Non-Variceal Upper Gastrointestinal Haemorrhage

- Clinical Profile and Application of Rockall Prognostic Score

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CERTIFICATE

This is to certify that the dissertation titled **"Non-variceal Upper Gastrointestinal Haemorrhage - Clinical Profile and Application of Rockall Prognostic Score"** is the bonafide original work of **Dr. Raja Yogesh K.**, in partial fulfillment of the requirements for M.D. Branch– I (General Medicine) Examination of the Tamilnadu Dr. M.G.R Medical University to be held in MARCH 2009. The Period of study was from January 2008 to September 2008.

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DECLARATION

I hereby solemnly declare that the dissertation titled **"Non –variceal Upper Gastrointestinal Hemorrhage – Clinical Profile and Application of Rockall Prognostic Score"** was done by me at Madras Medical College and Government General Hospital, Chennai-3, during January 2008 – September 2008, under the guidance and supervision of my unit Chief Prof. M. Jubilee, M.D.

The dissertation is submitted to the Tamilnadu Dr. M.G.R. Medical University towards the partial fulfillment of requirements for the award of M.D degree (Branch-1) in General Medicine.

Place: **SIGNATURE OF THE CANDIDATE**

Date :

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INTRODUCTION

Upper Gastrointestinal haemorrhage is one of the common emergencies met in clinical practice. The mode of presentation is diverse and depends on the cause and the amount of blood loss. It can present with a spectrum of clinical severity that ranges from trivial and insignificant bleeds to fulminant and lethal exsanguinations. It is considered a potentially fatal emergency with a mortality rate as high as 14%[1]. It accounts for up to 20,000 deaths annually in the United States. The overall incidence of acute gastrointestinal haemorrhage has been estimated to be 50 to 100 per 100,000 persons per year, with an annual hospitalisation of approximately 100 per 100,000 hospital admissions[1].

The incidence of UGIH has been stable since the mid-1980s. On one hand, the introductions of proton pump inhibitors and efforts to eradicate H.pylori infection have reduced the incidence of ulcer bleeding. On the other hand, with the increasing average age expectancy of the population and with it the increasing age-related co-morbid conditions may predispose patients to UGIH. The increasing use and abuse of NSAIDs might perhaps tend to shift the balance towards an

increased incidence. The risk of UGIH appears to be increased in certain group of patients, particularly those with underlying cardiovascular disease, chronic renal failure and perhaps patients older than 65 years of age.

Historically, the most common cause of UGIH has been gastroduodenal ulcer disease, although other upper gastrointestinal mucosal lesions account for a substantial proportion of cases. Whatever the lesion be, the initial step in the management of a patient with UGIH is to assess the severity of bleeding. The patient's hemodynamic status is the initial focal point and the basis for assessing the patient's overall clinical condition. Unstable vital signs indicate that the patient is bleeding from a major vascular source and indicate a poorer prognosis.

Resuscitation is of paramount importance in a bleeding patient and should be proportional to the severity of the bleed. In an actively bleeding patient, crystalloids or colloids need to be infused as rapidly as the patient's cardiovascular system will allow. Virtually all patients with unstable vitals should be transfused.

As the patient is being resuscitated and stabilised, the patient's history and physical examination might be analysed to ascertain the cause and site of the bleed. History of duration of the patients symptoms need to be carefully analysed. The historical features important to

determine include the presence of abdominal pain (PUD), history of retching (MW tear), or a change in bowel habits, or weight loss – pointing towards a GI malignancy. History of alcohol intake, smoking habits and history of NSAID ingestion should also be enquired into. Previous history of UGIH and abdominal surgeries may throw light on the problem at hand. History and physical examination to detect the presence of existing co-morbid conditions should be carried out since they are adverse prognostic indicators in patients with UGIH.

The primary diagnostic modality for evaluation of patients with UGIH is oesophagogastroduodenoscopy. The endoscopic appearance of the lesions and the presence of stigmata of bleed have an important role in predicting the course and outcome of the patients.

The major goal of treatment is to stop the bleeding and prevent rebleeding. The major forms of therapy include 1) pharmacological, 2) endoscopic therapy, 3) angiographic and 4) surgical. The use of each of these modalities depends on the cause of the cause of bleeding. For patients with significant bleeding, the mainstay of treatment of bleeding lesions is endoscopic therapy. Indeed, it is the major justification for oesophagogastroduodenoscopy in those with hemodynamically significant acute upper gastrointestinal bleeding, since endoscopic therapy unquestionably improves prognosis[3] . For this reason, such

patients should undergo the procedure as early as possible. In addition, the endoscopic appearance of certain lesions may help triage patients, and thereby reduce costs of hospitalization. A proposed approach to patients with UGIH is as follows...

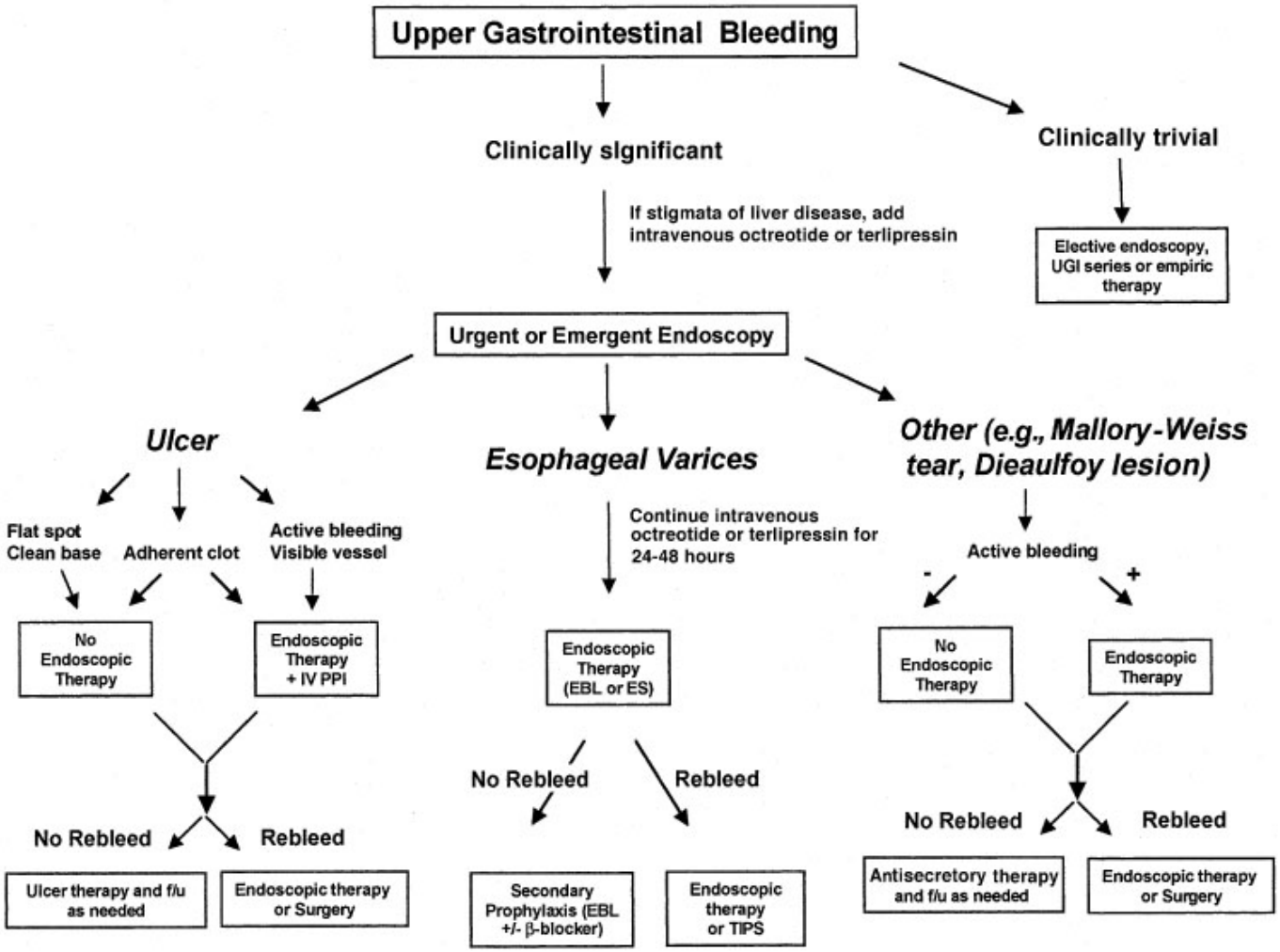


Chart 1: Approach to patients with UGIH

The prognosis of patients with UGIH is as varied as the presentation. A number of prognostic scoring systems have been developed to predict the outcome and course of the patients. One such prognostic scoring system is ***The Rockall Scoring System***.

Government General Hospital, Chennai, a tertiary referral centre, receives approximately 800-1000 patients/year with UGIH. In these patients, hospital admission is regarded as compulsory since there is a chance of these patients having continued bleeding or rebleeding. A very large proportion of these patients are actually at a very low risk for rebleeding and mortality. Identifying these patients and stratifying them according to their risks would help in their appropriate management. In those with a low risk of an adverse outcome, an early discharge can be planned and in those with a high risk, a more intensive monitoring can be provided. It is aimed that such stratification would lead to a reduction of hospital expenses in the former group (by means of an early discharge) and more intensive management in the latter group. It is believed that this would result in substantial resource savings both for the patient (in-hospital expenses), as well as the State, without compromising on the standard of health care. In this study, ***The Rockall Scoring System*** is used for stratifying patients with NVUGIH and the usefulness of the same in predicting the outcome is analysed.

AIMS OF THE STUDY

- To study the Clinical presentation, etiologies, and outcome of patients admitted with Upper Gastrointestinal bleeding of a Non-Variceal cause.

- To correlate the ***Rockall Score*** of these patients with
    1. The event of Re-bleed,
    2. Transfusion requirements,
    3. Duration of hospitalisation and
    4. Their final Outcome.

REVIEW OF LITERATURE

DEFINITION

Upper gastrointestinal haemorrhage is defined as any bleeding from a site in the gastrointestinal tract proximal to the ligament of Treitz (fore-gut).

PRESENTATION

Patients with Upper gastrointestinal haemorrhage present with a wide range of clinical severity ranging from trivial bleeds to fulminant and lethal exsanguinations. They can present with one or more of the following symptoms...

Haematemesis : It is defined as the vomiting of blood which can be fresh and bright red in colour or it may be old and take the form of coffee grounds. It indicates an upper gastrointestinal source (defined as the gastrointestinal segment proximal to the ligament of Treitz). Spurious haematemesis can occur due to vomiting of swallowed blood from a source in the respiratory tract.

Melaena: Melaena is defined as the passage of black, tarry, and foul smelling stools. The black tarry nature is due to the degradation of the blood to haematin and other haemochromes by the colonic bacteria.
A minimum of at least 50ml of blood is required to produce melaena

though volumes up to 100 ml may be clinically silent. For melaena to develop, blood has to be present for at least 14 hours within the GI tract. Thus, the more proximal the bleed, the more likely melaena would occur. Melaena, unlike haematemesis, is not specific for UGIH since melaena can also occur due to bleeding from the small bowel as well as slow bleeds from the ascending colon. Melaena should be differentiated from dark stools secondary to ingested iron or bismuth. These stools though dark are not tarry or offensive.

Haematochezia: It refers to the passage of bright red blood from the rectum that may or may not be mixed with stools. Although haematochezia is predominantly a symptom of lower gastrointestinal bleeding, it can occur in up to 10% of patients with UGIH, especially when the haemorrhage is brisk and haemodynamically significant.

Occult bleeding is defined as bleeding from the GI tract that is not apparent to the patient. It usually results from small amounts and haemodynamically insignificant bleed. Bleeding of obscure origin can be occult or obvious (e.g., manifest by haematemesis, melaena, or haematochezia), but from a source that is difficult to pinpoint on routine examination. These two forms of presentation are not considered in the study for reasons that are obvious.

Non-variceal bleeding forms the majority (90%) of the cases with UGIH[3,4]. The causes of NVUGIH are varied, and are listed below...

CAUSES OF NVUGIH

COMMON	LESS COMMON	RARE
Gastric ulcer Duodenal ulcer Mallory-Weiss tear	Gastric erosions/gastropathy Oesophagitis Cameron lesions Dieulafoy lesion Telangiectasias Portal hypertensive gastropathy Gastric antral vascular ectasias Gastric varices Neoplasms	Oesophageal ulcer Erosive duodenitis Aortoenteric fistula Haemobilia Pancreatic disease Crohn's disease

OESOPHAGITIS

Significant bleeding from oesophagitis accounts for up to 8% of patients with UGIH. Bleeding from oesophagitis more commonly causes occult blood loss than acute bleeding. Clinically obvious bleeding is most likely in those with extensive ulcerative disease or with an underlying coagulopathy. Specific therapy is directed at the cause of the underlying lesion (usually reflux oesophagitis), and typically involves high-dose proton pump inhibitors. Endoscopic treatment of bleeding lesions may benefit patients with oesophageal ulcerations and visible vessels, but because of the risk for perforation, it should be performed with caution.

MALLORY-WEISS TEAR

Mallory-Weiss tears are lacerations in the region of the gastroesophageal junction that typically occur in gastric mucosa, although 10% to 20% can occur in oesophageal mucosa. They are an important cause and account for approximately 5% to 10% of cases of upper UGIH.[1,3,5,6] Although they are thought to be caused by retching, a history of this is obtained in only 29% of patients.[7] Bleeding from Mallory-Weiss tears stops spontaneously in 80% to 90% of patients, and less than 5% of patients rebleed. Patients not bleeding during endoscopy and without other medical problems that require hospitalization are usually managed with supportive care only and can be discharged promptly. Endoscopic therapy with coagulation methods, injection, or banding effectively stops bleeding and should be performed on bleeding lesions or patients with bleeding stigmata. Angiographic therapy, with intra-arterial infusion of vasopressin, or embolization is successful in a high proportion of patients. Surgical therapy is rarely required.

DUODENAL AND GASTRIC ULCERS

Ulcer disease is the most common cause of acute UGIH. In a study done by Skok P et al., it was shown that gastroduodenal ulcers were responsible for UGIH in nearly 50% of cases.[8] The incidence of bleeding from duodenal ulcers is approximately twofold that of gastric

ulcers. The hospitalization rate for ulcer-related gastrointestinal bleeding appears to be constant at approximately 40 to 60 cases per 100,000 patients.[1,4,9]

Ulcers bleed when they erode into the lateral wall of a vessel. Ulcers located high on the lesser curve of the stomach or on the posteroinferior wall of the duodenal bulb are most likely to bleed (and rebleed), presumably due to the rich vascular supply in these areas. The precise pathophysiology of ulcer bleeding is unclear, but is likely to encompass factors related to the bleeding blood vessel itself, as well as factors related to the ulcer environment.

PREDISPOSING FACTORS FOR ULCER BLEEDING

A number of risk factors predispose to ulcer disease and its bleeding, the most prominent being acid, Helicobacter pylori, and nonsteroidal anti-inflammatory drugs (NSAIDs). In addition, underlying medical and clinical factors predispose to ulcer disease and bleeding. In a case control study of 1122 patients and 2231 controls done by Lanas et al[10], cardiovascular and cerebrovascular disease were independent predictors of peptic ulcer–related UGIH. Chronic pulmonary disease and cirrhosis also are associated with peptic ulcer disease. Pharmacologic agents besides aspirin and NSAIDs may predispose to ulcer disease. Glucocorticoids historically have been associated with an increased risk

of peptic ulcer, although newer data raise doubt about this association. Recent data link alendronate to the development of gastric ulcers and perhaps upper gastrointestinal bleeding. Also, ethanol may potentiate the damaging effects of NSAIDs in the mucosa, and, as expected, anticoagulants will facilitate bleeding.

Hospitalization appears to be an important risk factor for development of ulcer bleeding (duodenal greater than gastric). Bleeding tends to occur after prolonged hospitalization and is most common in patients with severe co-morbidities. Such "nosocomial" gastrointestinal bleeding is associated with poor outcome. A study done by Terdiman JP et al showed that such patients had a mortality rate of 34%.[11] It also showed that nosocomial ulcer bleeders were less likely to have a history of previous ulcer disease, to have H. pylori infection, or to be taking NSAIDs than those hospitalized for ulcer bleeding.

Gastric Acid

The evidence for a role of gastric acid in peptic ulceration is overwhelming and includes the hypersecretory disorder Zollinger-Ellison syndrome, in which patients develop ulcers with high frequency. The ability of antacid therapy alone to heal upper gastroduodenal tract ulceration also supports the role of acid. However, controversy exists surrounding the role of acid in inducing bleeding in non-

Zollinger-Ellison ulceration. Perhaps the best evidence of a role for acid in acute upper gastrointestinal haemorrhages comes from data indicating that acid reduction by proton pump inhibitors in patients with active or recent bleeding from upper gastrointestinal ulcerative lesions reduces the risk of bleeding and rebleeding[12-16].

Helicobacter pylori

As with acid, the link between H. pylori and peptic ulceration is firm. However, the role of H. pylori in ulcer bleeding is controversial.

Aspirin and Nonsteroidal Anti-Inflammatory Drugs (NSAIDS)

Aspirin and NSAIDs are probably the most important predisposing factors for ulcer bleeding. The mechanism of injury and ulceration is complex but appears to involve reduced production of cyclooxygenase-generated cytoprotective prostaglandins. Further, the risk of bleeding is increased by NSAIDs or aspirin, in part because of platelet dysfunction. The risk of gastrointestinal bleeding secondary to NSAIDs appears to be dose related.

The evidence for an increased risk of NSAID-induced upper gastrointestinal bleeding is derived largely from case-control studies. Although the data from these studies are not entirely consistent, the following points appear to be reliable: (1) the risk for gastric ulceration

is greater than for duodenal ulceration, although both are increased; (2) the risk of bleeding varies with the individual NSAID; for example, the relative risk of bleeding is greatest with azapropazone and piroxicam, but less with ibuprofen; (3) the risk of bleeding is dose dependent; and (4) multiple cofactors contribute to NSAID risk.

Cofactors important in NSAID-induced ulceration are diverse. For example, age and previous upper gastrointestinal bleeding appear to be important predictors of NSAID-associated bleeding. In a study by Silverstein et al[17], it was found that age greater than 75 years, history of heart disease, history of peptic ulcer, and history of previous gastrointestinal bleeding were independent predictors of NSAID-induced complications. These data are highly consistent with other data emphasizing the importance of age as an independent risk factor for NSAID ulceration. In addition, H. pylori may be a risk factor for ulcers, although the degree of risk is controversial.[18,19] Finally, corticosteroids, the bisphosphonate alendronate, and ethanol appear to potentiate the ulcerogenic effect of NSAIDs and may predispose to UGIH.

Ethanol

The role of ethanol as a predisposing factor for ulcer-related acute upper gastrointestinal bleeding is difficult to assess. It is important to note that patients who ingest ethanol chronically may have alcohol

induced liver disease and secondary portal hypertension, which is an important risk factor for nonulcer upper gastrointestinal haemorrhages. Nonetheless, ethanol is well known to induce gastric mucosal injury, and thus may cause or potentiate ulcer bleeding. Deleterious effects of NSAIDs are further increased among drinkers[20].

Anticoagulation Therapy

Anticoagulation increases the risk of bleeding from ulcer disease. The relative risk of hospitalization for bleeding ulcer in anticoagulated patients is about 3, and anticoagulants further increase the risk of bleeding in those taking NSAIDs[23]. In a randomised control study done by Shorr RI et al[21], it was found that compared with subjects who took neither anticoagulants nor NSAIDs, the relative risk of hemorrhagic peptic ulcer disease among users of both drugs was 12.7 (95% confidence interval, 6.3 to 25.7). This data emphasizes the risk of anticoagulants, particularly for those who use NSAIDs.

PROGNOSTIC FACTORS IN ULCER BLEEDING

Most ulcer bleeds are self-limited, and in these patients recovery is uneventful. However, a subset of patients have continued or recurrent bleeding, which is associated with a poorer prognosis. The prognostic factors emphasized in upper gastrointestinal bleeding apply particularly

to bleeding ulcers since they comprise the majority of upper gastrointestinal bleeding lesions. The three most important factors are age, existing co-morbid conditions and the nature of type of lesion found at endoscopy.

The seminal observation of Griffiths and colleagues that a visible vessel in an ulcer base was predictive of uncontrolled or recurrent bleeding established the importance of the endoscopic appearance of ulcers.[22] The most critical endoscopic features in ulcer bleeding include the following stigmata of active/recent bleeding: active arterial spurting, oozing of blood, a visible vessel, and fresh or old blood clot. Visible vessels are described endoscopically as elevated, dark red or purple lesions that protrude from the ulcer crater. A number of studies have examined endoscopic features as predictors not only of rebleeding but also of outcomes.[23]

Despite the difficulty in assessing stigmata of ulcer bleeding, it is accepted that certain characteristics of the ulcer at the time of endoscopy provide important prognostic information. For example, increasing ulcer size (>1cm) is associated with an increased rate of rebleeding and mortality[24]. Endoscopic haemostasis is less often successful in ulcers larger than 2 cm[24]. The appearance of the ulcer base is also important and may be one of the following: (1) a clean base only, (2) an ulcer base

with a flat, pigmented spot, (3) an ulcer base with an adherent clot, (4) an ulcer base with a visible vessel (also called a pigmented protuberance or sentinel clot that appears raised and rounded and is resistant to washing); or (5) an ulcer base containing a visible vessel or an adherent clot that is actively oozing or spurting. Although there is general consensus about management of patients with active bleeding, visible vessels, flat spots, and clean bases, controversy surrounds management of adherent clots, particularly after vigorous attempts to remove the clot. A prospective outcome study done by Laine et al[25] showed that vigorous irrigation was useful in this population and that endoscopic findings after washing may help triage endoscopic management at initial endoscopy.

GASTRIC EROSIONS

Although hemorrhagic and erosive gastritis refer to findings at endoscopy, a definite association between gastritis and significant bleeding has not been demonstrated. However, gastritis, most often erosive, remains a time-honoured cause of upper gastrointestinal bleeding, reported by endoscopy to be the cause of bleeding in 16% of patients.[26] Gastritis or erosive gastric injury rarely causes haemodynamically significant bleeding. When it does, patients usually have an underlying coagulopathy.

Subepithelial erosions develop in the following clinical situations: (1) after ingestion of NSAIDs, (2) in stress-related medical illnesses, and (3) with ethanol. The most common of these is NSAID ingestion. Of patients who ingest NSAIDs chronically, 40% to 60% have erosions at any given time, and 15 to 30% have ulcers.

Stress-related gastric mucosal injury occurs in extremely ill patients with serious trauma, extensive burns, major surgery, major medical illness (respiratory failure, sepsis, renal failure), and major neurologic trauma or intracranial disease. Indeed, some degree of stress-related gastric injury can be found in virtually all patients admitted to the intensive care unit, prompting the widespread use of prophylactic regimens for these patients.

Prophylaxis for stress-induced gastric injury in specific subsets of patients is warranted. Two strong independent risk factors for bleeding are respiratory failure and coagulopathy. Patients with respiratory failure on mechanical ventilation were studied in a randomized controlled trial by Cook D et al comparing ranitidine which sucralfate.[27] Clinically significant gastrointestinal bleeding developed in 10 of 596 (1.7%) patients receiving ranitidine, compared with 23 of 604 (3.8%) of those receiving sucralfate (relative risk, 0.44; 95% CI, 0.21 to 0.92;P = 0.02). There was no difference in ventilator-associated pneumonia in the two

groups. These data suggest that routine prophylaxis is beneficial in this population. Whether ranitidine is superior or more cost effective compared with proton pump inhibitors is unknown.

It is commonly believed that ethanol ingestion causes gastric erosions and gastrointestinal bleeding. However, support for this position is largely derived from experimental animal studies that infused extremely high concentrations of ethanol into the stomach. The term hemorrhagic gastritis is frequently applied to the subepithelial haemorrhages seen at endoscopy in alcoholic patients. However, histologic extravasation in such patients is typically superficial, and concomitant mucosal oedema is a prominent feature in adjacent, nonhaemorrhagic mucosa.

Alcohol consumption is a risk factor for upper gastrointestinal haemorrhage only in those with excessive ethanol consumption (4 or more drinks per day)[43]. An endoscopic study by Wilcox CM et al in alcoholics found that upper gastrointestinal haemorrhage in most of these patients was the result of peptic ulcer disease or disorders related to portal hypertension.[44]

Endoscopic therapy is generally not useful for treatment of gastritis of any etiology, although it can be attempted if a small number of isolated erosions appear to be the source of bleeding. Selective

arterial infusion of vasopressin has been reported to stop bleeding in patients with gastritis,[28] but its use requires considerable expertise, and it has not been rigorously studied.

DUODENITIS

Although duodenitis is often included in differential diagnosis of upper gastrointestinal haemorrhage, it is a rare cause of acute bleeding. Risk factors for severe erosive duodenitis are similar to those found in patients with bleeding peptic ulcers, like NSAIDs and H. pylori, and often is associated with anticoagulation therapy. The bleeding is usually self-limited and rarely requires intervention.

MALIGNANCY

Neoplasms of the oesophagus, stomach, and upper small intestine cause acute upper gastrointestinal haemorrhage infrequently. The majority of tumours associated with clinically significant acute UGIH are malignant[45]. Of the many tumours that cause UGIH, the most common is advanced gastric adenocarcinoma[45]. A small proportion of bleeding lesions have been managed with injection or coagulation therapy, and bleeding polypoid lesions can sometimes be snared, but large and/or sessile bleeding lesions typically require surgical intervention. These patients with upper gastrointestinal bleeding from tumours have a 1-year survival rate of 11%.

DIEULAFOY'S LESION

Dieulafoy's lesion, also termed *exulceratio simplex Dieulafoy*, refers to an abnormally large artery that retains the large calibre of its feeding vessel as it approaches the mucosa. This large vessel is thought to compress the mucosa and cause a small erosion with rupture of the vessel into the lumen. Dieulafoy's lesions are not uncommon, accounting for up to 6% of cases of upper gastrointestinal hemorrhage[47]. Dieulafoy's lesions are typically found in the proximal portion of the stomach, usually within 6 cm of the gastroesophageal junction, but they may be located anywhere in the gastrointestinal tract.

Bleeding is often massive and recurrent; it is often difficult to identify the lesion, unless it is actively bleeding or is associated endoscopically with stigmata of recent bleeding. Endosonography may be useful in the detection of Dieulafoy’s disease in patients with unexplained upper gastrointestinal bleeding[47]. Therapy with injection techniques, coagulative therapy, haemoclips, and banding can all control bleeding and prevent rebleeding in over 95% of cases. The long-term prognosis of patients with Dieulafoy's lesions, in the absence of concomitant medical illness, is excellent.

VASCULAR LESIONS

Vascular Ectasia

Vascular lesions are an uncommon, but important, cause of upper gastrointestinal tract bleeding. A number of vascular disorders can cause upper gastrointestinal haemorrhages, but the most common vascular lesions are vascular ectasias, which are most often found in the stomach or duodenum. Vascular ectasias more commonly cause lower gastrointestinal and occult bleeding than upper gastrointestinal tract bleeding. They are found in a variety of conditions, including renal failure, cirrhosis, scleroderma, the CREST syndrome, radiation injury, collagen diseases such as pseudoxanthoma elasticum and Ehlers-Danlos syndrome, and von Willebrand's disease. Vascular ectasias appear to be most often associated with chronic renal failure. The prevalence of vascular ectasia as a cause of upper gastrointestinal bleeding is related to the duration of renal failure and the requirement for hemodialysis.

The treatment of vascular ectasias is difficult because they are rarely found in isolation. Patients with lesions that are readily identified or are actively bleeding are best treated endoscopically with laser, bipolar electrocoagulation, banding, injection therapy, or argon plasma coagulation; each technique appears to be effective and safe in this setting. Perforation of the gastrointestinal tract, however, is a risk,

particularly with electrocoagulation or laser therapy. Massive bleeding may be stopped by angiographic therapy. Recurrent bleeding from a specific bleeding lesion after endoscopic or angiographic therapy is uncommon; surgical therapy is reserved for low-risk patients who have lesions that are clearly identified as the bleeding source.

Arteriovenous Malformations

True arteriovenous malformations, which may appear as raised or nodular lesions at endoscopy, are rare. These lesions are probably congenital in origin and, in contrast to vascular ectasias, usually involve the submucosa; they may be large and involve any portion of the gut wall; the primary treatment is resection of the involved bowel.

Hereditary Haemorrhagic Telangiectasia

Hereditary haemorrhagic telangiectasia is an autosomal dominant disorder characterized by telangiectasias of the skin, mucous membranes, and gastrointestinal tract. The peak incidence of bleeding is in the 6th decade of life, and can originate from any site in the gastrointestinal tract. Lack of telangiectasias on the lips, oral and nasopharyngeal membranes, tongue, and periungual areas should cast doubt on the diagnosis. Of many forms of treatment, endoscopic therapy is most effective in stopping haemorrhage from actively bleeding lesions. Surgical therapy is reserved for those with discrete lesions

identified as the source of the bleeding. Hormonal therapy, typically with an oestrogen and progesterone combination, has met with mixed results.

Haemangioma

Haemangiomas causing upper gastrointestinal haemorrhage are most commonly identified in the upper small intestine. These benign vascular tumours made up of proliferating vessels, almost all of which are cavernous haemangiomas, appear as single or multiple red, purple, or blue nodular lesions. These lesions generally should not be treated endoscopically. Angiographic therapy may stop bleeding; however, the most effective treatment is surgical.

Gastric Vascular Ectasia

Gastric vascular ectasia constitutes a group of recently recognized entities that rarely cause acute upper gastrointestinal haemorrhage. This lesion is characterized by aggregates of red spots. When the aggregates are arranged in a linear pattern in the antrum of the stomach, the term gastric antral vascular ectasia (GAVE), or watermelon stomach, is used. Its pathogenesis is unknown. Although originally thought to be portal hypertensive in etiology, recent work casts doubt on this hypothesis. Neither use of beta-blockers nor standard portal decompression has proved effective for treatment of gastric vascular ectasia, nor has

endoscopic thermal therapy or antrectomy been effective. One small trial, ethinyl estradiol (30 μ g) and norethisterone (1.5 mg) daily led to a significant decrease in transfusion requirements in the majority of patients[29]; however, these results have not been confirmed.

PROGNOSIS IN NVUGIH

Many studies have addressed the factors that predict outcome in patients with upper gastrointestinal hemorrhage.[30-34] Because upper gastrointestinal bleeding is most commonly caused by ulceration, prognostic factors for it tend to reflect those for bleeding peptic ulcer. Approximately 80% of upper gastrointestinal bleeding episodes are self-limited and require only supportive therapy.[35] The two most important prognostic variables appear to be the cause of bleeding and the presence of underlying comorbidity.

The major factors that determine the prognosis, course, and the outcome are

1. Age
2. Presence/Absence of Comorbid Conditions
3. Endoscopic Diagnosis
4. Presence of Stigmata of Bleeding on Endoscopy

Several scoring systems have been designed to identify patients with a high risk of adverse outcomes; the measures have generally been ascertained from mathematical models of risk of death or rebleeding.

ROCKALL SCORE

In 1996,a prospective study done by T A Rockall et al[36] on patients admitted with UGIH, attempted to identify patients who had negligible risk of further bleeding or death and for whom early discharge or even out patient management would be possible. It was also intended to identify patients with a high risk of adverse prognosis and thereby subject them to more intense monitoring.

In this study by, a prospective audit of the management and outcome of 4201 patients from 74 hospitals in four health regions in the UK were identified over a 4-month period during 1993. The participants for this analysis consisted of 2531 patients who were admitted as acute emergencies with acute upper gastrointestinal bleeding. A numerical scoring was devised based on three clinical variables and two endoscopic variables.

The clinical variables were:

A. Age (score 0 – 2),

B. Presence/absence of shock (score 0 – 2) and

C. Presence/absence of co-morbid conditions (score 2 – 3)

The two endoscopic variables included

A. Endoscopic diagnosis (0 – 2) and

B. Presence or absence of stigmata of recent bleed (0 and 2).

The maximal possible score was 11.

The patients' scores were correlated with the event of rebleeding, mortality and the duration of hospital stay. The results showed that the score identified a large proportion of patients with a low risk of further bleeding or death. It was also found that the length of the hospital stay also increased with increasing score.

In our study the usefulness of the Rockall score as a prognostic score analysed. Its correlation with the event of rebleed, transfusion requirements, duration of hospitalisation and the final outcome is studied.

MATERIALS AND METHODS

SETTING

The study was conducted on patients admitted in the wards of the Institute of Internal Medicine, Madras Medical College and Government General Hospital, Chennai.

COLLABORATING DEPARTMENTS

Institute of Internal Medicine, Government General Hospital

Department of Medical Gastroenterology, Government General Hospital

ETHICAL APPROVAL

Obtained

STUDY DESIGN

Single centre, non-interventional, comparative prospective study.

STUDY PERIOD

January 2008 to September 2008

SAMPLE SIZE

76 patients

SELECTION OF STUDY SUBJECTS

Inclusion Criteria

- ❖ Patients admitted in the general medical wards for Upper Gastrointestinal bleeding in whom endoscopy reveals a non-variceal cause for the bleeding.
- ❖ Patients of age greater than 12 years from both the sexes were included in the study.

Exclusion Criteria

- In-patients who were admitted for a problem other than a Gastro-intestinal bleed who develop UGI bleed during hospital stay.
- Out-patients who undergo endoscopy for Upper Gastrointestinal bleed.
- Patients admitted with Upper Gastrointestinal bleeding who do not undergo endoscopy.

CONSENT

The study was carried out with the informed written consent of each of the participants.

METHODOLOGY

Patients with Upper Gastrointestinal bleeding in whom endoscopy shows a non- variceal etiology for the bleed, are included in the study. The findings at endoscopy, the lesion and the presence or absence of stigmata of recent bleeding are noted. Data are collected from these patients regarding the nature and duration of their symptoms, co-morbid medical illnesses, history regarding NSAID use, past history of Upper Gastrointestinal bleeding, their alcohol and smoking habits. Their clinical examination findings, pulse rate, blood pressure, and the presence/absence of abdominal tenderness noted at the time of admission are collected from their medical case record. Relevant laboratory data like haemoglobin, urea, creatinine and tests for liver function are also noted.

A numerical score – *Rockall score* is calculated based on the clinical findings at presentation and their endoscopy findings. Three clinical variables are analysed, namely,

A. Age (score of 0-2),

B. Presence of Shock (0-2),

C. Co morbid conditions (2-3)

The two endoscopic variables included in the scoring are

A. The endoscopic diagnosis (0 – 2) and

B. Presence of stigmata of recent haemorrhage (0 and 2).

The Rockall score is obtained by the sum of the individual scores for the variables analysed. Based on the score the patients are stratified as

A. Low risk (score ≤2),

B. Intermediate risk (3 – 5) and

C. High risk (≥6).

The Rockall score is correlated with the 1) *occurrence of re-bleeding or prolonged bleeding,* 2) *the duration of hospitalisation,* 3) *requirement of transfusions and* 4) *the final outcome.*

These data are collected on the tenth day after the initial presentation, either by a direct interview or by a telephonic interview in the event of the patient being discharged before the tenth day.

The data collected are analysed statistically for correlation with the Rockall score. Microsoft Windows' Excel was used to tabulate the data and SPSS – Statistical Package for Social Sciences was used to analyse the data.

THE ROCKALL SCORING SYSTEM

VARIABLE	SCORE			
	0	**1**	**2**	**3**
AGE	<60	60-79	≥80	-
SHOCK (SBP mmHg, HR- beats/min)	'No shock': SBP≥100 and HR<100	'Tachycardia': SBP≥100 and HR≥100	'Hypotension': SBP<100	-
COMORBIDITY	No major morbidity	-	Cardiac failure, ischemic heart disease any major comorbidity	Renal failure, liver failure, disseminated malignancy
DIAGNOSIS	Mallory-Weiss tear, no lesion identified and no SRH/blood	All other diagnosis	Malignancy of the upper GI tract	-
MAJOR SRH	None or dark spot only	-	Blood in upper GIT, adherent clot, visible or spurting vessel	-

D E F I N I T I O N S

UPPER GASTROINTESTINAL HAEMORRHAGE

Haematemesis or melaena or other firm clinical or laboratory evidence suggesting a site of bleeding in the gastrointestinal tract proximal to the ligament of Treitz.

HAEMATEMESIS

It is defined as the vomiting of blood or blood clots and indicating an upper gastrointestinal site of the bleeding.

MELAENA

Melaena is defined as the passage of black, tarry and offensively foul-smelling stools.

HAEMATOCHEZIA

Haematochezia is defined as the passage of bright red blood from the rectum which may or may not be mixed with stool.

REBLEEDING

In this study, rebleeding was defined as a new episode of bleeding within the first ten days after the initial presentation, which manifested as recurrent haematemesis, haematochezia, fresh blood in the naso-

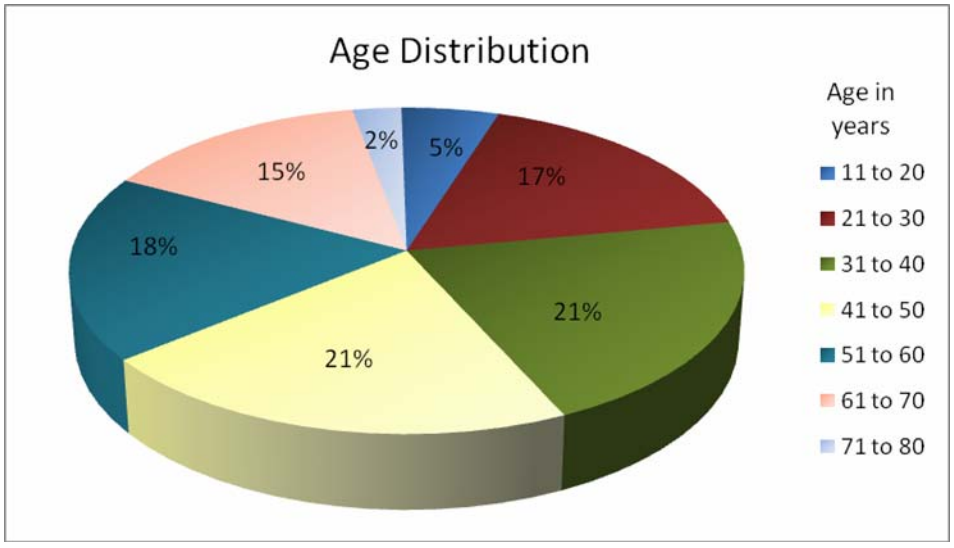
gastric aspirate, or circulatory instability occurring after initial hemodynamic stabilisation. Melaena if continuing even after the fourth day after first day of melaena is taken as ‘continuing’ bleed; and for analytical purposes in this study is grouped under ‘Rebleed’.

RESULTS

AGE DISTRIBUTION

The study population comprised of 76 patients. Their age ranged from 14 years to 80 years. The mean age of the group was 44.14years (±14.9).

Chart 2: Age Distribution of patients with NVUGIH



AGE AND DURATION OF HOSPITALISATION

The average duration of hospital stay in the study population was 5.72 days. The duration of hospitalisation increased with the age of the patient. It was longest (6.3 days) for those >50 and shortest (5.2days) for those ≤30 years.

Table 1: Age and duration of hospitalisation

AGE GROUP (years)	DURATION OF HOSPITALISATION (days)
≤30	5.29
31-50	5.41
>50	6.37
Total Mean	5.72

AGE AND INCIDENCE OF REBLEED

The incidence of re-bleed in this study was found to increase with increasing age, as is evident from the following data.

Table 2: Age and incidence of re-bleed

AGE GROUP (YEARS)	REBLEED
<30	29%
30-50	37%
>50	55%
AVERAGE	42%

AGE AND DIAGNOSIS

The average age of the patients against each diagnosis was the highest in patients with malignancy (58.14 yrs) while it was the least in patients with mucosal erosions (39.12). The average age of patients with PUD was 45.37 yrs.

Table 3: Endoscopic diagnosis and the average age

DIAGNOSIS	AVG. AGE IN YEARS
CA	58.14
MW	45.67
PUD	45.37
Normal Study	41.67
Mucosal Erosions	39.21
Grand Total	**44.14**

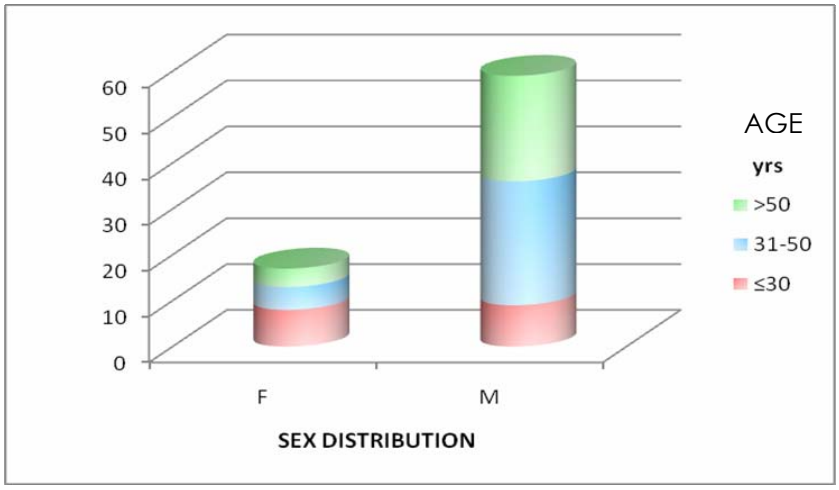
SEX DISTRIBUTION

Males constituted 77.63% of the study population and females 22.53 percent. The mean age of the female population in the study group was 38.23 years and that of males was 45.84 years. The most common diagnosis at endoscopy in females was Erosive Mucosal disease constituting 58.8% of the total diagnosis in females while in males the most common diagnosis was Peptic Ulcer disease accounting for 48.8% of the diagnosis.

Table 4: Sex and age distribution

	A G E			
Sex	**≤30yrs**	**31-50 yrs**	**>50 yrs**	**Grand Total**
F	8	5	4	17
M	9	27	23	59
Total	**17**	**32**	**27**	**76**

Chart 3: Sex and age distribution



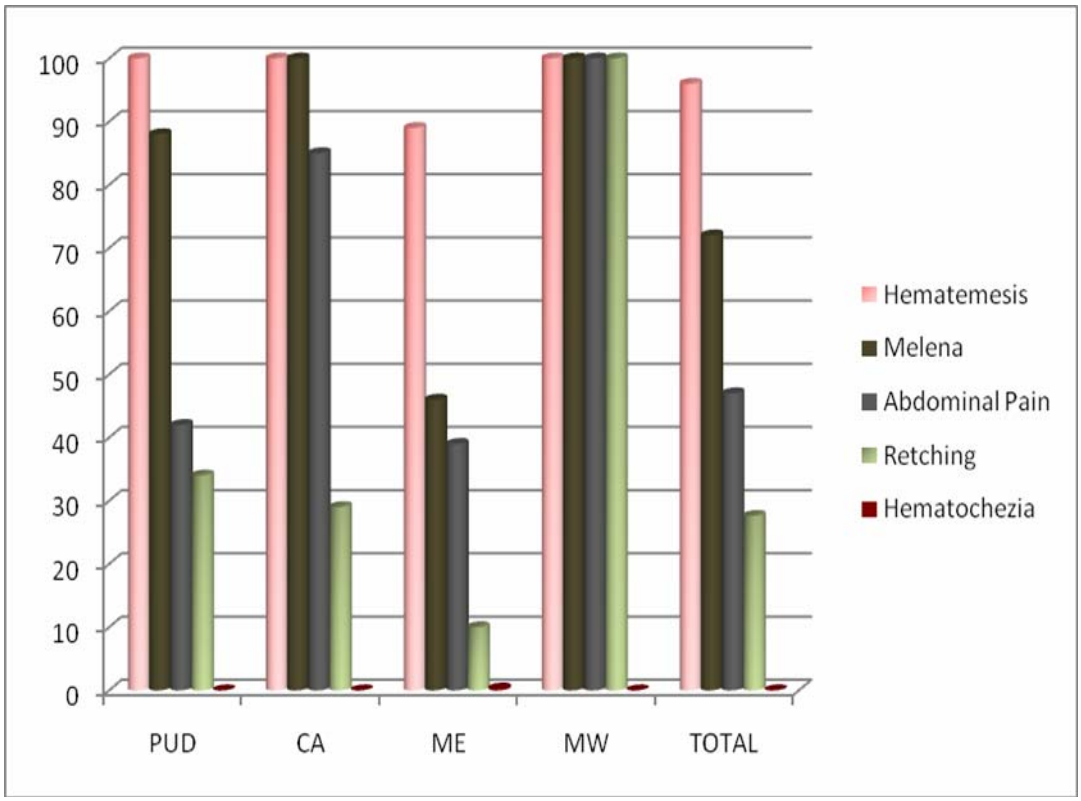
SYMPTOM ANALYSIS

In this study 96% presented with haematemesis and 72% with melaena. Retching was present in 27.6% of the entire study population and all patients with Mallory Weiss tear had a positive history. Forty seven percent of the population had abdominal pain. Abdominal pain was more common in patients with malignancy than in patients with Peptic Ulcer Disease.

Table 5: Symptom Analysis

DIAGNOSIS	HAEMATEMESIS %	MELAENA %	HAEMATOCHEZIA %	ABDOMINAL PAIN%	RETCHIN %
PUD	100	88	0	42	34
CA	100	100	0	85	29
ME	89	46	0.3	39	10
MW	100	100	0	100	100
TOTAL	96	72	0.01	47	27.6

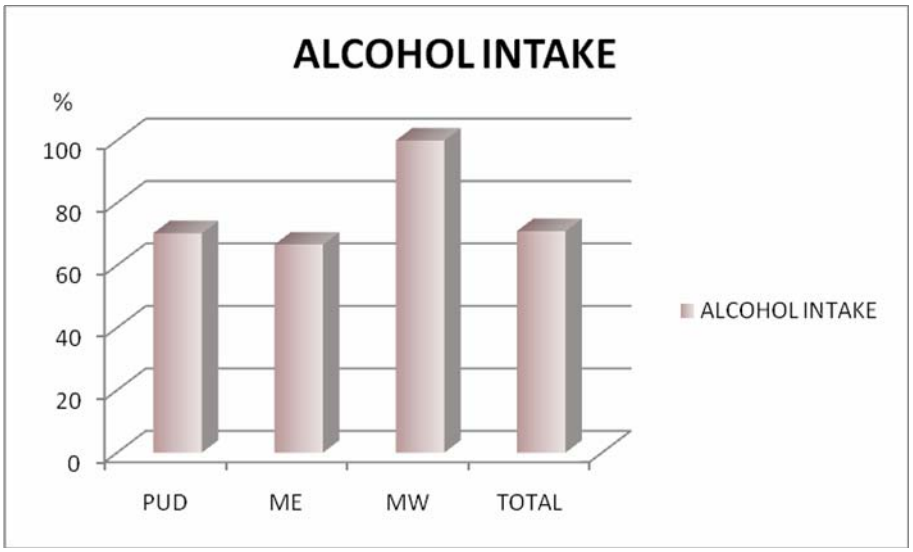
Chart 4: Symptom Analysis of patients with NVUGIH



ALCOHOL INTAKE

In this study population alcohol consumption was restricted to the males. Seventy one percent of the males had history of alcohol consumption on the day or the day before the day of presentation of the bleeding. History of alcohol intake was present in 70.3% of the males with PUD, in 66.7% of patient with Mucosal erosive disease and in 100% of patients with Mallory Weiss tear.

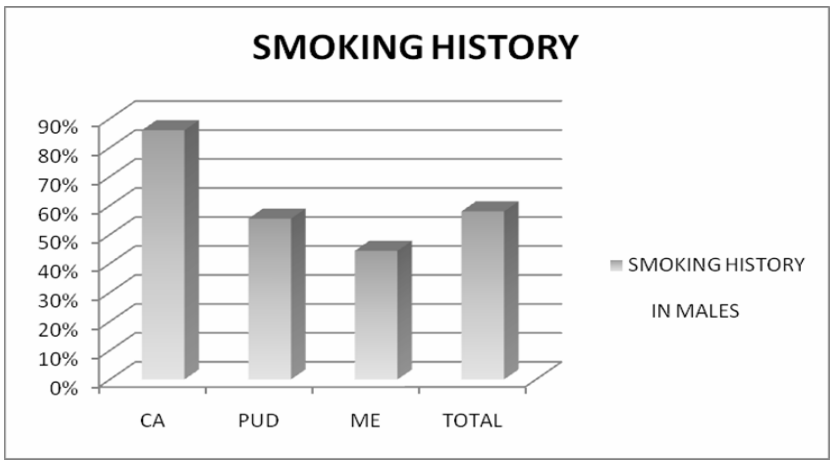
Chart 5: Alcohol Intake in males and diagnosis



SMOKING

Smoking was restricted to the males in the study population. Fiftyeight percent of the males with UGI bleed were smokers. Eighty six percent of the patients with UGI malignancy, 55.5% of male patients with PUD and 44.4% of patients with Mucosal Erosive lesions were smokers.

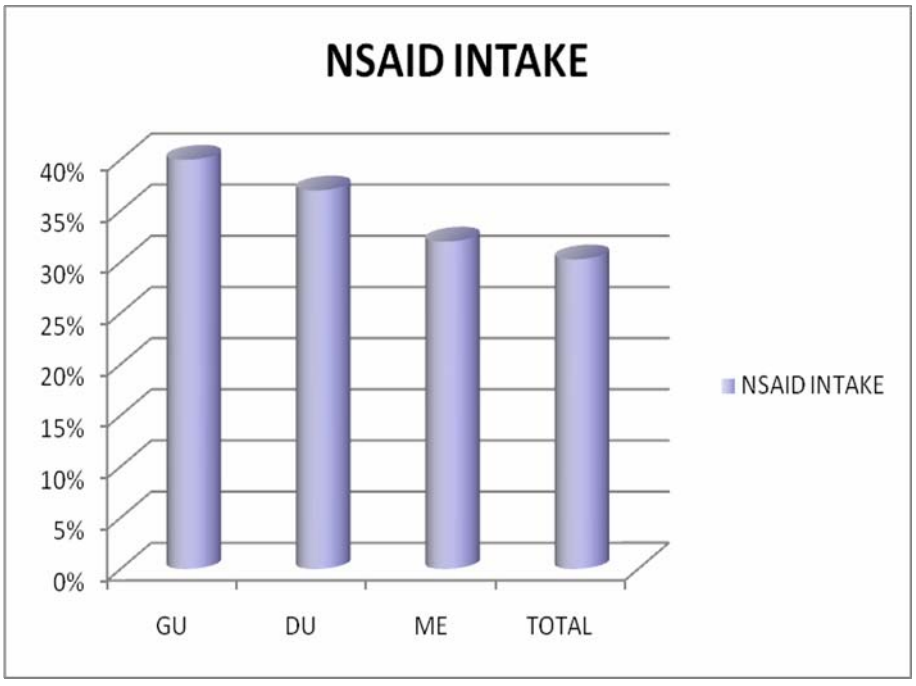
Chart 6: Smoking and Etiology of bleed



NSAID INTAKE

NSAID intake was associated in 30.26% of the patients. NSAID intake was relatively more common in females than in men. A positive history was present in 25% of the male patients and in 47% of the female patients. In patients with Gastric ulcer and Duodenal ulcer a positive drug history for NSAIDS was present in 40% and 37% respectively while in patients with erosive mucosal disease, 32% gave a positive history. *Ref Chart 7*

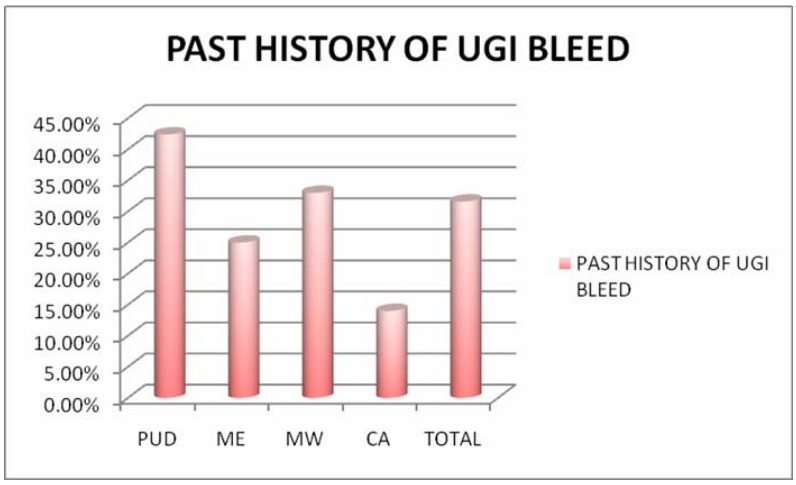
Chart 7: NSAID intake and diagnosis



PAST HISTORY OF UGIH

Out of the seventy six patients in the study, 24(31.6%) had a past history of UGI bleed. Patients with PUD had the highest past incidence of UGI bleed (42.4%) and patients with gastric carcinoma (CA) had the least (14%).

Chart 8: Etiology of Bleed and past history of Bleed



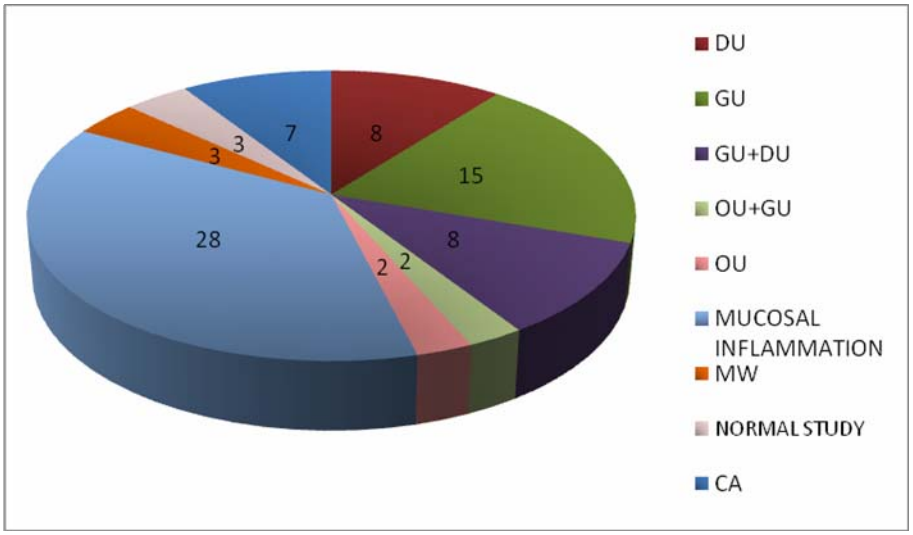
DIAGNOSIS

At endoscopy the most common diagnosis was PUD (46%), followed by Mucosal erosions (36.8%) followed by GI malignancy (9%). Among the patients with PUD the most common lesion was gastric ulcer (19.7%) and 10.5% had both gastric and duodenal ulcers. Three patients had a normal study at endoscopy. *Ref Chart 9*

Table 6: Etiology of bleed – Distribution

ENDOSCOPIC DIAGNOSIS	TOTAL	PERCENTAGE
DU	8	10.50%
GU	15	19.70%
GU+DU	8	10.50%
OU+GU	2	2.60%
OU	2	2.60%
Mucosal Erosions	28	36.80%
Mallory Weiss	3	3.90%
Normal Study	3	3.90%
Gastric Malignancy	7	9.00%
Grand Total	76	100%

Chart 9: Endoscopic diagnosis – Distribution



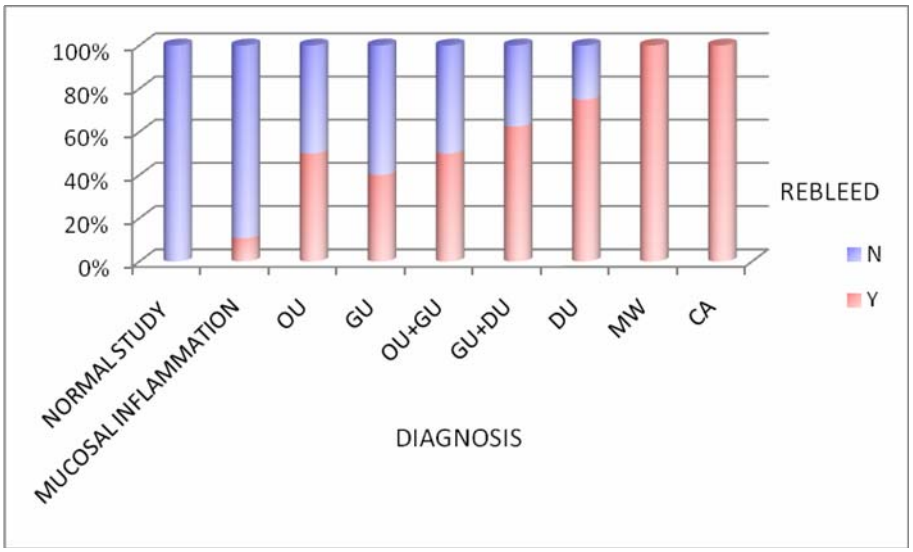
DIAGNOSIS AND REBLEED

In this study population, 42% of the patients had rebleeding. The rebleeding incidence was lowest for the patients with a normal study at endoscopy and highest for the patients with GI malignancy and those patients with Mallory-Weiss tear. *Ref. Chart 10*

Table 7: Endoscopic Diagnosis and Re-bleed

ENDOSCOPIC DIAGNOSIS	REBLEED PERCENTAGE
NORMAL STUDY	0%
MUCOSAL INFLAMMATION	10.70%
OU	50%
GU	40%
OU+GU	50%
GU+DU	62.50%
DU	75%
MALLORY-WEISS	100%
GASTRIC CA	100%
ALL	42%

Chart 10: Diagnosis and Re-bleed



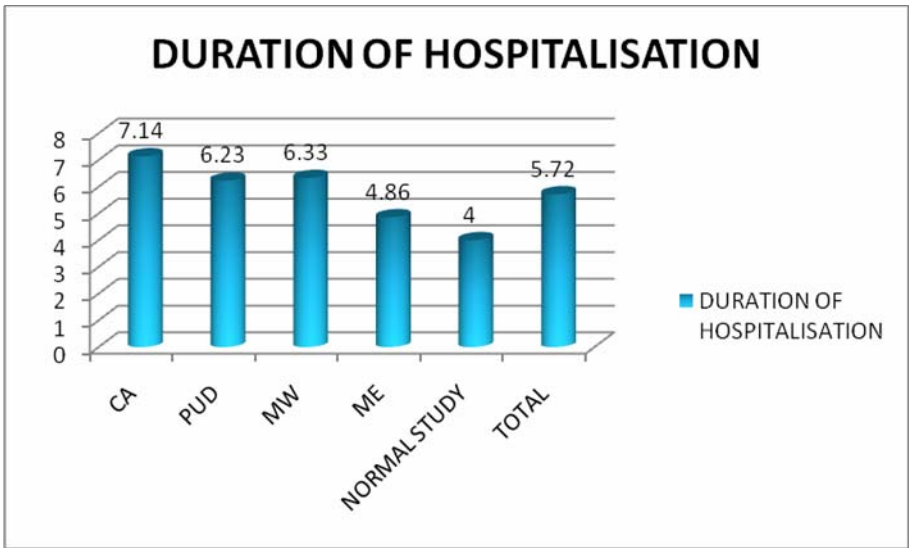
DIAGNOSIS AND DURATION OF HOSPITALISATION

The average duration of hospitalisation was 5.72 days. The longest in-hospital stay period (in-medical ward stay in case of malignancy) was for patients with gastric carcinoma whereas patients with a normal study at endoscopy had the shortest hospital stay. *Ref. Chart 11*

Table 8: Diagnosis and duration of Hospitalisation

DIAGNOSIS	DURATION OF HOSPITALISATION (days)
Gastric CA	7.14
PUD	6.23
Mallory Weiss	6.33
Mucosal Erosions	4.86
Normal Study	4
TOTAL	5.72

Chart 11: Diagnosis and Duration of Hospitalisation



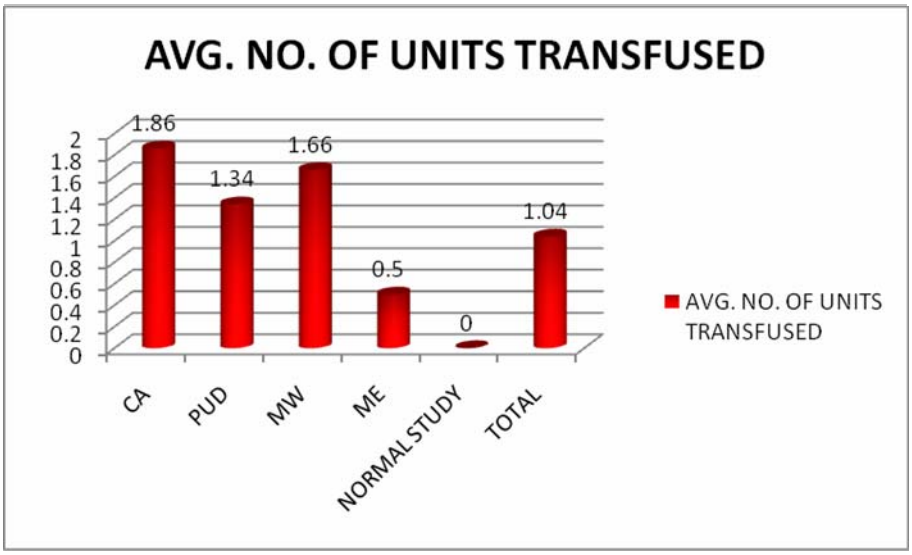
DIAGNOSIS AND TRANSFUSION REQUIREMENTS

Sixty two percent of the patients required blood transfusion. Patients with Gastric Carcinoma required the highest no of transfusions.The average number of units transfused in this study is 1.04. *Ref. Chart 12*

Table 9: Diagnosis and Transfusion Requirements

DIAGNOSIS	AVG. NO. OF UNITS TRANSFUSED
Gastric Carcinoma	1.86
PUD	1.34
Mallory Weiss	1.66
Mucosal Erosions	0.5
Normal Study	0
TOTAL	1.04

Chart 12: Diagnosis and Transfusion requirements



OUTCOME

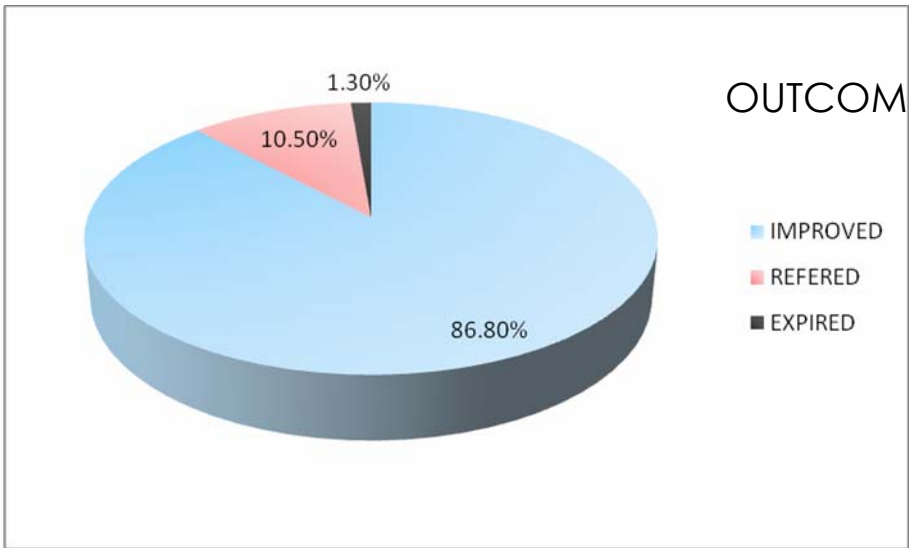
In this study 86.8% of the study population improved and were discharged. Five patients required endoscopic intervention constituting 8% of the patients in the 'improved' group. Eight patients (10.5%) were taken up for surgical management. All these patients had gastric carcinoma. There was one death, accounting for 1.3% of the population. *Ref. Chart 13*

Table 10: Outcome of the patients

OUTCOME	PERCENTAGE
Improved	86.80%
Refered	10.50%
Expired	1.30%

Grand Total	100%

Chart 13: Outcome of the patients



ROCKALL SCORE

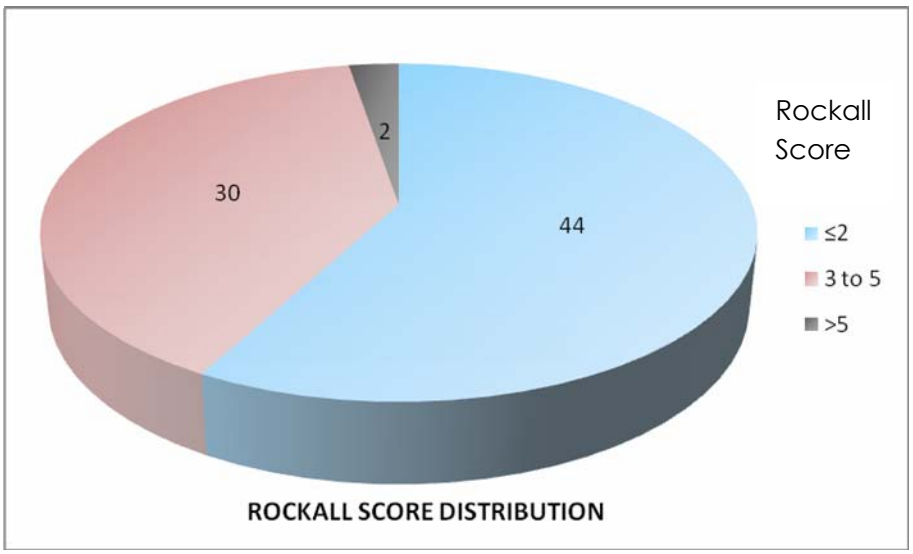
Rockall score was calculated for each patient in the study population. The majority of patients had a low Rockall score. Patients with a high Rockall score of >5 constituted only 2.6% of the study population. The average Rockall score of the study population was 2.63. *Ref. Chart 14.*

Table 11: Distribution of patients over Rockall score

ROCKALL SCORE	NO OF PATIENTS	PERCENTAGE
≤2	44	57.90%
3 to 5	30	39.50%
>5	2	2.60%

Grand Total	**76**	100%

Chart 14: Distribution of patients over Rockall score



ROCKALL SCORE AND THE EVENT OF REBLEED

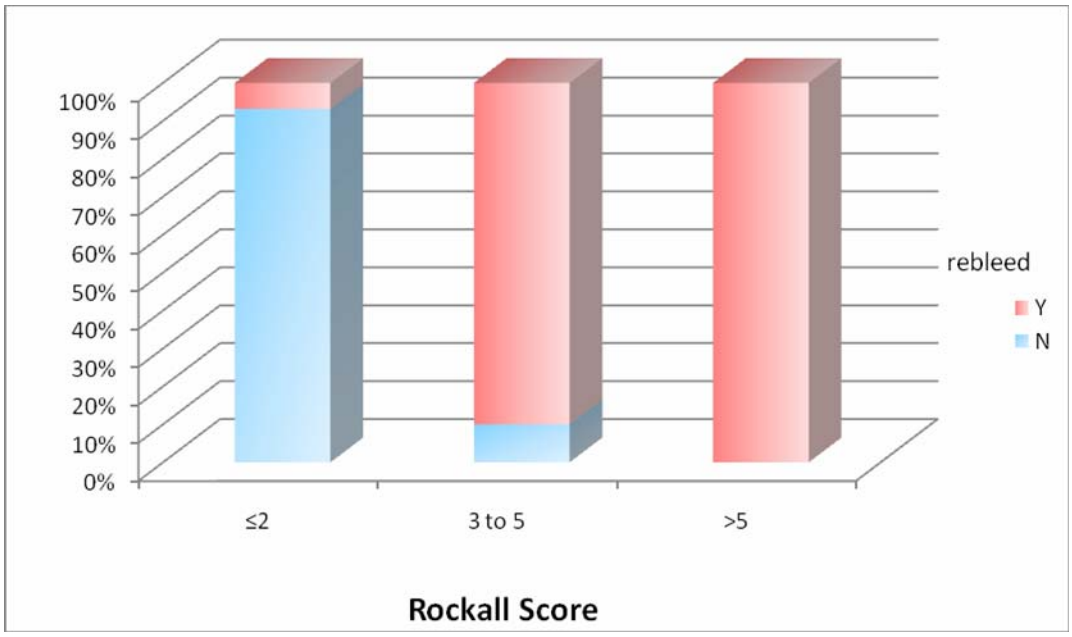
The value of Rockall score was correlated with the event of the patient having a rebleed. It was found that as the score increased there was a greater percentage of patients rebleeding. The correlation was highly significant with a p value of 0.000001. *Ref. Chart 15.*

Table 12: Rockall Score and correlation with re-bleed

REBLEED	ROCKALL SCORE			TOTAL
	≤2	3-5	>5	
YES	6.8% (3)	90.0% (27)	100% (2)	42.1%
NO	93.2% (41)	10.0% (3)	0%	57.9%

TOTAL	57.9% (44)	39.5% (30)	2.6% (2)	100%
p value < 0.000001				

Chart 15: Rockall Score and incidence of re-bleed



ROCKALL SCORE AND DURATION OF HOSPITALISATION

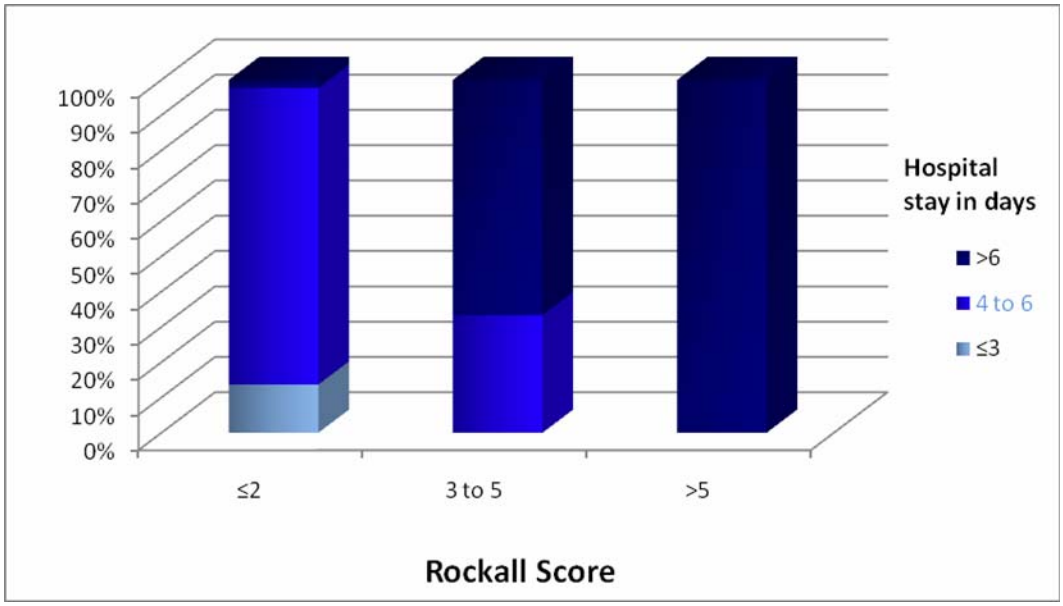
It was found in this study that there was a direct proportional relation between the value of the Rockall score and the duration of hospital stay of the patients. There was a positive correlation between the two with a highly significant p value of 0.000001. *Ref. Chart 16*

Table 13: Rockall score and correlation with duration of Hospitalisation

HOSPITAL STAY	ROCKALL SCORE			TOTAL
	≤2	3 to 5	>5	
≤3	13.6% (6)	0%	0%	7.9% (6)
4-6 days	84.1% (37)	33.3% (10)	0%	61.8% (47)
>6	2.3% (1)	66.7% (20)	100% (2)	30.3%(23)

TOTAL	57.9% (44)	39.5% (30)	2.6% (2)	100% (76)
p value< 0.000001				

Chart 16: Rockall score and duration of Hospitalisation

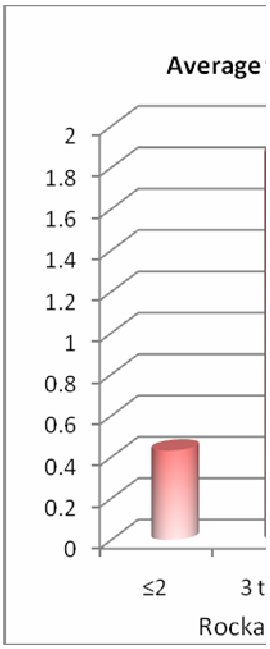


ROCKALL SCORE AND TRANSFUSIONS

There was an increasing need for transfusing the patients with increasing Rockall score. The average quantity of blood transfused for the patients in the study was 1.03 units. *Ref. Chart 17*

Table14: Rockall Score and correlation with transfusion requirements

Chart 17: Rockall score and transfusion requirements



ROCKALL SCORE	Average of No. of units transfused
≤2	0.432
3 to 5	1.87
>5	2
Mean transfusion req.	**1.03**
p value – 0.0001	

ROCKALL SCORE AND DIAGNOSIS

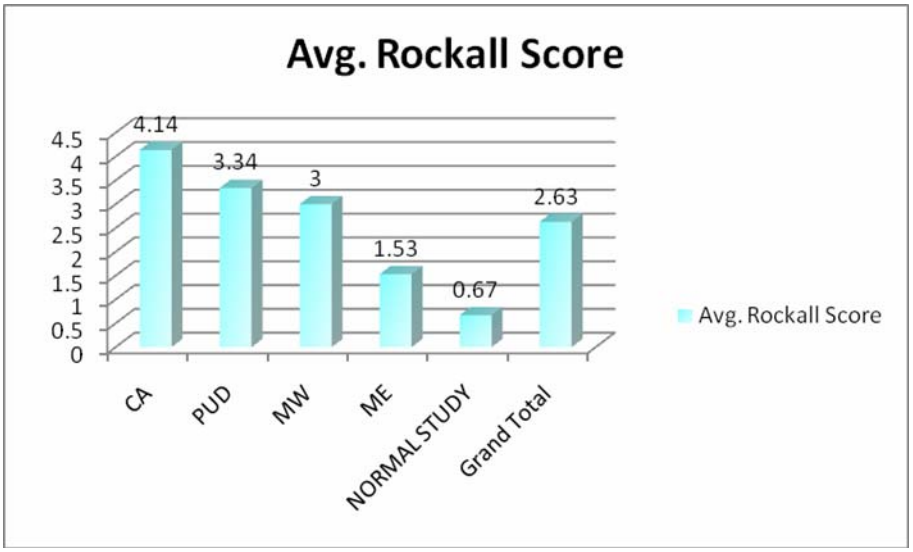
In this study, the average Rockall score was 2.63. Patients with GI malignancy had the highest Rockall score of 4.14, followed by PUD. Patients with a normal study at endoscopy had the least Rockall Score of 0.66. *Ref. Chart 18*

Table 15: Diagnosis and the Mean Rockall Score

Diagnosis	Avg. Rockall Score
CA	4.14
PUD	3.34
MW	3
ME	1.53
NORMAL STUDY	0.66

Total Avg. score	2.63

Chart 18: Diagnosis and the mean Rockall Score



ROCKALL SCORE AND HAEMOGLOBIN

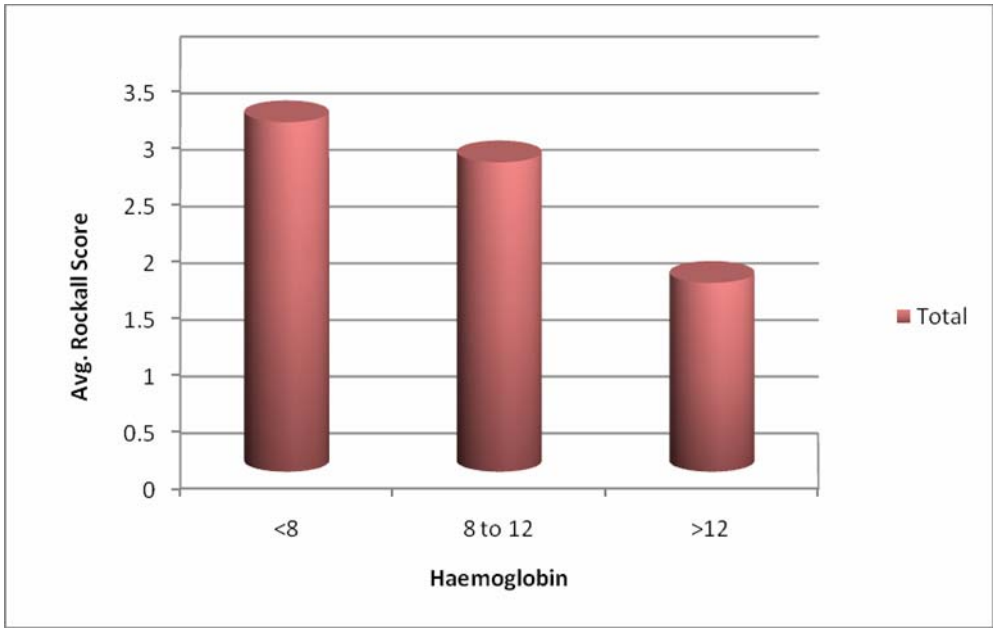
Rockall score also reflected the Haemoglobin levels of the patients in the study. It was found that with as the haemoglobin level decreased the Rockall score increased. It had a significant inversely correlation with a p value of 0.003. *Ref. Chart. 19*

Table 16: Hemoglobin and Rockall Score

HAEMOGLOBIN	AVG. ROCKALL SCORE
<8	3.08
8 to 12	2.72
>12	1.66
Grand Total	**2.63**

p value – 0.003

Chart 19: Haemoglobin and the Average Rockall Score



ROCKALL SCORE AND UREA

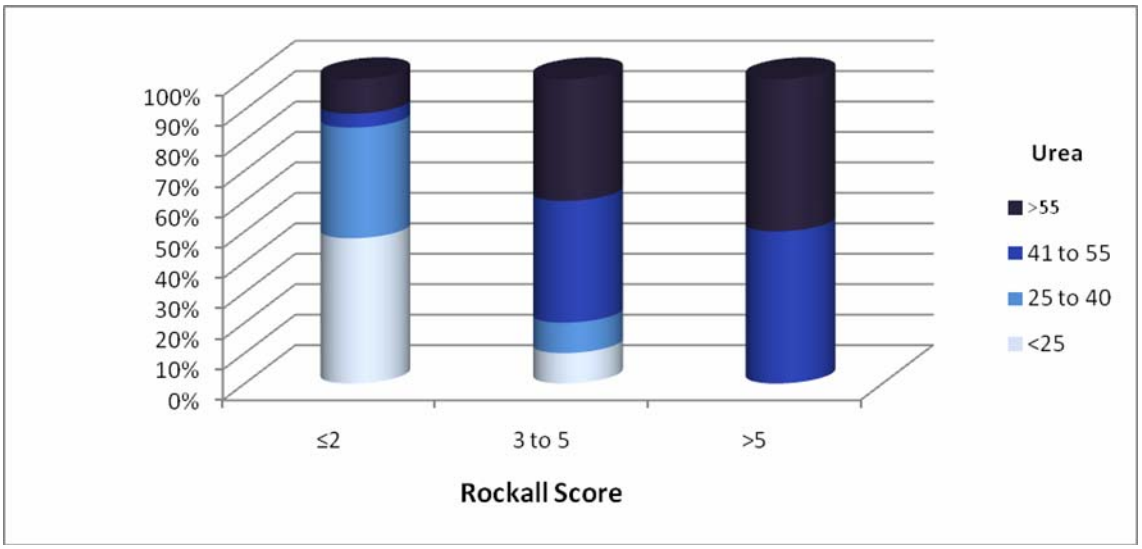
In this study it was found that patients with a high Rockall score also had elevated urea levels. The positive correlation was highly significant with a p value of 0.0001. *Ref. Chart 20*

Table 17: Rockall score and correlation with Urea value

Rockall Score	U R E A				
	<25	**25 to 40**	**41 to 55**	**>55**	**Grand Total**
≤2	47%(21)	36%(16)	5%(2)	11%(5)	100%(44)
3 to 5	10%(3)	10%(3)	40%(12)	40%(12)	100%(30)
>5			50%(1)	50%(1)	10%(2)
Grand Total	24	19	15	18	76

P value = 0.0001

Chart 20:Rockall Score and Urea levels



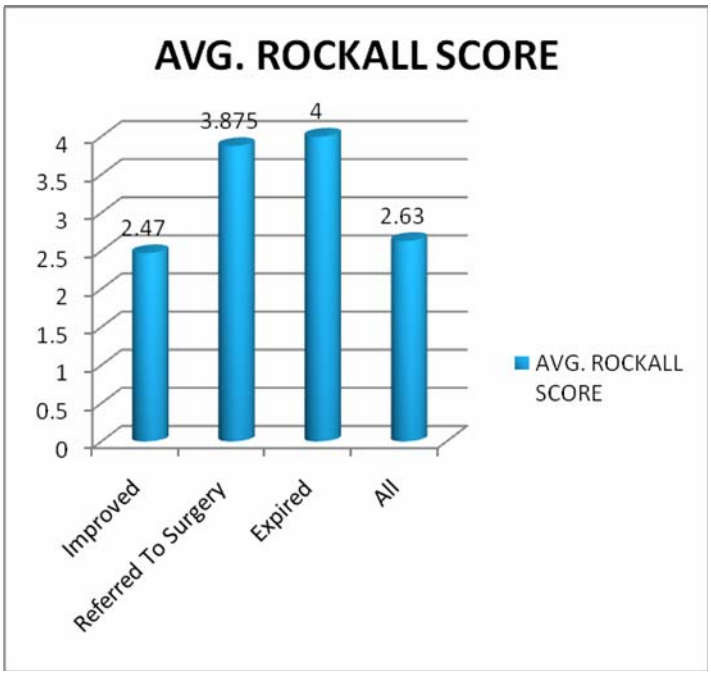
ROCKALL SCORE AND OUTCOME

The Rockall score of the patient who expired was 4. The average Rockall score of the patients who were referred to surgery was 3.87. The average Rockall score was the least (2.47) in the patients who had improved and were subsequently discharged..*Ref. Chart 21*

Table 18: Outcome and Mean Rockall score

OUTCOME	AVG. ROCKALL SCORE
Improved	2.47
Referred To Surgery	3.875
Expired	4
All	2.63

Chart 21: Final Outcome and Mean Rockall Score



DISCUSSION

Non-variceal upper gastrointestinal haemorrhage is a common medical emergency in everyday clinical practice. It can present with a varied clinical spectrum ranging from the insignificant to the catastrophic. Hence it is imperative that these patients need to be stratified according to their risks into those who are likely to have a complication (rebleed, mortality) and those in whom a less dramatic outcome is expected. This not only serves as a tool in better management of the former group but can also cut in-hospital costs of the

latter. Several adverse prognostic variables are known to affect the outcome. This study attempts to analyse these variables and the clinical profile of the patients admitted with NVUGIH. It also attempts to correlate the outcome with a prognostic scoring system - The Rockall Score.

AGE DISTRIBUTION

Age is an independent adverse prognostic variable in patients with NVUGIH[38]. In this study the mean age was 44.14 years (±14.9) as compared to 67.17±16.7 years reported by Marco Soncini[37] et al, in a study done in Italian population. The study-population of Vreeberg EM et al had a mean age of 71 years[34]. In our study, the majority of the population was middle aged (30-60years) and there was a trend of increasing duration of hospital stay with increasing age. The study also showed a greater incidence of rebleed with increasing age.

Out of the 76 patients who formed the study population only one patient was ≥80 years. The Rockall score stratifies the age into age <60, 60-79 and age ≥80. In this study the majority of patients fell into the first group with hardly any patients in the third group. This might be because the average life expectancy in Indian population is 62.8 years[52] as compared to western population who have a higher life expectancy [75.2

years in the U.K.]. Hence in the Indian scenario it might be appropriate to scale down the age groups to ones appropriate to our population.

SEX DISTRIBUTION

Gastrointestinal bleeding is more common in the males[1,2]. Males constituted 77.63% of the study population. This could probably be attributed to the fact that in the Indian setting men expose themselves more commonly to ethanol and smoking as compared to women. Though in this study both alcohol intake and smoking was restricted only to men, women had a higher incidence of NSAID intake compared to men. The most common diagnosis at endoscopy was PUD in the males. Both Mallory-Weiss tear and gastric malignancy were restricted to males. It is well known that both PUD and gastric carcinoma are more common in men[39.] The occurrence of Mallory Weiss tear only in males is partially explained by the fact that alcohol intake, a well known risk factor for its development[38], was restricted to the males, though there are reservations, owing to the small sample size. The most common diagnosis in females was mucosal erosive disease.

CLINICAL PRESENTATION

In this study, abdominal pain, thought to be a classical feature in PUD, was present only in 42% of the patients with PUD. Endoscopic studies have shown that peptic ulcerations are often asymptomatic and

in one such study duodenal acid perfusion produced pain in only 38% of the individuals[40]. Pain was more common among patients with gastric carcinoma occurring in 85% of these patients. Retching was present in all three of the patients with diagnosis of MW tear.

Out of the 76 patients, based on the presentation, a definitive clinical etiological diagnosis could be made in only 63 patients; and among these patients the endoscopic diagnosis concurred with the clinical diagnosis in only 56% of the patients. It can thus be inferred that reliance on purely signs and symptoms is neither sensitive nor specific means to arrive at the etiological diagnosis. It is therefore imperative that patients with significant NVUGIH undergo endoscopic to ascertain the etiology as well as for the management if then indicated.

DIAGNOSIS

Peptic Ulcer Disease : PUD has been reported by Skok P et al. to account for nearly 50% of the cases with UGIB[48]. In our study PUD accounted for nearly half the cases diagnosed at endoscopy. The most common single lesion in this group was gastric ulcer (45% of PUD) followed by duodenal ulcer. There were a considerable proportion of patients with combined lesions (30% of PUD).

There is a greater risk of gastric ulceration as compared to duodenal ulcers with NSAIDS[38]. In this study NSAID intake was associated with 40% of the patients with gastric ulcers as compared to 37% of patients with duodenal ulcer.

Incidence of bleeding from DU is approximately twice that of gastric ulcers.[38] Though in this study GU was commoner than DU, rebleeding was more common in patients with DU (75%) compared to GU (40%). The actual incidence of patients with gastric and duodenal ulcer who develop UGIB cannot be commented upon with this study.

The duration of hospitalisation was also higher in patients with DU patients requiring on an average 7.6 days of hospitalisation as compared to 5.6 days for the patients with GU. The number of transfusions required was slightly more for DU (avg. 1.8 units) compared to GU (1.3 units).

Table 19: Comparison between Gastric Ulcer and Duodenal Ulcer

	Gastric Ulcer	**Duodenal Ulcer**
Re-bleed	40%	75%
Avg. duration of hospitalisation	5.6 days	7.6 days
Avg. no. of transfusions	1.3 days	1.8 units

From this study it can be concluded that in a PUD patient with UGIB, DU has a more protracted course than a patient with GU.

MUCOSAL EROSIVE DISEASE

In this study isolated mucosal erosion were detected in 36.8% of the study group as compared to 11% reported by TA Rockall et al[41]. The three known risk factors for mucosal erosions are alcohol, stress and NSAIDS[44]. In this study there was a history of alcohol intake in 42% of the patients. Thirty two percent of the patients had a history of NSAID intake. A much lower incidence of 61% was reported by Marco Soncini for drug induced UGIB[37]. Stress an ill-defined abstract risk factor was not assessed in this study.

The incidence of rebleeding was 10.7% much less compared to those with PUD. The average duration of hospitalisation was 4.86 days and the average no of units of blood that needed to be transfused was 0.5. Clearly the clinical course was more benign compared to those with PUD.

GASTRIC CARCINOMA

Gastric Ca accounted for 9% of the patients presenting with NVUGIH in this study. The incidence is quite high compared to the 4% reported by TA Rockall et al[41]. It could be explained by the fact that many of the patients in this group were initially under the diagnosis of PUD but later re-allotted to the CA group after the biopsy taken on suspicion during endoscopy confirmed malignancy. Adenocarcinoma

was reported in all their biopsies. After the biopsy report these patients were subsequently referred to surgical units for further management. Since there was at least some delay between the time of endoscopy and the time of arrival of the biopsy report after which they were referred, their duration of stay might not be exactly indicative of their actual requirement. Moreover these patients were not followed up in their respective surgical wards after transfer. Nevertheless these patients had the highest requirements for transfusions and the highest incidence of rebleed in the entire study group.

MALLORY WEISS TEAR

According to literature, they account for approximately 5% of cases of UGIH[42]. In our study it accounted for 4% of the total group. All of them had a contributory alcohol history and all gave a history of retching. In our study all three patients had rebled and were transfused. Since this group was small, no generalisations are attempted made based on this study.

Three patients in the study had a normal UGI endoscopy. It is possible that these patients actually presented with haemoptysis and not haematemesis. Distinction between these two distinct symptoms is often blurred to patients. It is not uncommon to face a patient who is sure only of the fact that the portal of exsanguination was his mouth but can

contribute nothing more. Their inebriated state (two out of three in our study) only makes their history less clear and less reliable. However on a less accusative note, these patients might just have had a lesion (e.g. a Dieulafoy lesion) that was missed during endoscopy.

OUTCOME

The mortality rate for patients with UGIH in most studies was between 8% and 10%[1,42]. In this study the mortality rate was only 1.3%. There is a high possibility of underestimation of the mortality rate in our study since patients who died even before endoscopy, are not taken into consideration. The fact that an arbitrary cut of period (10 days) was chosen as the endpoint to record the outcome, would have also contributed to the low estimate, since delayed deaths (>10 days) due to rebleeding are not accounted for.

ROCKALL SCORE

The mean Rockall score of the entire population was 2.63± 1.72. The study by Marco Soncini et al had a mean Rockall score of 4.6±2.2[37] and that by Robert A Enns had a mean score of 4.8±1.9[49].In our study, the low risk group formed the greater part of the population and the high risk group constituted just 2.6%. This is because moribund patients

(potentially "high risk") who either died or were taken against medical advice before endoscopy, were not included in the study population. On the same lines, the fact that most endoscopies were done the morning after the presentation and not on an emergency basis could have contributed to the same effect.

Table 20: Comparison of Rockall Scores among studies

	Present Study	**Robert A Enns' study**	**Morco Soncini et al study**
Mean Rockall score	2.63±1.72	4.8±1.9	4.6±2.2
Low risk	57.9%	13%	17.8%
Intermediate risk	39.5%	53%	48.7%
High risk	2.6%	34%	33.5%

RE-BLEED

Using the Chi square test, it was found that the Rockall score had a positive correlation with the event of rebleed. The correlation was highly significant with a p value of 0.000001. Just 6.8% of the patients with a low score rebleed. Rockall score can be used to reliably predict

the event of rebleed. The occurrence of rebleeding in our study is considerably higher compared to the 5.3% reported by Marco Soncini[37]. In our study qualifying greater than 4 days of melaena as 'continuing bleed/rebleed' could have resulted in a possible overestimate of the actual rebleed. The exact duration of melaena that a single episode of UGIH would cause, is not defined and depends largely on the amount of bleed. "Fresh melaena" taken in a few studies[41] as an event of rebleed is a loosely defined term and hence was not considered.

DURATION OF HOSPITALISATION

The Rockall score was correlated with the duration of hospitalisation and there was a highly significant correlation (p value 0.000001). The average duration of hospitalisation was 6.01 days. The average duration of stay of patients in the high risk group was 8 days as compared to 4.6 days of the low risk group. The mean duration of hospitalisation in similar studies is compared below.

Table 21: Duration of Hospitalisation with respect to Rockall scores among studies

Mean duration of hospitalisation	**Our Study**	**Marco soncini[37]**	**Robert A Enns[49]**
Total	6.01 d	6.06d	5.47d
Low risk	4.6d	5.1d	3.6d

Intermediate risk	7.16d	5.9d	5.6d
High risk	8d	7.2d	7.2d

TRANSFUSION REQUIREMENTS

Sixty two percent of the patients in this study required transfusion as compared to 54.1% reported by Marco Soncini's study[37]. The no of transfusions was also a reflection of the calculated Rockall score. The correlation was significant with a p value of 0.0001. The Rockall score also reflected the urea value of the patients (p value0.000). Urea values are good indicators of the patients intravascular volume provided intrinsic renal failure is ruled out. BUN/creatinine ratio are indicators of the patients volume status[50] and probably are better indicators of the patients intravascular volume status than the patient's heart rate which can be influenced by the patients anxiety levels and haematemesis is indeed a terrifying event to the patient. The usage of the patients' blood pressure is also not without reservations since the patients hypertensive status is not always known and hence an arbitrary level of <100mmHg to account for shock may actually underestimate the number of patients in hypovolemia. This is even more relevant in the Indian setting where the proportion of 'undetected hypertensives' is probably higher[51] compared to developed countries. Incorporating the patient's BUN/creatinine or fractional excretion of Na (FE_{Na}) as a measure of the

volume status instead of the heart rate and blood pressure into a prognostic scoring system could probably solve these issues. Further studies are needed to see if incorporating these changes could increase the accuracy of such scoring systems.

Tabulating the endoscopic diagnosis against the average Rockall score, showed that the value was highest in patients with Gastric carcinoma, followed by those with PUD, Mallory-Weiss tear and mucosal erosions, in that order. Since Rockall score is a prognostic indicator reflecting the chance of rebleed, outcome and duration of hospitalisation, it follows that the prognosis of the patients follows the same order. As expected, among the patients with Peptic Ulcer Disease the ones with Duodenal ulcer had a higher Rockall score than patients with Gastric Ulcer.

ROCKALL SCORE AND OUTCOME

There were three different outcomes in the patients under the study. The average Rockall score of the ones that were discharged after improvement was 2.47 and for the ones which were referred for surgical

management the score was 3.87. The single patient who died had the highest Rockall score of the three groups.

Based on the above results and observations, this study thereby concludes that *Rockall score* serves as a useful prognostic indicator in patients with non-variceal upper gastrointestinal bleeding. It correlates well with the *rebleed*, *transfusion requirements, duration of hospitalisation,* and *their final outcome*. Hence, stratification according to this scoring system may aid in the better monitoring and managements of patients with Non-variceal Upper Gastrointestinal Haemorrhage.

CONCLUSION

1. **Peptic Ulcer Disease** is the *most common* cause of Nonvariceal Upper Gastrointestinal Haemorrhage.

2. Though among patients with peptic ulcer disease the commonest lesion found was gastric ulcer, duodenal ulcers had a greater chance of re-bleed and having a protracted course.

3. Increasing age was associated with increased occurrence of re-bleed and an increased duration of hospitalisation.

4. Non variceal bleeding was more common in males.

5. NSAID intake and alcohol are preventable predisposing factors for Non-variceal Upper Gastrointestinal Haemorrhage.

6. **Rockall score** is useful in predicting the prognosis of the patients with NVUGIH. It correlates well with the *re-bleed*, *duration of hospitalisation*, *transfusion requirements* and *outcome*.

SUGGESTIONS

1. The age criteria in Rockall score could be scaled down to suit the life expectancy of the Indian population.

2. Including the FE_{Na} or BUN/creatinine as a measure of the volume status in a prognostic system might improve its accuracy.

3. Stricter laws preventing the indiscriminate dispensing of drugs 'over-the-counter' might decrease the incidence of drug induced GI bleeding.

4. Increasing the Health awareness on the ill effects of alcohol might reduce the incidence of alcohol related causes of UGIH.

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ABBREVIATIONS AND ACRONYMS

UGIH : Upper Gastrointestinal Haemorrhage

NVUGIH	:	Non-Variceal Upper Gastrointestinal Haemorrhage
NSAID	:	Non Steroidal Anti-inflammatory Drugs
PUD	:	Peptic Ulcer Disease
MW	:	Mallory Weiss
GI	:	Gastro Intestinal
SRH	:	Stigmata of Recent Haemorrhage
CA	:	Carcinoma of the stomach
ME	:	Mucosal Erosions
GU	:	Gastric Ulcer
DU	:	Duodenal Ulcer
OU	:	Oesophageal Ulcer

NON-VARICEAL UGI BLEEDING -PROFORMA

Name**:** Age**:** Sex**:**

Occupation**:**

I.P.No: Unit & Ward No.: Address:
Ph:

DOA:

DOE: Days of Hospital Stay:

DOD:

Diagnosis – Clinical :
Endoscopic :

Clinical Presentation:

Haematemesis	
Melaena	
Haematochezia	
Retching	
Abdominal pain	
Giddiness / LOC	
Palpitation	
Other systemic bleed	

Drug history:

Anticoagulants	
Antiplatelet	
NSAIDs	

Personal history:

Alcohol	
Smoking	

Past history:

Previous UGI Bleed	
IHD	
CCF	
CKD	

DCLD	
Known UGI malignanacy	

Clinical Findings**:**

Pallor	
Tachycardia [HR>100]	
Systolic Hypotension [SBP<	
Altered sensorium	
P/A tenderness	
P.R - Blood / melaena	
Blood in NGT aspirate	

Laboratory data**:**

Hb	
Haematocrit	
Platelet count	

Bilirubin	
SGOT	
SGPT	
Total protein	
Albumin	

Urea	
Creatiniine	

No of units of Blood transfused**:**

Oesophago-gastro-duodenoscopy**:**

Rebleed during hospital stay**:**

Outcome at discharge**:**

Improved	
AMA	
Death & Cause	

Status 10 days post presentation**:**

Rockall Score:

68

INSTITUTIONAL ETHICAL COMMITTEE
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K.Dis.No.16328 P & D3/Ethies/Dean/GGH/08 Dated: 8/9/2008

Title of the work : "Evaluation of patients with non-variceal upper GI Bleed and the application of Rockall index with respect to the outcome"

Principal Investigator :

Department : Dr. Raja Yogesh K
General Medicine, MMC, Ch.3.

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 10th Sep 2008 at 2 P.M in GGH [illegible], Deans, Chamber, Chennai-3.

The members of the Committee, the Secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The principal investigator and their term are directed to adhere the guidelines given below:

1. You should get detailed informed consent from the patients/participants and maintain confidentiality.
2. You should carry out the work without detrimental to regular activities as well as without extra expenditure to the Institution or Government.
3. You should inform the IEC in case of any change of study procedure, site and investigation or guide.
4. You should not deviate form the area of the work for which I applied for ethical clearance.
5. You should inform the IEC immediately, in case of any adverse events or serious adverse reactions.
6. You should abide to the rules and regulations of the institution(s)
7. You should complete the work within the specific period and if any extension of time is required, you should apply for permission again and do the work.
8. You should submit the summary of the work to the ethical committee on completion of the work.
9. You should not claim funds from the Institution while doing the work or on completion.
10. You should understand that the members of IEC have the right to monitor the work with prior intimation.

SECRETARY
IEC, GGH,CHENNAI

CHAIRMAN
IEC, GGH,CHENNAI

DEAN
GGH &MMC, CHENNAI

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