeding denotes bleeding that is

not apparent to the patient and is caused by small amounts of bleeding. Obscure gastrointestinal bleeding refers to obvious (eg, manifest by hematemesis, melena, or hematochezia) bleeding, but from a source that is not easily identified on routine examination.

The epidemiology of UGI bleed has changed in the last 15 years. Recent development in the diagnosis and treatment of UGI bleed have been offset by the wide spread use of NSAIDS which increase the risk of Acute UGI bleed by almost 4 times overall in the general population. The net effect of these contrasting forces have caused large swings in the origin of UGI bleed.

AIM OF THE STUDY

1. To analyse various parameters that carry higher risk in patients with UGI bleed.

2. To develop a clinical scoring to identify patients with high risk UGI bleed (HRB).

3. To find out the correlation of our clinical scoring with endoscopic findings.

4. To determine the sensitivity of clinical scoring in identifying patients with high risk.

5. To assess the short term outcome (Rebleeding & Mortality) in those patients who had UGI bleed.

REVIEW OF LITERATURE

Aetiology of Upper Gastrointestinal Bleed

1. Duodenal Ulcer
2. Gastric Ulcer
3. Esophageal Varices
4. Esophagitis
5. Mallory-Weiss Tear[7,8]
6. Erosive Gastritis due to
    a) Alcohol[9]
    b) Non-Steroidal Antiinflammatory Drugs (NSAIDS), Aspirin, Steroids, Antimetabolites likie 5-FU, Caffeine.
    c) Chemicals - Kerosene, Ammonia, Acetone, Terpentine, Arsenic, Bromides, Copper Sulphate, Corrosive acids.
    d) Stress Ulcers - Due to Burns, Trauma, Prolonged medical illness, Post-operative complications.
    e) Thermal Injury
    f) Gastric Irradiation
    g) Infections

7. Neoplasms - Carcinoma Esophagus, Gastric Carcinoma, Leiomyoma, Haemangioma, Melanoma, Lymphoma, Polyps

8. Aorto-Enteric Fistula[10,11]

9. Vascular Anamolies - Rendu-Osler Weber syndrome[12], CREST syndrome, Angiodysplasias[13]

10. Haematological disorders - Leukaemia, Thrombocytopenia, Haemophilia, DIC, Polycythemia vera, Von-Willebrands disease.

11. Vasculitis - Pan arteritis nodosa, Henoch Schonlein purpura.

12. Occult UGI Bleed

Esophagus	-	Cameron erosions
Stomach	-	Portal hypertensive gastropathy, Dieulafoy's disease[14], GAVE - Watermelon stomach, AVM - Angiodysplasia
Biliary Tree	-	Haemobilia (trauma or calculus)
Pancreas	-	Aneurysm (Haemosuccus Pancreaticus)
Small Intestine	-	Portal hypertensive intestinal vasculopathy, Neoplasias (leiomyoma, leiomyosarcoma adenocarcinoma), Arteriovenous malformation (angiodysplasia), Aortoenteric fistulas.

Drug induced mucosal lesions (NSAIDS)

Of all the causes listed, the common causes are the following,

1. Peptic Ulcer Disease
2. Erosive Gastritis
3. Variceal Bleed
4. Mallory-Weiss Tear

Peptic Ulcer Disease - Peptic Ulcers are mucosal defects in the duodenum or stomach caused by a breakdown in the normal mucosal defences. It accounts for approximately 45% of UGI Bleed. Duodenal ulcer accounts for 25% and Gastric Ulcer for 20% of the bleed. NSAIDS and alcohol have been implicated as contributing factors alongwith excess of stomach acids. H.Pylori is another major contributing factor and there has been a formidable amount of interest and research in this organism and its clinical effects[15]. It is now known that nearly 100% of chronic superficial gastritis, 90% of Duodenal ulcer and 80% of Gastric ulcer are caused by H.Pylori. Multiple studies show that eradication of the organism markedly decreases the rate of ulcer recurrence.

Stress induced ulcer caused due to Head injury are Cushing's ulcers and that due to Burns are Curling's ulcers.

Erosive Gastritis - Bleeding assocaited with this comes from diffuse superficial erosions in the gastric mucosa which are usually caused by local irritants such as NSAIDS, alcohol, or due to stress. Approximately 20% of UGI Bleed is caused by this.

NSAIDS decrease the synthesis of protective prostaglandins by inhibiting cyclo-oxygenase and may have direct effects on the gastric mucosa causing irritation and superficial lesions[16]. Alcohol ingestion is a frequent cause of gastropathy. The gastric mucosa produces leukotrienes when exposed to alcohol and these may be responsible for the vascular stasis, engorgement and increased vascular permeability which leads to haemorrhage.

Major physiologic stressors like, burns, sepsis, trauma are often associated with gastritis. Decreased splanchnic blood flow during times of severe stress may cause a decrease in the mucus production, bicarbonate secretion and prostaglandin synthesis leading to breakdown in the normal mucosal defence.

Variceal Bleed - Varices are spontaneous venous dilatations of porto systemic collaterals that develop from portal hypertension. Approximately 10% of UGI Bleed is caused by this.1/3rd of patients with cirrhosis will bleed from varices[17]. Child Class C cirrhotics have 50% mortality rate for the first haemorrhage. Death is frequently not directly caused by variceal haemorrhage, but by other end organ failure such as aspiration, hepatic failure, sepsis or renal failure. Varices develop when there is a hepatic vein pressure gradient of more than 12 mm of Hg[18]. This is an indirect way of measuring portal vein pressure. Bleeding is most likely to develop when the Hepatic vein pressure gradient is greater than 18mm of Hg. Prediction of bleeding is difficult and influenced by the size and thickness of the varices. Esophageal varices are the most common venous collaterals but many other porto systemic collaterals can exist and are difficult to treat. Most gastric varices are associated with esophageal varices

and surgical intervention is frequently required. Hence the use of TIPS is becoming more common now[19].

Mallory-Weiss Tear - This occurs at the distal esophagus at the Gastroesophageal junction presumably after retching or vomiting although there is a lack of this antecedent history. This accounts for 7% of UGI Bleed. Bleeding occurs when the tear involves the underlying Esophageal venous or arterial plexus. It appears as an elliptic ulcer at the Gastroesophageal junction within a Hiatal hernia or in the gastric side just below the Gastroesophageal junction endoscopically. The majority heal within 24-48 hours. But patients with Portal hypertension are at an increased risk of massive bleeding from this compared to Non-Portal Hypertension patients. Endoscopic therapy is reserved for tears with active bleeding. Contiguous varices can be treated with sclerotherapy, band ligation or combination therapy. Angiography can be performed to embolise a bleeding vessel.

APPROACH TO THE PATIENTS WITH UGI BLEED

INITIAL PATIENT ASSESSMENT

When a patient is found to have one of the previously mentioned manifestations of gastrointestinal bleeding the first step in management should be to assess the severity of bleeding. Assessment of the patient's hemodynamics should be emphasized (Table 1). This hemodynamic assessment forms the basis for further management. Ongoing assessment of the vital signs further focuses resuscitation efforts, and also provides important prognostic information. Finally, ongoing and careful assessment of the patient's hemodynamic status helps triage appropriate intervention. For example,

patients with obviously unstable vital signs are often bleeding from major vascular sources, such as an ulcer with a visible vessel or gastroesophageal varices[20]; the prognosis of these patients is poorer than that of those with normal vital signs. and their clinical condition mandates more aggressive and timely intervention than patients with normal vital signs.

RESUSCITATION

The more severe the bleeding (ie, unstable vital signs and evidence of ongoing bleeding), the more vigorous the resuscitation efforts should be. In patients who have any evidence of hemodynamic instability, two large -bore intravenous catheters should be placed immediately. Colloid (normal saline or lactated Ringer's solution) should be infused as rapidly as the patient's cardiovascular system allows to restore the vital signs towards normal. ICU monitoring is indicated in hemodynamically unstable patients. Supplemental oxygen by nasal cannula or facemask should be given liberally. Vital signs and urine output should be monitored closely, and in selected situations (for patients with underlying cardiopulmonary disease) central venous monitoring is helpful. The importance of aggressive ICU monitoring and resuscitation has been emphasized by investigation suggesting that it may decrease mortality[21].

In addition to colloidal solution, patients must typically undergo blood transfusion. Decision about when and how much to transfuse the patients with gastrointestinal bleeding is often complicated and requires integrations of multiple aspects of the clinical situation. Virtually all patients with unstable vital signs have had significant blood loss and require blood transfusion. If the patient has subnormal tissue oxygenation, transfusion should be aggressive.

Patients with continued instability in vital signs, continued bleeding, symptoms of poor tissue oxygenation, or persistently low hematocrit value (20% - 25%) likewise should probably be transfused continuously. It is most appropriate to raise the hematocrit to a level of 30% in elderly patients, whereas in younger, otherwise healthy patients, hematocrit values in the 20% to 25% range may be satisfactory; in those with portal hypertension, it should not be above 27% to 28%. Transfusion should be with packed red blood cells, except in rare circumstances where whole blood transfusion may be used in those who cannot be cross-matched in a timely fashion. In those with specific defects in coagulation factors or platelets, these substances can be replaced. Patients requiring greater than 10 units of packed red blood cells should receive Fresh-frozen plasma or platelets or both. Warmed blood should be administered to patients requiring massive transfusion. (ie, > 3000 m L). The hematocrit should be monitored serially (typically after a specific transfusion). Serial hematocrits are not a substitute, however, for ongoing clinical assessment of the hemodynamics. Frequently, patients with chronic bleeding may have a low hematocrit, but no evidence of hemodynamic instability. In this situation, blood transfusion should be slow and deliberate, regardless of the hematocrit value.

TABLE - 1

HEMODYNAMICS, VITAL SIGNS, AND BLOOD LOSS

Hemodynamics vital sign	**% Blood loss (fraction of intravascular volume)**	**Bleed type**
Shock (resting hypotension)	20-25	Massive
Postural (orthostatic tachycardia or hypotension)	10-20	Moderate
Normal	<10	Minor

HISTORY, SYMPTOMS, AND SIGNS

Once the patient's hemodynamics and overall condition has been assessed and stabilized, attention should turn to the clinical history. The history helps the clinician assess the severity of bleeding and make a preliminary assessment of the site and cause. Historical features important in assessing the etiology of gastrointestinal bleeding are shown in Box 1.

Box 1 : Historical features in the assessment of gastrointestinal bleeding
Age
Prior bleeding
Previous gastrointestinal disease
Previous surgery
Underlying medical disorder (especially liver disease)
Nonsteroidal anti-inflammatory drugs (NSAIDs), Aspirin (ASA)
Abdominal pain
Change in bowel habits
Weight loss or anorexia
History of oropharyngeal disease

Simple demographic charecteristics are an essential part of the history. For example, elderly patients may bleed from a number of diseases less common in younger persons (ie, vascular ectasia, diverticula, ischemic colitis, cancer), whereas bleeding in younger patients is more likely to be from esophagitis, varices, or Meckel's diverticula (typically lower gastrointestinal bleeding in patients under 30 years of age). A past history of previous gastrointestinal disease or previous bleeding should focus the differential diagnosis immediately on related bleeding (eg, hereditary hemorrhagic telangiectasia, ulcer disease, diverticular bleeding). A history of previous

surgery is likewise important. For example, a history of previous aortic surgery should increase the suspicion for aortoenteric fistula. A history of liver disease raise the possibility of bleeding associated with portal hypertension. Ingestion of medications, such as aspirin or other nonsteroidal anti-inflammatory drugs, makes bleeding from ulceration more likely. Patients taking anticoagulant medications may be more likely to bleed from ulcers or vascular ectasias. Gastrointestinal bleeding in the setting of anticoagulation therapy, even in patients taking warfarin and who have a supratherapeutic international normalized ratio, is most often caused by underlying gastrointestinal tract pathology and should not be ascribed to over anticoagulation[22]. Other important historical features include abdominal pain (which suggests peptic ulcer disease, mesenteric or colonic ischemia) retching (Mallory - Weiss tear); or change in bowel habits, anorexia, or weight loss, all of which point to malignancy. Interestingly, elderly patients may be less likely to report abdominal pain associated with bleeding ulcers[23]. The history is also critical in ascertaining whether nongastrointestinal sources may be the casue of reported or witnessed bleeding, especially from the lungs or nasopharynx.

Physical examination should focus on putative evidence of liver disease (splenomegaly, ascites, caput), which increases the likelihood of portal hypertension and related cause of bleeding. The skin may also reveal evidence of chronic liver disease (eg, cutaneous spider angiomata, Dupuytren's contractures). Acanthosis nigricans may reflect underlying cancer (especially gastric cancer). Cutaneous telangiectases of skin or mucous membranes and lips raise the possibility of hereditary hemorrhagic telangiectasia (Osler - Weber - Rendu disease); pigmented lip lesions are seen with Peutz - Jeghers

syndrome; cutaneous tumors suggest neurofibromatosis; and purpura is consistent with vascular disease (Henoch - Schonlein purpura or polyarteritis nodosa). Abdominal tenderness (peptic ulcer, pancreatitis, and ischemia), abdominal masses, lymphadenopathy (malignancy), and splenomegaly (cirrhosis, splenic vein thrombosis) are all important to detect.

Hematemesis, melena, and hematochezia are classic symptoms and signs of gastrointestinal bleeding. It requires at least 50 mL of blood in the upper gastrointestinal tract for melena to become clinically apparent, although volumes of up to 100 mL of blood when infused into the stomach may be clinically silent[24]. When bright red blood is vomited, this typically signifies upper gastrointestinal bleeding that is significant, and is often caused by varices or an arterial lesion. Smaller amounts of bleeding that is significant, and is often caused by varices or an arterial lesion. Smaller amounts of bleeding from many other lesions are alarming to patients, however, and are often reported. Careful inquiry about the volume of vomited blood is essential. Witness of the amount of and character of blood may also be useful in ascertaining the character of bleeding. Patients with coffee ground emesis are not usually bleeding actively but have had a recent or even remote bleed. Although hematochezia can be caused by bleeding from many different sites in the gastrointestinal tract, the higher the site of bleeding the more hemodynamically significant. Small amounts of hematochezia are often reported by the patient, and care should be taken to ascertain the volume of blood. Chronic occult blood loss may lead to end-organ symptoms, such as lightheadedness, dyspnea, angina pectoris, or even myocardial infarction.

Bedside examination of the character of the stool is an essential and mandatory part of the physical examination. This part of the examination provides critical information about the site of bleeding, and also about the acuity of bleeding. For example, patients who are passing stools containing red blood, maroon-colored blood, or melena have active bleeding. In contrast, patients with brown stools are unlikely to have aggressive bleeding. Likewise, patients with infrequent stools are unlikely to have active bleeding, and those with a history of coffee ground emesis only and normal appearing stools, whether positive for occult blood or not, have usually had a trivial bleed.

DIAGNOSIS

Endoscopy

This has become accepted as the diagnostic study of choice in UGI Bleed[25]. It is well known that the majority of UGI Bleed will stop spontaneously and it is unlikely that urgent endoscopy will improve care or survival. However, 20% of the patients will have continued bleeding or have high risk lesions that will benefit from endoscopic evaluation and therapy.

Major therapeutic decisions will also be made based on endoscopic findings in patients with portal hypertension, prior gastric surgery and suspected aorta enteric fistula. Sclerotherapy of varices, treatment of actively bleeding lesions and treatment of high risk lesions in ulcers have been shown to improve survival, lower transfusion requirements, and decrease morbidity. Endoscopy may be most helpful in determining which of the several potential lesions are actually bleeding and in predicting which patients may rebleed as

evidenced by a visible vessel in an ulcer crater. Diagnostic accuracy is highest if performed within first 24 hours of bleeding[26].

Hazards of Endoscopy

1. Erroneous Diagnosis-With an experienced person errors are minimal.
2. Complications of Medication
    a) Local anaesthesia
        i. Acute cardiac rhythm disturbance
        ii. Circulatory failure
    b) Diazepam
        i. Increased salivation
        ii. Tendency to pulmonary aspiration
        iii. Amnesia
3. Perforation
4. Pulmonary aspiration
5. Cardiovascular complications
    - ✓ Changes in cardiac rhythm
    - ✓ Supra ventricular ectopics
    - ✓ Ventricular ectopics
    - ✓ ST-T changes

The morbidity and mortality in endoscopy is very low (0.3% to 0.03% respectively).

Endoscopic therapy

The choice of modality is generally not important. Institutional preference, the experience of the endoscopist and the availability of resources are the most important determining factors. For treatment of bleeding ulcers, laser therapy is useful but less effective than the other modalities, but due to high cost, immobility, and other technical reasons, laser therapy is not used for UGI Bleed.

Active haemorrhage can be controlled using heater probe. The heater probe delivers energy as heat to the distal, coated tip. Haemostasis is achieved by the use of direct pressure to the bleeding site with heat.

Injected substances include 1% polidocanol, thrombin, Absolute ethanol, epinephrine (1:10,000 dilution), and sodium tetradecyl sulfate. Injection therapy was atleast as effective as thermal therapy at preventing surgery and decreasing mortality in patients with bleeding ulcers.

Endoscopic Sclerotherapy (EST) has become widely available in the last two decades and is now the most common definitive therapy for bleeding esophageal varices[27]. Multiple agents have been used including sodium tetradecyl sulfate (1% to 3% solution), ethanolamine 5% and polidocanol. The type of sclerosant used does not appear to be important and intravariceal

injection is preferred to paravariceal injection, but both methods are effective. For acute control of haemorrhages, EST has 66-100% success rate and has proved more effective than balloon tamponade or medical therapy.

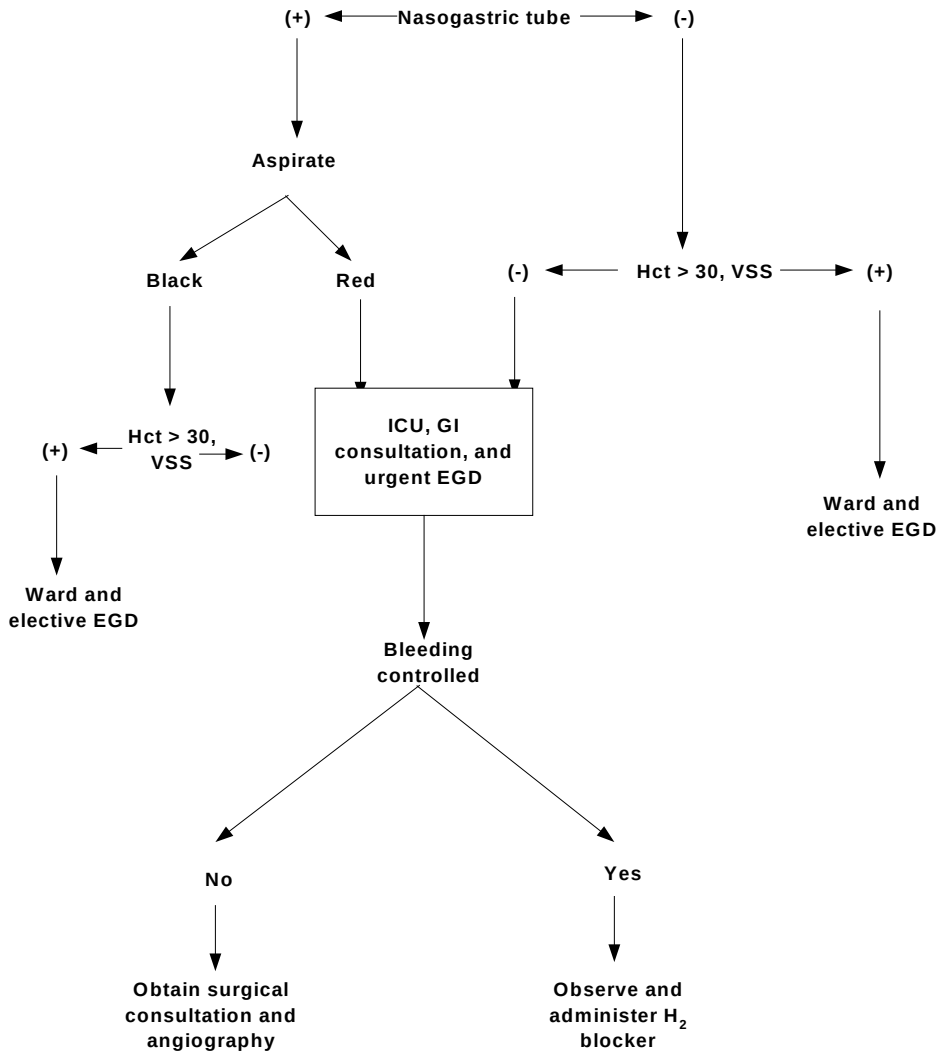
In 1986 Van Stiegmann and associates reported a new technique for endoscopic therapy of varices. It involved the placement of a small rubber band over the varices and has been termed Esophageal Band Ligation (EBL). EBL showed a significantly lower complication rate than EST (2% vs 22%) including less mortality[28]. Acceptance of EBL has been limited for several reasons. Multiple bandings per session are required and the present devices can only place one band before requiring reloading. Due to frequent removal of the scope for reloading, an overtube is required. The overtube may be difficult to place and many gastroenterologists are not trained to perform the procedure. Clear distal sleeves (which will make visualization of the varices easier), multiple banding devices are under development and may facilitate acceptance of this new technique and more widespread use.

THERAPY OF SPECIFIC LESIONS WITH UGI BLEED

The following algorithms are followed for management of suspected non - variceal and variceal UGI Bleed in general.

Fig. 1

I. Treatment algorithm for suspected nonvariceal upper gastrointestinal bleeding

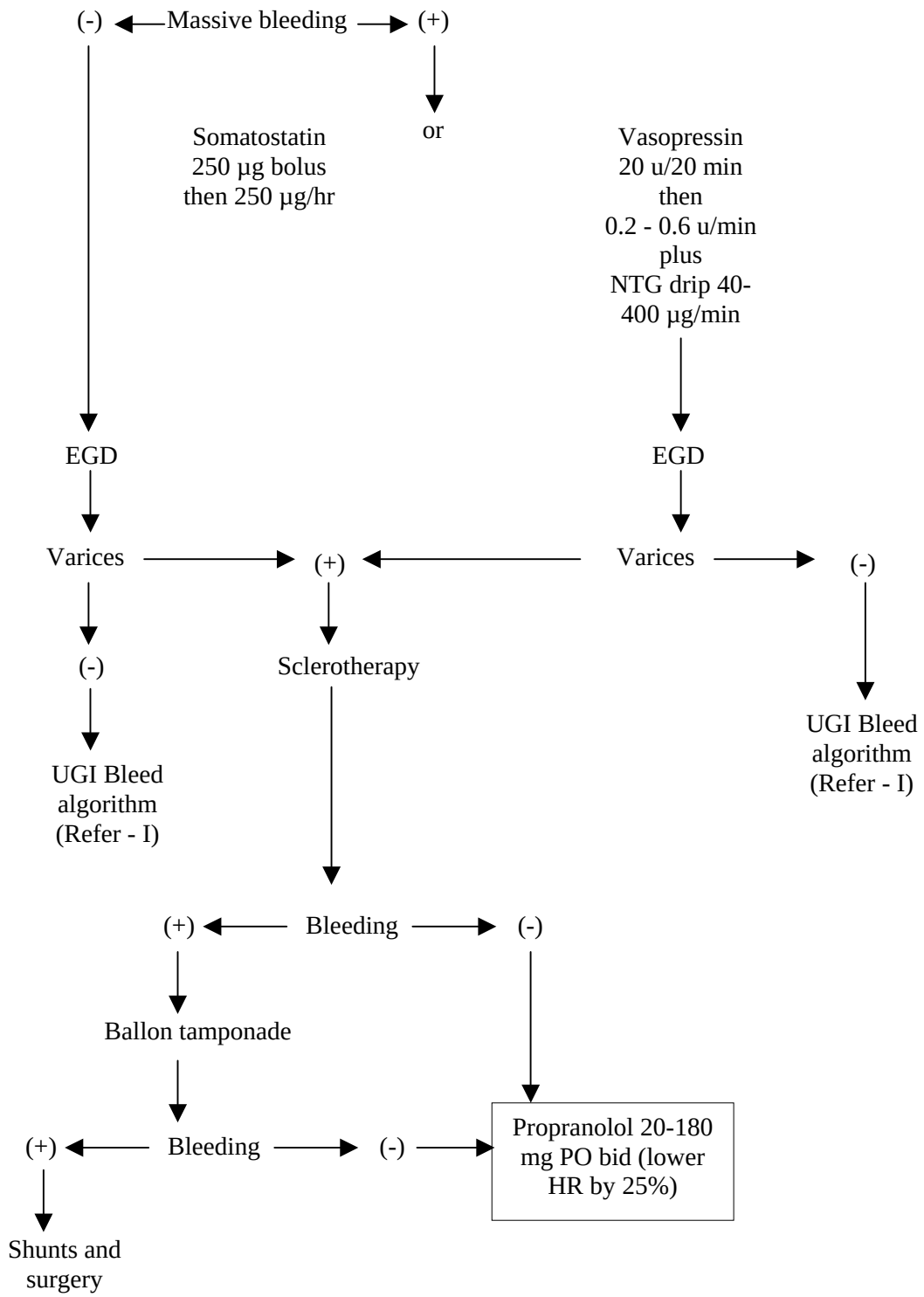


EGD - Esophagogastro - duodenoscopy
ICU - Intensive care unit

VSS - Vital signs stable
Hct - Haematocrit

Fig. 2

II. Treatment algorithm for suspected variceal bleeding



EGD - Esophagogastro - duodenoscopy
NTG - Nitroglycerin

Peptic Ulcer disease: The goals of Ulcer therapy include relief of symptoms and prevention of recurrence and complication.

a) **Gastric Ulcer**: In patients with Gastric Ulcer follow up with an upper endoscopy should be pursued until complete healing is documented. Surgical therapy should be considered for non-healing gastric ulcers as 5% turn out to be malignant.

b) **Duodenal Ulcer**: They are never malignant. Therefore, demonstration of ulcer healing in the absence of symptoms is unnecessary.

c) **H-pylori**: This can be cultured from the stomach of approximately 90% of Duodenal Ulcer patients and 70% of Gastric Ulcer patients. Determination of Duodenal Ulcer or Gastric Ulcer by endoscopy should be followed by confirmation of H-Pylori infection. Ulcer patients with H-pylori infections require anti-microbial therapy.

Reduction of stomach acidity and treatment of H-Pylori infection are the main goals of therapy in Peptic Ulcer Disease.

I. Medical Treatment

1) H-Pylori regimens: Triple therapy with the following combinations.

Bismuth subsalicylate, Metronidazole, Tetracycline (or)

Lansoprazole, Clarithromycin, Amoxicillin (or)

Metronidazole, Amoxicillin, Omeprazole are used in appropriate dosage for about 2 weeks for eradication of H-pylori infection.

2) H2-Receptor antagonists

a) Duodenal Ulcer

- **Acute Therapy** - Cimetidine, Ranitidine, Famotidine, Nizatidine, Roxatidine are all effective.

- **Prevention Therapy** - Therapy to prevent recurrent ulcer disease includes eradication of H-pylori infection and maintenance of H2 receptor antagonist therapy. There is no consensus on length of treatment.

- **Benign Gastric Ulcer**-Cimetidine, Ranitidine and Famotidine are all effective in dealing gastric ulcer.

- **Proton Pump inhibitors**-Omeprazole, Lanzoprazole profoundly decrease gastric acid secretion by inhibiting Hydrogen-potassium adenosine triphospates.

- **Sucralfate** - As effective as H2 receptor antagonist or high dose antacids in healing Duodenal Ulcer. It acts locally on the mucosal surface.

- **Antacids** - Used best as supplemental therapy for pain relief. Choice is determined by buffering capacity, sodium content and side effects. In general liquid antacids are more effective than tablets.

6) Other therapeutic measures

- **Dietary modification** - Avoidance of food that are reproducibly associated with dyspeptic symptoms. However, there is no evidence that a bland diet, improves symptoms or promotes ulcer healing.

- **Cessation of smoking** - Smoking is associated with an increased risk of peptic ulcer disease, delayed ulcer healing and an increased rate of recurrence[29].

- **NSAIDS and Aspirin** - These should be avoided as they are toxic to the gastric mucosa and are associated with dyspepsia and mucosal ulceration. Concomitant therapy with proton pump inhibitors, H2 receptor antagonist or sucralfate may ameliorate these symptoms. Misoprostal can help prevent NSAIDS associated gastric ulcer[30].

- **Alcohol** - Should be avoided as it damages the gastric mucosal barrier and is associated with gastritis.

II. Surgery

Indications:

Absolute : Massive bleeding

Combined perforation and bleeding

Relative : Presence of persistent shock

Rebleeding after admission

Obvious arterial bleeding at the time of endoscopy

Demonstration of large chronic ulcers, continued slow bleeding

III. Arterial angiotherapy

Can be used to control massive UGI Bleed in patients considered at high risk for surgical interventions.

1) Arterial Vasopressin produces cessation of bleeding in some gastric lesions and duodenal ulcer. But patients with known cardiac disease are at increased risk for complications of vasopressin.

2) Arterial embolization with absorbable gelatin sponge (Gelfoam). By this, control of bleeding is obtained in 70% of patients.

Specific treatable lesions include

- Mallory Weiss tear (100%)
- Duodenal Ulcer and bleeding gastritis (67%)
- Gastric ulcer (50%)
- Stress ulcer (55%)

Other agents used for arterial embolization include cyanoacrylate and thrombin.

3) Intravenous Vasopressin-The IV route is not recommended in bleeding lesions other than esophageal varices because vasopressin is ineffective in arterial bleeding and has no proven effect in other bleeding lesions.

Esophageal Variceal Haemorrhage

This is a medical emergency associated with high morbidity and mortality. If the diagnosis is confirmed and active bleeding persists several therapeutic options exist.

1) **Endoscopic Therapy**: Treatment of choice for acute variceal bleed. After the acute variceal haemorrhage has been controlled by endoscopic therapy it is also used to prevent variceal rebleeding.

a) **Sclerotherapy** - Effective for controlling primary haemorrhage and obliterating varices after the initial bleeding episode but is associated with significant complications. Recurrent bleeding occurs in upto 50% of patients that can be controlled with further sclerotherapy.

Complications-Ulceration, strictures, perforation, sepsis, fever.

b) **Variceal ligation or banding** - As effective as sclero therapy in controlling active haemorrhage and achieves variceal eradication

more rapidly with low rates of rebleeding and few complications[31].

Complications - Superficial ulceration, dysphagia, esophageal strictures, transient chest discomfort.

2) **Pharmacologic Therapy** : Less effective than endoscopic therapy. Nevertheless ready availability, ease of administration and the potential for success makes it a reasonable alternative when other therapies are unavailable.

   a) **Octreotide acetate** - Long acting synthetic analogue of somatostatin. Given as 50-100 micro gram. IV bolus followed by an infusion of 25-50 micro gm/hour.

   b) **Vasopressin** - Until recently, was the most widely used agent. A standard mixture of 100 units of vasopressin in 250ml of D5W. Starting dose 3 units/minute IV for 30 minutes followed by increments of 3 units/minute every 30 minutes until haemostasis is achieved. Concomitant infusion of nitroglycerin may decrease the cardiovascular side effects of vasopressin and provide more effective control of bleeding[32].

3) Balloon tamponade - Effective in temporary control of variceal bleeding before more definitive therapy can be undertaken[33]. The Sengstaken-Blakemore tube, Minnesota tube, Linton tube can be used. But this is

associated with a high rate of major complications and mortality from tube displacement. Endoscopy should confirm the site of bleeding and direct tube selection.

4) Surgery - Portacaval or Distal spleno renal shunt[34]. This controls variceal bleeding in 95% of the patients. In patients with good hepatic reserve, shunts should be considered if the patient,

- fails endoscopic or pharmacological therapy
- unable to return for follow up
- at high risk for death from recurrent bleeding

5) **Tips** - Radiologic alternative to a surgical shunt. An expandable metal stent is placed between the hepatic veins and portal vein. TIPS effectively decompresses the portal vein and is used for the following patients.

- one who fails endoscopic therapy
- has gastric variceal bleeding
- poor surgical candidates
- awaiting liver transplantation

- recurrent variceal haemorrhage who fails to respond to endoscopic/pharmacological therapy[35].

6) **Pharmacologic Prophylaxis** - Beta adrenergic antagonist has shown to decrease Portal hypertension. Propranolol and Nadolol in dosages sufficient to decrease the resting heart rate by 25% are effective prophylactic therapy for first variceal bleeding in patients with large varices, and are effective in preventing recurrent variceal bleeding, though no benefit in overall survival has been shown[36]. The addition of oral nitrates, improves the therapeutic effect of beta adrenergic antagonist in preventing variceal haemorrhage and may be as effective as sclerotherapy.

7) Hepatic Transplantation - In selected patients, this can reverse Portal hypertension and variceal haemorrhage.

Mallory-Weiss tear - Most of them stop bleeding spontaneously and seldom have recurrent bleeding. Some require therapeutic endoscopic technique to control the bleeding. Selective infusion of vasopressin into the left gastric artery has been successful for patients who continue to bleed. Rarely surgery to oversew the bleeding vessel may be necessary.

Erosive Gastritis - Identification of patients at risk allows for treatment to prevent ulcer formation and thus to prevent bleeding. At risk patients are those with

- major trauma
- head injury
- hypovolemic shock
- sepsis
- burns

In medical ICU, significant UGI Bleed-frank blood or coffee grounds NG aspirate or melena occurs in 6% of patients and occult bleeding in 14%. Prophylactic therapy to prevent stress bleeding includes H2 antagonist, high potency antacids through NG tube. Patients with erosive gastritis due to alcohol or drugs are advised to abstain from alcohol and stop the offending drugs. They can be treated with H2 antagonists and antacids.

ENDOSCOPY

The flexible endoscopy is a complex tool. It consists basically of a control head with eye piece and controls, and flexible shaft which has a maneuverable tip. The head is connected to a light source via connecting "umbilical" cord through which pass other tubes transmitting air, water, and suction, etc. Accessories include flexible biopsy forceps for passage through the instrument, a side arm for assistant's viewing and cameras. The image is transmitted either by fibre optics or electronically from a video chip.

At the heart of any fibre optic instruments are the viewing and light carrying bundles. In most modern instruments the distal lens which focusses the image onto the bundle is fixed and transmitted to the eye via a focussing lens.

First generation video endoscopes are mechanically identical to fibre endoscopes with a video chip and supporting electronics mounted at the tip, to and fro wiring, replacing the optical bundle and further electronics and switches occupying the site of the optical lens on the upper part of the control head. The ease of stances, brighter view and the natural visual field makes video endoscopes exceedingly relaxing to use which is beneficial to good endoscopy and patient communication.

The instruments used in our set up are

1. Clympus CV-20 2. Pentax FG2QP 3. Pentax Video System

The whole apparatus consists of the following

- The scope
- Biopsy needles
- Teaching scope
- The light source
- Brush
- Video monitor

Procedure - Patients were advised nil oral from 8 pm the previous night until the commencement of the procedure the next day. Ryle's tube aspirtation was done in the morning one hour before the commencement of the procedure. Normally no medication is given to the patient. The patient was put on left lateral position, the scope smeared with xylocaine jelly and introduced under direct vision past the epiglottis into the esophagus. The patient was encouraged to swallow the tube and the mucosa was constantly visualised through the eye piece, at the same time keeping a watch over the distance traversed by the scope. At no time was undue force exerted to pass the instrument.

In the esophagus the mucosal pattern and presence of varices were looked for. The level of esophago-gastric junction and its patulence were observed. The tube was then passed into the stomach (by the J maneuver) and the body, antrum and fundus were visualised. The instrument was further passed into the duodenum and lesions if any were visualised. Then the scope was carefully withdrawn seeing the mucosal pattern once again to confirm the findings as well as to see if any traumatic lesions were produced by the instrument. During the entire procedure the patient's vitals were frequently monitored.

MATERIALS AND METHODS

50 consecutive patients admitted in medical, surgical wards of Government General Hospital, Chennai, were taken into the study.

Study design : Prospective study

Venue : Govt. General Hospital, Chennai.

Study period : January 2006 to June 2006

All patients were examined at the time of admission. Accurate history with emphasis on relevant past history, NSAIDS, past UGI bleed, were taken in all patients. Complete physical examination was carried out as per protocol. Relevant simple investigations were carried out in each patient within 2 hrs of admission. All the results were noted.

All patients with UGI bleed were subjected to upper GI endoscopy within 24hrs.

Endoscopy was done in Olympus CV-20 apparatus by experienced Gastroenterologists and the findings were recorded.

Endoscopic interventions like injection therapy, Endoscopic sclero therapy, EVL were done as needed.

The patients were treated with resuscitation, transfusion, vasoconstrictors, PPI, H_2RI, antibiotics as needed.

All patients were observed for 1 week in hospital and evidence of rebleed, and death were recorded. All data were tabulated.

Each parameter was assigned a score, and patients with high risk were identified as those with score > 20.

Statistics

Statistical observations were done by Chi-Square chart with Pearson logistic regression analysis.

Based on quartiles the scores above 3rd quartile (75%) were called High risk and below 75% were called low risk. Patients above the score 20 (75%) were called high risk.

The values were recorded.

OBSERVATION

UGI bleed is a common medical emergency. As our hospital, the Government General Hospital, Chennai, being a tertiary care centre, we receive lot of cases of upper Gastrointestinal bleeding. we studied 50 cases of upper Gastrointestinal bleed, prospectively, and the observations are given below.

AGE

The age of the patients admitted was from 15 - 75 years. We divided the patients into three age groups. The number of patients in each age group is given below.

TABLE - 2

AGE WISE DISTRIBUTION

Age in years	No. of Cases	Total No. of Cases	Percentage (%)
< 40	21	50	42
40 - 60	24	50	48
> 60	5	50	10

Predominant age group is 40 - 60 yrs, accounting for 48%.

GENDER

The incidence of UGI bleed in both Male and Female sex is as follows.

TABLE - 3

SEX WISE DISTRIBUTION

Sex	No. of Cases	Total No. of Cases	Percentage (%)
Male	40	50	80
Female	10	50	20

Males were predominantly affected with UGI bleed. (80%). The ratio of Male : Female is 4 : 1.

MODE OF PRESENTATION

In our study, all the 50 patients, presented with Hemetemesis and none of them presented with melena alone. Of them, 45 patients presented with melena. Six patients had hematochezia. The mode of presentation is as follows.

TABLE - 4

MODE OF PRESENTATION

Presentation	No. of Cases	Total No. of Cases	Percentage (%)
Haematemesis	50	50	100
H & Melena	45	50	90
Haematochezia	6	50	12

SEVERITY OF HAEMETEMESIS

One of the major determining factors of outcome is quantity of bleed. In our study 5 (10%) patients presented with massive haemetemesis.

TABLE - 5

SEVERITY OF HAEMETEMESIS

Haemetemesis	No. of Cases	Total No. of Cases	Percentage (%)
< 500 ml	32	50	64
500 - 1000 ml	13	50	26
> 1000 ml	5	50	10

HAEMOGLOBIN

Haemoglobin percentage of the patients admitted with UGI Bleed, was calculated within 2 hrs of admission. We divided patients into 3 groups depending upon the severity of anemia. The results are given below.

Hb% AT INITIAL PRESENTATION

TABLE - 6

Hb% AT INITIAL PRESENTATION

Hb% (in grams%)	No. of Cases	Total No. of Cases	Percentage (%)
<5	5	50	10
5 - 9	15	50	30
≥ 10	30	50	60

5(10%) patients presented with very severe anaemia with Hb% < 5 gm%.

RYLE'S TUBE ASPIRATE

Ryle's tube aspirate was bright red in 15 (30%) patients, coffee - brown in 27 (54%) patients and was clear in 8(16%) patients. Bright red indicates current bleeding while coffee brown indicates recent or even old bleed.

RECTAL EXAMINATION

Bright red colour noted in 5(10%) patients, maroon in 10 patients and black in 25 patients. Rectal examination was normal in the remaining patients. Bright Red Rectal examination is a high risk clinical feature indicating significant bleeding.

VITAL PARAMETERS

Pulse, blood pressure and consciousness level play an important role in prognostication. The results are as follows.

TABLE - 7

VITALS

Parameter	Grade	No. of patients	Total no of cases	Percentage (%)
Pulse	< 100	32	50	64
	100 - 120	10	50	20
	> 120	8	50	16
Blood pressure	Normal	33	50	66

	Postural Hypotension	11	50	22
	Resting Hypotension	6	50	12
Consciousness level	Normal	30	50	60
	Dizziness	16	50	32
	Unconscious	4	50	8

Pulse > 120, Hypotension, unconsciousness are high risk features.

HISTORY

* Of the 50 patients, 24 (48%) patients were alcoholics.

* 23 (46%) patients were smokers.

* NSAIDS intake history was present in 9 (18%) cases, in the previous 48 hours.

* Previous history of PUD was present in 10 (20%) cases.

* Previous history of UGIB was present in 10 (20%) cases.

COMORBIDITIES

Comorbidities are major determinants of adverse outcome in UGI bleed.

* In our study, 7 patients had ascites.

* Hepatic encephalopathy was present in 3 patients.

* Two patients had been admitted in intensive care unit.

* Renal Failure was present for various reasons in 4 cases.

* Three patients had coronary artery disease.

ENDOSCOPIC FINDINGS

After initial resuscitation and hemodynamic stabilisation, all the 50 patients were subjected to upper Gastrointestinal endoscopy. The results are as follows :

TABLE - 8

ENDOSCOPIC FINDINGS

Lesion	No. of Cases	Total No. of Cases	Percentage (%)
Peptic ulcer	23	50	46
Gastric	4	50	8
Duodenal	14	50	28
Gastric + Duodenal	5	50	10
Mallory weiss	6	50	12
Erosions	10	50	20
Ca stomach	2	50	4
Variceal	9	50	18

TABLE - 9

FORREST GRADING OF ULCER BLEED

Lesion	No. of Cases	Total No. of Cases	Percentage (%)
Active bleeding	2	50	4
NBVV	2	50	4
Adherent clot	15	50	30
Clean base or pigmented ulcer	22	50	44

Peptic ulcer was the most common cause accounting for 46% of cases of UGIB. With duodenal ulcer, being the most frequent lesion (28%). High risk ulcers like, Ulcer with active bleeding and non bleeding visible vessel were diagnosed in 4 (8%) patients.

TABLE - 10

VARICEAL FINDINGS

Lesion		No. of patients
Grade	Mild	1
	Moderate	3
	Severe	5
Column	Mild	3
	Moderate	3
	Severe	3
Colour	Blue	5
	White	4
Gastric varices	Present	5
	Absent	4
PHT Gastropathy	Present	6
	Absent	3
Red Colour signs (RCS)	Present	5
	Absent	4

High grade varices, Red colour lesions, bluish varices are high risk features for Rebleed and mortality. 8 (16%) patients had high grade varices.

BLOOD TRANSFUSION

Of the 50 patients, 22 (44%) patients required blood transfusion and of them 9 (18%) patients required more number of blood transfusions. (>3 units). Blood transfusion was not required in 28 (56%) patients. High blood transfusion requirement is a high risk factor for adverse outcome.

TABLE - 11

BLOOD TRANSFUSION REQUIREMENT

Blood transfusion		**No. of Patients**	**Total no. of Patients**	**Percentage (%)**
Required	Total	22	50	44
	< 3 units	13	50	26
	>3 units	9	50	18
Not required		28	50	56

ADVERSE OUTCOMES

After taking clinical and endoscopic scoring, we studied the clinical course of the patients. The results are as follows :-

TABLE - 12

ADVERSE OUTCOME

Outcome	Rebleed	Percentage (%)	Death	Percentage (%)	Total No. of cases
Non variceal	6	12	1	2	41
Variceal	6	12	3	6	9
Total	12	24	4	8	50

Rebleed was noted in 12 (24%) patients.

Of the 41 cases of non - variceal bleed, 6 patients had rebleed (12%) and of the 9 cases of variceal bleed, 6 patients had rebleeding. Only one patient out of 41 patients of non-variceal bleed died while in the variceal side 3 out of 9 died.

DISCUSSION

One of the major challenges of managing UGIB involves identifying patients who are at high risk of rebleeding and death. Identification of patients who are suitable for early discharge and out patient endoscopy is also important for effective resources use.

We studied, 50 patients admitted with upper gastrointestinal bleeding, in medical and surgical wards in our Government General Hospital.

We assessed the clinical severity by careful history, thorough bed side examination and basic blood investigations and compared it with endoscopic severity.

We assessed the high risk patients by looking for several factors like **age above 60 years, massive hemetemesis, tachycardia**, **shock, melena, haematochezia, severe anaemia at presentation, bright red blood in Ryle's tube aspirate, red colour in rectal examination, presence of altered consciousness and comorbidities like Liver, renal, cardiac disease.**

In our study of 50 patients, 5 patients (10%) were above 60 years of age. 40 patients (80%) were males and 10 patients (20%) were females with male : female ratio of 4:1.

Moderate blood loss was found in 13 patients (26%), severe blood loss in 5 patients (10%).

Tachycardia with a pulse rate of more than 120 / min. was found in 8 (16%) patients.

Postural hypotension was present in 11(22%) patients and resting hypotension, in 6(12%) patients, dizziness in 16 (32%) patients, unconsciousness in 4 (8%) patients. Some of the patients had co-morbidities. Liver failure in 7 (14%) cases, renal failure in 4(8%), coronary artery disease in 3(6%) cases.

Based on the above parameters, we divided the cases into 2 groups, high risk and low risk groups. Thirty six patients came under low risk group and 14 patients came under high risk group based on logistic regression analysis. Patients who were above the 3rd quartile (>75%) with a clinical score above 20 were called high risk group (HRB).

After the clinical risk scoring, we subjected all 50 patients for early endoscopy. Endoscopic results are as follows.

Peptic ulcer was the most common cause, accounting, for 23 (46%) cases. Mallory - weiss lesions were found in 6(12%) cases. Gastric erosions were found in 10 (20%) cases, carcinoma of stomach in 2(4%) cases, which on subsequent histopathological examination proved to be adenocarcinomas.

Esophageal varices were found in 9 (18%) cases. And of them associated features, indicating severity like Red colour signs were present in 5 cases, gastric varices were found in 5 cases. Portal hypertension gastropathy in 6 cases.

In the non - variceal cases, endoscopic severity of ulcers was assessed based on Forrest Classification. Active bleeding with spurting and oozing was present in 2 cases. NBVV in 2 cases, Adherent clot in 15 cases, and clean base or blue pigmented ulcer in 22 cases.

Our study accurately predicted those patients who had high risk lesions for massive bleeding like, spurting, oozing or NBVV in endoscopy belonged to the group with high risk clinical scoring (CS).

High risk clinical scoring correlated accurately with endoscopic high grade lesions.

Chi-square	Value	DF	P<0.001
Pearson	25.25	4	

Several prognostic indicators in upper GI bleeding have been identified. The most important is the cause of bleeding. Variceal hemorrhages have much higher rebleeding and mortality rates than other conditions. Mortality from variceal hemorrhage during the initial hospitalization is at least 30%, with rebleeding rates of 50% to 70%[37]. A reduction in the mortality rates associated with variceal bleeding would lower the overall mortality of upper GI bleeding because varices account for approximately 10% of all bleeding episodes[38].

Stigmata of recent bleeding that can be visualized at endoscopy, such as active arterial spurting, oozing of blood, a visible vessel, or a fresh or old blood clot, are important predictors of outcome in peptic ulcer bleeding. A visible vessel is described endoscopically as an elevated, dark red, blue, or gray mound that protrudes from the ulcer crater and is resistant to washing. The

endoscopic diagnosis of a visible vessel is actually an organizing clot plugging a side hole in the bleeding artery located just below the ulcer base[39]. The evolution of the endoscopic appearance of visible vessels, or sentinel clots, has been described as an initial large, red clot that becomes darker and smaller with time, eventually replaced with a white plug of fibrin and platelets that finally disappears[40]. The dark, small sentinel clot that is not oozing, also described as a bare visible vessel, and older stigmata in the form of a flat, black eschar or white clot, have lower rates of rebleeding[41]. Controversy regarding the evolution of vessel colour and the variable risks associated with the color of the visible vessels continues[42].

Despite the lack of uniform endoscopic descriptions and risks for rebleeding depending on the type of visible vessel, the presence of a visible vessel in an ulcer crater at endoscopy predicts an increased risk for required surgical intervention and increased mortality[43]. The incidence of rebleeding in patients who have ulcers with visible vessels is up to 50%, whereas no rebleeding is observed in patients with no stigmata of recent bleeding[44]. When endoscopy is performed within 6 to 24 hours after admission, visible vessels are found in 20% to 50% of bleeding ulcers.

Other prognostic indicators include the following:

Severity of the initial bleed. Severity is assessed by the transfusion requirement, the presence of bright red blood in the nasogastric aspirate, or the presence of hypotension[45].

Age of the patient. Patients older than 60 years have been shown to have higher mortality rates than their younger counterparts, although this indicator may not be independent from concomitant disease[3].

Concomitant disease. Diseases such as chronic renal failure and severe cardiopulmonary disease affect the ultimate outcome.

Onset of bleeding during hospitalization. Patients who begin to bleed while hospitalized have a mortality rate of 25%, whereas the rate is only 3.7% for patients who start to bleed before admission[46].

In our study, in variceal bleeding, all the patients with high risk clinical scoring had high grade oesophageal varices and high risk clinical scoring could be correlated with endoscopic severity.

Similar study conducted by Rajesh Kashyap et al.,[47] at Indira Gandhi Medical College Shimla, (A clinical profile of Acute Upper Gastrointestinal bleeding, the original article was published in JIACM 2005) concluded that the clinical assessment was significantly correlating with endoscopic findings ($p < 0.001$). Their study revealed that the commonest cause of UGIB was peptic ulcer (63%) followed by variceal bleeding (11%), Erosions (11%) MW syndrome (10.8%), other (6.3%). Ten percent of patients had stigmata of Recent hemorrhage.

Several population based and prospective studies, support peptic ulcer disease being the most common cause of UGIB, approximately in 50% of cases.

However in a recent analysis of Clinical Outcomes Research Initiative (CORI) database found peptic ulcer was responsible for 20.6% of cases of UGIB[48]. The recent declining trend of PUD, in the western world could be attributed to widespread use of Proton Pump Inhibitors and H.Pylori eradication protocol and increasing use of Cox - 2 inhibitors instead of conventional NSAIDS.

In our study, 9 patients (18%) had a history of NSAIDS or aspirin in the previous 48 hours, which could be a precipitating factor. NSAIDS can cause bleeding ulcer and also increases the risk of bleeding from a pre existing ulcer. Study of Indhira Gandhi Medical College, showed that NSAIDS intake history was present in 37% of patients.

In our study 24 (48%) patients were alcoholics and 6 (12%) patients, had consumed alcohol prior to the onset of UGIB, and alcohol could be probably the precipitating factor among them.

In our study, we analysed the outcomes of the patients with UGIB, and found that adverse outcomes like early rebleeding was present in 12(24%) cases. Of the 41 non-variceal cases, 6 had rebleeding and of them peptic ulcer was the diagnosis in 4 cases, carcinoma stomach in 1 case and erosive mucosal lesions in 1 case.

Endoscopic instillation of 1in 10000 adrenaline to cause tamponade was performed and bleeding controlled in 4 patients. One patient with carcinoma of stomach was subsequently transferred to surgery dept. and there he underwent gastrectomy. One patient, admitted with massive UGIB with shock and

syncope, who had active bleeding during endoscopy with spurting of blood, subsequently died. That patient had coronary artery disease with an old Anterior wall myocardial infarction, a significant co-morbidity that increases mortality risk.

In variceal category, the risk of rebleeding was very high. Out of 9 patients, 6 patients had rebleeding. Banding and Endoscopic sclerotherapy were performed to control bleeding.

In both variceal and non-variceal patients, those with high risk clinical score had greater chances of rebleeding, and high risk clinical scoring was accurate in predicting Rebleed.

Chi-square	Value	P<0.001
Pearson	23.9	

4 (8%) patients died, variceal bleeding was the major cause of death, accounting for 3 (6%) cases and bleeding peptic ulcer was responsible for 1 (2%) case.

In variceal bleeding, all the three patients had high risk clinical scoring, and had co-morbidities.

Of the three patients died of variceal bleeding, one patient had coronary artery disease with old Anterior wall myocardial infarction, one patient had renal failure, and one had both CAD and renal failure. Two patients had been treated in ICU.

Among the 4 deaths, 3 of them were more than 60 yrs of age, died due to variceal bleeding and 1 patient was between 40 and 60 yrs of age, died due to peptic ulcer bleeding and co-morbidity (CAD).

In our study, those patients who died with high risk clinical scoring were very much in correlation with adverse outcomes, namely death.

Chi-square	Value	Significant
Pearson	11.18	P<0.001

In a similar study conducted by Rajesh Kashyap, et al, at Indhira Gandhi Medical College, Shimla, adverse outcomes like rebleeding was present in 10% of cases, and death due to UGIB was 6.3% with variceal bleeding being the most common cause of mortality and rebleeding.

Another Indian study conducted by Rajnish Monga et al, GB Pant Hospital, New Delhi concluded that the predictors of rebleed, Morbidity and mortality, were dependent on age, presence of shock, fresh blood in Ryle's tube, Hb%, packed cell transfusions, co-morbid condition, stigmata of recent bleed on endoscopy and the underlying diagnosis. The mortality increased with an increase in number of co-morbid conditions. Apart from endoscopic stigmata of recent ulcer bleeding many independent factors predict the rebleeding risk.

Similar study conducted by Vreeburg EM, et al[49], Department of Gastro enterology, Academic Medical Centre, Amsterdam, concluded that the risk scoring system is a clinically useful scoring system for stratifying patients with

acute UGIB into high and low risk categories for mortality and predicting rebleeding.

Another study conducted by Church et al[50], department of Gastroenterology, Middle Sex Hospital, London concluded that clinical Risk scoring system based on Rockall score, can identify patients at high risk of death, but it is inadequate for the prediction of Rebleeding.

Sanders DS et al[51], department of Histopathology, Royal Hallamshire Hospital, Sheffield, UK, conducted a similar study and concluded that the clinical risk scoring system based on Rockall, was predictive of both rebleeding and mortality in patients with variceal hemorhage and peptic ulcer.

Another study, conducted by Oh VJ et al[52], Department of Medicine, Sungkyunkwan University School of Medicine, Seoul, Korea, concluded that the clinical risk scoring based on Rockall is useful to predict poor outcomes such as rebleeding and death in patients with bleeding peptic ulcer.

CONCLUSION

1. Clinical risk scoring is an inexpensive tool that can identify patients who are at higher risk of morbidity and mortality from UGI bleed.

2. There is an excellent correlation between high risk clinical scoring (HRB) and endoscopic severity.

3. The clinical risk scoring can be used as a prognostic marker to assess Rebleeding, transfusion requirement and early mortality in patients with UGI bleed.

SUMMARY

In our hospital, the Government General Hospital, Chennai, we started this study with the objectives of identifying high risk clinical features in upper Gastrointestinal bleeding and testing the sensitivity of high risk clinical features in predicting endoscopic severity and adverse outcome like Rebleed and Mortality.

We studied 50 consecutive patients admitted with UGI bleed in Medical and Surgical wards prospectively. Clinical assessment was done by taking careful history, bedside examination and basic blood investigation.

We assigned a score for each parameter with due emphasis laid over quantity of bleed, continued bleed, unstable hemodynamics, altered consciousness level and presence of co-morbidities.

After clinical stabilisation, all the patients were subjected to early upper GI endoscopy. And endoscopy revealed those patients with high risk clinical score, had high grade ulcer bleed in the non - variceal category based on Forrest classification and high grade esophageal varices, in the variceal category.

Endoscopy also provided an excellent correlation between high risk clinical scoring and early adverse outcome like Rebleed and Mortality.

We found that clinical risk scoring was a sensitive tool in identifying patients who are at higher risk of morbidity and mortality from UGI bleed and in predicting endoscopic severity.

PROFORMA

Name: Age: Sex : M ☐ F ☐

Hospital No: Occupation Address

Ward: Unit:

DOA: DOD:

MODE OF PRESENTATION

Hemetemesis : Yes ☐ No ☐ Number of Bouts
Amount of Blood (Total)
Fresh ☐ Altered Blood ☐

Melena : Yes ☐ No ☐ Number of Episodes

Bleeding : Mild ☐ Moderate ☐ Severe ☐

Associated Loss of consciousness : Yes ☐ No ☐

Blood transfusion needed : Yes ☐ No ☐

SYMPTOMS PRIOR TO BLEED

Nausea ☐ Vomiting ☐ Regurgitation ☐ Flatulence ☐ Dysphagia ☐

Heart burn ☐

Pain : Yes ☐ No ☐ Site
Character
Radiation
Nocturnal Pain
Periodicity
Rhythmicity

Weight: Lost ☐ Stable ☐ Gained ☐

Appetite : Good ☐ Poor ☐

Bowel Habits : Normal ☐ Diarrhoea ☐ Constipation ☐

Recent Changes in Bowel habits : Yes ☐ No ☐

DRUG HISTORY

Aspirin ☐ NSAIDS ☐ Steroids ☐ Others

Ingestion within 48 Hrs of symptoms - Yes ☐ No ☐

PAST HISTORY

History of HT ☐ IHD ☐ DM ☐ PT ☐ Jaundice ☐ Renal Failure ☐
Pulmonary disease ☐

Any other specific diseases:

History of Umbilical Sepsis : Yes ☐ No ☐

FAMILY HISTORY

Relevant ☐ Nil Relevant ☐

PERSONAL HISTORY

Smoking Habits

Smoker : Yes ☐ No ☐ No.of Cigarettes...../day
No.of Years.....

Alcohol Consumption
Alcoholic : Yes ☐ No ☐ Type of Beverage.....
Amount...../day/week
No.of Years.....

Diet : Veg ☐ Non-Veg ☐

GENERAL EXAMINATION

Mental State: Conscious ☐ Dizziness ☐ Unconscious ☐

General Appearance

Nutrition : Well nourished ☐ Moderately nourished ☐ Malnourished ☐

Dehydration : Yes ☐ No ☐ Febrile / Afebrile

Anaemia ☐ Icterus ☐ Cyanosis ☐ Clubbing ☐

Gen.Lymphadenopathy ☐ Pedal edema ☐

Periphery : Normal ☐ Cold & Clammy ☐

K-F ring Yes ☐ No ☐

CARDIOVASCULAR SYSTEM

Blood Pressure — Supine: mm/Hg
Standing mm/Hg
Sitting: mm/Hg

Pulse: /min

JVP: Normal ☐ Elevated ☐

Auscultation: Heart Sounds:

Added Sounds:

RESPIRATORY SYSTEM

Respiratory Rate: /min

Auscultation: Bilateral Air Entry: Good ☐ Decreased ☐
Added sounds:

ABDOMEN

Tenderness : Yes ☐ No ☐ Site:

Visible Veins : Yes ☐ No ☐

Ascites : Yes ☐ No ☐

Splenomegaly : Yes ☐ No ☐ Rectal examination

Hepatomegaly : Yes ☐ No ☐ Ryle's tube Aspirate

Mass : Yes ☐ No ☐

CENTRAL NERVOUS SYSTEM

Conscious / Oriented: Yes ☐ No ☐

Metabolic flap: Yes ☐ No ☐

Any focal neurological deficit: Yes ☐ No ☐

CLINICAL DIAGNOSIS

CLINICAL PREDICTION: On the basis of the evidence obtained, I predict that the Patient will Settle ☐ Continue bleeding ☐ Rebleed ☐

SUMMARY OF INVESTIGATIONS AND PROGRESS

Day	**Hb%**	**PCV**	**No.of units of Blood transfused**	**Clinical status**

INVESTIGATIONS

ROUTINE

Complete Hemogram:	Hb%	Urine:
	TC	Albumin
	DC	Sugar
	ESR	Deposits
	Platelet count	
Blood:	Urea	Urea / Creatinine=
	Sugar	
Serum:	Creatinine	Electrolytes

SPECIFIC

Blood Group / Rh typing	BT:
PCV:	CT:
LFT	ECG:
	CXR:

Stool : Occult Blood - Present ☐ Absent ☐
USG : Abdomen

ENDOSCOPY

Ulcer: Gastric ☐ Duodenal ☐ Esophagus

Site: Varices:

Size: Grade:

Number: Columns:

Clot ☐ Visible Vessel ☐ Oozing ☐ Extent:

Contents : Fresh ☐ Altered Blood ☐

Any Growth: Treatment:

Biopsy report: Follow up:

BIBLIOGRAPHY

1. Rockall TA, Logan RF, Derlin HB, et al. Incidence and Mortality from UGIB in UK. BMJ. 1995; 331 : 222 - 6.

2. Blatch ford O, Davidson LA et al, Acute UGIB, in West of scotland. Case ascertainment study. BMJ 1997; 315 : 510 - 4.

3. Yavorski, RT, et al. Analysis of UGIB in military medical facilities, Am.J. Gastroenterol, 1995; 90 : 568.

4. Fleischer D, Etiology and prevalence of severe persistent UGI Bleeding. Gastroenterology 1983; 84 : 538.

5. Shift L. et al, observations on the oral administration of citrate blood in man. Am.J.Med. Sci. 1942; 203 : 409.

6. Ebert RA, Stead EA, et al, Response of normal subjects to Acute blood loss. Arch.Intern.Med. 1940; 68 : 578.

7. Shepard HA, Harry et al. Recurrent retching with gastric mucosal prolapse. Dig. Dis.Sci; 1984; 29 : 121.

8. Knaner CM. Mallory weiss syndrome. Gastroenterology 1976; 71 : 5.

9. Kelly JP, Kaufman DW, Koft RS et al. Alcohol consumption and UGIB, Am.J. Gastroenterol. 1995, 90-1058.

10. Champion MC, Sullivan SN, Coles JC, et al. Aortoenteric fistula. Ann. Surg. 1982; 195 : 314.

11. Steffes BC, Leary JP. Primary Aorto duodenal fistulas. Ann.Surg.1980;46 : 121.

12. Naveau S.Aubert et al. Vascular malformations. Dig. Dis.Sci. 1990; 35 : 821.

13. Angiodysplasia of upper Gastrointestinal tract. J.Clin. Gastroenterol. 2000; 95 : 415.

14. Juler GL et al. The pathogenesis of Dieulafoy's gastric erosion. American.J.Gastroenterol, 1984; 79 : 195.

15. El-omar et al : Helicobacter pylori infection and abnormalities of Acid scretion in patients with duodenal ulcer disease. Gastroenterology. 109 : 681 - 691. 1995.

16. Graham Dy, Smith JL Aspirin and Stomach Ann. Internal Medicine 1986; 104 : 390.

17. Snady, Feinman, Prediction of variceal hemorrhage; Am.J. Gastroenterol 1988; 83 : 519.

18. Grace. H.ELTA; Yamada, Textbook, Gastroenterology, Chapter 33, 705.

19. Barange et al. Hepatology, 1999; 30 : 1139.

20. Don.C.Rockey. Gastroenterol. Clin. N.Am. 34, 2005, 581 - 588.

21. Gilbert DA. Epidemiology of upper gastrointestinal bleeding Gastrointest. Endosc. 1990; 36 : 58.

22. Cellular RE, Garaler et al Gastrointestinal Tract bleeding value of nasogastric Aspirate. Arch. Intern. Med. 1990; 150-1381.

23. Peterson WL, Barnett CC, Smoth HJ, et al. Routine early endoscopy in upper gastrointestinal bleeding ; a randomized, controlled trial. N.Engl.J. Med 1981; 304 : 925.

24. Luk GD et al. Gastric Aspiration in localization of gastro intestinal hemorrhage. JAMA. 1979; 241 : 576.

25. Dronfield, et al. Br.Med.J. 1982; 284; 545.

26. Leinicke, et al. Gastrointest Endosc. 1976;22:228.

27. Hashizume, et al. Hepatology. 1992 ; 15 : 69.

28. Steigmann, et al. N.Engl.J.Med. 1992; 316 : 1527.

29. Piper, et al. Scand.J.Gastroenterol. 19 : 1015 - 1021.

30. Hawkey, et al. N.Eng.J.Med. 338;727 - 734. 1998.

31. Hou, et al Hepatology 1995;21 : 1517.

32. Fort, et al. Hepatology, 1990; 11 : 678.

33. Panes, et al. Dig.Dis.Sci. 1988; 33 - 454.

34. Galambos, et al. N.Eng.J.Med. 1976; 295 : 1098.

35. Ring, et al. Ann.Int.Med. 1992; 116 : 304.

36. Conn, et al, Hepatology, 199; 13 : 902.

37. Graham, et al Gastroenterology 1981; 80 : 800.

38. Silverstein, et al. Gastrointest Endosc. 1981; 27; 73.

39. Swain et al. Gastroenterology 1986; 90 : 595.

40. Johnson et al. Gastrointest. Endosc. 1990; 346 : 516.

41. Ware et al. Gastroenterology. 1985;88 : 1209.

42. Lin et al. Gut 1994; 35 : 1389.

43. Griffith et al. N.Engl.J.Med. 1979;300 : 1411

44. Storey et al N. Eng. J.Med. 1981; 2305 : 915.

45. Corley et al. Ann.J.Gastroenterol 1998; 93 : 336.

46. Longstreth, et al. Am.J.Gastroenterol. 1995; 90 : 206.

47. Rajesh Kashyap, et al. JIACM 2005; 6(3) : 224 - 8.

48. Boon pongmanee, et al. Gastro. Endosc. 2004; 59 : 788 - 94.

49. Vreeburg, et al. Gut 1999 Mar; 44(3) : 331-5.

50. Church, et al. Gastro, Endosc. 2006 apr; 63(4) : 606 - 12.

51. Sanders, et al. Am.J.Gastroenterol. 2002 March; 97(3) : 630 - 5.

52. Oh VJ, et al. Korean.J.Gastroenterol. 2004 Aug; 44(2) : 66 - 70.

MASTER CHART

Sl. No.	Age & Sex	Past History		H/ Emesis	Melena	H.chezia	Hb% gm%	RTA	Rec.E	Pulse per.mt	BP mmHg	Cons	Liver Failure Sign.	HE	Co-mor	NSAIDS	Alco	Smoking	Trans.REQ
		UGIB	PUD																
1.	39/M	-	+	1	+	-	11	2	3	88	3	3	-	-	-	-	+	+	-
2.	16/M	+	-	3	+	-	4	1	2	122	2	2	-	-	-	-	-	-	4
3.	54/M	-	+	2	+	-	8	2	3	128	1	2	-	-	-	+	-	+	2
4.	25/M	-	-	1	-	-	13	3	3	86	3	3	-	-	-	-	+	+	-
5.	33/F	-	-	1	+	-	10	2	-	90	3	3	-	-	-	+	-	-	-
6.	62/M	+	-	3	+	+	4	1	1	7	2	1	+	+	ARF, CAD	-	+	-	4
7.	38/M	-	-	1	+	-	10	2	3	80	3	3	-	-	-	-	-	-	-
8.	46/M	-	-	1	-	-	12	3	3	84	3	3	-	-	-	+	+	+	-
9.	52/M	+	-	2	+	-	8	1	2	126	1	2	+	-	-	-	+	-	2
10.	36/F	-	-	1	+	-	9	2	3	90	3	3	-	-	-	+	-	-	-
11.	44/F	-	-	1	+	-	12	2	3	86	3	3	-	-	-	-	-	-	-
12.	28/M	-	-	1	+	-	12	2	3	84	2	2	-	-	-	-	+	+	-
13.	57/M	-	-	1	+	-	8	2	3	86	3	2	-	-	-	-	-	-	1
14.	26/F	-	-	1	-	-	12	3	3	80	3	3	-	-	-	-	-	-	-
15.	75/M	-	-	2	+	-	10	1	3	106	3	2	-	-	-	-	+	+	1
16.	44/F	-	-	1	+	-	11	2	3	88	3	3	-	-	-	+	-	-	-
17.	38/M	+	+	2	+	+	8	1	2	124	2	2	-	-	-	-	+	+	4
18.	48/M	-	-	1	+	-	11	2	3	90	3	3	-	-	-	-	+	+	-

Sl. No.	Age & Sex	Past History		H/ Emesis	Melena	H.chezia	Hb% gm%	RTA	Rec.E	Pulse per.mt	BP mmHg	Cons	Liver Failure Sign.	HE	Co-mor	NSAIDS	Alco	Smoking	Trans.REQ
		UGIB	PUD																
19.	42/M	-	-	2	+	-	8	2	2	112	2	2	-	-	-	-	-	+	2
20.	46/M	+	-	3	+	+	4	1	1	126	1	1	-	-	CAD	-	+	+	5
21.	36/F	-	-	1	+	-	11	3	3	90	3	3	-	-	-	-	-	-	-
22.	71/M	+	-	3	+	+	3	1	1	132	2	1	+	+	ARF	-	+	-	4
23.	74/M	-	-	1	+	-	7	2	3	88	1	3	-	-	-	-	-	-	2
24.	30/M	-	-	1	+	-	11	3	3	90	3	3	-	-	-	+	-	-	-
25.	36/M	-	-	1	+	-	12	2	3	86	1	3	-	-	-	-	+	+	-
26.	44/M	-	-	1	+	-	14	2	3	92	3	3	-	-	-	-	+	-	-
27.	33/F	-	-	1	+	-	12	2	3	90	3	3	-	-	-	-	-	-	-
28.	48/M	-	-	2	+	-	8	1	2	106	3	2	+	-	-	-	+	+	2
29.	29/M	-	+	2	+	-	10	2	3	108	3	3	-	-	-	-	-	+	2
30.	40/M	-	+	1	+	-	14	3	3	86	3	3	-	-	-	-	+	+	-
31.	52/M	-	-	1	+	-	9	2	3	90	2	3	-	-	-	-	+	-	1
32.	46/M	-	-	1	+	-	10	2	3	86	3	3	+	-	-	-	+	+	1
33.	48/M	+	-	2	+	-	8	1	2	106	3	2	-	-	-	-	+	-	2
34.	36/M	-	-	1	+	-	12	2	3	80	3	3	-	-	-	-	+	-	-
35.	38/F	-	+	1	+	-	12	2	3	86	3	3	-	-	-	-	-	-	-
36.	42/M	+	+	2	+	-	7	1	2	112	2	2	-	-	-	+	-	+	3
37.	42/M	-	-	1	+	-	12	2	3	88	3	3	-	-	-	-	+	+	-

Sl. No.	Age & Sex	Past History		H/ Emesis	Melena	H.chezia	Hb% gm%	RTA	Rec.E	Pulse per.mt	BP mmHg	Cons	Liver Failure Sign.	HE	Co-mor	NSAIDS	Alco	Smoking	Trans.REQ
		UGIB	PUD																
38.	28/M	-	-	1	+	-	13	3	-	80	3	3	-	-	-	+	-	+	-
39.	52/F	-	+	2	+	-	10	1	2	126	2	2	-	-	-	-	-	-	2
40.	38/M	-	=	2	+	-	6	1	2	116	2	2	-	-	CKD	-	-	-	1
41.	40/M	-	=	1	-	-	12	3	-	80	3	3	-	-	-	-	-	+	-
42.	58/M	+	=	2	+	-	6	1	2	106	2	2	-	-	-	-	-	-	4
43.	46/M	-	+	1	+	-	9	2	3	90	3	3	-	-	-	-	-	+	-
44.	34/M	-	+	1	+	-	12	2	3	86	3	3	-	-	-	-	+	+	-
45.	56/M	+	-	2	+	-	6	1	2	116	2	2	+	-	-	-	+	-	4
46.	28/F	-	-	1	+	-	10	2	3	80	3	3	-	-	-	+	-	-	-
47.	38/M	-	+	1	+	-	12	2	3	82	3	3	-	-	-	-	+	+	-
48.	40/M	-	-	1	+	-	4	2	3	78	3	3	-	-	-	-	+	+	-
49.	70/M	-	-	3	+	+	4	1	1	136	1	1	-	+	CAD, ARF	-	+	-	4
50.	44/F	-	-	1	+	-	10	2	-	92	3	3	-	-	-	-	-	-	-

Haemetemesis

1. - 500ml
2. - 500-1000ml
3. - > 1000ml

RTA (Ryle's tube Aspirate

1. - Bright Red
2. - Coffee brown
3. - Clear

Rectal Examination

1. - Bloody
2. - Maroon
3. - Melena
4. - Normal

BP

1. - Resting hypotension
2. - Postural hypotension
3. - Normal

CAD : Coronary artery disease
ARF : Acute renal failure
CKD : Chronic kidney disease
COMOR : Comorbidities
ALCO : Alcohol

Consciousness

HE - Hepatic Encephalopathy

1. - Unconsciousness 2. - Dizziness
3. - Normal

Sl. No.	NON VARICEAL						VARICEAL					Rebleed	Death	Remarks
	Gastric Ulcer	Duodenal Ulcer	Forrest	Mallory Weiss	Erosions	Others	Grade	Colour	Gastric Varices	PHT. Gastropathy	Red colour signs			
1.	-	+	3	-	-	-	-	-	-	-	-	-	-	-
2.	-	-	-	-	-	-	III	White	-	-	-	-	-	Banding done
3.	-	+	3	-	-	-	-	-	-	-	-	-	-	-
4.	-	-	3	+	-	-	-	-	-	-	-	-	-	-
5.	-	-	4	-	+	-	-	-	-	-	-	-	-	-
6.	-	-	-	-	-	-	IV	Blue	+	+	+	+	+	Endoscopic sclerotherapy PD done
7.	-	+	4	-	-	-	-	-	-	-	-	-	-	-
8.	-	-	4	-	+	-	-	-	-	-	-	-	-	-
9.	-	-	-	-	-	-	III	Blue	+	+	+	+	-	Endoscopic ligation done
10.	+	-	4	-	+	-	-	-	-	-	-	-	-	-
11.	-	+	3	-	-	-	-	-	-	-	-	-	-	-
12.	-	-	4	+	+	-	-	-	-	-	-	-	-	-
13.	-	-	4	-	-	Ca Stomach	-	-	-	-	-	+	-	Transferred to surgery dept
14.	-	-	4	-	+	-	-	-	-	-	-	-	-	-
15.	+	-	3	-	-	-	-	-	-	-	-	-	-	-

Sl. No.	NON VARICEAL						VARICEAL					Rebleed	Death	Remarks
	Gastric Ulcer	Duodenal Ulcer	Forrest	Mallory Weiss	Erosions	Others	Grade	Colour	Gastric Varices	PHT. Gastropathy	Red colour signs			
16.	-	-	4	-	+	-	-	-	-	-	-	-	-	-
17.	-	+	4	-	-	-	-	-	-	-	-	-	-	-
18.	-	+	4	-	-	-	-	-	-	-	-	-	-	-
19.	+	+	3	-	-	-	-	-	-	-	-	-	-	-
20.	-	-	1	-	-	-	-	-	-	-	-	+	+	Endoscopic injection done
21.	-	+	4	-	-	-	-	-	-	-	-	-	-	-
22.	-	-	-	-	-	-	IV	Blue	+	+	+	+	+	Peritoneal dialysis done
23.	-	-	3	-	-	Ca Stomach	-	-	-	-	-	-	-	Transferred to surgery
24.	-	-	4	-	+	-	-	-	-	-	-	-	-	-
25.	-	+	4	-	-	-	-	-	-	-	-	-	-	-
26.	-	-	4	+	-	-	-	-	-	-	-	-	-	-
27.	-	-	4	-	-	-	-	-	-	-	-	-	-	-
28.	-	-	-	-	-	-	III	White	-	-	-	-	-	Banding done
29.	-	+	4	-	-	-	III	Blue	+	+	+	+	-	Variceal ligation done
30.	-	+	4	-	-	-	-	-	-	-	-	-	-	-

Sl. No.	NON VARICEAL						VARICEAL					Rebleed	Death	Remarks
	Gastric Ulcer	Duodenal Ulcer	Forrest	Mallory Weiss	Erosions	Others	Grade	Colour	Gastric Varices	PHT. Gastropathy	Red colour signs			
31.	-	-	-	-	-	-	II	White	-	-	-	-	-	-
32.	-	+	4	-	-	-	-	-	-	-	-	-	-	-
33.	-	-	-	-	-	-	III	White	+	+	+	+	-	Endoscopic sclero therapy
34.	-	-	3	+	-	-	-	-	-	-	-	-	-	-
35.	-	+	3	-	-	-	-	-	-	-	-	-	-	-
36.	+	+	2	-	-	-	-	-	-	-	-	+	-	Endoscopic injection
37.	-	-	4	+	-	-	-	-	-	-	-	-	-	-
38.	-	-	4	-	+	-	-	-	-	-	-	-	-	-
39.	+	+	2	-	-	-	-	-	-	-	-	+	-	Endoscopic injection done
40.	+	-	3	-	-	-	-	-	-	-	-	-	-	-
41.	-	-	4	-	-	Eso erosion	-	-	-	-	-	-	-	-
42.	+	-	3	-	-	-	-	-	-	-	-	+	-	Endoscopic injection done
43.	-	+	4	-	-	-	-	-	-	-	-	-	-	-
44.	+	-	3	-	-	-	-	-	-	-	-	-	-	-
45.	-	-	1	-	-	-	-	-	-	-	-	+	-	Endoscopic injection done

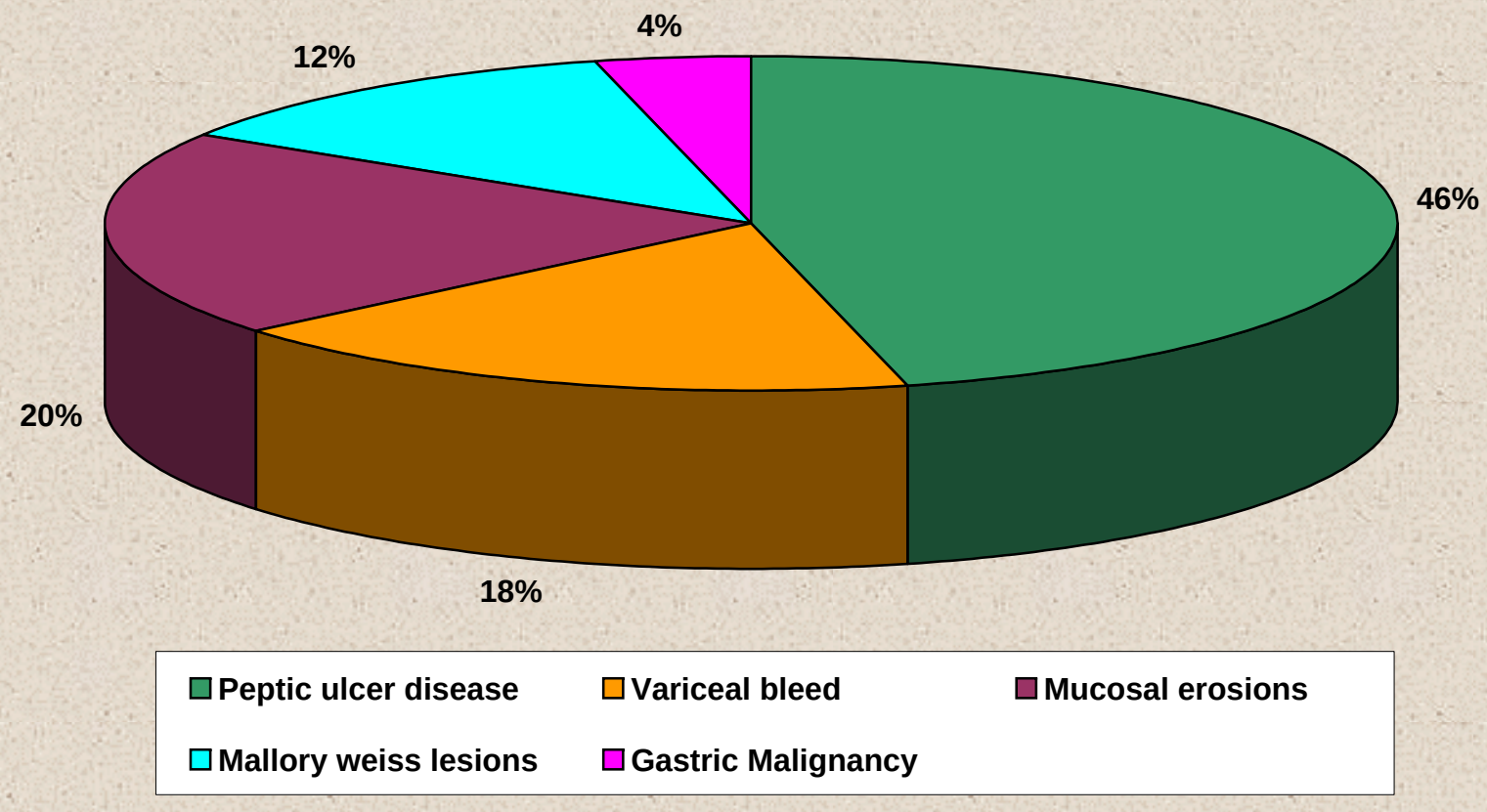
Sl. No.	NON VARICEAL						VARICEAL					Rebleed	Death	Remarks
	Gastric Ulcer	Duodenal Ulcer	Forrest	Mallory Weiss	Erosions	Others	Grade	Colour	Gastric Varices	PHT. Gastropathy	Red colour signs			
46.	-	-	3	-	+	-	-	-	-	-	-	-	-	-
47.	-	+	4	-	-	-	-	-	-	-	-	-	-	-
48.	-	-	-	+	-	-	-	-	-	-	-	-	-	Endoscopic sclerotherapy & PD done
49.	+	-	-	-	-	-	III	Blue	+	+	+	+	+	-
50.	-	+	-	-	-	-	-	-	-	-	-	-	-	-

FORREST

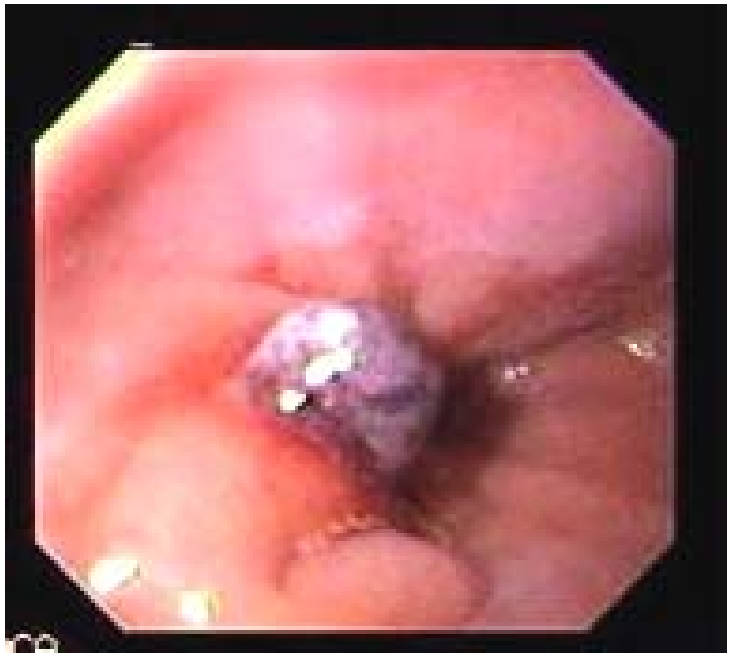
I - Active Bleeding
II - Non Bleeding Visible Vessel
III - Adherent clot
IV - Clean base (or pigment ulcer)

PD - Peritoneal Dialysis
ESO - Esophageal

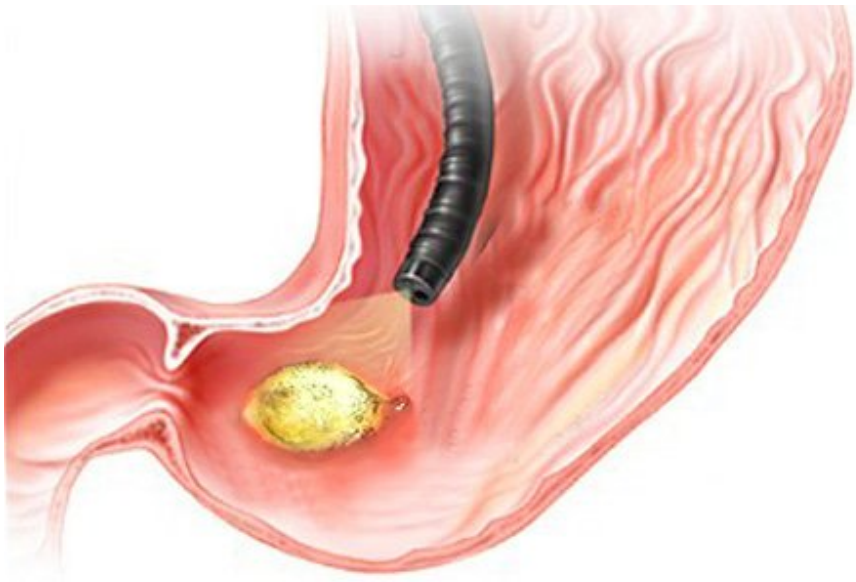
Fig. 3. INCIDENCE OF VARIOUS CAUSES OF UGI BLEED IN OUR STUDY



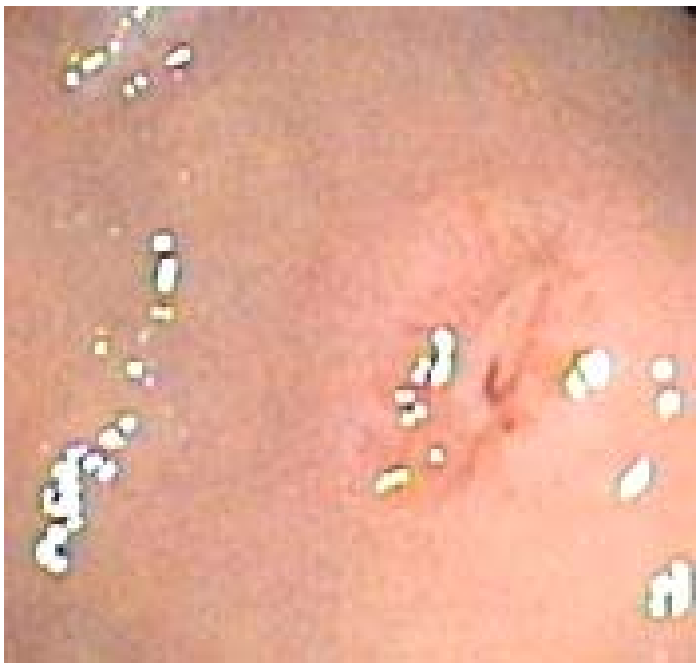
MALLORY - WEISS LESION



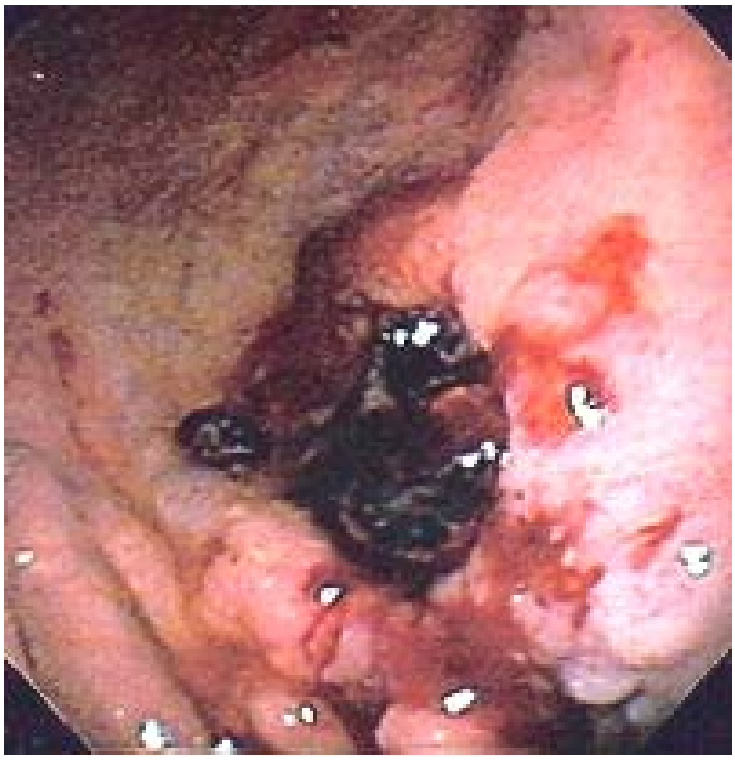
ENDOSCOPE VISUALISING GASTRIC ULCER



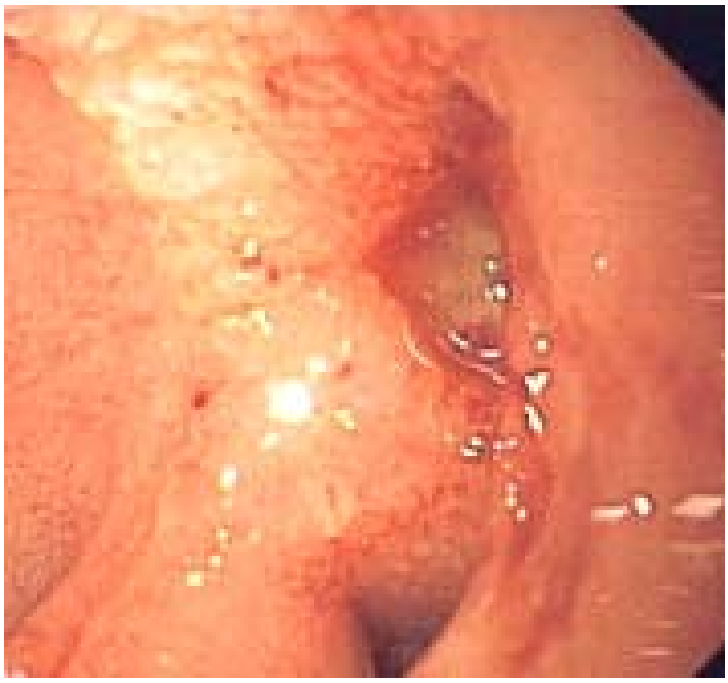
GASTRIC ULCER WITH VISIBLE VESSEL



GASTRIC ULCER WITH ADHERENT CLOT



DUODENAL ULCER IN POSTERIOR WALL



OESOPHAGEAL VARICEAL BLEEDING