CLINICAL AND ETIOLOGICAL PROFILE OF CHILDREN WITH UPPER GASTROINTESTINAL BLEED AND ENDOSCOPIC CORRELATION

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CERTIFICATE

This is to certify that the dissertation titled, **"Clinical and Etiological profile of children with Upper gastrointestinal bleed and Endoscopic correlation"** submitted by **Dr.R.V.Dhakshayani,** to the Faculty of Pediatrics, The Tamilnadu Dr.M.G.R Medical University, Chennai, in partial fulfillment of the requirements for the award of M.D. Degree (Pediatrics) is a bonafide research work carried out by her under our direct supervision and guidance, during the academic year 2008-2011.

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DECLARATION

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CONTENTS

CLINICAL AND ETIOLOGICAL PROFILE OF UPPER GASTRO INTESTINAL BLEED IN CHILDREN WITH ENDOSCOPIC CORRELATION

Upper gastrointestinal bleed in children is a fairly common symptom encountered in paediatric practice. UGI bleed is an alarming symptom to the patients and parents and a challenge to the physician. Gastrointestinal bleed in children accounts for 10-15% of referrals to paediatric gastroenterology services[1]. Most aetiologies are self limiting and benign, but it is crucial not to miss conditions that may lead to severe consequences, if undiagnosed.

UGI bleed constitutes 54%[2] of all children presenting with gastro intestinal bleeding. Some causes span the entire paediatric range, while some others are common in particular age groups. Over the years of advancements in endoscopy, radiology and newer therapeutic modalities, it has been possible to narrow down to the cause of bleed more accurately and treat it more effectively.

Acute gastro intestinal bleed usually presents suddenly, often unheralded by any symptom; in a few, the bleed may be slow and continuous, manifesting as anaemia or hemodynamic instability. In quite a number of these patients, there is no identifiable cause despite extensive investigations, posing diagnostic challenge and therapeutic dilemma to the treating physician.

Mode of presentation

Upper gastro intestinal bleed suggests a bleeding site proximal to the ligament of Trietz and can present as hematemesis, melanemesis, melena, hematochezia[3] or occult blood with or without hemodynamic changes. Melena usually indicates bleeding above the ileo-caecal valve, which is acted upon by intestinal bacteria [4]. In general, the darker the blood, the higher is its origin from the gastrointestinal tract.

Assessment of severity

Fresh hematemesis alone, melanemesis with melena or fresh hematemesis with melena predicts major bleeding and requires hospitalisation, compared to a minor bleed, as evidenced by melanemesis or melena alone[5,6].

Hematochezia is usually a manifestation of lower gastrointestinal source of bleeding but can be a manifestation of UGI bleed in 11%[7] due to very rapid transit of blood through the GIT and is suggestive of approximately 20%[8] of loss of blood volume proximal to the ligament of Trietz

The presence of hemodynamic instability in children with upper gastrointestinal bleeding indicates massive bleeding.

Etiology of upper gastrointestinal bleeding

Age is an important factor in determining the most likely cause of bleed[9, 10,11].The presence or absence of other symptoms like significant abdominal pain, vomiting, diarrhoea, dysphagia etc. and signs of a surgical condition may help in narrowing down to the diagnosis, though a precise diagnosis needs further work up.

Erosive damage to the gastrointestinal mucosa is the most common cause of bleeding in the western world[9], although, variceal bleed secondary to portal hypertension also occurs very frequently to warrant consideration in most cases with massive bleeding. Vascular malformations are rare causes in children and more difficult to identify[12.]

In India, as reported in a few studies, varices secondary to extrahepatic portal hypertension is the most common cause of upper gastrointestinal bleeding[10,13].

Table - I

Etiological factors of UGI bleed in children according to age[11].

Age category	Etiological Factors
Infants	Gastritis
	Esophagitis
	Stress ulcer
	Mallory Weiss tear
	Vascular Malformation
1 year to 12 years	Esophageal varices
	Peptic ulcer disease
	Stress ulcer
	Mallory Weiss tear
	Esophagitis
Adolescents	Esophageal varices
	Peptic ulcer disease
	Gastritis
	Foreign body
	Mallory Weiss tear
	Esophagitis
	Stress ulcer

Table - II

Etiological factors of UGI bleed in children- according to site[14]

I. GI Causes

Site	Common	Uncommon

Esophagus	• Varices
	• Esophagitis
	  • Acid reflux
	  • Pill induced
	• Mallory-Weiss tear[12]
		• Esophagitis
		Viral (herpes Cytomegalovirus)
		Allergic
		Fungal
		Caustic ingestion
		• Aorto esophageal fistula
		• Foreign body
		• Duplication cyst.

Stomach	• Gastritis Aspirin NSAIDs H.Pylori Viral • Ulcer Stress Acid Peptic disease • Portal gastropathy	• Gastritis Crohn's disease • Portal hypertension • Ulcer Zollinger-Ellison Syndrome[15] • Varices • Dieulafoy's disease • Duplication cyst • Leiomyoma • Vascular malformations • Eosinophilic gastropathy
Duodenum	• Ulcer H.Pylori related • Duodenitis Crohn's disease	• Duplication cyst • Poly • Foreign body • Varices • Lymphoid hyperplasia • Vascular malformations • Hemobilia

II. SYSTEMIC DISORDERS

Bleeding diathesis

Disseminated intravascular coagulation

ITP

Malignancies

Leukemia

Vasculitis

Henoch- Schonlein purpura

Connective tissue disorders

Ehler's Danlos Syndrome

Cutis laxa

III. SPURIOUS HEMATEMESIS

Swallowed blood from ear, nose, throat, dental region

Intra cranial hemorrhage through cribriform plate

Hemosiderosis

IV. MUNCHAUSEN BY PROXY

Grossly, the common causes of upper gastrointestinal bleed can be categorised into variceal and non variceal bleed

Clinical presentation of common causes of upper gastro intestinal bleed

Variceal bleed

Upper gastro intestinal bleeding associated with portal hypertension is mostly a dramatic event and is associated with large volumes of blood loss. It can be potentially fatal in 25- 40% of cases[16].

Portal hypertension in children is ideally considered in two groups, intra hepatic and extra hepatic. In children, extra hepatic portal hypertension is atleast 50% more prevalent than intra hepatic portal hypertension[6,9]. Nearly 80% of these children have atleast one episode of bleeding by the age of 10 years[7,9,17].

UGI bleed associated with PHT is usually a dramatic event associated with loss of large volumes of blood, usually presenting as spontaneous onset of frank hematemesis, in a previously healthy child , triggered by a minor intercurrent infection or NSAIDs. Variceal bleed may also present as a slow ooze with melena, rather than a sudden hematemesis.

In predicting the probability of re bleeding in variceal bleeds, the variables considered are color of the varices, cherry red spots, red wheal markings, hematocystic spots, Conn's grading of variceal size[18] and intra variceal pressure.

The spleen is always enlarged in EHPVO and symptomless splenomegaly may be a presentation[19,20]. The liver is normal in

consistency and stigmata of hepatocellular disease are absent. Classically the frequency of variceal bleeding decreases after adolescence[21].

Non cirrhotic portal fibrosis is an important cause of portal hypertension in eastern India and occurs in older children and adolescents[22]. Portal hypertension related to cirrhosis may be associated with bleeding any time after 6 months of age but is most prominent in children older than 2 years of age.

Non variceal bleed

Peptic ulcer disease encompassing gastric ulcer, and duodenal ulcer is the most common in this group of disorders[23,24,25,26]. Peptic ulcer disease can be primary associated with bile reflux and H.pylori or secondary to antibiotics, auto immune gastritis, eosinophilic gastritis, corrosives, stress, Crohn's, Menetrier's disease, Zollinger Ellison's[15] disease etc.

Usually presenting as minor hematemesis, esophagitis[5,6,27] may be associated with bronchospasm, eczema or allergic rhinitis and a family history of food allergy or asthma is often seen in children with eosinophilic esophagitis.

Erosive gastritis accounts for 5-25% of cases of upper GI bleeds[27].Common in infants and young children. The usual presentation is a minor hematemesis, precipitated by trauma, burns, major surgery, hypoxemia, acidosis, shock or most commonly NSAIDs.

Drugs are the next important cause of mucosal damage in children. Drugs such as aspirin, NSAIDs, and steroids are associated with mucosal erosion, ulceration and bleeding[22,27,28,29,30].

Mallory-Weiss tears [31]are common in children who have a hiatus hernia[32].The tear occurs at the site of the greatest intraluminal diameter subjected to a sufficiently great transluminal pressure gradient the usual site being the gastric mucosa near the gastro esophageal junction[33,34,35,36] .

Vascular malformations[37,38] like hemangiomas, venous, arterial, arteriovenous or lymphatic malformations rarely present as UGI bleed in children. Hemobilia[39] occurring due to abdominal trauma or rupture of hepatoduodenal artery is a rare cause.

UGI bleed can be rarely due to multisystem diseases like collagen vascular diseases, coagulopathy, malignancies etc.

Spurious hematemesis

Upper GI bleed can be due to swallowed blood, the source of which could be from the nose (epistaxis), nasopharynx, oropharynx, dental region and rarely from intracranial bleed through the fracture of cribriform plate.

Hemosiderosis can present as upper GI bleed. Examination of the blood for hemosiderin is diagnostic.

MANAGEMENT OF UPPER GASTRO INTESTINAL BLEED

1. Assessment of the severity of bleed and steps for stabilization.

The severity is assessed and the child is resuscitated accordingly. Blood component replacement is done if required[40]. NG tube is introduced and gentle lavage is done with saline at room temperature[41]. The urine output is to be monitored.

Treatment goals in patients with upper gastro intestinal bleeding are:

Step I : Restore and maintain the intra vascular volume

Step II : Re-establish oxygen carrying capacity by transfusion with whole blood or packed red blood cells

Step III : Determine source and site of bleeding

Step IV : Stop gastrointestinal bleeding.

II. Detailed history and meticulous examination of the child

The child is allotted into either a first bleed or recurrent bleed category. Detailed history regarding the bleed - volume, color, presence of clots or food, pain, retching, drugs etc. is obtained. History of jaundice, abdominal distension, pedal edema, neonatal umbilical sepsis, bleeding from other sites is obtained. Markers of chronic liver disease, connective tissue disorders and splenomegaly are looked for.

III. Investigations

Essential investigations done initially in a case of UGI bleed

All patients

- Complete blood count
- Blood grouping and cross matching
- Renal function tests, Serum electrolytes
- Liver function tests

Suspected /Proven Liver Disease

- Prothrombin time and partial thromboplastin time
- Serum ceruloplasmin
- Ophthalmological evaluation

Ultrasonography including Doppler is an important tool in the diagnosis of various causes of portal hypertension[42,43].

Endoscopy plays a very important role in the diagnosis and therapy of UGI bleed[44-53] of variceal and non variceal etiology. With the advent and aid of endoscopy, it has become possible to prognosticate the outcome, based on the grading systems available for the common causes of gastrointestinal bleed.

Grading of varices based on size of varices[54].

Grade I(F_1) - Varices can be depressed by endoscope

Grade II-(F_2) - Varices cannot be depressed by endoscope

Grade III-(F_3) - Varices are confluent around the circumference of the esophagus.

Classification of gastric varices[55]:

GOV - GOV_{1-} gastric varices in continuity with esophageal varices extending 2-5 cms below the gastro esophageal junction.

GOV2 - esophageal varices extending into the fundus

IGV - IGV_1- gastric varices located in the fundus

IGV2 -gastric varices located in the body or antrum

Conn's Endoscopic grading of esophageal varices[18]:

Grade I - Visible only on inspiration

Grade II - Visible both on inspiration and expiration

Grade III - Varices projecting into the lumen but less than 50%.

Grade IV - Projecting into the lumen > 50%

Endoscopic prognosticators of re bleeding in variceal bleed:

Color of varices (blue varices), cherry red spots, red wheal markings, hematocystic spots, Conn's grading of varices (Grade Ill/IV) and high intra variceal pressure are found to be good predictors of variceal re bleeding[56].

Endoscopic grading of esophagitis

Grade	Hetzel &Dent, et al.1988[57]	Savary & Miller, et al.1978[58]
0	Normal esophageal mucosa; no abnormality noted	
1	No macroscopic erosions visible; erythematous, hyperaemic & friable esophageal mucosa	1 or more longitudinal non-confluent mucosal lesions with erythema often covered with exudates above or extending from the GE junction.
2	Superficial erosions or ulcerations involving <10% of the last 5 cms of the esophageal mucosa.	Confluent erosive & exudative mucosal lesions that do not cover the entire circumference of the esophagus.
3	Superficial erosions or ulcerations involving 10% to 50% of the last 5 cms of the esophageal mucosa.	Circumferential mucosal lesions covering the whole esophageal mucosa.
4	Deep erosions or confluent erosions or ulcerations involving > 50% of of the last 5 cms of the esophageal mucosa.	Chronic mucosal lesions such as ulceration with or without stricture formation.

Endoscopic appearance in gastritis:

Erosive gastropathy commonly involves the gastric antrum; erosions are superficial, multiple and do not perforate. Mild gastritis presents as a mosaic pattern and severe gastritis with multiple cherry red spots and a confluent hemorrhagic appearance.

Endoscopic prognosticators of re bleeding in non variceal bleed:

The stigmata of a recent hemorrhage from an ulcer like active bleeding (oozing or spurting), an adherent clot, a visible vessel, "sentinel clot", or flat pigmented spots (red, black, purple, brown) are the most reliable prognostic indicators of re bleeding[59,60,61].

Forrest classification and stigmata of recurrent bleed:

Grade			**Risk of re bleeding (%)**
Grade III	-	Clean base	0-12
Grade IIC	-	Pigmented flat spot	0-8
Grade IIB	-	Adherent clot	25-35
Grade IIA	-	Non bleeding visible vessel	50-60
Grade IB	-	Oozing hemorrhage	10-30
Grade IA	-	Spurting hemorrhage	80-90

In 10 to 20% cases, endoscopy may not reveal a source of bleeding. If endoscopy is normal, a possibility of spurious hematemesis should be considered and the child referred to ENT surgeon or dental surgeon to exclude lesion in the respective areas.

In certain circumstances, specialised investigations such as selective arteriography and radionucleide studies may be necessary. Barium studies are less sensitive in this regard.

IV. Specific Therapy

Treatment of non variceal bleeds (Mucosal lesions of the GIT):

The aim of therapy is to neutralize or prevent release of gastric acid

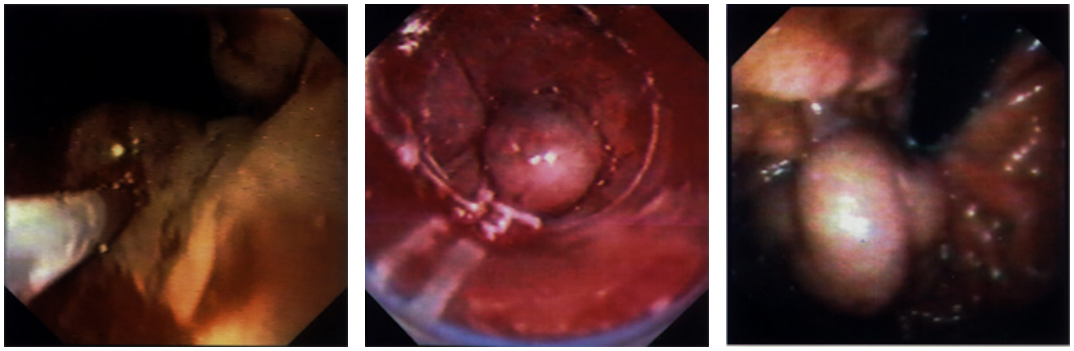
- Gastric lavage with Normal saline or nor adrenaline
- H_2 receptor antagonists like Ranitidine (3-5 mg/kg).
- Antacids (1-2ml/kg / dose).
- Cytoprotective agents like Sucralfate as in esophageal and gastric ulcers.
- Proton pump inhibitors like Omeprazole as in severe esophagitis and GI bleed in older children (0.7 mg/kg/dose).
- Drugs that increase LES pressure like metoclopramide can be tried in reflux esophagitis and esophageal varices.
- Endoscopic management: For bleeding peptic ulcers and vascular malformations, thermal coagulation with a heater probe, electro coagulation and photocoagulation are the techniques employed.
- Hemoclips are also being employed in the management of variceal bleed.

Treatment of variceal bleed

Bleeding from esophageal varices is a life- threatening event. Hence, the primary objective of therapy is immediate cessation of bleeding. Different options available to meet this objective are as follows:

- Endoscopic techniques
  - Sclerotherapy
  - Band ligation
- Pharmacological therapy
  - Vasopressin[62,63,64,65]
  - Terlipressin
  - Glypressin
  - Somatostatin[66]
  - Octreotide[67,68]
  - H_2 receptor antagonists
  - Antacids
- Balloon tamponade
  - Sengstaken - Blackmore tube
  - Minnesota tube
- Surgical Techniques
  - TIPS
  - Porto- caval shunt
  - Distal spleno- renal shunt
  - Devascularisation

Endoscopic sclerotherapy (EST) and Endoscopic variceal band ligation(EVL) are the accepted modalities of treatment for variceal bleed and can be performed either as an emergency or elective procedure.EST produces an intimitis followed by thrombosis and fibrosis of the vessels.



EST Band Ligation Glue injection

Initially, it is done once in 3 weeks till adequate obliteration of varices, followed by EST once in 3 months upto 1 year and yearly once for 3 years. The sclerosants accepted universally are 3% polidocanol and 3% sodium tetra decyl sulphate[69,70]. Cyano acrylate (Histacryl- glue) [71] can be used for fundal varices.

In Endoscopic variceal ligation (EVL)[72], the esophageal varices are banded with elastic rings. As efficacious as EST and with fewer complications, EVL can be done only for varices more than Grade II. EST and EVL can be combined for better results.

Prevention of gastrointestinal bleed:

- Avoidance of ulcerogenic drugs like NSAIDs and others in high risk patients. Use of Cox-2 inhibitors (cyclo-oxygenase-2) may have a vital role in the near future.
- Better umbilical care in the newborn period will decrease the incidence of EHPVO.
- The non selective β blocker propranolol helps to reduce portal venous pressure and is used as a preventive measure, in those EHPVO patients who have not bled and also to prevent re bleed in others[73].
- Regular EST/EVL can be used to prevent re bleeding.
- Endoscopic variceal ligation is advised as a prophylactic measure in those who have not bled.
- Maintenance treatment with Ranitidine to decrease recurrent ulcer bleeds.
- Anti H.pylori therapy in cases of duodenal ulcer.
- Liver transplantation is the therapeutic modality of choice for preventing recurrent bleeding in children with end stage liver disease[74].

REVIEW OF LITERATURE

Zahid Afrain, et al (1999), in their article, state that 10% to 15% of referrals to paediatric gastroenterologists were due to gastrointestinal haemorrhage in infants and children[6].

Daniel, et al (1939), in their observations found that melena can result when as little as 50 to l00ml of blood is present in the upper gastrointestinal tract[4].

Wara ,et al (1985) correlated various patterns of UGI bleed with clinical outcome and found that dark hematemesis or melena alone would predict massive bleeding 5 to 10% of the times. On the other hand, fresh hematemesis alone, dark hematemesis with melena, or fresh hematemesis with melena predicted massive bleeding in 21%, 24% and 27% respectively[1].

Silverstein, et al (1981), in their survey on UGI bleed, correlated mortality rate with NG aspirate and stool color[5].

Nasogastric aspirate	Stool color	Mortality rate (%)
Clear	Red, Brown, Black	10
Coffee Ground	Brown or black	10
	Red	20
Red blood	Black	10
	Brown	20
	Red	30

Martin Ulshen (2000), states that erosive mucosal lesions are the commonest causes of bleeding, although variceal bleeding secondary to portal hypertension occurs frequently enough to require consideration. Vascular malformations are rarer causes of bleeding in children and are difficult to identify[9].

Ted A Williams (1993), has quoted that stress ulcer, esophagitis, gastritis and Mallory- Weiss tear are the common causes of upper GI hemorrhage in infancy and pre- school children, whereas in older children along with stress ulcer and gastritis, esophageal varices contribute to the majority of cases observed[10].

Robert H Squires JR, (1998), states in his study that erosive lesions of the GIT like esophagitis, gastritis, Mallory- Weiss tear are the common causes of upper GI hemorrhage in infancy and preschool children, whereas in older children stress ulcer, gastritis and esophageal varices contribute to the majority of cases observed[14].

Mittal SK, et al (1978), in his review of 70 cases with extrahepatic portal hypertension states that 70% of children who bleed for the first time are more than 6 years of age, in contrast to European studies where 80% of children who bleed for the first time do so before 6 years of age. Splenomegaly was present in almost all children with EHPVO. Splenomegaly may occur by one month of age and is present before 3 years in a majority of children with EHPVO and may be the presenting

complaint in 18-40% of patients. Hypersplenism is associated in upto 40% of cases[16].

Webb U and Sherlock S, (1979), in their study on the etiology, presentation and natural history of EHPVO, observed that the frequency of variceal bleeding, classically decrease after adolescence but it can occur at any age and can result in massive bleeding. They also observed that ascites is a rare finding in EHPVO, though it may occur at the time of onset of disease or after a massive bout of bleeding and is a transient phenomenon .Dilated superficial porto systemic collaterals in the form of abdominal veins are rarely seen. The liver is normal in consistency with no evidence of liver cell failure[21].

Alvarez F, Bernard O, Brunelle F et al (1983), in their study on portal obstruction in children, found that the two most common causes of prehepatic portal hypertension were portal vein thrombosis and splenic vein thrombosis, with the former being the most frequent cause in the paediatric population. They also reported that bleeds occur in 80% of children with extrahepatic portal hypertension, compared to 30% in children with intrahepatic portal hypertension[17].

Tanner S, (1989), reported that the commonest cause of major upper gastrointestinal bleed in children is from the ruptured esophageal varices, most commonly due to extrahepatic portal hypertension (60%)[76].

Ganguly S, et al (1997), in their study of portal hypertension in 50 children of North-east India, identified non-cirrhotic portal fibrosis as an important cause of portal hypertension. They observed it in 24 (48%), followed by EHPVO in 18(36%) and cirrhosis of liver in 8(16%) children[22].

Burt CAV, et al (1931), **Kinsella TJ,** et al (1948), **Powel**l,et al (1984), in their studies on Mallory-Weiss tear have documented that the GIT in children, including the esophagus, has greater tensile circumferential strength than the adult GIT and this, perhaps, is responsible for the lesser rates of occurrence of this lesion in the paediatric population[33].

Cotton PB, et al (1973), did early endoscopy in patients with hematemesis and melena and found that erosive gastritis accounts for 15% of all cases of UGI bleeds[24].

Watts HD, (1976), in his study on the effect of hiatus hernia on the site of injury in a patient with vomiting, reported that Mallory-Weiss tear occurs more frequently in patients with an established hiatal hernia[32].

Bergman GF, et al (1976), conducted a study on the occurrence of gastrointestinal hemorrhage after therapeutic doses of aspirin in normal children and found that there is significant incidence of mucosal erosion, ulceration and bleeding after aspirin ingestion[28].

Abraham Bogoch, (1995), in his study on esophagitis, reports that massive hematemesis is an uncommon complication of this condition when the frequency of gastroesophageal reflux disease is considered. Bleeding, if occurs is more often relatively slight and intermittent[27].

Livoti G, et al (1997), evaluated the relationship between drug intake and upper gastrointestinal bleeding and found that about 50% of cases showed gastric erosions secondary to drug intake[31].

Chaibou M, et al (1998), in a prospective cohort study of 1006 children,observed the complications of upper gastrointestinal bleed as severe anemia (10 cases), transfusion requirement (10), hypotension (3) surgery (1), and concluded that clinically significant upper GI bleeds are rare in children[40].

John J Herbst, (2000), quotes that stress ulcer is an important cause of upper gastrointestinal bleeding in children[37].

De Giacoma C, Tomasi G, Gattic, et al (1989), in their study on ultrasonographic prediction and severity of esophageal varices in children, found that if the ratio of lesser omental thickness to aortic diameter is greater than 1.9, its prediction of the presence of esophageal varices is good[42].

Westra SJ, et al (1995), in a study using Doppler ultrasound in children suggests that portal vein pulsatility is a sensitive and specific finding indicative of portal hypertension in children[43].

Moare AM (1975), did a comparative study between endoscopy and radiology in acute upper gastrointestinal hemorrhage in 158 patients and reported that an emergency endoscopy of the upper GIT provides a more precise method of diagnosis than emergency radiology[46].

Cadranel, et al (1977), in a series of 100 upper GI scopies and colonoscopies in infants and children, using standard fibre optic endoscopy proved that the endoscopy is more reliable than roentgenography in the diagnosis of superficial mucosal erosions and gastro intestinal bleeding[47].

Gleason WA, et al (1974), conducted a study on the safety of fibre optic GI endoscopy in children and found it safe to be used in children[45].

Kenneth Cox and **Marvin E Ament** (1979), in a retrospective study of upper gastrointestinal bleed in 68 children and adolescents, has observed, in descending order of frequency, the five most common causes of UGI bleed as duodenal ulcers (20.5%), gastric ulcers (17.6%), esophagitis (14.7%), gastritis (13.2%) and esophageal varices (10.2%). Further they have stressed that endoscopy is the most reliable method of identifying the site of bleeding [48].

Graham, et al (1978), using fibre optic endoscopy observed that the commonest lesion in children with UGI bleed as duodenal ulcer in infancy, gastric ulcer in 1 to 6 years of age and duodenal ulcer in children more than 7 years of age[49].

Charles B Hargrove, et al (1984), in their study of 46 paediatric patients aged between 18 days and 24 months, evaluated the diagnostic usefulness and safety of UGI scopy for various indications. Procedures were performed without sedation in 45% of all children studied, including 87% of infants less than 3 months of age and the procedure was well tolerated. Endoscopy proved to be an accurate and efficient means of establishing the site of bleed in UGI tract hemorrhage. The commonest lesions observed were duodenal ulcer, gastric erosion and duodenitis. As this study was restricted to children below 2 years and as the sample size was small, varices were not observed[51].

Sarin SE, Sundaram KR and Ahuja RE (1989), analysed the clinical, endoscopic and hemodynamic variables in 126 patients with PHT to find out the predictors of variceal bleeding. Of the six variables used, variceal size indicated 35% and intra variceal pressure accounted for 12% of the explained variability between bleeders and non-bleeders. The presence of three red color signs and the color of varices accounted for only 3% and 1% respectively of the total explained variation. Using discriminant analysis, 85% of the bleeders and 81% of the non-bleeders were correctly identified[54].

Mittal SK, Kalra MC and Vyom Aggarwal (1994), New Delhi, in their study on upper GI endoscopy in 236 children presenting with hematemesis, identified varices as the commonest lesions on

endoscopy (39.41%), followed by esophagitis (23.73%),gastritis (8.47%) and gastric erosions (7.2%). Only 3 children had well defined gastric ulceration, while duodenal and esophageal ulcers were observed in 1 each. UGI endoscopy was essentially normal in 65 (27.54%) cases[53].

Grace H Elta, Tadataka Yamada et al (1996), have given several factors that predict a poor prognosis in upper GI bleeding. On analysing the stigmata of hemorrhage, the authors observed that spurting arterial bleeding, non bleeding visible vessel and adherent clot (no visible vessel) have a risk for re bleeding in 85% - 100%, 18%-55% and 24-41%, respectively[61].

Chan ACW, et al (1993), in a double blinded randomized controlled trial compared sodium tetra decyl sulphate and ethanolamine oleate in the sclerotherapy of esophageal varices and concluded that both are equally effective in controlling variceal hemorrhage, but sodium tetra decyl sulphate obliterates the varices in significantly fewer sessions[70].

STUDY JUSTIFICATION

Upper gastrointestinal bleeding in children is a common problem encountered by paediatricians and is potentially fatal. Accurate and proper diagnosis can be a challenge and demands an overview of this ailment, the profile & complications of which are amendable to intervention and possible prevention in this era.

Children with massive bleed who are seriously ill require timely, focussed and immediate intervention. Children with minor bleeds too require assessment to establish a diagnosis and to plan on further management.

With only a limited number of studies available in India in this domain, this study is undertaken to analyse the etiological, clinical and investigative profile of children with UGI bleed, to assist the physician in determining the urgency with which to proceed, to correlate clinical features with endoscopic findings and to educate the parents on preventable factors that lead to bleeding per se or its recurrence.

OBJECTIVES OF THE STUDY

To analyse

1. Etiology
2. Clinical presentation
3. Clinical and endoscopic correlation

of upper gastrointestinal bleed in children aged 3 months to 12 years.

SUBJECTS AND METHODS

1. Methodology

Study Design

Descriptive study.

Place of Study

Paediatric Medical Units & Department of Paediatric Gastroenterology, Institute of Child Health and Hospital for Children, Chennai.

Study Period

September 2008 - September 2010

Study Population

Children aged between 3months to 12 years of age of either sex presenting with upper gastrointestinal bleed.

Inclusion Criteria

Children in the age group of 3 months - 12 years who attended the hospital with the prime symptom of overt upper GI bleed during the study period.

Exclusion Criteria

1. Children with bleeding secondary to systemic infections or diagnosed bleeding diathesis.
2. Children with bleeding secondary to systemic diseases like vasculitis and others.
3. Those with a re bleed during the study period were excluded.

Sample Size

All children with the above inclusion criteria who presented during the study period (155 cases).

Ethical committee clearance was obtained from the Institutional review board.

2. Manoeuvre

Sampling method - Consecutive sampling method

All consecutive children with hematemesis or melena admitted in the Institute of child health and hospital for children or attending the department of gastroenterology with hematemesis or melena were included in the study.

History

Detailed history was taken regarding the bleed, color, volume, number of episodes during present admission, presence of clots, admixture of food and melena. Specific attention was given to the intake of food additives and drugs like NSAIDs known to precipitate gastrointestinal bleeding.

History of abdominal pain, distension, mass abdomen, and symptoms related to chronic liver disease, history of fever and features suggestive of systemic infection was sought. Neonatal history suggestive of umbilical sepsis and umbilical vein catheterisation was also obtained.

The details were entered in a pre structured proforma.

Clinical Examination

A detailed clinical examination of the child was done .Pallor, icterus, petechiae, purpura, epistaxis, evidence for chronic liver disease and systemic disorders were looked for. A thorough abdominal examination was done for presence of organomegaly especially spleen, prominent veins over abdomen and free fluid.

Ear, Nose, throat and dental regions were examined for probable bleeding sources. A classification of major and minor bleeds were made based on the color of bleed, quantity, presence of combinations of hematemesis and melena and the presence or absence of hemodynamic instability on presentation.

Major bleeds

a. Fresh hematemesis alone.

b. Fresh hematemesis with melena.

c. Dark hematemesis with melena.

d. Fresh hematemesis, dark hematemesis with melena

e. Hematochezia

Minor bleeds

a. Only dark hematemesis (coffee ground).

b. Only melena.

Any of the above, with hemodynamic instability was considered as massive bleed [5,6,75].

With the above history and clinical examination, a provisional clinical diagnosis was made. All necessary investigations were done including ultrasonogram of the abdomen.

Endoscopy

Upper GI endoscopy was done within 24 hours of bleeding or after stabilisation of the child as the condition warranted. Olympus N30 Upper gastrointestinal fibre optic scope fitted with video adapter was used. All the endoscopies were done by paediatric gastroenterologist.

Procedure

Endoscopy was done after 6 hours of fasting before the procedure.

The esophagus, esophago-gastric junction, stomach and duodenum upto D_2 were visualised. The endoscopic findings were recorded in detail and variceal lesions were graded and other predictors of variceal bleeding were recorded. Mucosal changes like erythema, erosion and hemorrhage were looked for.

The sclerosant, 3% sodium tetra decyl sulphate was used. A volume of 1-2 cc per site was injected per site. The sclerotherapy sessions were repeated with a 3-4 weeks interval till the varices were downgraded to Grade I. In addition they were advised to continue tablet propranolol 1mg/kg/day in 2 divided doses.

Children who had non variceal lesions like esophagitis, gastritis and Mallory-Weiss tear were treated with H_2 receptor antagonists and proton pump inhibitors.

If the endoscopy turned out to be normal, with the probability of spurious hematemesis, expert opinion for the source of bleeding in ENT, dental region and haematological disorders was obtained from the specialists concerned.

All the findings were entered in the pre structured proforma, which is annexed.

3. Statistical analysis

The data were analysed using SPSS 15.0 version.

Descriptive statistics like number, proportion, percentage ,range and inferential statistics like p value was arrived at using Chi square test.

p value<0.05 was considered for statistical significance

OBSERVATIONS

155 children in the age group of 3 months - 12 years were studied.

The total number of variceal bleeds was 65 and non variceal bleeds was 90.

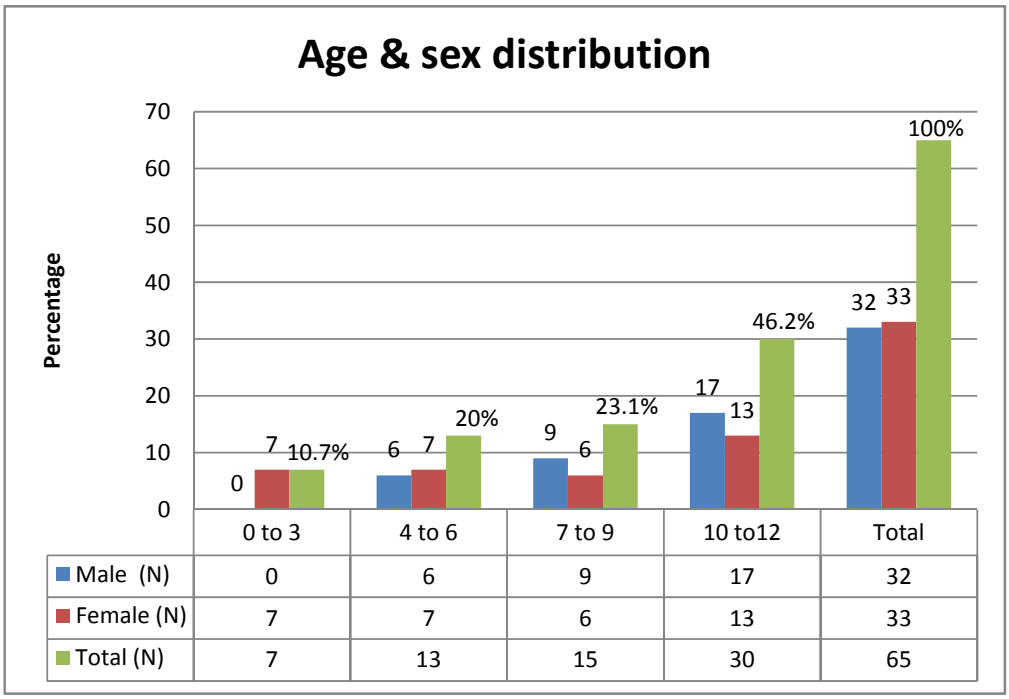
Demographic characteristics of children with upper GI bleeds.

Total no. of males = 81(52.25%)

Total no. of females = 74(47.75%)

No predilection to sex was observed (M:F -1.1:1)

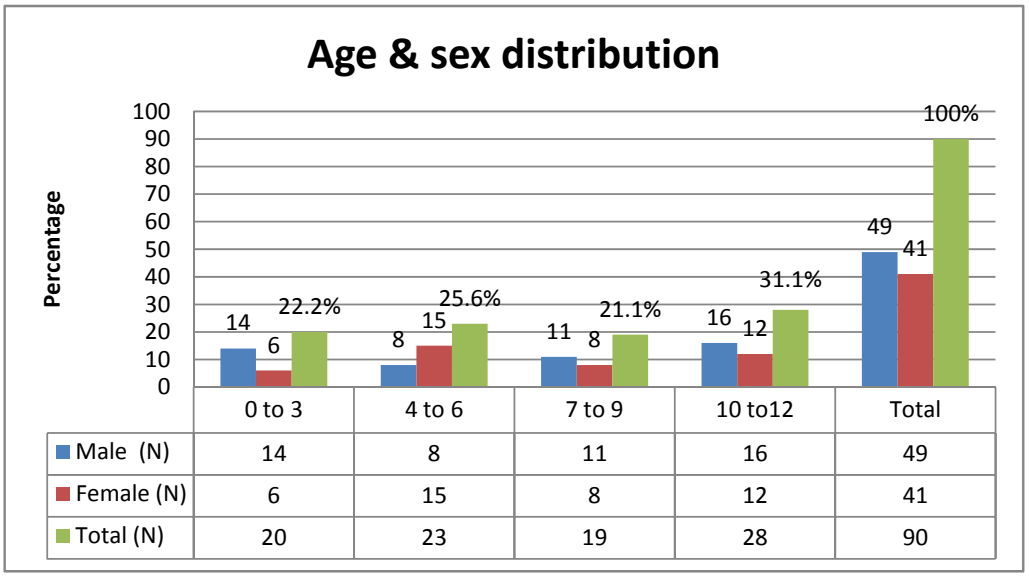
Age and sex distribution of children with upper GI bleed due to variceal etiology



	0 to 3	4 to 6	7 to 9	10 to12	Total
Male (N)	0	6	9	17	32
Female (N)	7	7	6	13	33
Total (N)	7	13	15	30	65

Figure - 1

Age and sex distribution of children with upper GI bleed due to non variceal etiology



	0 to 3	4 to 6	7 to 9	10 to12	Total
Male (N)	14	8	11	16	49
Female (N)	6	15	8	12	41
Total (N)	20	23	19	28	90

Figure - 2

The frequency of upper gastrointestinal bleeding due to variceal cause seems to increase with increasing age. Of those variceal bleeders, 69.2% of cases are above 6 years of age.

UGI bleeding due to non variceal causes tends to be almost same in all age groups. 47.7% of the cases are below 6 years and 52.3% above 6 years of age.

The difference observed is not statistically significant.

[p value = 0.48]

60 children out of total 155 cases had atleast one episode of upper gastrointestinal bleed before the study period.

51 of them were diagnosed to have varices as the source of upper gastrointestinal bleed. Two children were cases of chronic liver disease secondary to Wilson's, one had duodenal ulcer, and six re bled due to gastritis.

Of the non -variceal bleeders, 35 children out of the 90, had multiple episodes of bleed, mostly minor, on presentation.

Of these children, 22% had received medical management, either IV fluids or blood transfusion prior to admission at ICH.

About 73% of children attended the hospital within 24 hours of having the UGI bleed.

Table - III

Presentation as major bleed

SI No.	Group	Mode of presentation	No. of children	Percentage (%)
1	Group I	Fresh hematemesis alone	53	63.8
2	Group I+III	Fresh hematemesis with melena	9	10.8
3	Group II+III	Dark hematemesis with melena	20	24.1
4	Group I+ II +III	Fresh hematemesis, dark hematemesis with melena	1	1.2
Total			**83**	**100.0**

Total no. of major bleeds = 83 /155 (53.54%)

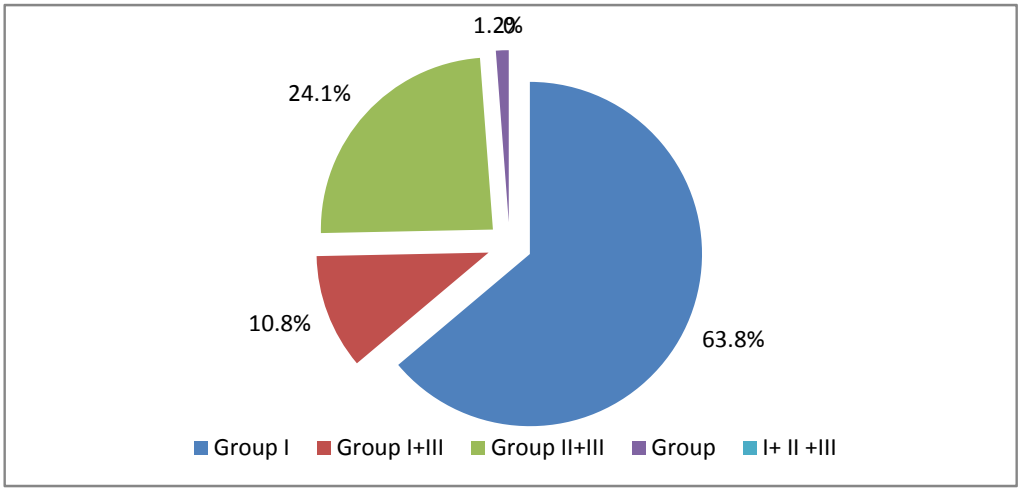


Figure -3 : Major bleed - mode of presentation

Table - IV

Presentation as minor bleed

SI No.	Group	Mode of presentation	No. of children	Percentage (%)
1	Group II	Dark hematemesis	55	76.3
2	Group III	Melena	17	23.7
Total			**72**	**100**

Total no of minor bleeds= 72/155 (46.45%)

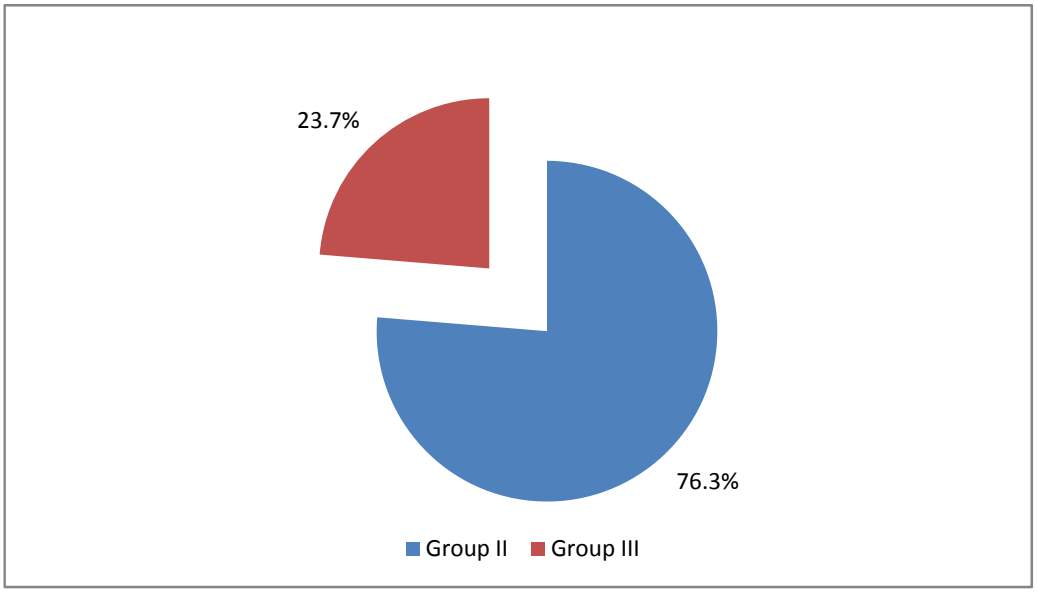


Figure – 4 : Minor bleed- mode of presentation

In 72 children with UGI bleeds (46.45%), the bleed was minor , of which 55(76.3%) presented as dark hematemesis and 17(23.7%) presented with melena alone.

Table- VII

Categorization of bleed and correlation with etiology

	Variceal	Non variceal	**Total**
Major	54	29	**83**
Minor	11	61	**72**
Total	**65**	**90**	**155**

Grossly, 65 children were variceal bleeders, of which 54 had major bleeds(83.1%) and 11(16.9%) had minor bleeds. Among the non variceal bleeders, 29(32.2%) and 61(67.8%) children had bled in major and minor volumes respectively.

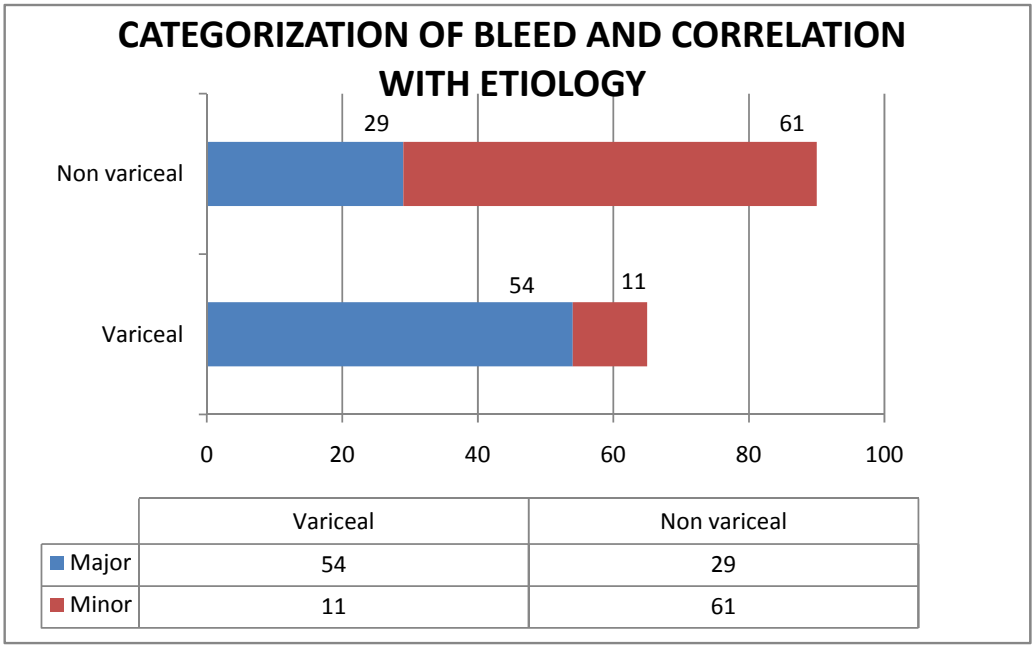


Figure – 5

Table - V

Major Bleeds as per Etiology

Sl. No.	Etiology	No. of children	Percentage (%)
I	**Variceal**	54	**65.1**
	a. EHPVO	52	62.7
	b. Intrahepatic	2	2.4
II	**Non variceal**	29	**34.9**
	a. GIT lesions	28	33.7
	b. Spurious hematemesis	Nil	0
	c. Not known	1	1.2

53.54% of UGI bleeds presented with major bleed. Of the major bleeds 65.07% were of variceal and only 34.93% were of non variceal etiology.

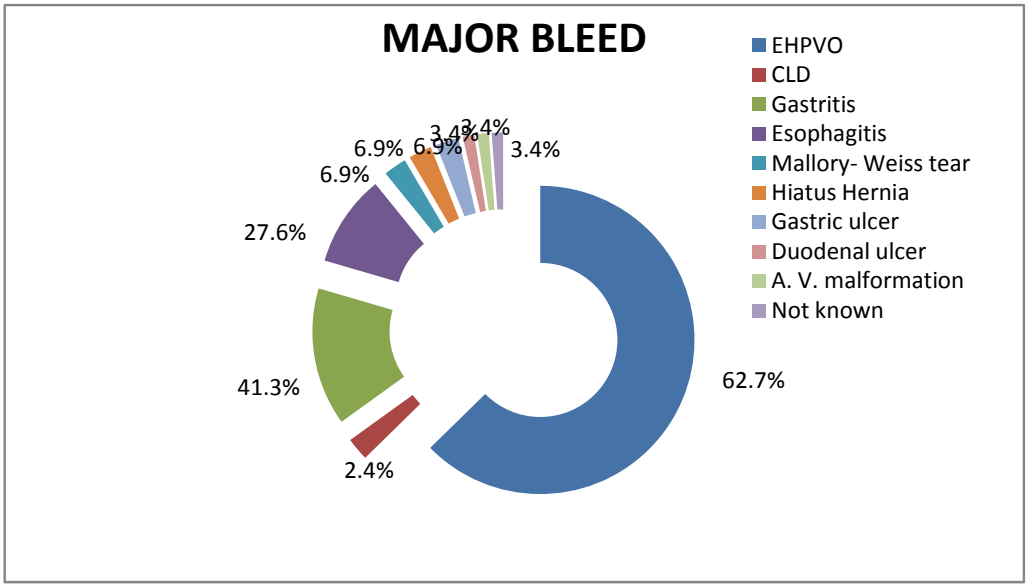


Figure - 6

Table -VI

Non variceal causes of major bleed

SI No.	Etiology	No. of children (N)	Percentage (%)
I	**GIT lesions**	28	**96.6**
	Gastritis	12	41.3
	Esophagitis	7	24.1
	Mallory- Weiss tear	2	6.9
	Hiatus Hernia	2	6.9
	Gastric ulcer	2	6.9
	Duodenal ulcer	1	3.4
	Duodenal polyp	1	3.4
	A. V. malformation	1	3.4
II	**Spurious hematemesis**	Nil	0
III	**Not known**	1	**3.4**
Total		**29**	**100**

Of the non variceal causes of major bleed, mucosal lesions of the GIT form the predominant cause. Among them, gastritis formed the commonest cause followed by esophagitis. 2 cases each of hiatus hernia, Mallory-Weiss tear, and gastric ulcer presented with major bleed. One case of Histo pathologically proven Brunneroma involving the second part of the duodenum was identified in the course of the study.

One child with history of accidental paracetamol ingestion, presented with fresh hematemesis, dark hematemesis and melena. Another toddler had sustained blunt injury abdomen and presented with dark hematemesis.

The cause of bleed could not be ascertained in one child.

Table – VII

Minor Bleed as per etiology

SI No.	Etiology	No. of children (N)	Percentage (%)
I	**Variceal**	11	**15.3**
	EHPVO	11	**15.3**
	Intra hepatic	Nil	**0**
II	**Non variceal**	61	**84.7**
Total		**72**	**100**

Minor bleeds were the mode of manifestation in 46.45% in of children with UGI bleed. Only 11(15.27%) of those children had varices, the rest of them 61(84.73%), had bleed due to non variceal causes.

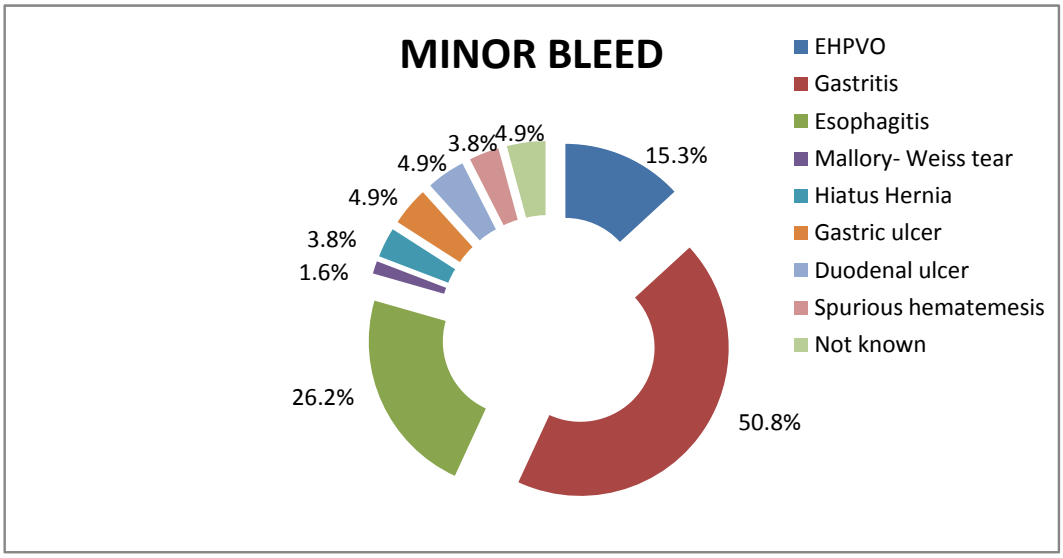


Figure - 7

Table - VIII

Non variceal causes of minor bleed

SI No.	Etiology	No. of children (N)	Percentage (%)
I	**GIT lesions**	56	**91.8**
	Gastritis	31	50.8
	Esophagitis	16	26.2
	Mallory- Weiss tear	1	1.6
	Hiatus Hernia	2	3.8
	Gastric ulcer	3	4.9
	Duodenal ulcer	3	4.9
	A. V. Malformation	Nil	0
II	**Spurious hematemesis**	2	**3.8**
III	**Not known**	3	**4.9**
Total		**61**	**100**

Out of the 61cases of non variceal minor bleeds, 88% were due to mucosal lesions of GIT. The cause was spurious in 3.8% and not identifiable in 4.9% of cases, with the endoscopic picture turning out to be normal.

Major bleeds were predominantly variceal bleeds (Table V), whereas the minor bleeds were predominantly non variceal bleeds (Table VII). This observation was found to be statistically significant. p value <0.001.

74(47.7%) children had taken ulcerogenic drugs, of which 46(62.1%) had UGI bleed due to non variceal causes and in another 28(18.7%) cases, the drugs were a trigger factor for bleed in previously diagnosed cases of EHPVO. In non- variceal group, in both the major and minor categories gastritis was the predominant lesion involved. The ulcerogenic drug was predominantly paracetamol in most of the cases. 2 of these children had taken steroids in addition to antipyretics. However no significant difference in clinical and endoscopic finding were noticed in those two children.

Pre treatment either in the form of IV Fluids or blood transfusion was given to 28 of these children. Resuscitation was required in an overall of 95(61.3%) children; and incidentally all the 83(87.3%) cases of major UGI bleed required some level of resuscitation. 12 cases of minor bleed also needed supportive measures for stabilization at admission.

Of these 95 children, 72 children improved with crystalloids while 23 required blood transfusion. In those who needed resuscitation, the cause was variceal bleeding in 62 children and non variceal in the remaining 33 children.

Table - IX

Clinical Presentation of UGI bleed based on etiology

SI No.	Symptom	Variceal	Non variceal	Total No.	Percentage (%)
1	Abdominal distension	40	2	42	27.1
2	Lt. quadrant mass	33	1	34	21.9
3	Abdominal pain	5	56	61	39.3
4	Fever	28	46	74	47.7
5	Pallor	53	36	89	57.4
6	Jaundice	4	0	4	2.6
7	Vomiting	24	34	58	37.4
8	Diarrhoea	2	0	2	1.2
9	Dysphagia	0	2	2	1.2
	Clinical signs				
1	Anaemia	56	42	98	63.2
2	Icterus	4	0	4	2.6
3	Prominent abdominal veins	6	0	6	3.8
4	Splenomegaly	61	2	63	40.6
5	Hepatomegaly	0	4	4	2.6
6	Free fluid	8	0	8	5.2

Of the 155 children, abdominal distension was the presenting complaint along with UGI bleed in 42 children, of which 38 were variceal bleeders and 4 non variceal bleeders. Abdominal pain was the complaint in 39.3% cases .47.7% of the children had fever on presentation. 37.4% had vomiting and 1.2% each had diarrhea or dysphagia.

A history of neonatal umbilical sepsis or UVC was present in 53(34.2%) cases and 52 among them were diagnosed with extrahepatic PHT. One child with history of UVC ,however did not develop features of EHPVO. Thus 82.5% (52/63) of children with EHPVO had a history of umbilical sepsis or UVC in the neonatal period.

Clinical Examination findings in children with UGI bleed

On examination 98 (6 1.5%) of children were found to be anemic; 4 children (2.4%) each had icterus and hepatomegaly, and prominent abdominal veins was found in 6(5.3%) children.

Splenomegaly was clinically evident in 63 (92.8%) cases. 61 of them were cases of EHPVO, with no evidence of liver cell failure. 2 children with non variceal causes for bleeding too had splenomegaly.

A clinical diagnosis of non variceal bleed was made in 35 children with a history of ulcerogenic drug ingestion and first episode bleed, usually of minimal quantity. Abdominal pain was an important accompanying symptom in 56 (58.33%), of non variceal bleeders (56/90), the commonest etiology of which was made out to be gastritis and esophagitis after UGI scopy.

Table – X

Clinical Presentation of UGI bleed based on category of bleed

SI No.	Symptom	Major	Minor	Total No.	Percentage (%)
1	Abdominal distension	38	4	42	27.1
2	Lt. quadrant mass	33	1	34	21.9
3	Abdominal pain	38	23	61	39.3
4	Fever	34	40	74	47.7
5	Pallor	60	29	89	57.4
6	Jaundice	4	0	4	2.6
7	Vomiting	42	16	58	37.4
8	Diarrhoea	2	0	2	1.2
9	Dysphagia	2	0	2	1.2
	Clinical signs				
1	Anaemia	65	33	98	63.2
2	Icterus	4	0	4	2.6
3	Prominent abdominal veins	6	0	6	3.8
4	Splenomegaly	53	10	63	40.6
5	Hepatomegaly	1	3	4	2.6
6	Free fluid	8	0	8	5.2

Abdominal distension was the predominant presenting complaint along with major UGI bleed in 42 (27.1%) children. Vomiting was yet another important symptom among major bleeders (37.4%). Anemia was present in 65 major bleeders and 33 minor bleeders (63.4%) and this carried statistical significance. ($p<0.01$) Splenomegaly was present in 63 children, 53 of them were major variceal bleeders.

Investigation Findings

Hemoglobin was less than l1gmldl in 63.2% of children. The total and differential leukocyte counts were elevated in 34.2% children and decreased in 27.2% children. The remaining children had counts within normal limits.

The serum bilirubin and liver enzymes was elevated in 2 patients with CLD secondary to Wilson's. These 2 children had in addition prolonged BT, CT and PT. The 2 children showed hypoalbuminemia and reversal of A:G ratio.The liver enzymes were significantly elevated in 14 children.

Ultrasonogram, was done in all 155 children. Among the 65 variceal bleeders, 63 had features of EHPVO and the remaining two showed evidence of a cirrhotic liver. The ultrasonogram findings were normal in most cases of non variceal bleed.

Table –XI

Endoscopy findings in UGI bleed

SI No.	UGI Scopy findings	Total No. (N)	Percentage (%)
I	**Variceal**	**65**	**41.9**
	Grade I Varices	4	2.6
	Grade II/III Varices	43	27.7
	Grade IV Varices	7	4.5
	Congestive gastropathy	6	3.8
	Sclerosed varix	5	3.2
II	**Non variceal**	**90**	**58.1**
	Gastritis	43	27.7
	Oesophagitis	23	14.8
	Mallory- Weiss tear	3	1.9
	Lax LES	4	2.6
	Gastric ulcer	5	3.2
	Duodenal ulcer	4	2.6
	Duodenal polyp	1	0.6
	A. V. Malformation	1	0.6
	Spurious hematemesis	2	1.3
	Normal study	4	2.6
Total		155	100

Of the variceal lesions, 27.7% had Grade 2 &3 varices. 4.5% had grade 4 varices with markers of impending bleed. Sclerosed varices and congestive gastropathy were the findings in 3.2% and 3.8% respectively.

Of the non variceal bleeders, gastritis was made out in 43 children (27.7%).Gastritis was noticed in body of the stomach in 22(51.6%), 11(25.6%) in antrum and in 10(23.2%) it was a diffuse lesion.

23 children showed evidence of esophagitis (14.2%), 3 (1.9%) children had Mallory- Weiss tear, 5(3.2%) children had gastric ulcer 4(2.6%) had duodenal ulcer. Duodenal polyp and A. V. malformation was present in one each (0.6%).

Endoscopy was normal in 6 cases and in these children ENT, dental and hematological assessments were done. After all investigations, spurious hematemesis was diagnosed to be the source of upper GI bleed in 2 cases, of which 1 was due to dental caries and the other was due to pharyngitis.

Despite all appropriate and extensive investigations, the cause of bleed could not be ascertained in 4 children.

DISCUSSION

In this study, 155 children in the age group of 3 months to 12 years, who presented with the primary symptom of overt upper gastrointestinal bleed during the study period were included and evaluated. We had 65 cases of variceal bleed and in the remaining 90 cases, the causes were non variceal, which included mucosal lesions of GIT, A- V malformations, Mallory-Weiss tear, spurious hematemesis etc. The cause could not be ascertained in a few children despite detailed and appropriate investigations.

No sex predilection was observed in this study. male:female -1.1:1. But the M:F ratio among children above 6 years of age showed male preponderance.(1.4:1)

About 60 children had atleast one episode of UGI bleed before the study period. Among them, 53(88.3%) had varices as the cause of recurrent bleed. This is consistent with a study by **Mittal SK,**[16] et al, who in his review of 70 cases of EHPVO, reported that these children have a minimum of 3 to 5 episodes of bleeding before presentation. Similar observation was made by **Fonkulsrud**[76].

Major bleeds were the mode of presentation in 53.54% of children, and minor bleeds in 46.46% of cases. About 65.06 % of cases of major bleeds were due to varices, consistent with observations made by **Tanner,** et al[9], **Boyle JT** et al[15], and **Robert Squires** et al[14].

Minor bleeds were mostly due to non-variceal causes (84.7%); most commonly encountered being the mucosal lesions of GIT. This is comparable to observations by **Robert Squires,** et al[14] and **Abraham Bogoch,** et al[21].

The difference observed in the incidence of major and minor GI bleeds among variceal and non variceal groups was found to be statistically significant.

Resuscitation was required in a total of 95 cases ,62 of them(65.2%) being variceal bleeders and 33 (34.8%) non variceal bleeders.

Left hypochondrial mass (splenomegaly) was the presenting complaint in 40.6%of cases and out of them, 96.8 % (61/63) had varices as the source of bleeding, the incidence is consistent with that observed by **Webb, LJ** et al[21].

Anemia (Hb < 11 gm/dl) was observed in 63.2% of children which could be presumed to be due to the blood loss. Prominent abdominal veins were seen in 6 cases of which 2 cases were due to chronic liver disease with portal hypertension. Prominent abdominal veins were seen only in 6.3 % (4/63) cases of EHPVO and this concurs with studies by **Tanner S[76]** and **Webb and Sherlock[21].**

History of ulcerogenic drug ingestion was present in 72 cases, of which 46 (68.16%) had evidence of mucosal lesions of GIT, while in 26 (31.84%) cases the drug intake was a trigger factor for bleeding in already existing varices. The role of ulcerogenic drugs in upper GI hemorrhage has been reported in many studies.

In this study, it was found that 65 cases were due to variceal hemorrhage. Of the variceal lesions, 27.7% had grade 2 &3 varices. 4.5% had grade 4 varices with markers of impending bleed. Sclerosed varices and congestive gastropathy were the findings in 3.2% and 3.8% respectively.

Of the non variceal bleeders, gastritis was made out in 43 children (27.7%).Gastritis was noticed in body of the stomach in 22(51.6%), 11(25.6%) in antrum and in 10(23.2%) it was a diffuse lesion.

23 children showed evidence of esophagitis (14.2%), 3 (1.9%) children had Mallory- Weiss tear, 5(3.2%) children had gastric ulcer 4(2.6%) had duodenal ulcer. Duodenal polyp and A. V. malformation was present in one each (0.6%).

Table - XII

Comparison of etiologies among various studies

Series	Yachha et al. 1996 Lucknow, India N=76	Mittal et al. 1994 New Delhi, India N=236	Cox et al. 1979 Los Angeles, USA N=68	This study
Type of population	Pediatric	Pediatric	Pediatric	Pediatric
Portal hypertension	95%	39.41%	10.29%	41.90%
Drug induced gastritis	1.30%	7.20%	13.23%	27.70%
Esophagitis	-	23.73%	14.70%	14.83%
Peptic ulcer disease	-	2.11%	38.22%	5.80%
Others	3.90%	27.54%	23.52%	9.77%

As with most other Indian studies, extra hepatic PHT was found to be the commonest cause of bleed in our study too.

Cadranel, et al, in their study of endoscopy of GIT in children found that GI bleeding was the commonest indication for endoscopy. Of the UGI bleeds, esophagitis (50%), was the most frequent lesion, followed by gastritis (14.2%), gastric ulcer (14.2%), duodenitis (14.2%) and varices (7.4%)[47], but our study showed gastritis as the predominant mucosal lesion.

Kenneth Cox, et al, in a retrospective study of upper GI hemorrhage in 68 children and adolescents who were less than 19 years reported the five most common causes of bleeding, in descending order of frequency as duodenal ulcer (20.5%), gastric ulcer (17.6%), esophagitis (14.7%), gastritis (13.2%) and esophageal varices (10.2%). However this marked difference in this study was due to the inclusion of patients upto 19 years of age and this could be the reason for higher incidence of mucosal lesions of GIT than that of varices[48].

Hargrove, et al, in their study of children below 24 months of age found that duodenal ulcer (52%), gastric erosion (16%) and duodenitis (16%) were the commonest lesion in infancy. In this study, endoscopy was performed in 45% of all children without sedation[51]. All the children in our study underwent UGI scopy under conscious sedation.

Table - XIII

Etiology of upper gastrointestinal bleeding in western studies

Series	Age group (years)		
	Infancy	**1 to 6**	**7 to 18**
Gleason, et al	GU	GU	DU/GE/Varices
Cox and Ament	GE	GU	DU
Graham, et al	Duo	GU	DU
Liebman, et al	GE	-	-
Gryboski, et al	GE	DU/GE	DU
Hargrove, et al	DU/GE	Duo/GU	-

* GU – Gastric Ulcer, DU – Duodenal Ulcer, Duo – Duodenitis, GE – Gastric Erosion

Mittal SK, et al, in a diagnostic study of upper GI endoscopy of 236 children (upto 12 years) from Northern India, who presented with a history of hematemesis, observed that varices were the commonest lesions (in 39.41%), followed by esophagitis (23.73%). Gastritis, gastric ulcer, duodenal ulcer and esophageal ulcers were identified in 7.20%, 12.7%, 0.42% and 0.42% cases respectively. Causes of bleeding could not be ascertained in 27.54% cases. This study is consistent with our observations[16]. However a higher incidence of gastritis was noticed in this study.

Table - XIV

Comparison of endoscopy findings among various studies

Endoscopic Findings	Western series (%)	North Indian study (%)	This study (%)
Normal	16.5	27.54	3.9
Varices	9.1	39.41	41.9
Esophagitis	14.1	23.73	14.8
Gastritis	32.5	8.47	27.7
Duodenal ulcer	24.1	0.42	2.6
Others	4.1	0.42	9.1

Thus, while western studies reported esophagitis, peptic ulcers and gastric erosions as the commonest lesions in upper GI bleeds, we observed varices as the commonest cause of upper GI bleed followed by gastritis and esophagitis, comparable to the **S.K. Mittal** study from north India. In our study peptic ulcer as a cause of bleed was found only in 2.6% of total cases.

In this study, we observed that esophageal varices were of grade II and III in 66.2% of cases, congestive gastropathy in 9.2% and sclerosed varices in 7.7% of cases. Cherry red spots and hematocystic spots were visualized in 8 children and these along with increased variceal sizes are the risk factors of variceal bleeding as reported by **S.K. Sarin,** et al and others[7].

By correlating the clinical and endoscopic findings, we observed, that varices secondary to PHT as the source of UGI bleed were diagnosed clinically in all the cases.

Non variceal lesions were diagnosed clinically in 72.2% (65/90) of cases. In the case of non variceal bleeds due to mucosal lesions of GIT, endoscopy diagnosed 84 lesions as against 65cases diagnosed clinically. The diagnostic yield for endoscopy was 1.3 fold than the clinical diagnosis.

Overall clinical diagnosis was possible in 83.3%. (130/155) of cases and this was especially due to the large number of variceal bleeds diagnosed, clinically.

After necessary investigations and endoscopy, the diagnosis was arrived at, in 96.1% (149/155) of cases. The difference in the percentage of diagnosis made clinically and after endoscopy, was mainly because of the effectiveness of endoscopy in diagnosing the mucosal lesions of GIT.

The, diagnosis could not be reached in 4(2.5%) of the cases, despite adequate investigations, which was comparable to other studies.

SUMMARY AND CONCLUSION

- A total of 155 children aged 3 months - 12 years with upper gastro intestinal hemorrhage were taken up for the study.
- This study showed a male female ratio of 1.1:1 among UGI bleeders.
- Most of the major bleeds were due to variceal etiology (65.1%), while majority of the minor bleeds were due to non-variceal causes (84.7%).
- Recurrent bleeds occurred most frequently with varices (92.8%).
- Ulcerogenic drug ingestion was present in 47.7% of the total cases, of which 62.11% showed evidence of GI mucosal changes and in another 18.73%the drugs were a trigger factor for bleeds in already existing varices.
- Splenomegaly in children with upper GI bleeds, was found to occur most commonly (in 96.8%) due to portal hypertension and was a reliable clue in clinical diagnosis.
- The etiology of upper GI hemorrhage was predominantly varices in 41.93%, of cases. The other causes were others were gastritis in 27.7%, esophagitis in 14.8%, Mallory-Weiss tear in 1.9%, hiatus

hernia in 2.5% and A.V.malformation and duodenal polyp each in 0.6%. Peptic ulcer disease was noticed only in 5.8%of total bleeds.

- Spurious hematemesis was the cause in 1.3% of these children.
- **Endoscopy plays a very important role in the diagnosis and therapy of gastrointestinal bleed of variceal and non variceal etiology. Hence, endoscopy is recommended in all cases of UGI bleed.**

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CLINICAL PROFILE OF CHILDREN WITH UPPER G I BLEED

Proforma

Name :

Age :

Sex :

I.P.No :

Address :

History :

Present illness :

Type of bleed :

Group I : frank blood in vomitus

Group II : coffee ground vomitus

Group III : melena

Amount of bleed :

No of episodes of bleed :

Time of bleed :

Time of presentation :

Time interval between episodes :

(in case of multiple episodes prior to the presentation)

Treatment of previous episodes : (if any)

Any trauma, head injury or recent surgery preceding the bleed:

ASSOCIATED GI SYMPTOMS

Vomiting :

Diarrhoea :

Abdominal pain :

Difficulty in swallowing :

ASSOCIATED SYSTEMIC SYMPTOMS:

Fever :

Rash :

Joint pains :

Palpitation :

Cool extremities :

Altered sensorium :

PAST HISTORY OF

Gastro Intestinal Diseases :

Liver Diseases :

Bleeding Disorders :

Use of medications :

(NSAIDS, warfarin, antibiotics)

Any abdominal surgery :

NEONATAL HISTORY OF

Umbilical sepsis :

Umbilical vein catheterisation :

FAMILY HISTORY OF

Gastro Intestinal Diseases :

Liver Diseases :

Bleeding Disorders :

PHYSICAL EXAMINATION

VITALS	:						
Pulse	:	BP	:	Temp	:	CRT	:
Nutritional status	:	Wt	:	Ht	:		

GENERAL EXAMINATION

Pallor	:
Jaundice	:
Petechiae, ecchymosis	:
Clubbing	:
Pedal edema	:
Generalised lymphadenopathy	:
Any evidence of shock	:
Any evidence of vasculitis	:

EXAMINATION OF THE ENT

Epistaxis	:
Nasopharyngeal congestion/oozing	:
Tonsillar enlargement and bleeding	:
Bleeding gums	:

EXAMINATION OF THE ABDOMEN

Distension	:
Dilated veins over the abdomen	:
Free fluid	:
Surgical scars	:
Hepatomegaly	:
Splenomegaly	:

EXAMINATION OF OTHER SYSTEMS :

CARDIOVASCULAR SYSTEM :

(any evidence of decompensation

of circulatory status)

RESPIRATORY SYSTEM :

(any possibility of hemoptysis)

CENTRAL NERVOUS SYSTEM :

(any evidence of hepatic encephalopathy)

INVESTIGATIONS :

CBC :

PS :

Blood grouping & Typing :

Renal Function Tests :

Liver Function Tests :

Coagulation profile :

Any others (specify) :

USG Abdomen :

CLINICAL IMPRESSION :

Upper G I Endoscopy :

ANNEXURE

ABBREVIATIONS

BP	-	Blood Pressure
BT	-	Bleeding Time
CLD	-	Chronic Liver Disease
CRT	-	Capillary Refill Time
CT	-	Clotting Time
DC	-	Differential Count
Hb	-	Hemoglobin
Ht	-	Height
DU	-	Duodenal Ulcer
DUO	-	Duodenitis
EHPVO	-	Extra Hepatic Portal Venous Obstruction
ENT	-	Ear, Nose and Throat
Eso	-	Esophagitis
EST	-	Endoscopic Sclerotherapy
EVL	-	Endoscopic Variceal Ligation
GE	-	Gastroenterology
GERD	-	Gastro-Esophageal Reflux Disease
GI	-	Gastrointestinal
GIT	-	Gastrointestinaltract

GU	-	Gastric Ulcer
IGV_1	-	Isolated gastric varices Grade I
IGV_2	-	Isolated gastric Varices Grade II
I.P.	-	In Patient
ITP	-	Idiopathic Thrombocytopenic Purpura
LCM	-	Left Costal Margin
Lt.	-	Left
OGV_1	-	Oesophagogastric Varices Grade I
OGV_2	-	Oesophagogastric Varices Grade II
PHT	-	Portal hypertension
PCV	-	Packed Cell Volume
PT	-	Prothrombin Time
RCM	-	Right Costal Margin
SAP	-	Serum alkaline Phosphatase
SGOT	-	Serum glutamate Oxalo Transferase
SGPT	-	Serum glutamate Pyruvate Transferase
SPSS	-	Statistical Package For Social Sciences
TC	-	Total count
TIPS	-	Transjugular intrahepatic portosystemic Shunt
UGI	-	Upper gastrointestinal
Wt	-	Weight