International Journal of Biomedical Research
ISSN: 0976-9633 (Online); 2455-0566 (Print)
Journal DOI: https://doi.org/10.7439/ijbr
CODEN: IJBRFA **Original Research Article**

Spectrum of upper gastrointestinal bleed in children

Sadhana R. Zope[1], Radha G. Ghildiyal[2] and Prachi S. Karnik[*3]

[1]Assistant Professor, [2]Professor and Head, [3]Assistant Professor,
Department of Pediatrics, Lokmanya Tilak Municipal Medical College and General Hospital, Sion, Mumbai – 400022

QR Code

***Correspondence Info:**
Dr. Prachi S. Karnik
Assistant Professor
Department of Pediatrics,
Lokmanya Tilak Municipal Medical College and General Hospital,
Sion, Mumbai – 400022

***Article History:**
Received: 22/11/2017
Revised: 24/11/2017
Accepted: 26/11/2017
DOI: https://doi.org/10.7439/ijbr.v8i11.4487

Abstract

Aims and Objectives: To study the etiology, occurrence and pattern of upper gastrointestinal haemorrhage and investigations this would aid in the early diagnosis and management of children with upper gastrointestinal haemorrhage.
Methods: This study was conducted over two years on 50 children below 12 years of age who presented with upper gastrointestinal bleeding, at a tertiary care hospital. All the cases were analysed by taking a detailed history and examination. An oesophagogastroduodenoscopy was done in indicated cases. The patients were treated according to standard guidelines and were followed up for a period of two year. Bleeding control was assessed during follow up based on check scopy findings.
Results: The majority of children who presented with upper gastrointestinal bleedwere in the age group of 6-10 years with male predominance with male to female ratio being 1.3:1. EHPVO was the most common cause. Among 15 cases of EHPVO, 12 required endoscopic interventions, with recurrent bleed in 3 patients (25%). Oesophageal varices were the commonest finding seen on endoscopy. Of the 14 patients (66.6%) who followed for check scopy, 78.5 % showed no bleed on follow up and 21.5% showed small varices not requiring any intervention. The mortality was 26% (13) in patients with UGIB. Patients who had an underlying hepatic failure and septicaemia had higher mortality as compared to other patients.
Conclusion: The outcome of children with EHPVO depends on the control of bleeding. Sclerotherapy and banding are effective in long-term variceal bleeding control. EHPVO was associated with better outcome inpatients with UGIB.
Keywords: Gastrointestinal haemorrhage, Oesophagogastroduodenoscopy, EHPVO, Oesophageal varices, Septicaemia, Sclerotherapy.

1. Introduction

Upper gastrointestinal bleed in children is a special challenge for the medical team and parents. The causes range from benign treatable to severe life threatening [1]. Seriously ill patients require timely, focused, and appropriate assessment and treatment. In contrast, patients with trivial bleeding who are not seriously ill require patience, reassurance, limited assessment and little if any treatment [2].

Hematemesis means bloody vomitus. It is the most common manifestation of upper gastrointestinal bleeding [3]. It is usually associated with passage of altered blood per rectum; sometimes with shock and death. It is quite alarming for the patient as well as care takers. Though most cases are self-limiting and benign it may turn into medical and /or surgical emergency [4].

The most important complication of haemorrhage is circulatory impairment and tissue hypoxemia. A 15% loss of blood volume is usually readily tolerated and compensated by contraction of large veins and recruitment of fluid from extra vascular sites. As volume depletion becomes greater, constriction of arterioles, shunting of cardiac output from non-vital areas as skin, bone, tachycardia, and orthostatic hypotension occur. After 40 - 50% of depletion of blood volume, complete loss of ability

to compensate occurs with shock, impaired flow of blood to vital organs, tissue hypoxemia, lactic acidosis and ultimately death [5]. Rapid correction of blood volume is essential. The goal of therapy is to decrease blood loss and protect each link in delivery of oxygen to areas by restoring blood volume. Medical management is the mainstay of treatment along with preventive measures. If needed, surgical management depending on the etiology and severity of bleeding may be needed.

The present research was undertaken to study the etiology, occurrence and pattern of upper gastrointestinal haemorrhage in children and investigations which would aid in the early diagnosis and management of children with upper gastrointestinal haemorrhage.

2. Materials and Methods

After obtaining Institutional Ethical Committee approval and parent's written informed consent, this prospective cohort study was conducted in the Department of Pediatrics at a Tertiary Care Hospital in Mumbai, over a period of two years. The study group included total 50 children below 12 years of age who presented with upper gastro intestinal bleed, either admitted in the pediatric ward or attending the outdoor pediatric clinic. The parents who were not willing to give written informed consent were excluded from the study.

All the cases were analyzed by taking a detailed history with special emphasis on the age of presentation of the bleed, etiology, associated symptoms, various blood investigations, requirement of urgent medical or surgical treatment, modalities of treatment required, scopy findings on admission and on follow up and their outcome after treatment. A thorough general and clinical examination was done for all the patients. The evaluation of vital parameters associated bleeding manifestations, evidence of liver cell failure, and a systemic examination done.

An oesophagogastroduodenoscopy was done in indicated cases in our hospital on Gastro video scope GIFV 70, Olympus. Pre-medication like midazolam was given in few patients, if required. All patients tolerated the procedure well with no post-procedure complications. Endoscopic ligations were performed with endoscopic ligation device, multi-band ligator with V grip band. In patients bleeding actively, ligations were performed at and around the site of bleeding. For endoscopic sclerotherapy, sclerosant used was 3% sodium tetradecyl sulfate diluted with saline to 1%. The varices were injected with 25 G needles with up to 2 ml sclerosant at the site. In addition, other investigations like antibody titres for autoimmune hepatitis, dengue titres, viral hepatitis titres, blood cultures were also done where relevant. Many of these cases were treated with medical line of management according to standard guidelines. In a majority of cases, two drugs and endoscopic interventions were required in controlling bleeding.

A regular follow up of the study cases was maintained during which detailed gastrointestinal examination, growth assessment along with check scopy, for need of repeat intervention if required, was done at 6 months, 12 months and 18 months.

2.1 Statistical Analysis

The data were analyzed for statistical significance using chi-square test (including the one-way classification of chi-square test, with or without Yate's correction for continuity) and Fischer's exact test. The results were considered statistically significant if the p value was less than 0.05 (Note- at some places, although p value was in the significant range it could not be interpreted well due to a small number of cases).

3. Observations and results

A total 50 children below 12 years of age with upper gastro intestinal bleed were included in the study. The majority of children who presented with UGIB were in the age group of 6-10 years with males (58%) presenting more frequently than females (42%) with the ratio being 1.3:1. EHPVO (30%) was the most common cause of UGIB in children, followed by drug induced gastritis (18%) and hepatic encephalopathy secondary to various etiologies (18%), (Table 1).

Table 1: Case distribution of patients presenting with UGIB

Diagnosis	No. of Cases	%
Aplastic anaemia	2	4.0%
Budd-Chiari syndrome	2	4.0%
Dengue h'gic fever	3	6.0%
Drug induced gastritis	9	18.0%
EHPVO	14	28.0%
EHPVO with hypersplenism	1	2.0%
Gastritis	1	2.0%
Hepatic encephalopathy in *P. falciparum* malaria	1	2.0%
Hepatic encephalopathy in viral hepatitis	1	2.0%
Drug induced hepatic encephalopathy	1	2.0%
Hepatic encephalopathy in Auto Immune hepatitis	2	4.0%
Hepatic encephalopathy in viral hepatitis	1	2.0%
Hepatic encephalopathy in Wilson disease	3	6.0%
Mallory Weiss tear	1	2.0%
Oesophagitis	1	2.0%
P. falciparum	2	4.0%
Septicaemia With DIC	5	10.0%

Of the cases who presented with drug induced gastritis, non-steriodal anti infammatory drugs was found to be the contributing factor in 55% of patients. The spectrum of endoscopic findings seen in the study cases were oesophageal varices (85.7%), antral gastritis (4.8%), Mallory Weiss tear (4.8%) and portal hypertension gastropathy (4.8%). Thus oesophageal varices were found to be the most common cause of UGIB. Out of 14 patients (66.6%) who followed for check scopy, 78.5 % showed no bleed on follow up and 21.5% showed small varices not requiring any intervention. Of the 50 patients of UGIB, 8 patients (16%) required blood transfusion and supportive hemodynamic care.

Out of 15 patients of EHPVO, 12 required endoscopic intervention with recurrent bleed in 3 patients (25%). Of the 12 patients who needed surgical treatment, 9 patients (75%) underwent banding and 3 patients (25%) underwent sclerotherapy. Of the 9 patients who underwent banding, 7 patients (77.8%) had no bleeding on follow up scopy. 100% of patients who underwent sclerotherapy had no bleeding on follow up. This difference was not statistically significant, (Table 2).

Table 2: Correlation of endoscopic treatment and follow up scopy findings

Endoscopic treatment	Follow up check Scopy		Total
	Insignificant bleeding	No bleeding	
Post Banding	2 (22.2%)	7 (77.8%)	9 (100.0%)
Post Sclerotherapy	0 (0.0%)	3 (100.0%)	3 (100.0%)
No intervention	1 (33.3%)	2 (66.6%)	3 (100.0%)
Total	3 (21.4%)	11 (78.6%)	15 (100.0%)

The mortality was 26 % (13) in patients presenting with UGIB. Patients who had an underlying hepatic failure and septicaemia had a higher mortality as compared to other patients. Prolonged prothrombin time and high serum bilirubin levels were associated with poor prognosis.

4. Discussion

The present study was carried out on a total of 50 cases of hematemesis, to determine causes of UGIB and to analyze demographic, endoscopic findings and outcome. Mean age of the patients was 7.14 yearswith maximum cases ranging from 6-10 years (42%). UGIB was commonly observed in males (58%) as compared to 42% femaleswith a male: female ratio of 1.3:1.The reason for a male preponderance could be because parents were more concerned about a male child as compared to a female child and males were brought more frequently for medical aid than females. Our study was comparable with other studies [1, 6,7].

On analysing the etiology of hematemesis in our study, we observed a predominance of EHPVO (30%), followed by drug induced gastritis (18%), hepatic failure (18%) and septicaemia (10%). There was 1 case (2%) of Mallory Weiss tear, 2 (4%) cases of *P. falciparum* presenting as UGIB. Also, we observed 2 cases (4%) of aplastic anaemia and 9 patients (18%) presented with UGIB secondary to hepatic failure due to various etiologies. Hence the commonest cause of hematemesis was oesophageal varices. The severity of bleeding was more with varices secondary to EHPVO than other causes like drug-induced gastritis. Hence, the patients with EHPVO were referred more often and earlier for medical care at our tertiary centre. Hepatic failure was a major cause of hematemesis in our study. The reason for UGIB in these children was deranged liver function, leading to a deranged coagulation profile causing bleeding from upper gastrointestinal tract. Thus, a coexisting other life threatening disease has a high morbidity and mortality in patients presenting with UGIB. Etiology of hematemesis in this group was comparable with previous studies [1,6,8-11].

Of the 50 patients who presented with UGIB, 37 (74%) had associated melena at the time of presentation. About 100 to 200 ml of blood in the upper GI tract is required to cause melena, which may persist for several days after bleeding has ceased. An associated coagulation disorder is also responsible for melena in patients presenting with UGIB. Total 15 cases were diagnosed as EHPVO, with46% patients being in the age group of 6-10 years with males more commonly presenting with UGIB as compared to females which was comparable to other studies [12,13].

We observed maximum duration of hospital stay in patients with septicaemia with a mean hospital stay of 18 days, followed by *P. falciparum* malaria (13days) and EHPVO (7 days). The least duration of hospital stay was of 2 days, in the patients who presented with UGIB secondary to drug induced gastritis. Thus, variceal causes of UGIB were associated with long hospital stay as compared to non variceal causes. The reason for prolonged hospital stay in patients with septicaemia and *P. falciparum* malaria was because of severity of infection, associated abnormal coagulation profile, multi organ involvement and need for a longer course of treatment.

The present study showed that 18% cases of UGIB were because of drug-induced gastritis. Of these, in 55% cases, NSAIDS were the contributing factor, followed by one case each of Chloroquine and Sodium Valproate induced gastritis, which was comparable with other studies done by Leca E *et al*, Kalyoncu *et al*[14-16]. We recommend that NSAIDS should be used judiciously in pediatric patients.

Total 21 patients (42%) underwent oesophagogastroduodenoscopy (OGD scopy), 18 of them presented with UGIB had oesophageal varices on scopy followed by one each of antral gastritis, Mallory Weiss tear and portal hypertension with gastropathy. Our study was comparable with studies done by Haung IF *et al*, Yachha SK *et al* [17,18]. Being a tertiary referral centre, cases of esophageal varices were referred. We observed that oesophageal varices secondary to EHPVO was a more frequent presentation of UGIB in our institute. Eight patients (16%) presented in shock, requiring blood transfusion. Of these, 3 were of EHPVO and septicaemia with DIC each. In EHPVO, since the severity of bleed was more as compared to other causes of UGIB, patients required urgent hemodynamic support. The cause of shock in patients with septicaemia was because of generalized septicaemia and deranged coagulation function requiring repeated blood and platelets transfusions.

Out of 15 patients of EHPVO, 3 (20%) required medical therapy alone and 12 (80%) required medical and endoscopic intervention both. Of these, all patients were given oral propranolol as primary and secondary prophylaxis and 5 patients (33.3%) were given injection somatostatin infusion also. Of 21 patients (42%) who underwent OGD scopy, 14 (66%) of them followed for check scopy, of these 78.5% had normal findings and 21.5% showed small varices which not needing any intervention. Total 12 cases underwent endoscopic treatment, of these 9 patients underwent banding, 7 patients (77.8%) had no bleeding on follow up scopy and 2 (22.2%) showed small varices on follow up scopy. Out of the 3 who underwent sclerotherapy, none had a bleed on follow up. The difference was not statistically significant as our sample size was too small to arrive at a conclusion.

During the study, total 13 (26%) patients expired; the cause of high mortality in these patients was associated co-morbid conditions, multi organ involvement, and not UGIB per se. The maximum mortality was with aplastic anaemia (100%), followed by patients with septicaemia with DIC (80%) and followed by those with hepatic encephalopathy secondary to various etiologies. Out of 2 patients diagnosed as aplastic anaemia, both expired, showing 100% mortality. The mortality in these patients was not because of UGIB per se but because of the underlying pancytopenia and secondary infections due to decreased immunity leading to poor outcome. Of the 5 patients diagnosed as septicaemia with DIC presenting as UGIB, 4 (80%) expired during the study and only one survived (20%). The high mortality in these patients was because of generalized infection, consumption coagulopathy leading to bleeding from all sites, with multi organ failure and not the upper gastro intestinal bleeding. Patients diagnosed as drug induced gastritis (18%), *P. falciparum* with or without hepatic encephalopathy (4%), dengue haemorrhagic fever (2%) and Mallory Weiss tear (2%) had a 100% cure rate. Patients diagnosed as EHPVO, all (100%) were symptom free after treatment. There was a low mortality with these patients because of less severity of the disease, no multi organ involvement, early diagnosis and intervention. Of the nine patients diagnosed with hepatic encephalopathy secondary to various etiologies, 8 patients (88.9%) expired and 1 patient (11.1%) survived. Our study showed a high mortality among the patients with hepatic encephalopathy (80%) as compared to the other studies [11,13,19,20]. The reason for high mortality in these groups was late referral to our centre. All patients were treated symptomatically for hepatic encephalopathy.

Among the 18 patients showing abnormal serum bilirubin, 9 (50%) expired as compared to 4 patients (12.5%) of the 32 having normal levels and difference was statistically significant ($p<0.005$). Patients with abnormal prothrombin time (22/50) had a higher mortality (54.5%) as compared to a mortality of 3.6% in a group having normal prothrombin time. This association was also statistically significant. The cause of mortality in these patients was not UGIB per se, but the underlying deranged coagulation profile and liver function leading to multi organ damage and death. Our results compare with other studies [11,13,19].

5. Conclusion

We concluded that the outcome of children with EHPVO depends on the control of bleeding. Sclerotherapy and banding are effective in long-term variceal bleeding control. In EHPVO, there is no underlying hepatic dysfunction or abnormal coagulation profile; hence it is associated with a better long-term prognosis as well as associated with a better outcome of patients with UGIB.

Acknowledgement

The authors sincerely thank the Department of Pediatrics and Dean of the Topiwala National Medical College, Mumbai for permission to carry out this study.

Reference

[1]. Rafeey M, Khalafi J. Available from: URL: http://medicaljournal.tbzmed.ac.ir/PDF/bahar%2085/eng/10.pdf.Accessed on Dec 7 2010.

[2]. Heitlinger LA, McClung HJ. Gastrointestinal hemorrhage. In: Wyllie R, Hayms JS eds. Pediatric Gastrointestinal Disease. Pathophysiology, Diagnosis, and Management, 2nd edn. Philadelphia, WB Saunders; 1999: 64-72.

[3]. Okello TR. Upper gastrointestinal endoscopic findings in adolescents at Lacor hospital, *Uganda Afr Health Sci* 2006; 6(1): 39-42.

[4]. Fleisher G, Ludwig S, ed. Gastrointestinal bleeding. Pediatric Emergency Medicine, 4th ed. Philadelphia: Lippincott Williams and Wilkins; 2000; 30:275-282.

[5]. Hirschowitz BI. Development and application of endoscopy. *Gastroenterology* 1993; 104:337-42.

[6]. Dehghani SM, Haghighat M, Imanich MH, Tabebordbar MR. Upper Gastrointestinal Bleeding in Children in Southern Iran. *Indian Journal of Pediatrics* 2009; 76:635-638.

[7]. Zahavi I, Arnon R, Ovadia B, Rosenbach Y, Hirsch A, Dinari G. Upper gastrointestinal endoscopy in the pediatric patient. *Isr J Med Sci* 1994; 67-69.

[8]. Mittal SK, Kalra KK, Aggarwal V. Diagnostic upper GI endoscopy for hemetemesis in children: experience from a pediatric gastroenterology centrein north India. *Indian J Pediatr*1994; 61: 651-654.

[9]. Dass R, Barman H, Duwarah SG, Deka NM, Jain P and Choudhury V. Unusual Presentations of Malaria in Children: An Experience from a Tertiary Care Centre in orth East India. *Indian J Pediatr* 2010; 77(6):655-660.

[10]. Hussey S, Kelleher K, Ling SC. Prospective study of major upper gastrointestinal haemorrhage in children. *Clinical Pediatric Emergency Medicine*; 11; 207-16**.**

[11]. Aftab R, Iqbal I. Fulminant hepatic failure in children clinical features, etiology and outcome analysis. *Pak Paed J* 2007; 31(4):195-200.

[12]. Patrawala V, Das HS, Sawant P, Rathi P, Vyas K, Dhad Phale S, *et al*. A long term post sclerotherapy follow up of EHPVO children with upper gastrointestinal haemorrhage. *Bombay Hospital Journal*. 2001; 43(4): 514-7.

[13]. Poddar U, Thapa BR, Prasad A, Sharma AK, Singh K. Natural history and risk factor in fulminant hepatic failure. *Arch Dis Child* 2002; 87: 54-6.

[14]. Leca E, Grimaldi BL, Maurage C, Jonville-Bera AP. Upper gastrointestinal complications associated with NSAIDs in children. *Therapie*. 2007; 62(2):173-6.

[15]. Kalyoncu D, Urganci N, Cetinkaya F. Etiology of Upper Gastrointestinal Bleeding in Young Children. *Indian Journal of Pediatrics* 2009; 27:899-901.

[16]. Li Voti G, Acierno C, Tulone V. Relationship between upper gastrointestinal bleeding and nonsteroidal antiinflammatory drugs in children. *Pediatric Surg Int* 1997; 12: 264-265.

[17]. Huang IF, Wu TC, Wang KS. Upper gastrointestinal endoscopy in children with upper gastrointestinal bleeding. J Chin Med Assoc2003; 66: 271-275.

[18]. Yachha SK, Khanduri A, Sharma BC, Kumar M. Gastrointestinal bleeding in children. *J Gastroenterol Hepatol* 1996; 11:903.

[19]. Bendre SV, Bavdekar AR, Bhave SA, Pandit AN, Chitambar SD, Arankalle VA, *et al*. Fulminant hepatic failure: Etiology, viral markers and outcome. *Indian Paediatr* 1999; 36: 1107-12.

[20]. Lee WS, McKiernan P, Kelly DA *et al*. Etiology, outcome and prognostic indicators of childhood fulminant hepatic failure in the United Kingdom. *J pediatric gastroenterol Nutr*. 2005; 40(5): 575-81.