DISSERTATION

ON

A STUDY ON UPPER GASTROINTESTINAL ENDOSCOPIC FINDINGS IN PATIENT ADMITTED WITH UGI BLEED

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CERTIFICATE

This is to certify that dissertation entitled '**A STUDY ON UPPER GASTROINTESTINAL ENDOSCOPIC FINDINGS IN PATIENT ADMITTED WITH UGI BLEED'** submitted by **Dr. K SIVAKUMAR** to the faculty of General Medicine, The Tamil Nadu Dr. M.G.R. Medical University, Chennai in the partial fulfillment of the requirement of M.D Degree - Branch I (General Medicine) is a bonafide research work carried out by him under my direct supervision and guidance.

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CONTENTS

INTRODUCTION

The extensive clinical spectrum of gastro intestinal bleeding may encompass many different scenario.

The reason for this diversity is bleeding can occur from multiple different lesions and many sites in the gastro intestinal tract.

Gastro intestinal bleeding is a common clinical problem requiring more than 300,000 hospitalization annuals.

Upper Gastrointestinal bleeding, which most commonly arises from mucosal erosive diseases, account for upto 20,000 deaths annually.

The overall incidence of acute upper gastrointestinal hemorrhage has been estimated to be 50-100 per 1,00,000 person per year, with an annual hospitalization rate of approximately 100 per 1, 00,000 hospital admission.

Bleeding from upper gastrointestinal tract is approximately five times more common than bleeding from lower gastrointestinal tract. Bleeding may be massive or trivial, obvious or hidden.Gastrointestinal bleeding occurs clinically in one or more of the following four ways 1. Hemet emesis (from the Upper IT) 2. Hematochezia (from the lower GIT) 3. Occult (unknown to the patient) 4. Obscure (from an unknown site in the GIT).

The most important in the management of gastrointestinal bleeding is to determine 1. Source of bleeding 2. Stop active bleeding 3. Treat underlying abnormality 4. Prevent recurrent bleeding.

Historically, the most common cause of upper GI bleeding has been gastro duodenal ulcer disease, although other upper gastro intestinal tract mucosal lesion account for a substantial proportion of cases.

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AIM OF THE STUDY

1. To find out the prevalence of nature of lesion on Upper Gastro Endoscopy in patients admitted for UGI bleed.

2. To find out the prevalence of nature of lesion in patients with minor, moderate, major bleed.

REVIEW OF LITERATURE

UPPER GASTRO INTESTINAL BLEED

3.1 DEFINITION[: 1]

Vomiting of blood almost always proximal to the ligament of Treitz.

Manifestations:

Hemetemesis : Vomiting of blood, may be fresh and bright red color or old, with the appearance of coffee grounds.

Melena : Passage of black, tarry, and foul-smelling stool results from degradation of blood to hematin or other hemochromes by bacteria.

3.2 Symptoms :

Hemetemesis and Melena

Postural giddiness

Palpitation

Sweating

Signs:

BP: Resting hypotension and postural hypotension and tachycardia

Signs of liver disease: Jaundice,spider angiomata, palmar erythema,dupytren's contracture.

Signs of portal hypertension: Spleenomegaly , ascites and caput medusae
Acanthosis nigricans (underlying malignancy)

Cutaneous telengiectasias of skin, mucous membranes.
Abdominal mass and tenderness.

3.3 Historical Features That Help Assess the Etiology of Gastrointestinal Bleeding[1]

- Age
- Prior gastrointestinal bleeding
- Previous gastrointestinal disease
- Previous gastrointestinal surgery
- Underlying medical disorder (especially liver disease)
- Use of nonsteroidal anti – inflammatory drugs, including aspirin
- Use of anticoagulation and / or anti – platelet therapy
- Abdominal pain
- Change in bowel habits
- Weight loss
- Anorexia
- History of orophyrangeal disease.

3.5 ADVERSE PROGNOSTIC VARIABLE IN PATIENTS WITH ACUTE UPPER GASTROINTESTINAL BLEEDING[2,3]

Increasing age

- Increasing number of comorbid conditions (especially renal failure, liver failure, heart failure, cardiovascular disease, disseminated malignancy).

- Variceal bleeding (as compared with non variceal bleeding)
- Shock or hypotension on presentation
- Red blood in the emesis or stool
- Increasing number of units of blood transfused
- Active bleeding at the time of endoscopy
- Bleeding from a large (> 2.0cm) ulcer
- Bleeding from a visible or spurting vessel
- Onset of bleeding in the hospital
- Need for emergency surgery.

3.6 CAUSES OF ACUTE UPPER GASTROINTESTINAL BLEEDING[1]

Common

- Gastric ulcer
- Duodenal ulcer
- Esophageal varices
- Mallory – Weiss tear

Less Common

- Gastric erosions / gastropathy
- Esophagitis
- Cameron lesions / Dieulafoy lesion
- Telangiectasias
- Portal hypertensive gastropathy / gastric varices
- Gastric antral vascular ectasia (watermelon stomach)
- Neoplasm

Rare

- Esophageal ulcer
- Erosive duodenitis
- Hemobilia
- Pancreatic disease / crohn's disease
- Aortoenteric fistula

3.6 CAUSES:

3.6.1 Esophagitis[1]:

Common cause of upper gastrointestinal bleeding, occurring in nearly 15% of patients who undergo endoscopy bleeding.

Severe bleeding is less frequent. It causes occult blood loss more commonly than it causes acute bleeding

Obvious bleeding is most likely in patients with extensive erosive disease.

3.6.2 Mallory-Weiss Tear[4]:

Lacerations in the region of gastro esophageal junction that typically occur in the gastric mucosa. 10 to 20 % can occur in the esophageal mucosa.

Account for 5 to 10 % of cases of upper gastrointestinal hemorrhage. History of retching is obtained only 29% of patients.

Bleeding stops spontaneously in 80-90% of patients.

3.6.3Duodenal and Gastric ulcers[1, 7]:

Ulcer disease is the most common specifically identified cause of acute upper gastrointestinal hemorrhage. Peptic ulcer has been reported to be responsible for nearly 50% of cases of upper gastrointestinal bleeding.

The incidence rate of bleeding from duodenal ulcer is approximately twice that of gastric ulcers. An ulcer bleeds when it erodes into the lateral wall of a blood 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 because of rich vascular supply in these areas.

3.7 Predisposing factors to ulcer bleeding[7]:

- Gastric acid
- Helicobacter pylori infection
- Underlying medical and clinical factors
    - Cardiovascular & Cerebrovascular disease
    - Chronic Pulmonary Disease
    - Cirrhosis
- Drugs like NSAID, glucocorticoids, bisphosphonates alendronate, anticoagulants.
- Ethanol
- Prolonged hospitalization

3.7.1 Gastric acid[1,7] :

Zollinger-Ellison syndrome – hyper secretory disorder in which ulcers develop with high frequency. The ability of antacid therapy alone to heal upper gastro duodenal tract ulceration also supports the role of acid.

The best evidence of a role for acid in upper gastrointestinal hemorrhage comes from reduction of the risk of bleeding and rebleeding by a proton pump inhibitor.

3.7.2 Helicobacter pylori infection[5,74]:

The link between H.pylori infection and peptic ulceration is firm. Some studies suggest that H.pylori infection increases the risk of ulcer bleeding. The risk of Ulcer bleeding in NSAID users infected with H.pylori was nearly twice that of uninfected NSAID users. Eradication of H.pylori in patients who are starting long-term NSAID treatment has been shown to reduce the risk of an ulcer.

3.7.3 Aspirin and other Non steroidal Anti-inflammatory drugs[5,6,74]:

The most important predisposing factors for ulcer bleeding. The mechanism of injury involve reduced production of cyclo oxygenase generated cytoprotective prostaglandins. The risk of bleeding also is increased because of platelet dysfunction.

The risk of gastro intestinal bleeding caused by NSAIDs appears to be dose related. The risk of gastric ulceration is increased to a greater extent than that of duodenal ulceration.

The risk of bleeding varies with the individual NSAID; for example, the relative risk of bleeding is greatest with azapropazone and piroxicam and less with ibuprofen.

The risk of bleeding is dose dependent. Multiple cofactors contribute to the risk of ulcer bleeding associated with NSAIDs. Age >75 yrs, history of heart disease, gastrointestinal bleeding were independent predictors of NSAID-induced complications

Glucocorticoids, alendronate, ethanol appear to potentiate the ulcerogenic effect of NSAIDs and may predispose to upper gastrointestinal bleeding. Cyclooxygenase 2 inhibitors also are associated with an increased risk of ulcer bleeding but to a lesser degree than NSAIDs.

3.7.4 Ethanol[6]:

Patient who ingest ethanol chronically may have alcohol induced liver disease and secondary portal hypertension. Ethanol can induce gastric mucosal injury and cause or potentiate ulcer bleeding.

The relative risk of acute upper gastrointestinal bleeding rose with increasing amounts of alcohol consumed. The relative risk of acute upper gastrointestinal bleeding caused by aspirin was raised at all levels of alcohol consumption.

3.8.1 Endoscopic stigmata of active or recent bleeding[7,8,9]

1. Active arterial spurting
2. Oozing of blood
3. Visible vessel
4. Fresh blood
5. Blood clot

3.9.1 Prognostic Endoscopic characteristics of the ulcer[2,3]:

Large ulcer size >1 cm is associated with increased rates of rebreeding & mortality.

Endoscopic hemostasis is successful less often for ulcers larger than 2 cm than for smaller ulcers.

3.9.2 Ulcer base may have[1,7]:

1. Clean base

2. A base with a flat, pigmented spot

3. A base with a visible vessel

4. A base with an adherent clot

5. A base that contains a visible vessel or an adherent clot that is actively oozing or an adherent clot that is actively oozing or spurting.

3.10 Other causes:

3.10.1 Gastric Erosions[10]:

Gastropathy most often erosive has been reported to be the cause of bleeding in 16% of patients with upper gastrointestinal bleeding.

The bleeding is rarely hemodynamically significant unless the patient has an underlying coagulopathy

Sub epithelial gastric erosions develop in the following clinical situations[1, 10]

1. After ingestion of NSAIDs

2. Stress related mucosal disease

3. With consumption of ethanol

Stress related gastric mucosal disease occurs in[1, 10]:

1. Extensive trauma
2. Severe burns
3. Major surgery
4. Serious medical illness
    - respiratory failure
    - sepsis
    - renal failure
5. Major neurologic trauma or intra cranial disease

Ethanol[6]:

Ingestion of ethanol causes gastric erosions and gastrointestinal bleeding the term hemorrhagic gastritis is applied to the subepithelial hemorrhages seen at endoscopy in alcoholic patients.

Alcohol consumption is a risk factor for upper gastrointestinal hemorrhage only in persons with excessive ethanol consumption (4 or more drinks per day)

3.10.2 Duodenitis[1]:

Rare cause of acute bleeding Risk factors for severe erosive duodenitis include NSAID, H.pylori infection and anti-coagulation therapy.

The bleeding is usually self limited.

3.10.3 Malignancy[11]:

Neoplasms of esophagus, stomach, upper small intestine cause acute upper gastrointestinal hemorrhage infrequently.

Often associated with occult, asymptomatic bleeding usually self limited.

The most common tumor is advanced gastric adenocarcinoma.

3.10.4 Vascular lesions:

Dieulafoy lesion[12,69]: Also termed exulceration simplex Dieulafoy.

Abnormally large arteriole that retains the large caliber of its feeding vessel as it approaches the mucosa.

The large vessel compress the mucosa and cause a small erosion, with rupture of the vessel into the lumen.

They account for 6% of cases of upper gastrointestinal hemorrhage.

Dieulofoy lesions typically are found in the proximal portion of the stomach, usually within 6 cm of the gastroesophageal junction.

They may be located anywhere in the gastro intestinal tract. Bleeding is often massive and recurrent.

The lesion is difficult to identify unless it is actively bleeding.

Endoscopic ultrasonography may be useful for detecting a Dieulafoy lesion in a patient with unexplained upper gastrointestinal bleeding.

VASCULAR ECTASIA[12]:

Vascular lesions are uncommon but important causes of upper gastrointestinal tract bleeding.

The most common are vascular ectasias, most often are 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

- Renal failure
- Cirrhosis
- Scleroderma
- CREST syndrome (Calcinosis, Raynauds phenomenon, Esophageal dysmotility, Sclerodactyly, Telangiectasis)
- Radiation injury
- Pseudoxanthoma elasticum

- Ehlers-Danlos syndrome
- Von Willebrands disease

Vascular ectasia most often appear to be associated with chronic renal failure.

It was more often the cause of bleeding in patients with renal insufficiency than in those with normal renal function.

ARTERIO VENOUS MALFORMATION[12]:

True arteriovenous malformations appear as raised or nodular lesions at endoscopy They are rare. Congenital in origin and usually involve the submucosa.

They may be large and involve any portion of the gut.

HEREDITARY HEMORRHAGIC TELANGIECTASIA[13]:

Osler-Weber-Rendu disease is an autosomal dominant disorder.

Characterised by telangiectasias of skin, mucous membranes of gastrointestinal tract.

The peak incidence of bleeding is in the 6^{th} decade of life.

Bleeding can originate from any site in the gastrointestinal tract.

Epistaxis is the most common manifestations of hereditary hemorrhagic Telangiectasia and typically occurs before the 2nd decade. 80% of patients have a family history of bleeding.

Lack of telangiectasias on the lips, oral and nasopharyngeal membranes, tongue and periungual areas cast doubt on the diagnosis.

HEMANGIOMA:

Most commonly identified in the upper small intestine.

These benign vascular tumors are made up of proliferating vessels and appear as single or multiple red, purple or blue nodular lesions.

Blue rubber bleb nevus syndrome is characterized by cavernous hemangiomas of the skin, gastrointestinal tract and other viscera.

GASTRIC VASCULAR ECTASIA[14]:

Rarely causes acute gastro intestinal hemorrhage. Characterised by aggregates of ecstatic vessels that appear as red spots on the gastri mucosa. The aggregates are arranged in a linear pattern in the antrum of the stomach , the term gastric antral vascular ectasia(GAVE) or Watermelon stomach.

The red spots may be more diffuse and involve the proximal stomach, the term diffuse gastric vascular ectasia is used.

Most common in middle-aged and elderly women with associated achlorhydria,

Atrophic gastritis and cirrhosis.

This lesion is difficult to differentiate from portal hypertensive gastropathy.

Its pathogenesis is unknown.

3.11 TREATMENT OF PEPTIC ULCER BLEEDING:

Goals of therapy:

1. Treat the peptic ulcer & thus bleeding
2. To stop active bleeding
3. To prevent rebleeding

Pharmacologic Therapy:

Agents to treat active ulcer bleeding are

- Octreotide
- Stomastatin
- Vasopressin
- Secretin
- Histamine H2 receptor antagonists
- Proton pump inhibitors
- Anti fibrinolytics
- Prostaglandins

The greatest risk of rebleeding from an ulcer is within the first 72 hours after the Initial bleeding episode.

The acidic pH retards blood clotting and enhances clot dissolution by proteolytic Enzymes like pepsin.

Elevating intragastric pH may facilitate platelet aggregation, further supporting a role for acid lowering therapy.

Although H2 receptor antagonists are widely available, non toxic and inexpensive the available data donot support their use in patients with ulcer bleeding.

Proton pump inhibitors have significantly better acid reducing characteristics, Particularly at high doses, and effective in preventing ulcer bleeding in high risk Patients.

The use of nitrovasodilator(Glyceryl trinitrate, isosorbide dinitrate, isosorbide Mononitrate or transdermally administered nitroglycerin) was associated with a significantly decreased risk of upper gastrointestinal tract bleeding.

3.11 Assessment and resuscitation measures to protect the airway and maintain adequate tissue perfusion take priority over all endoscopic procedures. Ideally the patients should be haemodynamically stable with a heart rate of less than 100 beats/ min and systolic blood pressure greater than 100mm Hg. Initially evaluation should focus on determining whether the bleed is from the upper or the lower gastrointestinal source. A clear nasogastric aspirate may be seen in 14% of bleeding duodenal ulcers (DU). [39] Blood transfusions should be instituted in patients with postural symptoms and haemoglobin less than 10g / dl or patients with haemoglobin of less than 7 – 8 g / dl even without postural symptoms.

Recommendations of care for severe non – variceal UGI bleed. [40]	
1	Protect airway. Intubate for active bleeding or altered mentation
2	Medical resuscitation with fluids and blood products
3	Correct coagulopathies (goal : PT<15 sec, platelets > 50,000/cumm)
4	Lavage with an orogastric tube if blood obscures much stomach
5	Use therapeutic double – channel videoendoscope
6	Have therapeutic instrumentation ready before endoscopy
7	Have a trained nurse assistant available.

3.11.2

Rockall risk scoring system [41,43]				
	Score			
Variable	0	1	2	3
Age (years)	<60yrs	60 – 79yrs	> 80 yrs	
Shock	No shock	Tachycardia	Hypotension	
Systolic BP (mm Hg)	>100	>100	<100	
Pulse rate	<100	>100	>100	
Co – morbidity	Nil		Cardicfailure IHD, Other Major co-morbidity	Renal failure, Liver failure, Disseminated Malignancy
Diagnosis	Mallory – Weiss tear	All other diagnosis	Malignancy of upper GIT	
	Without SRH, no lesion			
Stigma of recent bleed (SRH)	None of dark spots		Blood in UGIT. Adherent clot , visible or spurting vessel	

3.11.3 PREDICTORS OF RE – BLEED, MORBIDITY AND MORTALITY[3,4,5]

The risk of in – hospital mortality is dependent on age, presence of shock, co – morbid condition, stigmata of recent bleed on endoscopy and the underlying diagnosis.

Multivariable Scoring System which have been validated for use in gastrointestinal bleed include the APACHE scoring system.[41] those proposed by Zimmerman et al [42] and Rockall et al [43].

Endoscopy provides important risk assessment for rebleed. Rockall Risk Score stratifies the risk of death and re – bleed, with a risk of rebleeding of 5% if the score is 0 and 40% if score is more than or equal 8. Mortality rate is below 1% and as high as 41% if the score is 0 -2 and 8 or more, respectively.

3.11.4

Modified Forrest Criteria [46]:

1. Actively bleeding ulcer.

   1a. Spurting

   1b. Oozing.

2. Non – actively bleeding ulcer

   2. a. Non – bleeding visible vessel

   2. b. Ulcer with surface clot.

   2. c. Ulcer with red or dark blue spots.

3. Ulcer with clean base.

Presence of shock and type 1 bleeding peptic ulcer carried a rebleed risk of 80% and presence of non – bleeding visible vessel predisposes the patient to a 50% risk of further bleed. Ulcer with a clean base (type 3) and ulcers with red or dark blue spots (type 2c) rarely re – bleed during hospitalization. Bleeding peptic ulcer in the posterior duodenal bulb and proximal gastric lesser curve are located near major vessels and are associated with higher re – bleed rates and are more likely to cause death [43,44].

3.11.5 Predictors of Mortality

Patients characteristic that have been reported to be associated with increase in mortality are[42,44]:

1. Age
2. onset of bleeding
3. co – morbidity
4. hypotension and shock at presentation
5. Fresh bleed in Ryle's tube aspirate
6. Haemoglobin level.at presentation and on serial follow – up
7. Number of packed cells transfusions
8. Corticosteroids
9. Combined use of aspirin and oral anticoagulants.

Patients who start bleeding during hospitalization (secondary bleeding) have a significantly higher mortality as compared to those who bleed prior to hospitalization (primary bleeding). [47] This is primarily because of presence of co – morbidity factors in hospitalized patients. Mortality is significantly higher in patients with comorbid illness which include CNS diseases, hepatic insufficiency, pulmonary diseases, cardiac diseases, renal failure, physiological stress and cancer. The mortality increases with increase of co – morbid conditions.[43,47]

Use of low dose aspirin is associated with moderately reduced risk of severe bleeding and a decreased mortality from GI bleeding. [48,49]. Possible explanation is the increased patient awareness and vigilance and propensity to bleed from minor lesions with aspirin therapy.

3.11.6 Predictors of Rebleeding[50]

As many as 10% patients rebleed after endoscopic therapy.

1. Failure of therapy and recurrent upper GI bleeding is associated with an increase in mortality. Apart from the
2. Endoscopic stigmata of recent ulcer bleed, many independent factors predict the rebleeding risk. These include
3. Age more than 65 yrs.
4. Tachycardia and shock at admission,
5. Obesity, haematemesis,
6. Specific ulcer location
7. Diameter more than 2 cm.

3.11.7 Helicobacter pylori and recurrence of ulcer bleed[54,56]

H. pylori eradication in patients with bleeding ulcers is known to reduce the recurrence of bleeding. In various small studies with a follow up period ranging from 4 to 48 months, the rate of duodenal ulcer relapse and rebleeding was significantly reduced in patients with successful eradication of H. pylori .

In patients with bleeding peptic ulcer associated with H. pylori, eradication should be confirmed by urea breath test or the biopsy urease test at endoscopy.

3.12 ENDOSCOPIC THERAPY

Peptic ulcer disease accounts for about two third of all cases of upper gastro intestinal haemorrhage as diagnosed on upper GI endoscopy. [57]. Forrest type 3 (clean base) ulcers on endoscopy are seen in approximately 30 – 50% of peptic ulcer bleeds. These along with type 2c ulcers don't require any further endoscopic therapy.

For ulcers with active bleed or evidence of stigmata of recent bleed, the endoscopic modalities available are listed below.

3.12.1 Endoscopic Therapies Used for Ulcer Bleeding

Injection:

- Epinephrine
- Saline
- Water
- Ehanol
- Sclerosants
- Fibrin glue
- Thrombin

Thermal methods

- Bipolar or multipolar electrocoagulation
- Heater probe
- Laser photocoagulation
- Monopolar electrocoagulation

Hemoclips

Argon plasma coagulation

Band ligation

Endoloops

Endoscopy has beyond doubt reduced morbidity, hospital stay, risk of recurrent bleeding and need for surgery in patients with non – variceal upper GI bleed. Early endoscopy (within 24 hours) in an intensive care setting in patients of NVUGIB significantly effects the transfusion requirement, duration of ICU stay and rebleed rate.[60].

3.12.2

Endoscopic stigmata of ulcer haemorrhage, their prevalence, risk of rebleeding and effect of endoscopic therapy on rebleed rates is listed out below.

Outcome of Endoscopic Therapy for Bleeding Peptic Ulcers According to Their Endoscopic Appearance. 1

Appearance	Frequency (%)	Rebleeding Rate (%)		Surgery Rate (%)		Mortality Rate (%)	
		No ET	ET	No ET	ET	No ET	ET
Active bleeding	18	55	20	35	7	11	<5
Visible vessel	17	43	15	34	6	11	<5
Adherent clot	15	22	5	10	2	7	<3
Flat spot	15	10	<1	6	<1	3	<1
Clean ulcer base	35	<5	NA	0.5	NA	2	NA

]

3.12.3 Haemoclips versus hypertonic saline – epinephrine injection.

Injection therapy with Epinephrine at concentration of 1:10,000 or 1:20,000 using 23 to 25 – guage sclerotherapy needle is widely used. It causes local tamponade, vasoconstriction and enhances platelet aggregation to achieve local haemostasis. Epinephrine is injected into the submucosa in 0.5 – 1.0 ml increments in all four quadrants around the bleeding site. Endoscopic metallic clips were compared in an RCT to hypertonic saline – epinephrine injection versus the combination of both by Chung et al[61]. The authors achieved intial haemostatis in 95% of patients with active bleeding or visible vessel with a tendency to produce less rebleed rates and surgeries with the use of haemoclips. Haemoclips have their technical and practical limitations that include need for special training, special devices, possibility of clip tearing the vessel wall and inability to stop bleed in arteries greater than 1 – 2 mm size.

3.12.4 Ethanol injection versus Haemoclips

Injection of alcohol carries a high risk of ulceration and perforation, thus only small volume (maximum of 2 ml) is recommended. A retrospective study reported similar haemostasis rates with haemoclip application and ethanol injection group.[62]. Alcohol injection produces dehydration and fixation of tissue and subsequent necrosis and ulceration. Because of this risk of ulceration and perforation only small dose of alcohol is used.

Other sclerosants such as sodium tetradecyl sulphate, polidocanol and ethanolamine have been used alone or in combination with epinephrine for injection therapy of peptic ulcer bleed.

3.12.5 Heater probe versus Haemoclips

Thermocogulation when compared to haemoclips for visible or bleeding vessel in the ulcer base revealed a similar death rate and surgery rates [63]. The same study reported the superiority of haemoclips in prevention of rebleed (2% rebleed rate with haemoclip and 21% rebleeding in the heater probe group) and lower transfusion requirement and hospital stay. Haemoclip application failure in six out or 56 patients was attributed to the ulcer site (proximal posterior gastric wall or posterior wall of duodenal bulb) or size of ulcer greater than 15mm.

Butyl 1 – 2 cyanoacrylate versus hypertonic saline – epinephrine injection

Butyl 1 – 2 cyanoacrylate is a liquid glue that polymerizes on contact with blood. Lee et al[63] reported similar initial haemostasis rates with both butyl 1-2 cyanoacrylate and hypertonic saline – epinephrine injection therapy in actively bleeding peptic ulcers but the glue injection group had a significantly lower rebleeding rate (14% vs 42%). The rebleeding rates were similar in non-bleeding visible vessel ulcers in both groups. Glue embolization and perforation are the two feared complications with glue injection.

3.12.6 Fibrin Glue and hypertonic saline – epinephrine injection

Theoretically the injection of fibrin glue to halt peptic ulcer bleeding may be safer than other sclerosing agents as it would not cause necrosis or degeneration. However, results from a prospective randomized control trial conduced by Song et al comparing fibrin glue and hypertonic saline – epinephrine injection suggested no statisfically significant difference between the two modalities of therapy[64.]

3.12.7 Combined modalities

There is trend towards combined use of two endoscopic modalities using injection and mechanical or injection and thermal probe therapy in actively bleeding peptic ulcer [61,66]. Rebleed rates with combined therapy using adrenaline injection and thermocoagulation showed a trend towards decreased rebleeding compared to injection alone when dealing with spurting peptic ulcer haemorhage.[66]

3.13.DRUG THERAPY

Intravenous high dose omeprazole (80 mg bolus followed by 8 mg / hr), after endoscopic haemostatis has been achieved in a peptic ulcer bleed reduces the risk of rebleeding and need for endoscopic re – treatment and surgery.[67] Use of H2 receptor antagonists is not supported by clinical trials.

Oral omeprazole in the dose of 40mg twice a day has been reported to decrease the risk of rebleeding in patients with stigmata of recent bleed without endoscopic therapy by Khuroo et al.[68]

3.14 RECURRENT HAEMORHAGE

Rebleeding after successful endoscopic therapy range from 10 – 20%. In a prospective, randomized study, Lau et al compared endoscopic retreatment with surgery after initial endoscopy for recurrent ulcer bleed.[53] In patients ulcer and recurrent bleeding after initial endoscopic control of bleeding, endoscopic retreatment reduced the rate of surgery and complications. Second endoscopic procedure avoided surgery in 73% of rebleed patients. Multivariate analysis showed two predictors of rebleed; ulcer diameter more than 2cm and hypotension during rebleeding. Patients who rebleed after second endoscopy should be subjected to either surgery or embolization for haemostasis.

3.15 NON – ULCER NVUGIB

3.15.1 Dieulafoy's lesions

A Dieulafoys lesions is marked by the presence of large, submucosal artery that protrudes through the mucose, is not associated with peptic ulcer and cause massive GI bleeding. Endoscopic therapy with injection therapy, thermal probes, clipping devices and band ligation has been evaluated. Narayan et al reported a rebleeding rate of 50% with single modality therapy.[31]. A combined used of epinephrine injection and heater probe, as recommended for active bleeding or adherent clots, achieves an intial haemostatis in 90% and a rebleed rate of less than 20% of patients [2]. Tattooing with India Ink around the arterial spurter assists the surgeon in identifying the bleeding site at laparotomy.

Endoscopic band ligation for Dieulafoy's lesion in a case series was successfully used in 3 out of 4 patients [32]. One out of three successfully banded patients had rebleeding from the post banding ulcer site, which was managed successfully with epinephrine injection.

3.15.2 Mallory – Weiss Tears

Mallory – Weiss mucosal laceration are usually secondary to vomiting and result in self limiting, small volume bleed. Haemorrhage is usually mild and self – limiting. Jensel et al prospectively evaluated endoscopic therapy for actively bleeding Mallory – Weiss tears, without portal hypertension. [33]. They achieved a 100% haemostasis rate compared with 40% for medical therapy. Twenty percent of patients with visible non-bleeding vessel or adherent clot rebleed. They recommend the use of low power settings of light touch bipolar coagulation, or ephinephrine injection only in actively bleeding tears. Patients with portal hypertension and actively bleeding. Mallory – Weiss tears should be subjected to therapy for the adjacent varix and not bipolar coagulation.

Haemclipping has been successfully used in actively bleeding Mallory – Weiss tears or tears with a visible vessel or fresh adherent clot. In a series of 58 patients of whom 26 had high – risk signs on endoscopy 2.8+1.6 clips were deployed with 100% technical success and haemostasis. No rebleeding was noticed in any of the patients on a 2 months follow – up [34].

Endoscopy is the key diagnostic tool for management of upper gastrointestinal bleeding. In addition it provides a unique therapeutic opportunity which has over the years reduced the need for emergency surgery, but the impact on survival is less dramatic with the mortality from severe UGI bleeding remaining fairly constant. Optional use of endoscopic therapeutic modalities shall continue to play a pivotal role in management of UGI bleed in the years to come.

3.16 Portal Hypertension:

3.16.1 Definition: Elevation of the hepatic venous pressure gradient (HVPG) to > 5mmHg.

3.16.2 Classification:

Prehepatic

- Portal vein Thrombosis
- Splenic vein Thrombosis
- Massive Splenomegaly (Banti's syndrome)

Hepatic

Pre sinusoidal

- Schistosomiasis
- Congenital hepatic fibrosis

Sinusoidal

- Cirrhosis – many causes
- Alcoholic hepatitis

Post sinusoidal

- Hepatic sinusoidal obstruction (Veno occlusive disease)

Post hepatic

- Budd-Chiari Sydrome
- Inferior vena cava webs
- Cardiac causes
    - Restrictive Cardiomyopathy
    - Constructive pericarditis
    - Severe Congestive heart failure

3.16.3 Factors of risk of variceal bleeding:

- Severity of cirrhosis – CHILD'S class
- Height of wedged hepatic vein pressure
- Size of the varix
- Location of the varix
- Endoscopic stigmata like
    - Red wale signs
    - Hematocystic spots
    - Diffuse erythema
    - Bluish color
    - Cherry – red spots
    - White – nipple spots

- Tense Ascites

3.16.4 SITES OF COLLATERAL FORMATION:

Distal Oesophagus & Proximal Stomach : Gastro esophageal varices are the Major collaterals formed between the portal venous system and systemic venous system .

Umbilicus : Vestigeal umbilical vein communicates with the portal vein and gives rise to prominent collaterals around the umbilicus (Caput Medusae)

Retroperitoneum : Collaterals communicate between the ovarian vessels and iliac veins.

Rectum : Inferior mesenteric vein connects with pudendal vein and result in Rectal varices.

Measurement of Portal pressure [17]

- Hepatic Vein Pressure Gradient – The most commonly used indirect method
- Splenic Pulp Pressure
- Portal Vein Pressure
- Endoscopic variceal Pressure [18]

3.16.5 Detection of varices :

- Upper gastrointestinal endoscopy [19]
- Ultrasonography
- Computed Tomography
- Magnetic Resonance Imaging
- Endoscopic Ultrasonography

3.16.6 Clinical assessment of patients with portal hypertension – related bleeding:

History of hemetemesis and Melena

Peripheral stigmata of liver disease like

- Jaundice
- Spider telangiectasias
- Palmar erythema
- Dupuytren's Contracture
- Parotid enlargement
- Testicular atrophy
- Loss of secondary sexual characters
- Ascites

- Encephalopathy
- Features of portal hypertension like splenomegaly & ascites
- Caput Medusae around the umbilicus
- Venous hum in the epigastrium due to the collateral flow in the falciparum ligament.

3.16.7 Laboratory studies[:1]

Evidence of hepatic synthetic dysfunction

- Prolongation of Prothrombin time
- Hypoalbuminemia
- Hyperbilirubinemia
- Anemia

Feautures of Hypersplenism

- Anemia ,
- Thrombocytopenia,
- Leukopenia

Abdominal imaging :

- Reveal splenomegaly,
- collateral vessels,
- abnormal liver echotexture
- Contour and ascites.

3.16.8 Treatment of Portal Hypertension:

Pharmacologic Therapy: [1,15,20,16]

Drugs that decrease portal blood flow

- Non-Selective beta adrenergic blocking agents
- Vasopressin
- Somatostatin and in analogs

Drugs that decrease intrahepatic resistence

- Nitrates
- Alpha1 adrenergic blocking agents (Prozosin)
- Angiotensin receptor blocking agents

3.16.9 Endoscopic therapy:

Sclerotherapy [21]

Available evidence does not support emergency sclerotherapy as first line treatment of variceal bleeding.

Sclerosants used include Sodium tetradecyl sulfate, Sodium morrhuate, Ethanolamine oleate, Absolute alcohol.

Complications include

- Retrosternal discomfort
- Sclerosant induced esophageal ulcer related bleeding
- Stricture and perforation

Variceal ligation: [25]

Preferred endoscopic modality for control of acute bleeding and prevention of rebleeding.

The procedure involves suctioning of the varix into the channel of an endoscope and Deplaying a band around the varix. The band strangulates the varix causing thrombosis.

Varices in the mid or proximal esophagus not need to be banded. Complications include Esophageal ulcers, strictures, dysmotility.

TRANS JUGULAR INTRA HEPATIC PORTA SYSTEMIC STENT SHUNT:[22]

Reduces elevated portal pressure by creating a communication between the hepatic vein and intrahepatic branch of the portal vein.

TIPS function as a side to side portacaval shunt. The procedure involves cannulation of Hepatic vein through a trans jugular approach and using a Rosch needle, the portal vein is cannulated. A guide wire is then passed to connect the hepatic vein and a branch of the Portal vein. Following dilation of the tract, a stent is placed and dilated as required to reduce the portacaval pressure gradient to below 12 mmHg.[22]

The stents used are Wall stent, Palmaz stent and coated stent with Polytetrafluoro - ethylene.

The most common indications for placement of a TIPS is refractory variceal bleeding when pharmoco and endoscopic therapies have failed [24], especially in patients with CTP class B or C cirrhosis.

Surgery:

Non – shunt Procedures:

- Esophageal Transection
- Devascularization Procedures

Porto systemic shunts

- Selective shunts
- Partial porto systemic shunts

3.17.1 Esophageal varices:[23]

Present in approximately 40% of patients with cirrhosis and upto 60% in cirrhosis with Ascites. New varices will develop at a rate of 5% per year.

Progression of small varices to large varices occurs at a rate of 10 % to 15% per year and is related to the degree of hepatic dysfunction.

Upto 25% of newly diagnosed varices will bleed at 2 years. The best predictor of bleeding appears to be variceal size. The risk of bleeding in patients with varices less than 5mm in diameter is 7% at 2 years and it is 30% if size is more than 5mm.

Risk of bleeding is virtually absent when HVPG is < 12mmHg.

Approximately one half of patients with a variceal bleed stop spontaneously because hypotension leads to Splanchnic vasoconstriction which results in decrease in Portal pressure. Excessive transfusions increase the chance of rebleeding.

Failure to control bleeding at 5 days occurs in [24]

- ✓ Active bleeding at endoscopy
- ✓ Lower initial hematocrit level
- ✓ Higher serum aminotransferases level
- ✓ CTP class
- ✓ Bacterial infection
- ✓ HVPG >20mmHg
- ✓ Portal vein thrombosis

One third of patients will rebleed within the next 6 weeks; 40% will take place within 5 Days of the initial bleed.

Predictor of rebleeding: [24]

- Active bleeding at endoscopy
- Bleeding from gastric varices
- Renal insufficiency
- HVPG >20mmHg

3.17.2 DETECTION OF ESOPHAGEAL VARICES THROUGH UPPER GASTRO INTESTINAL ENDOSCOPY:

Endoscokpic grading of esophageal varices compiled by the Japanese Research Society Portal Hypertension.

The descriptors include:

- Red color signs,
- color of the varix,
- form of the varix,
- location of the varix.

Red color signs:

- Red wale markings – Longitukdinal whip-like marks on the varix.
- Chery red spots – 2 to 3 mm or less in diameter.
- Hematocykstic spots – Blood filled blisters 4 mm or greater in diameter.
- Diffuse redness.

Colour:

- White or blue.

Form of the varix:

Grade I : Small and straight

Grade II : Tortuous and occupying less than one third of the esophageal lumen.

Grade III : Large and occupying more than one third of the esophageal lumen .

Location :

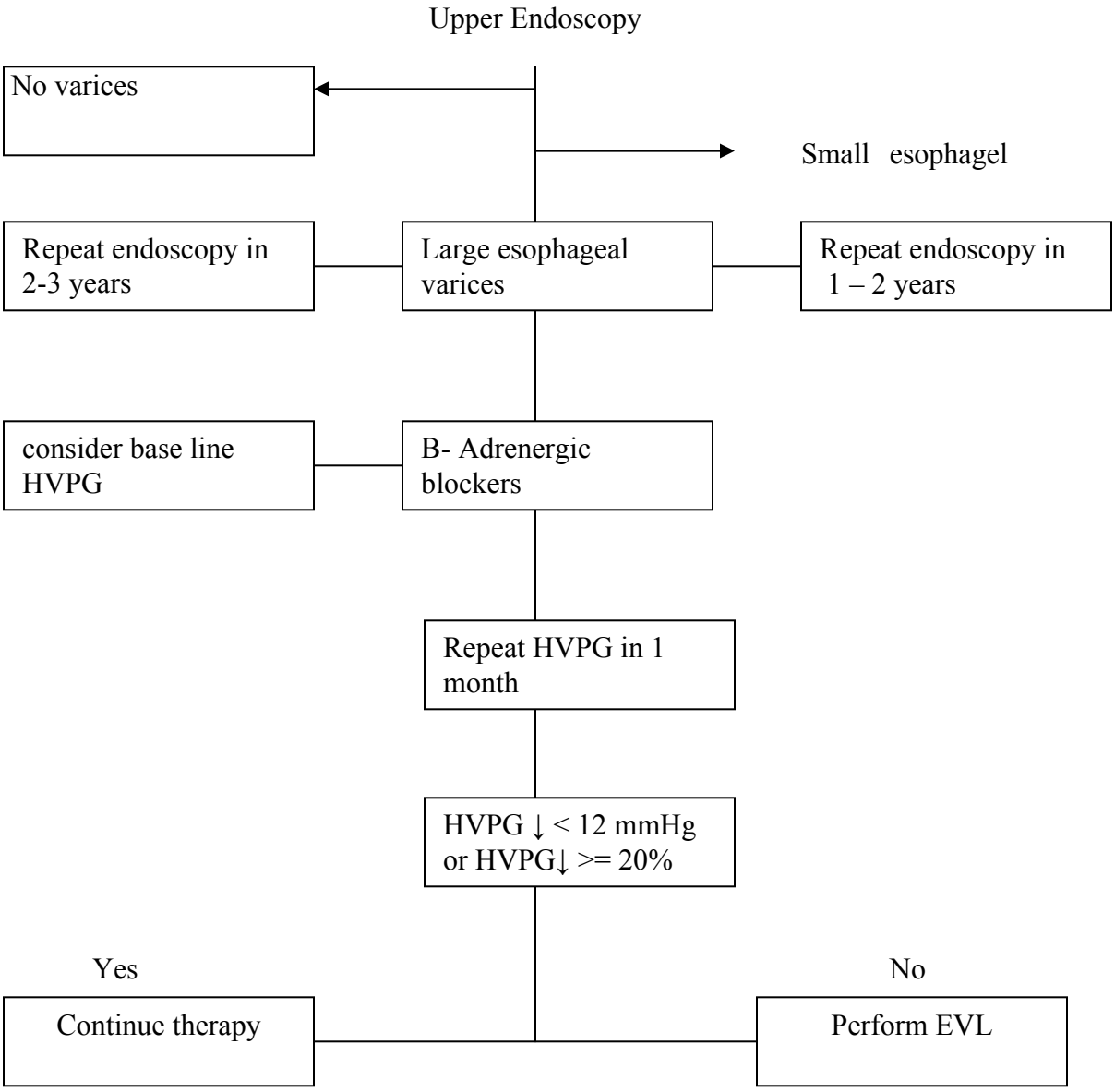
- Lower third, middle third, upper third of the esophagus

SIZE:

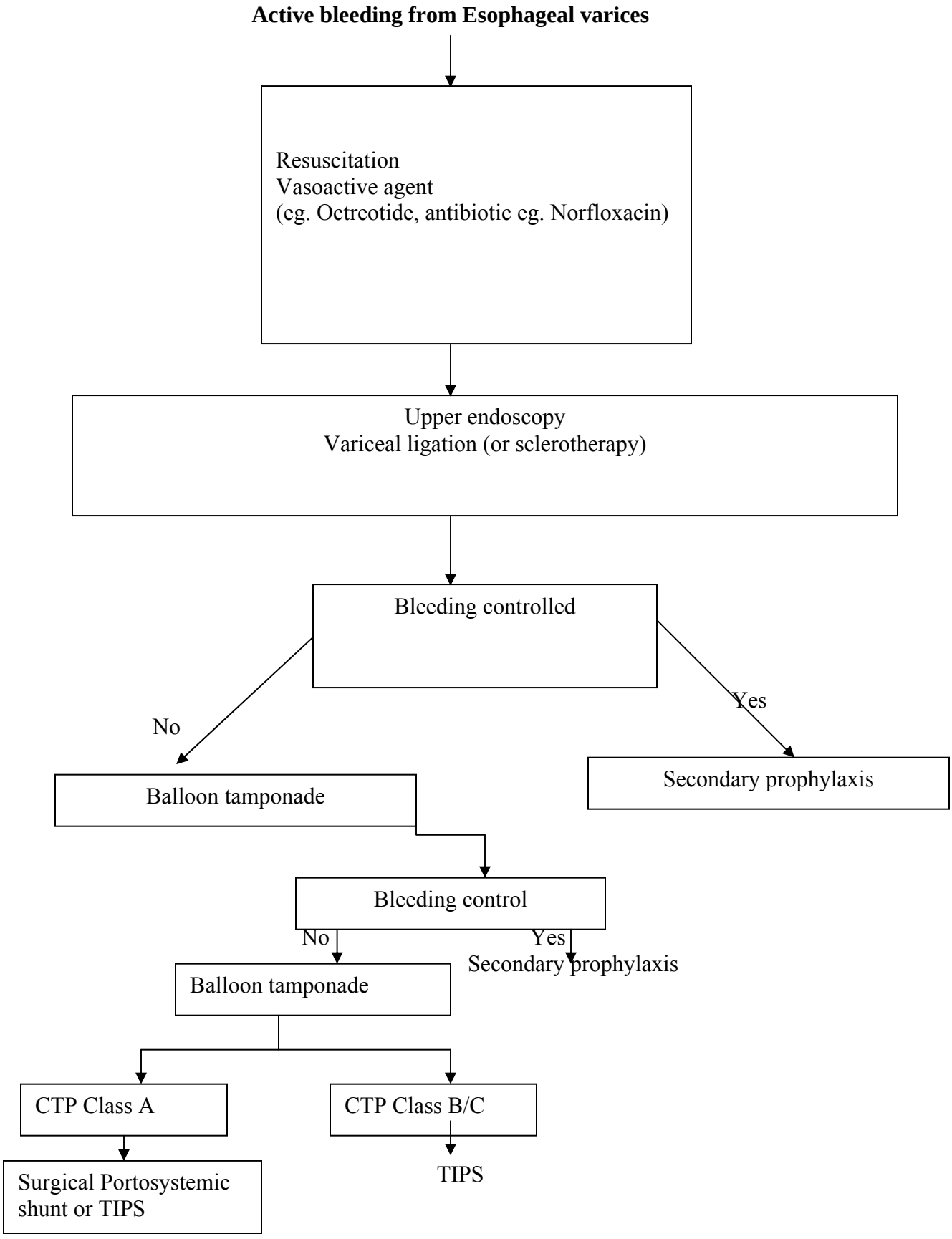
Size of the varices in the lower third of the esophagus is most important.

- Small varices – Occupying lesss than one third of the lumen <5mm in diameter.
- Large varices - >5mm in diameter.

3.17.3 Primary Prophylaxis to prevent first episode of variceal bleed[1]



3.17.4 Algorithm for the Management of acute variceal bleeding[1]



3.18.1 GASTRIC VARICES[27,28]

The most widely used classification of gastric varices is the sarin classification.

The type1 gastro oesophageal **GOV-I** extend 2 – 5 cm below the gastro oesophageal junction and are in continuity with oesophageal varices

Type 2 gastro oesophageal varices **(GOV2)** are in the fundus of the stomach and in continuity with oesophageal varices

IGV1: Varices that occur in the fundus of the stomach in the absence of oesophageal varices.

IGV2 – Varices that occur in the gastric body antrum, or pylorus

GOV1 comprises approximately 70% of all gastric varices.

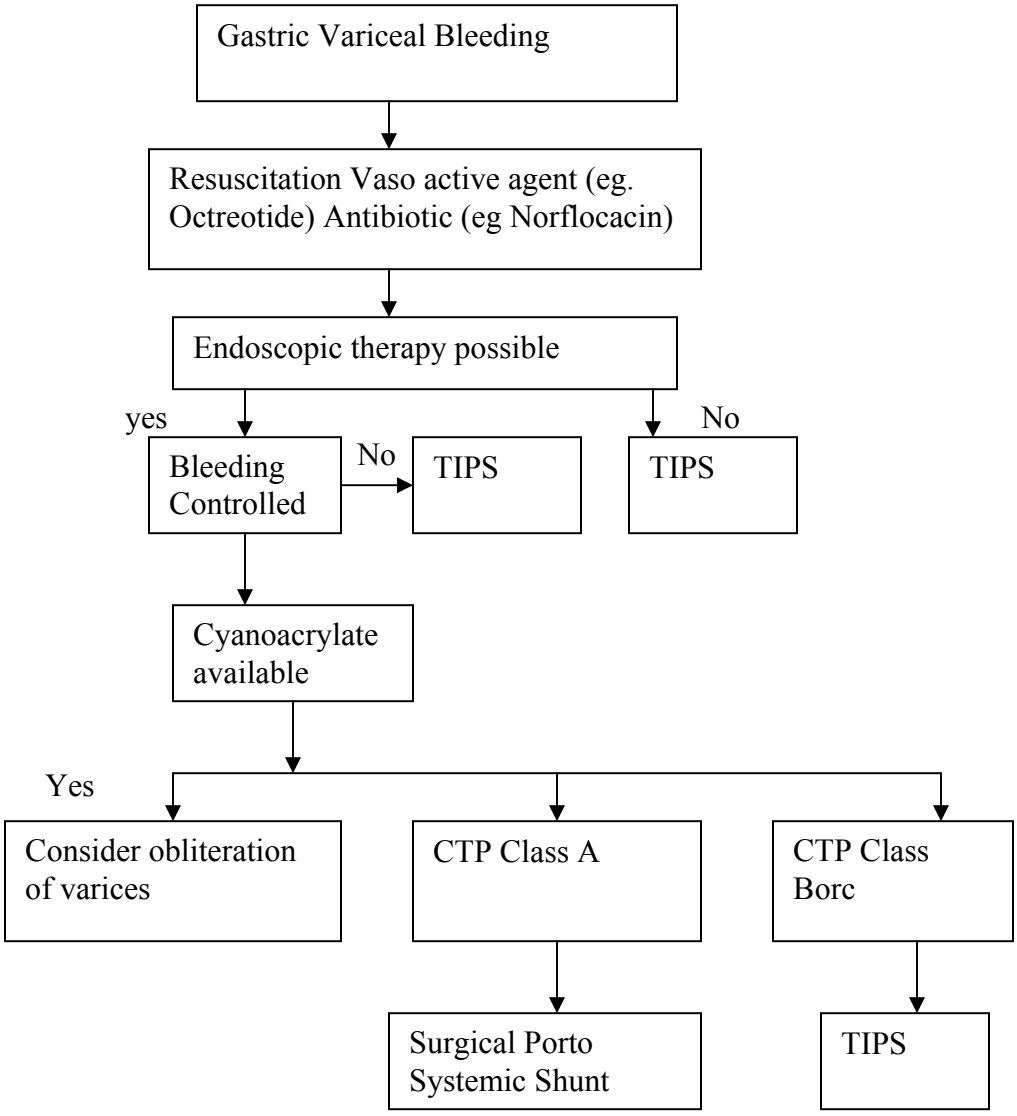
Approximately 25% of patients with portal hypertension have gastric varices, most commonly GOV1

Intrahepatic causes of portal hypertension maybe associated with both GOV1 and GOV2. Splenic vein thrombosis usually results in IGV1, but the most common cause of fundal gastric varices may be cirrhosis.

Bleeding is more common in patients with GOV2 and IGV1,then in those with other types of gastric varices.
Gastric Varices tend to the larger in diameter than oesophagheal varices.

Bleeding from gastric varices has been described with an HVPG less than 12 mmHg.

3.18.2 ALGORITHM FOR THE MANAGEMENT OF GASTRIC VARICEAL BLEEDING



4.1Materials and methods

Place of study	:	Department of Internal Medicine , Thanjavur Medical college hospital , Thanjavur
Type of study	:	Prospective study
Period of study	:	Dec 2007 to Nov 2008
Ethical Committee approval	:	The present study was approved by the Ethical Committee
Collaborating Department	:	Department of Medical Gastro Enterology
Consent	:	Informed consent was obtained from the Participants

4.2 Selection of patients

Hundred patients satisfying the following inclusion criteria and not having any of the exclusion criteria were taken up for the study.

4.2.1Inclusion criteria

(1) All adult patients of both sexes who were giving definite history of vomiting of frank blood or coffee ground coloured vomit and/ or passed dark coloured stools were chosen for this study.

(2) Inpatients admitted for other illnesses and who subsequently developed UGI bleeding following prescription with drugs like aspirin, other NSAIDS, steroids, anticoagulants and other gastro toxic drugs were also included

(3) Inpatients who developed UGI bleed after administration for severe medical illness like respiratory failure , sepsis.

4.2.2 Exclusion criteria

The following groups of patients were excluded from this study after detailed history taking, clinical examination and investigations because of the confounding factors which will interfere with the results.

(1) Patients with history of epistaxis and bleeding gums and subsequently developed spurious hemetemesis.

(2) Bleeding and clotting disorders

(3) Hematological disorders

(4) Critically ill patients those who could not be mobilized for UGI endoscopy

4.3 Study method

4.3.1 History

Patients characteristics like age and sex were noted. Detailed history regarding the UGI bleeding like number of times of hematemesis approximate quantity of blood vomited each time, associated with malena or presenting with malena alone were obtained. Symptoms of common diseases that can lead to UGI bleeding and detailed history of drug intake like aspirin, other NSAIDs , steroids and symptoms due to blood loss were recorded in the questionnaire.

Detailed history asked from the patients regarding the risk factors of UGI bleeding:

(1) Known peptic ulcer disease (diagnosed by a physician or a gastro enterologist)

(2) Alcoholism (those who are consuming alcohol at least 100ml/day Regularly for >3 months)

(3) Smoking (those patients who are smoking one or more beedies or cigarettes per day regularly for >3 months)

(4) Stress and serious systemic illnesses of the patients

(5) Intake of drugs that may cause UGI bleeding when taken like NSAIDs, steroids, bisphosphonates and chemotherapeutic agents were obtained.

4.3.2 Clinical examination

Routine general and systemic examination of the patients was carried out With the aim of

(1) Assessing the general condition of the patient

(2) Confirmation of UGI bleeding by Ryle's tube aspiration .

(3) Assesing severity of blood loss

(4) Assessing the common causes of gastro intestinal bleeding like Cirrhosis of liver with portal hypertension.

(5) Ruling out hematological disorders causing UGI bleed.

4.3.3 Laboratory investigations

Routine urine and blood investigations to find out hemoglobin status, blood Grouping and typing for transfusion, to find out renal failure, hepatic failure, bleeding and clotting disorders and hematological disorders were carried out.

4.3.4 Upper gastrointestinal endoscopy

Endoscopy was done for all the patients after overnight fasting, using PENTAX video endoscopic system , to directly visualize the mucosa of the esophagus, stomach and duodenum, like varices, ulcers , erosions.

The endoscopic stigmata of active or recent hemorrhage and endoscopic prognostic features like number of ulcers, site and location of ulcers, size of ulcers, bleeding or not healing or not, clean base of the ulcer or adherent blood clot, oozing of blood from the ulcer base and about visible blood vessel were studied.

The site, grading of varices were studied and search for rare causes for UGI bleed were made.

4.4 Study approach

Number of patients affected, were studied with respect to age group, approximate quantity of total blood loss, prevalence of endoscopic findings in UGI bleed.

RESULTS AND OBSERVATIONS

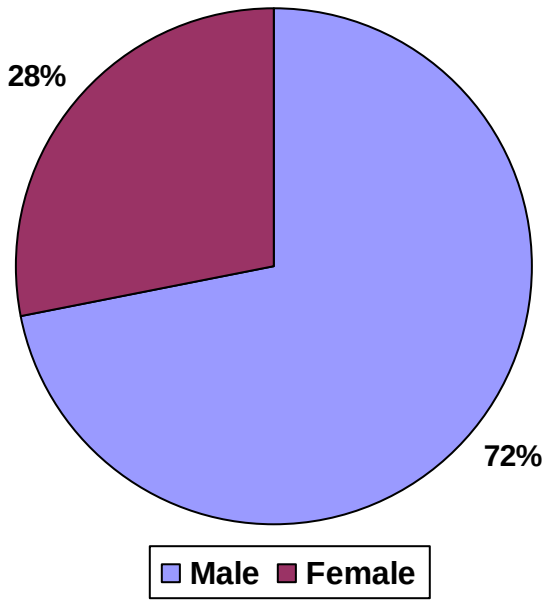
5.1. SEX DISTRIBUTION

In this Study of 100 cases of UGI Bleed the Sex distribution is observed as

Table-5.1

	Number of cases	%
MALE	72	72 %
FEMALE	28	28 %
TOTAL	100	100 %

Figure-5.1



In the study among 100 patients, 72 were males and 28 were females.

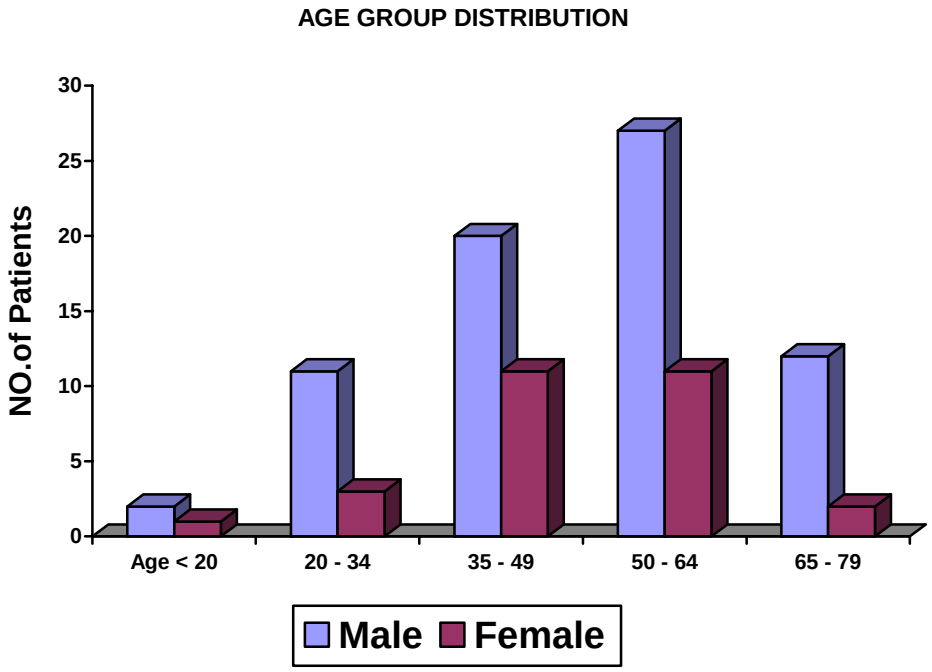
5.2. AGE DISTRIBUTION

In this Study of 100 cases of UGI Bleed the Age distribution in each Age Group is observed as

Table-5.2

S.No	Age Group	Male	Female	Total
1	Age<20	2	1	3
2	Age 20 -34	11	3	14
3	Age 35 – 49	20	11	31
4	Age 50 – 64	27	11	38
5	Age 65 - 79	12	2	14
Total		**72**	**28**	**100**

Figure-5.2



It was found that majority of patients (38%) were in the age group of 50- 64 years.

The highest age of patient participated in study was 75 years and lowest age of patient was 15 years.

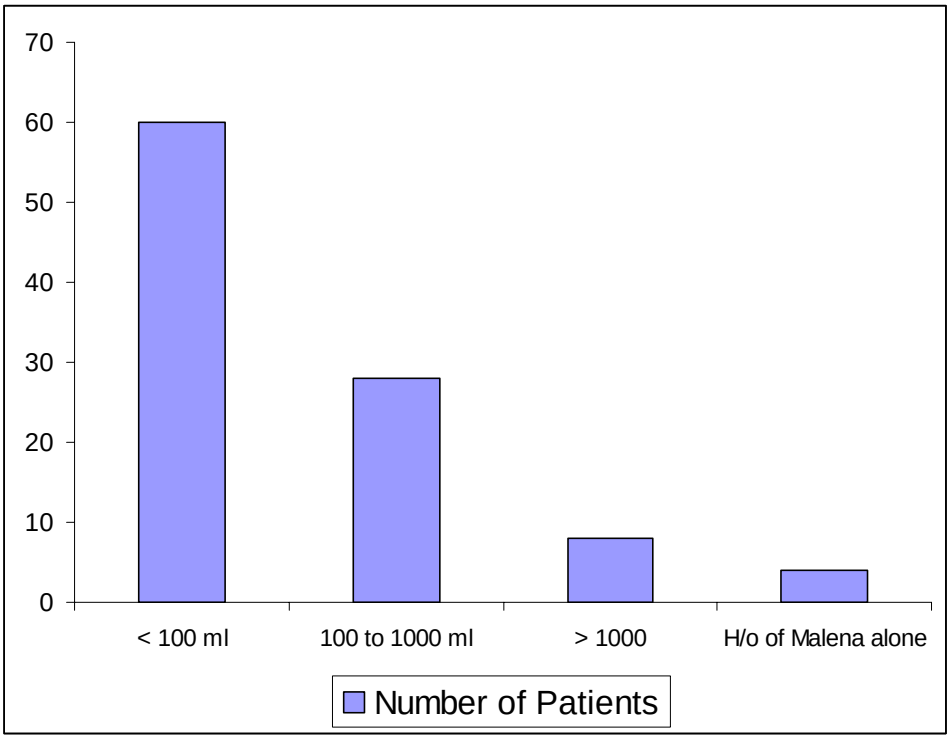
5.3. QUANTITY OF BLOOD LOSS

In this study UGI bleed the approximate quantity of blood loss is observed as

Table-5.3

S.No	Quantity of blood loss	Number of Patients
1.	< 100 ml	60
2.	100 to 1000 ml	28
3.	> 1000	8
4.	H/o of Malena alone	4

Figure 5.3



It was found that majority of patients 60 % were having minor UGI bleed with

< 100ml of blood less.

Only 8% of the patients had major UGI bleed and four patients had only malena.

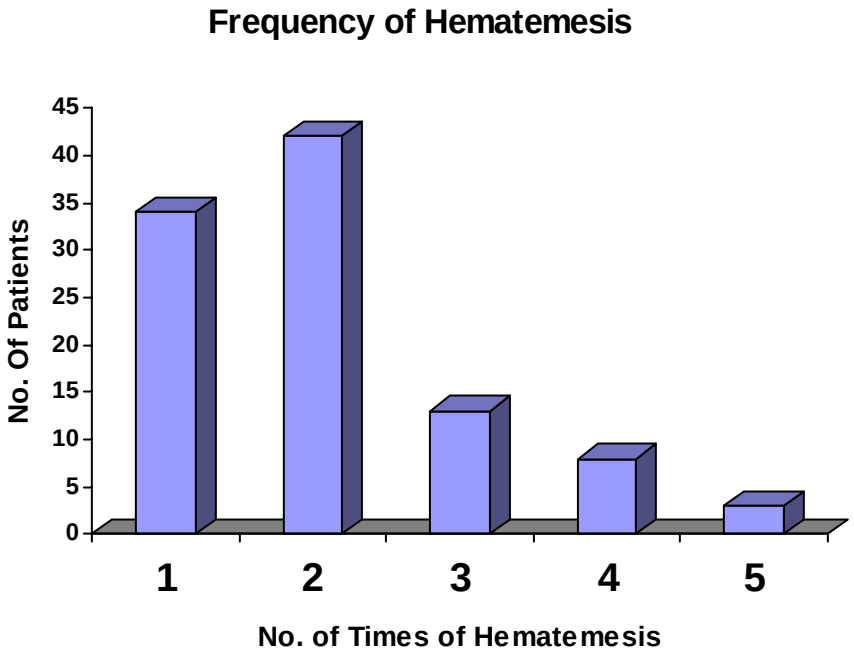
5.4 FREQUENCY OF HEMETEMESIS

In this Study of 100 cases of UGI Bleed the frequency of Hematemesis is observed as,

TABLE-5.4

S.No	Number of Bouts of Hematemesis	Number of Patients
1.	One	34
2.	Two	42
3.	Three	13
4.	Four	8
5.	Five	3

Figure 5.4



It was found that percentage of patients with two episodes of hematemsis was more with 42%.

The maximum frequency of hemetemesis was five.

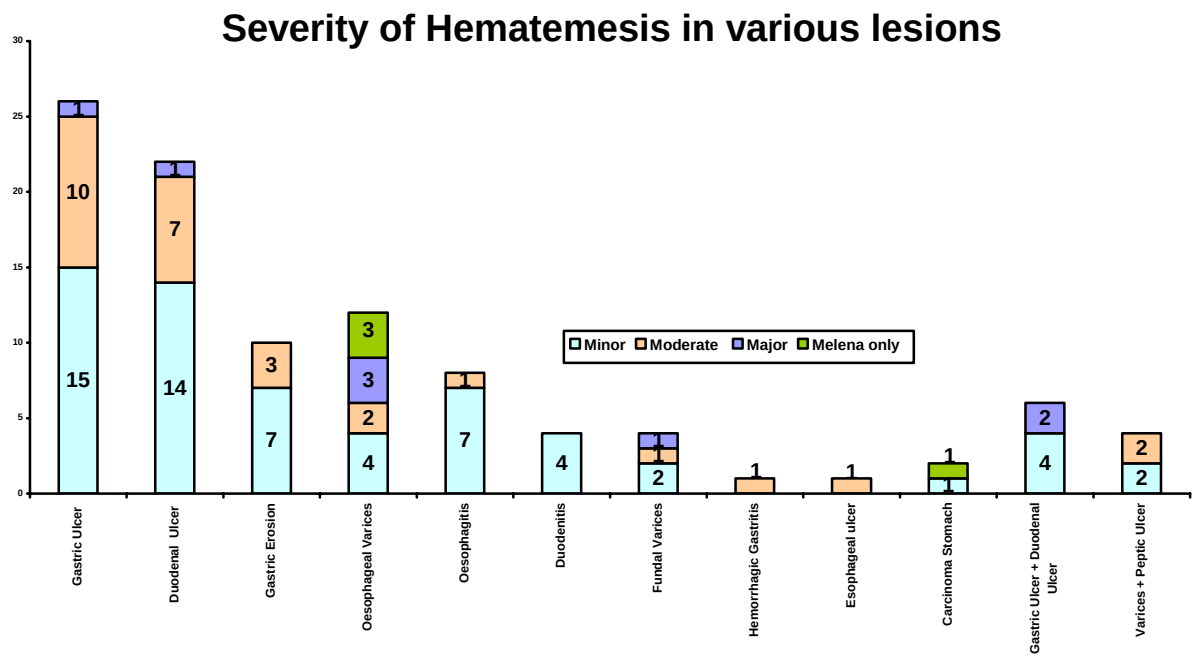
Single episode of hemetemesis was observed in 34 partients (34%)

5.5. SEVERITY OF LESION

In this Study in UGI bleed frequency of severity of clinical presentation is observed as **Table-5.4**

Sl.No	Nature of Lesion	Minor	Moderate	Major	Melena only
1	**Gastric Ulcer**	15	10	1	0
2	**Duodenal Ulcer**	14	7	1	0
3	**Gastric Erosion**	7	3	0	0
4	**Oesophageal Varices**	4	2	3	3
5	**Oesophagitis**	7	1	0	0
6	**Duodenitis**	4	0	0	0
7	**Fundal Varices**	2	1	1	0
8	**Hemorrhagic Gastritis**	0	1	0	0
9	**Esophageal ulcer**	0	1	0	0
10	**Carcinoma Stomach**	1	0	0	1
11	**Gastric Ulcer + Duodenal Ulcer**	4	0	2	0
12	**Varices + Peptic Ulcer**	2	2	0	0
	Total	**60**	**28**	**8**	**4**

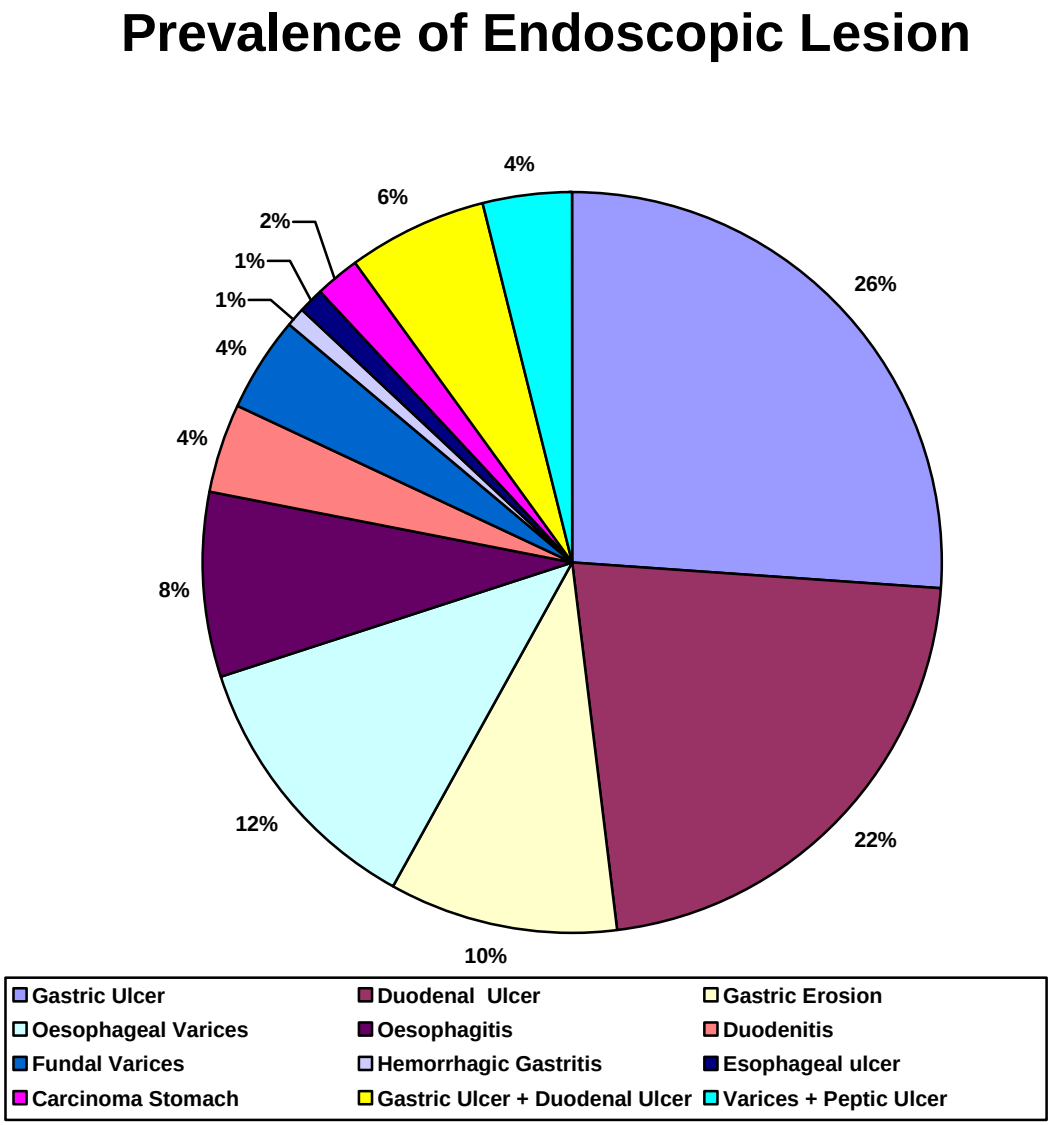
Figure-5.5



It was observed that 60% of lesions were presented as minor UGI belled and 28% as moderate UGI bleed.
Only 8% of lesions presented with major UGI bleed

Varices (50%) were the most common lesion found in major UGI bleed.

Figure-5.6


Prevalence of Endoscopic Lesion
4%
6%
2%
1%
1%
4%
4%
8%
12%
10%
22%
26%
Gastric Ulcer
Duodenal Ulcer
Gastric Erosion
Oesophageal Varices
Oesophagitis
Duodenitis
Fundal Varices
Hemorrhagic Gastritis
Esophageal ulcer
Carcinoma Stomach
Gastric Ulcer + Duodenal Ulcer
Varices + Peptic Ulcer

5.6. Prevalence of Endoscopic Lesion

Table- 5.5

In this study of UGI bleed the prevalence of nature of lesions on endoscopy is observed as

Sl.No	Nature of Lesion	< 20		20-34		35-49		50-64		65-79		Total %
		M	F	M	F	M	F	M	F	M	F	
1	**Gastric Ulcer Alone**	1	-	4	1	3	2	8	3	4	-	26%
2	**Duodenal Ulcer Alone**	-	1	3	1	4	3	5	2	3	-	22%
3	**Gastric Erosion**	-	-	1	-	2	1	3	1	1	1	10%
4	**Oesophageal Varices**	1	-	1	1	3	1	3	1	1	-	12%
5	**Oesophagitis**	-	-	1	-	2	1	1	1	2	1	8%
6	**Duodenitis**	-	-	-	-	1	-	2	1	-	-	4%
7	**Fundal Varices**	-	-	-	-	1	1	1	-	1	-	4%
8	**Hemorrhagic Gastritis**	-	-	-	-	-	-	1	-	-	-	1%
9	**Esophageal ulcer**	-	-	-	-	1	-	-	-	-	-	1%
10	**Carcinoma Stomach**	-	-	-	-	1	-	1	-	-	-	2%
11	**Gastric Ulcer + Duodenal Ulcer**	-	-	1	-	1	1	1	2	-	-	6%
12	**Varices + Peptic Ulcer**	-	-	-	-	1	1	1	-	-	1	4%
Total												**100%**

In this study among 100 patients , 26 patients (26%) had gastric ulcer alone, 22 patients (22%)had duodenal ulcer alone and 6 patients had both

gastric ulcer and duodenal ulcers (6%), collectively comprising most common cause of UGI bleed contributing 54% of the total .

It was found that Esophageal varices were the second most common lesion contributing 12% of UGI bleeding.

4 cases(4%) of fundal varices were noted

The other acid peptic disorder lesions observed were gastric erosion 10%, Esophagitis 8% Duodenitis 4%,Hemorrhagic gastritis 1% Esophageal ulcer 1%

Peptic ulcer were noted in 4 patients (4%) of varices.

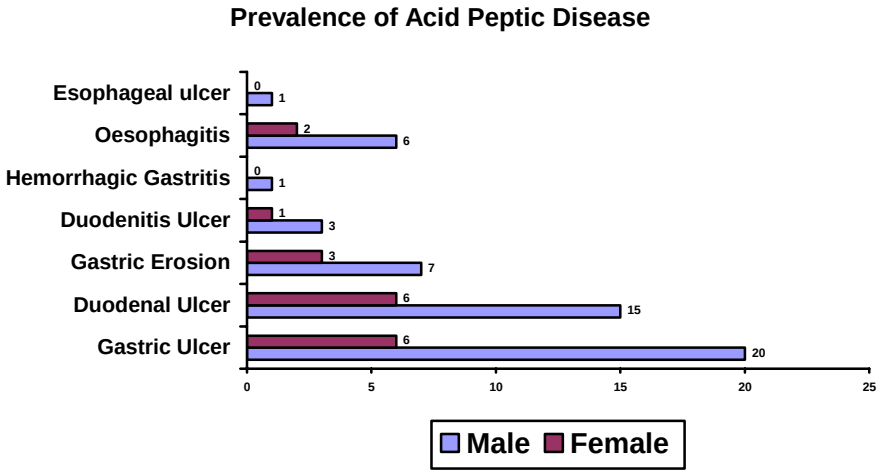
Multiple lesions were observed in 10% of cases

5.7. Prevalence of Acid Peptic Lesion

In this Study of UGI Bleed the prevalence of acid peptic lesion is observed as Table-5.6

Sl.No	Acid peptic disease	Male	Female	Total	%
1	**Gastric Ulcer Alone**	20	6	26	37%
2	**Duodenal Ulcer Alone**	15	6	22	31%
3	**Gastric Erosion**	7	3	10	14%
4	**Duodenitis**	3	1	4	5%
5	**Hemorrhagic Gastritis**	1	0	1	1%
6	**Oesophagitis**	6	2	8	11%
7	**Esophageal ulcer**	1	0	1	1%
			Total	72	

Figure-5.7



It was found that gastric ulcer was present in 26% patients (37%) among 72 patients of with single acid peptic disease lesion only.

Duodenal ulcer 31% was the second common lesion gastric erosion was seen in 14% of patients with acid peptic lesion.

Four cases were due to duodenitis

DISCUSSION

6.1. Sex

Out of one hundred patients studied, seventy two were male patients and twenty eight were female. In a Scandinavian study, it was found that incidence of UGI bleeding was twice as high among men as among women (oliver et al., 1997) .

6.2. Age

The percentage of number of patients in the age group of equal to or above 50 yrs of age was 52 % comprising more than ½ of all the patients. In this study it was found that elderly patients were bleed in a high incidence because the frequency of bleeding is directly related to the duration of the disease.

The increased incidence of UGI bleed in elderly individuals were also due to frequent prescription of NSAIDs and aspirin for their cardiac problems and the relative risk was 2.0 times higher than the others.

In the early study by Griffin et al., (1991) the relative risk for elderly patients with age group > 60 years was 3.8 times than the others.

6.3. Severity of hemetemesis

Percentage of patients with one or two episodes of hematemesis was 76%. 60% of the patients admitted for UGI bleed were having minor UGI bleed (<100 ml). Only 8 % of the patients had severe UGI bleeding (1000ml) in the present study and majority of the patients 50% were found to have oesophageal varices and fundal varices on endoscopy.

In this study among 8 cases of major UGI bleed , Esophageal varices (3 cases) and Fundal varices (1 case) contributes 50% of total number of major UGI bleed.

Rupture of varices is the most common cause of life threatening hemorrhage. Risk of bleeding is greatest when varices are large and when they are prominent in the gastric fundus. (Lebrec, 1980)[34].

6.4. Endoscopic findings

In the present study, patients had undergone delayed UGI endoscopy by three to five days . The prevalence of nature of lesions is as follows.

6.4.1. Peptic ulcer:

In this study, gastric ulcer alone 26 cases (26%), duodenal ulcers alone 22 cases (22%) and both lesions in a same patient 6% collectively remain the most common cause of UGI bleed with total of 54% .

In American Society for Gastrointestinal Endoscopy (Gilbert DA, Tedesco F J, et al[39]) Chronic gastric and duodenal ulcers collectively remain the most common cause of hematemesis and melena.

Rockall et al; GF Longstreth:[2,43]AMJ Gastroenterol 1995 reported peptic ulcer was the most common lesion on endoscopy in cases with UGI bleed.

The increased frequency of gastric ulcers bleed than duodenal ulcers is likely to be due to gastric ulcers have a slightly greater tendency to bleed than duodenal ulcers, 23.7% compared with 19.1%.

6.4.2. Oesophageal Varices:

In this study, both esophageal and fundal varices contributes 16% of cases of total UGI bleed.

Atkinson[33] compiled esophageal varices accounted for an overall 7.3% to 11.1% in his study.

In OMGE International upper GI Bleeding survey, 1978-86, Esophageal varices was the second most common cause of UGI bleed.

mVan Leerdam et al; AMJ gastro enterol 98:1494, 2003; quoted 7-20% cases of UGI bleed were due to varices.

6.4.3. Gastric erosion:

In this study, 10% of cases were due to gastric erosion.

DM Jensen et al: 2003[71], mentioned 2-7 % of UGI bleed were due to gastric erosion.

Erosive gastritis 7-22% were reported in Johnston SJ, Jones et al, Epidemiology and course of GIH, British Medical Journal 1973.

The Americal Society for gastrointestinal Endoscopy National Survey of UGI bleed (Gilbert DA, Tedesco FJ, et al), reported 23.4% cases of gastric erosions.

6.4.4. Esophagitis:

In this study, 8 % of cases were due to Esophagitis

Kc Thromopoulas et al[31]: European journal gastroenterol Hepatalol 16:2004 reported 1-13% cases of UGI bleed were due to Esophagitis.

The American Society for gastrointestinal endoscopy national survey (Gilbert et al[39]) reported 6.3 % cases of esophagitis as a cause for UGI bleed.

6.4.5. Carcinoma stomach:

In this study, 2% of cases of acute UGI bleed were due to Carcinoma Stomach.

In Jones FA Problems of alimentary bleeding 1970[37]; Gastric cancer accounted for 2.7% of cases of acute alimentary tract bleeding.

Alum et al described 4.1 cases of gastric carcinoma as a cause of acute GIT bleed.

Flme et al: Eur JGastroenterol 2005 reported 2-7% cases of gastric carcinoma.

6.4.6. Varices and Peptic Ulcer:

In this study, 4% UGI bleed had both varices and peptic ulcer findings on Endoscopy.

In this study, varices and peptic ulcer (4 cases) contributes 20% of total number of varices cases(20).

Dagradi AE, Mehler R, Tan et al, AMJ 1970[35]; mentioned other sources of bleeding in esophageal varices patients include gastric and duodenal ulcers in upto 20% of cases.

6.4.6. Duodenitis:

In our study, 4 cases of UGI bleed were found to have Duodenitis on Endoscopy. As evident as friable and punctuate erosion of a slightly nodular mucosa, mainly in the duodenal bulb, can be a source of blood loss. I In ASGE[39] Survey duodenitis was observed in 5.8% cases.

6.4.7. Other causes:

Other causes of UGI bleed that occurs in less frequency in this study includes one case of Esophageal ulcer, Hemorrhagic gastritis.

CONCLUSION

The study on endoscopic findings in upper gastro intestinal bleed concludes that

(1) The peptic ulcer disease was the most common lesion found on endoscopy with prevalence of 54%.

(2) Varices contributes second common lesion, next to peptic ulcer disease in UGI bleed with prevalence of 16%.

(3) Minor UGI bleed was the commonest presentation. majority of lesions (60%) presented with minor UGI bleed 28% lesions presented as moderate UGI bleed. Only 8% presented as major UGI bleed.

(4) Varices account for the most common cause for major UGI bleed contributing 50%.

(5) Gastric ulcer was commonest lesions accounting for 37 cases (37%) among 72 cases having single acid peptic lesions on endoscopy. The second most common is duodenal ulcer (31%).

(6) Multiple lesions were found in 10% of cases. Peptic ulcer lesions were found in 20% of total number of varices cases.

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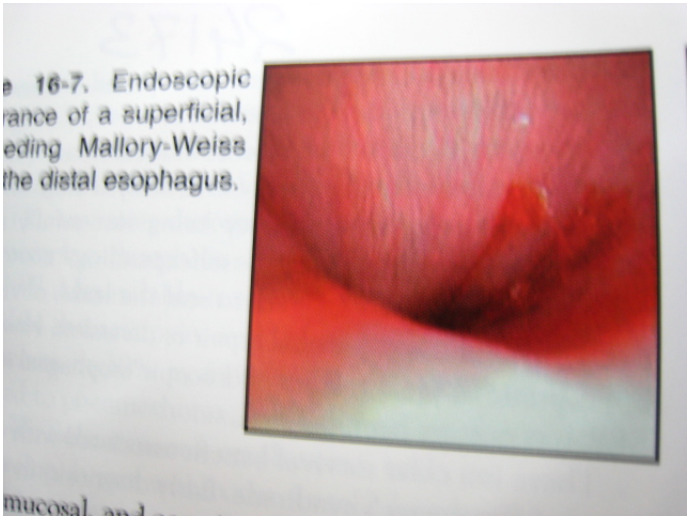
LIST OF ABBREVIATIONS

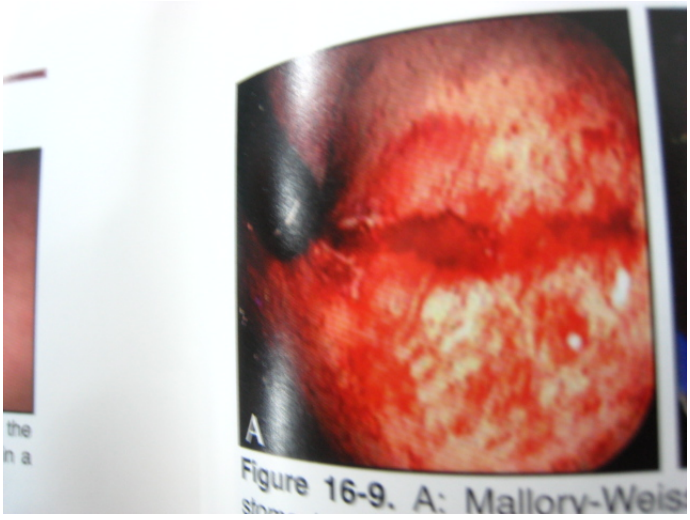
1)	UGI	-	Upper Gastro Intestinal
2)	NSAID	-	Non Steroidal Anti-Inflammatory Drugs
3)	H.pylori	-	Helicobacter Pylori
4)	NVUGIB	-	Non Variceal Upper Gastro Instestinal Bleeding
5)	HVPG	-	Hepatic Venous Pressure Gradient
6)	CTP	-	Child Turcotte Purg
7)	EVL	-	Endo Variceal Ligation

3.4

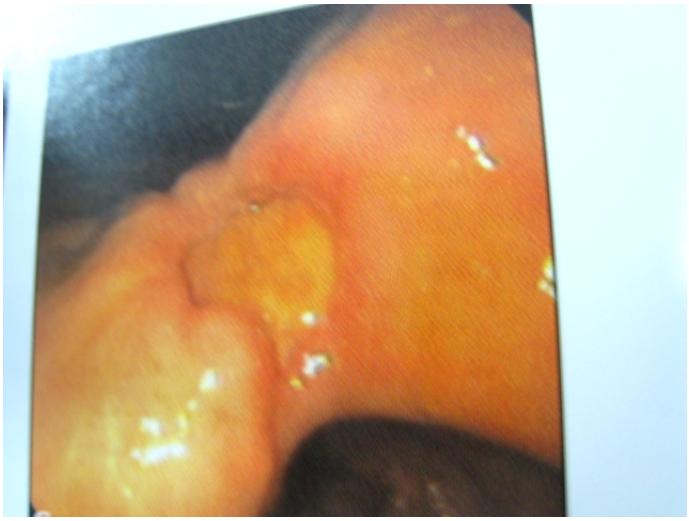
Patients Homodynamic Status, Degree of Blood Loss, and Severity of Gastrointestinal Bleedings. 1		
Patients Homodynamic Status (Vital Signs)	Blood Loss (%of Intravascular Volume)	Severity of Bleed
Shock (resting hypotension) Postural (orthostatic hypotension and Tachycardia) Normal	20 – 25 10 – 20 < 10	Massive Moderate Minor

Mallory weiss Tear

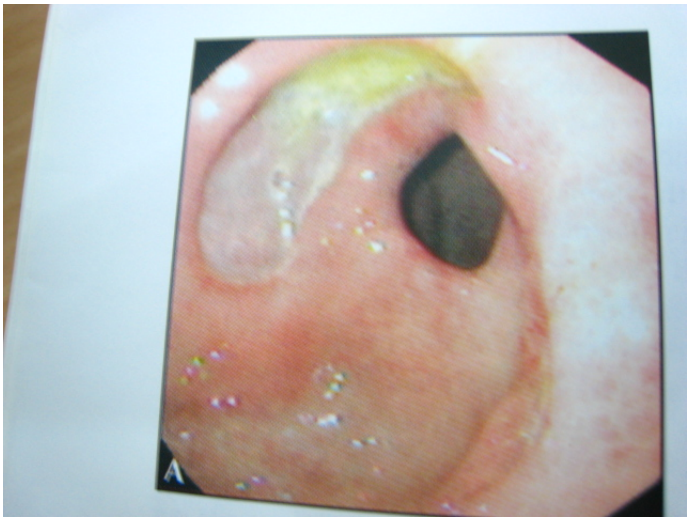




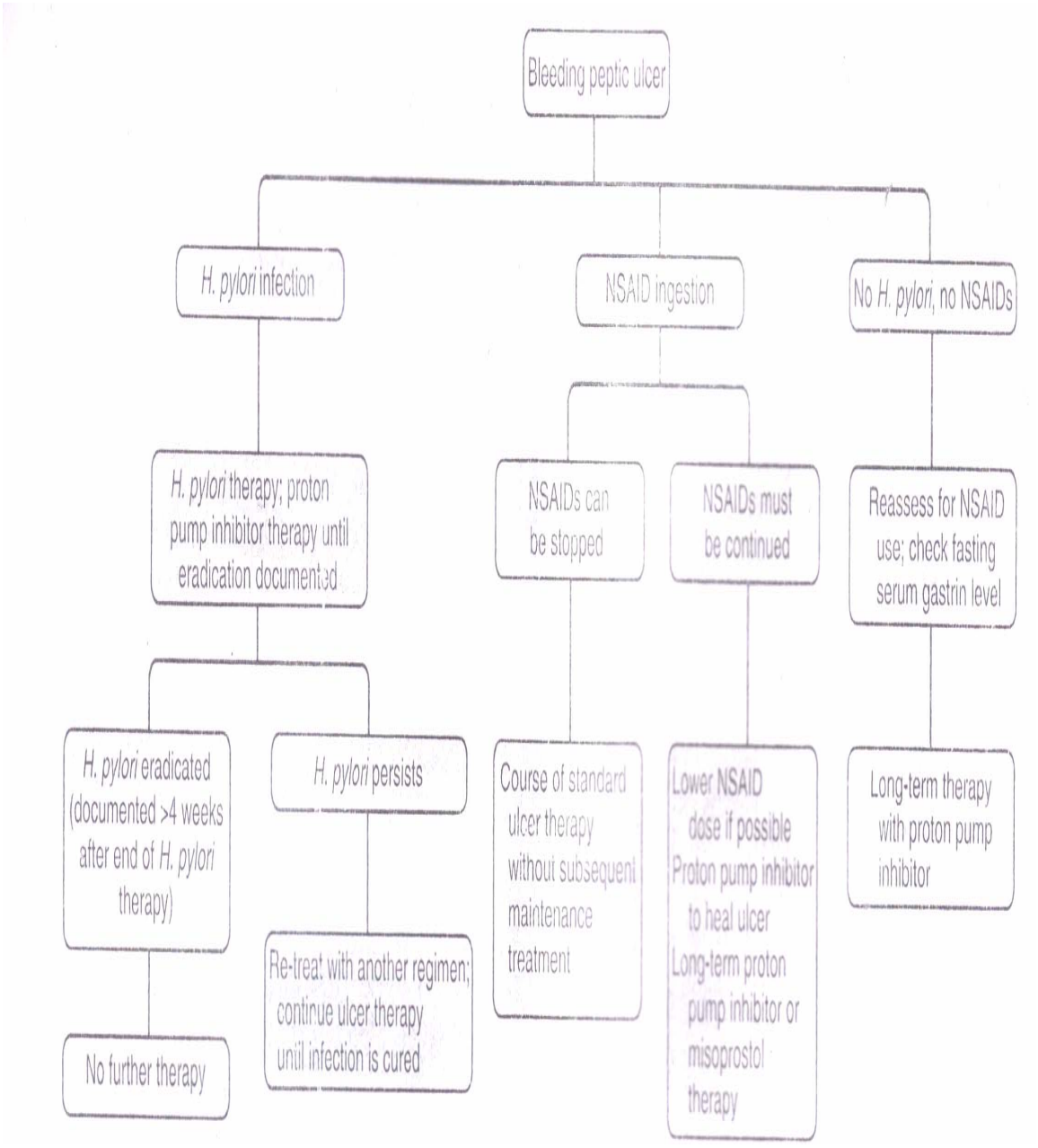
Duodenal ulcer



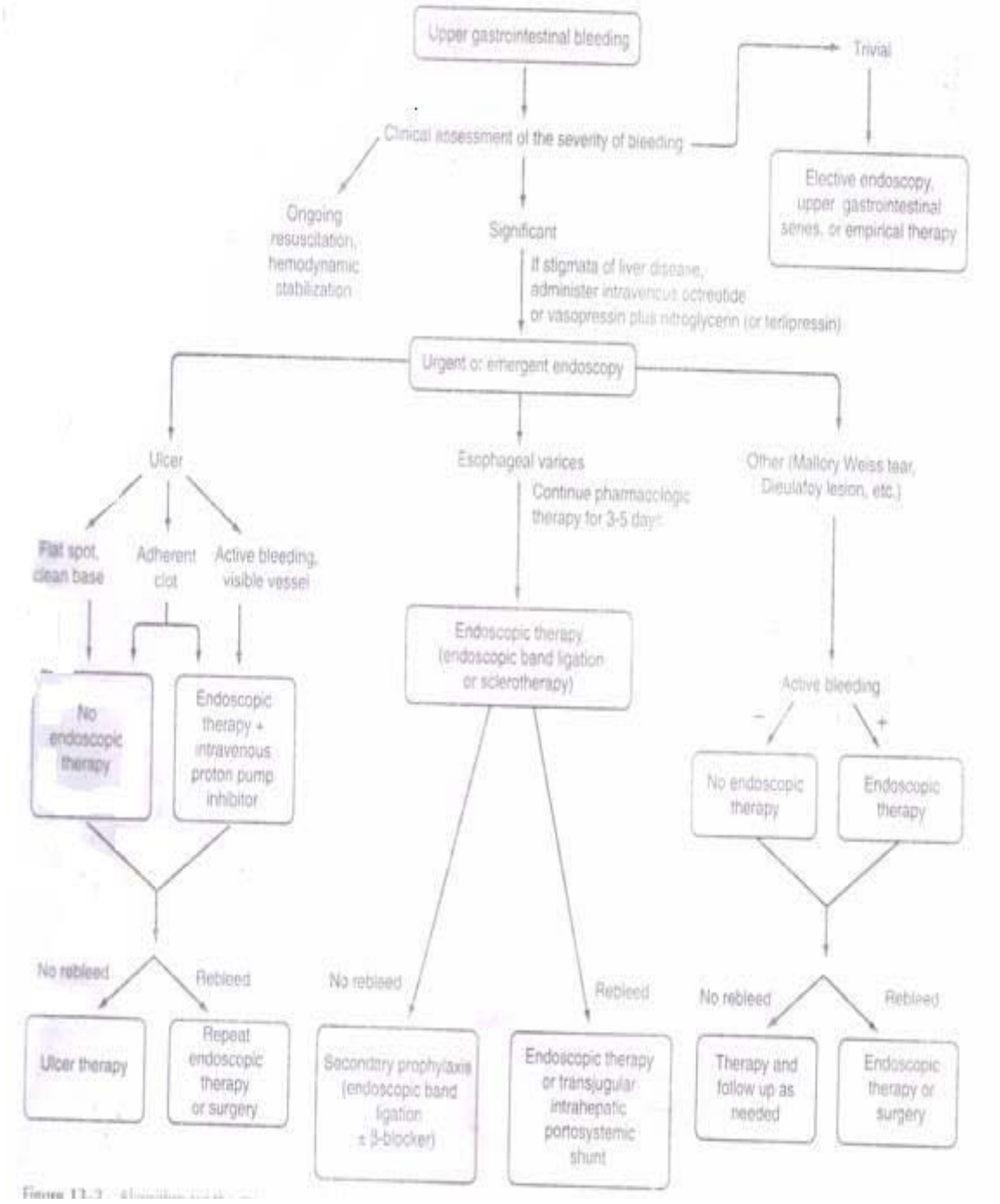
Gastric ulcer



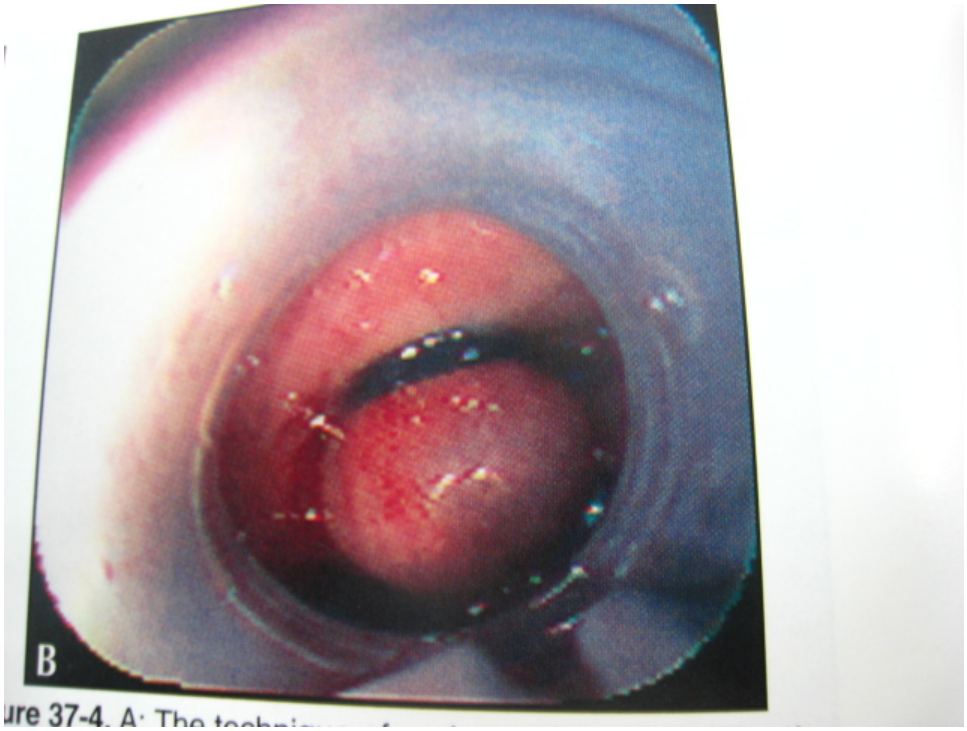
Algorithm for the management of bleeding peptic ulcer due to various etiology



Algorithm for the management of acute upper gastrointestinal bleeding



Endosopic variceal ligation



ure 37-4. A: The

Appendix A: Proforma – Study on Endoscopic finding in UGI Bleed

Department of Internal Medicine, Thanjavur Medical College

1. Name: 2. Age: Yrs: 3. Sex: M F

4. IP.No: 5. Ward: 6. unit:

7. Occupation: 8.Monthly Income:

9. Height(in cm) _____ 10. Weight (inKg) _____

12. Hematesis and malena

S.No	History	Details
(a)	No. of times blood vomited	
(b)	Quantity of blood loss each time (in milli litre)	(1) (2) (3) (4) (5) (6)
(c)	Total quantity of blood loss	(1) < 100ml (2) 100 to 1000ml (3) > 1000ml
(d)	H/o Malena	

13. Other Symptoms

(a) Abdominal Pain → (B) Heart burn →

(c)Dysphagia → (d) Abdominal lump →

(e) Waterbrash / Vomiting → (f) Dyspepsis →

(g) Jaundice → (h) Any Other bleeding tendency →

(i) Syncope → (j) Palpitation →

14. Triggers

(a) Known PUD (b) Alcoholism (c) Smoking (d) NSAID

(e) Steroids (f) Anti Coagulants

15. Past History

(a) HT (b) DM (c) CAHD (e) H/O Asthma/ COPD

(f) Others (Specify) _____

16. Clinical examination

(a) Level of consciousness (b) Dyspnea (c) Pallor (d) Icterus

(e)Pedal edema

(f) Purpura /Petechiae (g) Cold clammy extremities (h) Signs of liver cell failure (i) Sternal tenderness (j) RT aspiration to confirm GI bleed

17. Vital signs

(a) Pulse rate (supine): (b) Pulse rate (standing) :
(c) BP (supine): (d) BP (Standing):
(e) Respiratory rate: (f) Temp:

18. Examination of system:

(a) Abdomen :

(b) Respiratory system :

© Cardiovascular system :

(d) Central Nervous System :

19. Investigations:

(i) Urine: (a) Albumin (b) Sugar (c) Bile salt (d) Bile pigment

(ii) CBC (a) TC (b) DC (C) Hb:

(d) RBC: (e) Platelet:

(iii) (a) Bleeding time: (b) Clotting time: (c) Blood Group

(d)Blood sugar(R): (e) Blood Urea: (f) Serum Creatinine:

(g) Serum .Electrolytes:

(iv) LFT: (a) SGOT (b) SGPT (c)Serum. Bilirubin

(d) Alkaline. Phosphatase

(e) Serum. Protein (f) Serum Albumin

(v) Chest X – Ray:

(vi) ECG:

20. Upper GI Endoscopy: MGE No. : Date:

(a) Esophagus:
(b) Stomach:
(c) Duodenum: