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Etiology and Outcome of Endoscopic Band Ligation in Esophageal Varices in Bangladeshi Children

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ABSTRACT
Background: *Esophageal varices is an important cause of upper gastrointestinal bleeding. It is a life-threatening condition in children. Among different modalities of treatment, endoscopic band ligation is the most common treatment for esophageal varices.*
Objectives: *To find out the etiology of portal hypertension and to see the outcome of endoscopic band ligation of esophageal varices in children with portal hypertension.*
Methods: *This prospective observational study was done in the department of Gastroenterology, Hepatology and Nutrition, BSHI. 30 subjects were included in this study from July 2019 – June 2021. Every case was treated with endoscopic band ligation. Cases were followed up for a minimum period of 1 year after the band ligation. Statistical analyses of the results were be obtained by using window-based Microsoft Excel and Statistical Packages for Social Sciences (SPSS-24).*
Results: *Age of the children was 3-18 years with mean 7.27±2.21 years and male: female ratio was 2:1. Out of 30 children 17 (56.7%) were extra-hepatic and 13 (46.6%) were hepatic causes of portal hypertension. Maximum patients 14 (46.6%) had grade III varices followed by 9 (30%) patients had grade IV and 7 (23%) patients had grade II with red sign. Most of the patients, 14 (46%) required 2 sessions and 11 (36%) patients required 3 sessions for variceal obliteration in both group and 5 (16%) cases of extra-hepatic children required only 1 session whereas hepatic cases required multiple sessions. Among 30 patients, 25 (83%) patients developed re-bleeding where 12 (40%) patients had early re-bleeding and 13 (43%) had late re-bleeding. No complications were noted except nausea and vomiting.*
Conclusion: *Extrahepic was the most common etiology and endoscopic band ligation was safe as well as effective in treatment of esophageal varices.*
Keywords: *Portal Hypertension, Esophageal Varices, Band ligation.*

I. INTRODUCTION

Portal hypertension is a common clinical syndrome defined by the elevation of hepatic venous pressure gradient (HVPG) above 5 mm Hg and is caused by a combination of two simultaneous occurring hemodynamic processes: (1) increased intrahepatic resistance to passage of blood flow through the liver due to cirrhosis and (2) increased splanchnic blood flow secondary to vasodilatation within the splanchnic vascular bed. [1] Many liver and vascular diseases cause portal hypertension in children, which may give rise to severe life-threatening complications such as esophageal varices, ascites, hepatopulmonary syndrome, portopulmonary hypertension, and hepatic encephalopathy. [2]

Esophageal varices are an important cause of upper gastrointestinal bleeding. The incidence of oesophageal varices increases in approximately 5% per year in patients with CLD and the rate of progression from small to large varices are approximately 5 – 10% per year. [3] It is characteristically abrupt and massive with an accompanying high mortality. The first episode of variceal bleeding is not only associated with a high

mortality but also with a high recurrence rate in those who survive. [4] A study showed that about one-third of patients with cirrhosis and esophageal varices experience bleeding in the course of the disease and after an initial bleeding from esophageal varices, re-bleeding occurs in about 60% of the patients. [5]

It is estimated that approximately 50% of pediatric patients with chronic liver disease and 90% of those with extrahepatic portal vein obstruction (EHPVO) will experience gastrointestinal bleeding. Extrahepatic portal hypertension has been reported to be the most common cause of upper gastrointestinal bleeding (UGIB) in children. [6] However Chronic liver disease (CLD) is not uncommon in paediatric population. A study carried out in Paediatric Gastroenterology and General Paediatrics department of BSMMU showed that 4 % of hospital admissions were due to liver diseases and of those 40% were due to chronic liver diseases. [7] Esophageal varices are present in about 60% of patients with an end-stage cirrhosis and in about 30% of patients with compensated cirrhosis.

Clinically, significant portal hypertension is diagnosed when clinical manifestations of the disease appear or the portal pressure gradient exceeds 10 mmHg. [8] Portal hypertension may manifest as gastrointestinal bleeding, splenomegaly and ascites. [9] Therefore, early diagnosis, proper management and regular follow up are essential. The gold standard for the diagnosis of portal hypertension is direct measurement of portal pressure or hepatic venous pressure gradient. [10] These measurements can be obtained only by invasive methods, which are not feasible in most centers of the world. There are a number of indirect ways to assess the status of oesophageal varices, these are barium swallow of oesophagus, ultrasonography and upper gastrointestinal endoscopy. The upper gastrointestinal endoscopy is currently the best reliable way to diagnose oesophageal varices and hence, PHTN. [10]

The treatment of portal hypertension has undergone a dramatic evolution in the last decade. Therefore, therapeutic modalities with the lowest complication rate and best long-term success would be preferable for the children. [11] Treatment modalities for oesophageal varices include pharmacological and endoscopic therapies. Pharmacological therapies are non-selective beta blockers and nitrates. Endoscopic therapies include injection sclerotherapy and endoscopic band ligation. [12] Endoscopic band ligation has replaced injection sclerotherapy because of comparable results, less complications and lower risk of rebleeding. [12]

In Endoscopic band ligation, rubber bands are used to ligate oesophageal varices with subsequent necrosis and sloughing of the varix followed by fibrosis and re-epithelialization. This technique was first described by Stiegmann & Colleagues in 1986. The multiband ligator has made variceal ligation easier and more comfortable for the patient. With this technique, up to 6 varices can be ligated after a single insertion of the endoscope. [13]

Esophageal variceal band ligation is the best form of management for variceal bleeding until liver transplantation or surgical shunting is unavoidably required as a result of recurrent bleeding. [14] The frequency of esophageal variceal band ligation (EVBL) for the eradication of esophageal varices has no consensus. Some authors reported a minimum of one month between banding procedures while others perform EVBL on a weekly basis. [14] But little data is available on the number of sessions required to achieve complete variceal eradication.

II. METHODOLOGY

This Prospective observational study was carried out in the Department of Pediatric Gastroenterology, Hepatology and Nutrition of Bangladesh Shishu (Children) Hospital and Institute. during July 2019 to June 2021. A total of 30 patients were participated in the study. Among them 3-18 years old children with esophageal varices who were admitted at the department of Paediatric Gastroenterology, Hepatology and Nutrition of BSHI during the study period. After taking consent and matching eligibility criteria, data were collected from patients on variables of interest using the predesigned structured questionnaire by interview, observation. Statistical analyses of the results were be obtained by using window-based Microsoft Excel and Statistical Packages for Social Sciences (SPSS-24).

III. RESULTS

Table I: Baseline characteristics of subjects (n=30)

Variables	No. of patients (n = 30)	Mean±SD	Percentage (%)
Age (years)		7.27±2.21	
Range (min – max)		4-11 years	
Gender			
Male	20		66.7
Female	10		33.3
Age of first variceal bleeding (in year)		5.3±1.21	
Extrahepatic		5.26±1.45	
Hepatic		5.33±1.21	

Data were expressed as frequency, percentage, mean ± SD and range
Table I shows total 30 cases were included in this study and their age range was 4-11 years. The mean age was 7.27±2.21 years. Male: Female ratio was 2:1. The mean age at onset of the first variceal bleeding was 5.3 ± 1.21 years. In extra-hepatic cases it was 5.26 ± 1.45 years and in hepatic cases it was 5.33±1.21 years. So, in extra-hepatic cases first variceal bleeding occurs slightly earlier than hepatic cases.

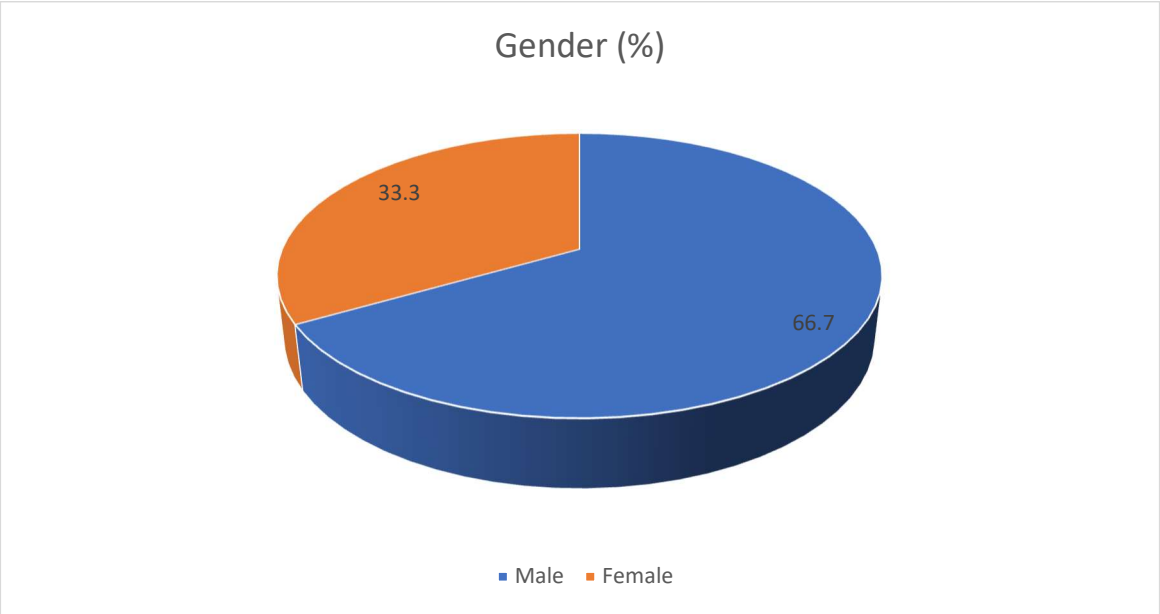

Figure-I: Gender distribution of subjects
Pie diagram shows that two third 20 (66.7%) patients were male and one third 10 (33.3%) were female.

Table II: Distribution of subjects by age (n=30)

Age (years)	Number of patients	Percentage (%)
≤5 years	9	30.0
6-9 years	14	46.7
≥10 years	7	23.3
Mean±SD	7.27±2.21	
	(4 – 11)	

Data were expressed as frequency, percentage, mean ± SD and range.
Figure in parenthesis indicate range.

Table II shows, among 30 study subjects maximum 46.7% patient in between 6-9 years followed by 30% patients age below 5 years and 23.3% patients more than 10 years. The age range of patients were between 4-11 years. The mean age was 7.27±2.21 years.

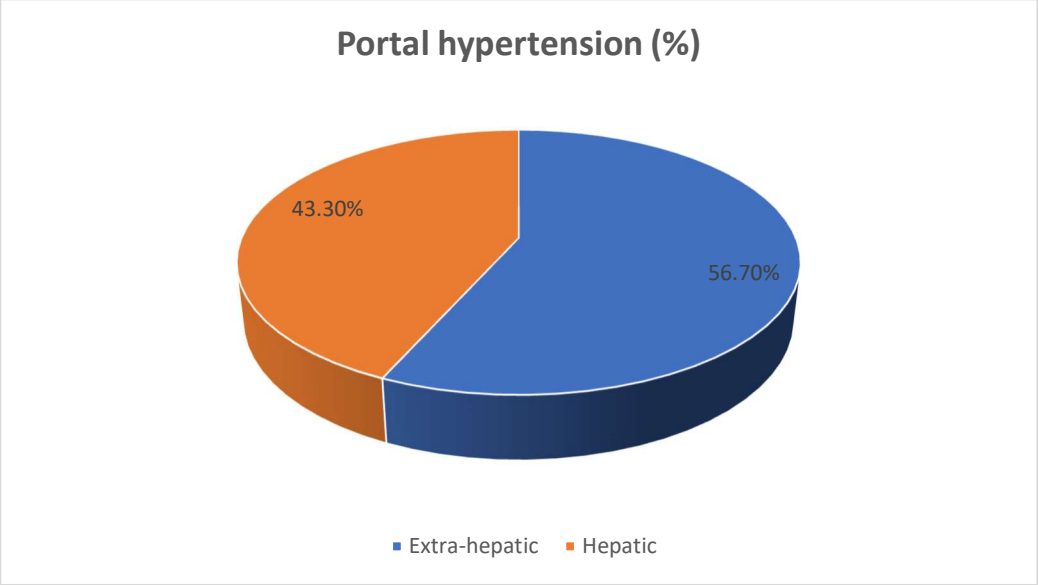

Figure- II: Etiology of portal hypertension (n=30)

Pie diagram shows that, the most common etiology of portal hypertension was extra hepatic 17(56.7%). Hepatic cause of portal hypertension was found 13 (43.3%)

Table III: Etiology of hepatic and extra-hepatic portal hypertension of studied subjects (n=30)

Etiology	Extrahepatic (n=17) No. (%)	Hepatic (n=13) No. (%)
Chronic Hepatitis B	0(0.0%)	1(7.7%)
Wilson disease	0(0.0%)	6(46.2%)
Biliary cirrhosis	0(0.0%)	1(7.7%)
Autoimmune hepatitis	0(0.0%)	2(15.4%)
Cystic fibrosis	0(0.0%)	1(7.7%)
Cryptogenic CLD	0(0.0%)	2(15.4%)
Portal vein thrombosis	3(17.6%)	0(0.0%)
Idiopathic cause	14(82.4%)	0(0.0%)
Total	17	13

Data were expressed as frequency, percentage

Table III showing: 17(56.7%) subjects had extra-hepatic portal hypertension, among them portal vein thrombosis 3(17.6%) and idiopathic cause 14(82.4%). Among 13(43.3%) patients in hepatic group of portal hypertension, maximum was Wilson disease 6(46.2%) followed by autoimmune hepatitis 2(15.4%), cryptogenic CLD 2(15.4%), chronic hepatitis B 1(7.7%), biliary cirrhosis 1(7.7%), cystic fibrosis 1(7.7%).

Table IV: History of studied subjects (n=30)

History	Extrahepatic (n=17) No. (%)	Hepatic (n=13) No. (%)	P value
H/O hematemesis			
Yes	17(100.0%)	11(84.6%)	0.094ns
No	0(0.0%)	2(15.4%)	
H/O melena			
Yes	12(70.6%)	10(76.9%)	0.697 ns
No	5(29.4%)	3(23.1%)	
H/O jaundice			
Yes	0(0.0%)	13(100.0%)	0.001s
No	17(100.0%)	0(0.0%)	
H/O consanguinity			

Yes	2(11.8%)	3(23.1%)	0.410 ns
No	15(88.2%)	10(76.9%)	
Family history of liver disease			
Yes	0(0.0%)	2(15.4%)	0.094 ns
No	17(100.0%)	11(84.6%)	
H/O blood and blood product transfusion			
Yes	0(0.0%)	1(7.7%)	0.245 ns
No	17(100.0%)	12(92.3%)	
H/O umbilical catheterization			
Yes	2(11.8%)	0(0.0%)	0.201 ns
No	15(88.2%)	13(100.0%)	

Chi-squared Test (χ 2) was done to analyze the data**.** *p≤ 0.05 was considered to be significant. s= significant, ns = not significant

Table IV shows maximum children of both groups had history of hematemesis and melena. H/O jaundice significantly more in hepatic group. Some children of both groups had H/O consanguinity, few children of hepatic group had family history of liver disease and H/O blood product transfusion, 2 children of extrahepatic group had H/O umbilical catheterization and there was no significant difference.

Table V: Comparison of physical examination between two groups (n=30)

Physical examination	Extrahepatic (n=17) No. (%)	Hepatic (n=13) No. (%)	P value
Anemia			
Mild	6(35.3%)	3(23.1%)	0.469ns
Moderate	11(64.7%)	10(76.9%)	
Jaundice			
Present	0(0.0%)	12(92.3%)	0.001s
Absent	17(100.0%)	1(7.7%)	
Stigmata of CLD			
Yes	0(0.0%)	6(46.2%)	0.002s
No	17(100.0%)	7(53.8%)	
Hepatomegaly			
Yes	1(5.9%)	13(100.0%)	0.001s
No	16(94.1%)	0(0.0%)	
Splenomegaly			
Yes	17(100.0%)	13(100.0%)	–
No	0(0.0%)	0(0.0%)	
Ascites			
Yes	3(17.6%)	12(92.3%)	0.245 ns
No	14(82.4%)	1(7.7%)	

Chi-squared Test (χ2) was done to analyze the data**.** *p≤ 0.05 was considered to be significant.
s= significant, ns = not significant

Table V shows, Jaundice, Stigmata of CLD, Hepatomegaly, Ascites were significantly higher in hepatic group ($p<0.05$).

Table VI: Comparison of laboratory test between two groups (n=30)

Physical examination	Extrahepatic (n=17) No. (%)	Hepatic (n=13) No. (%)	P value
Laboratory Test			
6-9 (gm/dl)	11(64.7%)	11(84.6%)	[a]0.278[ns]
>9 (gm/dl)	6(35.3%)	2(15.4%)	
Mean±SD	8.60±1.63	8.42±0.97	
Range	6.30-11.20	6.80-10.30	
Serum Bilirubin			
Normal	17(100.0%)	1(7.7%)	[b]0.001[s]
Raised	0(0.0%)	12(92.3%)	
Serum ALT			
Normal	17(100.0%)	0(0.0%)	[b]0.001[s]
Raised	0(0.0%)	13(100.0%)	
Serum Albumin			
Normal	16(94.1%)	2(15.4%)	[b]0.001[s]
Low	1(5.9%)	11(84.6%)	
INR			
Normal	17(100.0%)	13(100.0%)	–
Raised	0(0.0%)	0(0.0%)	

[a]P- value reached from unpaired t-test and [b]P- value reached from Chi-squared Test (χ^2). *p≤ 0.05 was considered to be significant. s= significant, ns = not significant

Table VI shows Hemoglobin level was found almost same in both groups. But liver function tests (serum bilirubin, serum ALT, serum albumin) were significantly altered in hepatic group (p<005).

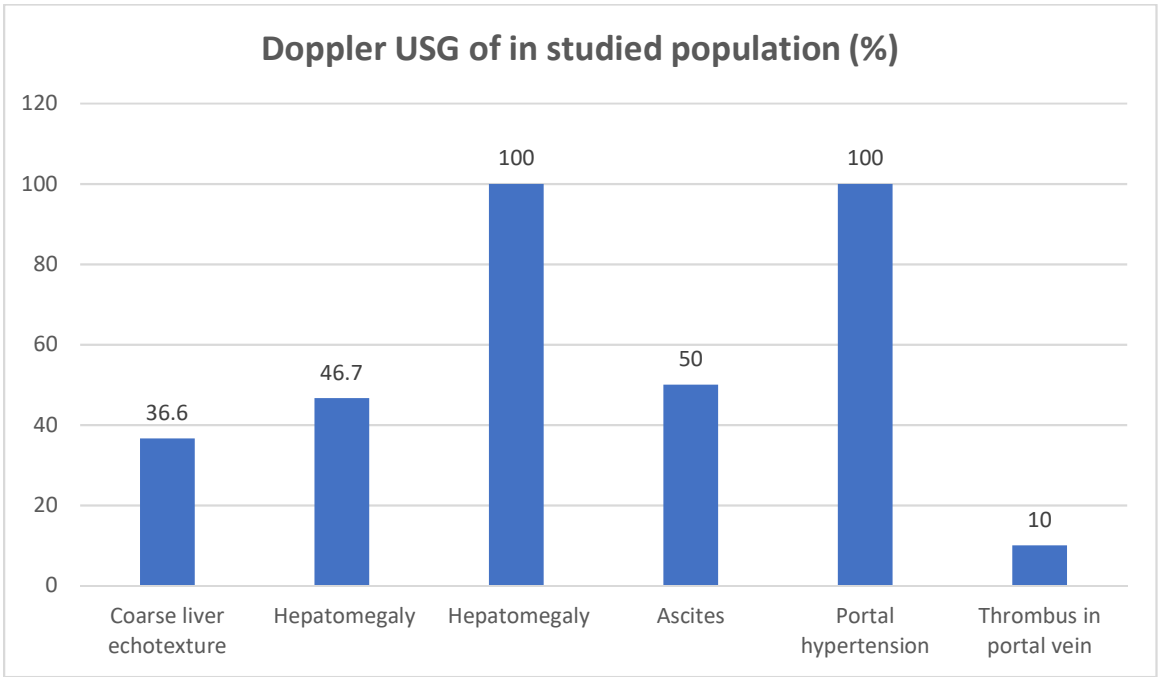

Figure-III: Doppler USG finding in studied population

Bar diagram shows that 100% patient had Portal hypertension was diagnosed by Doppler USG of hepatobiliary system. Others Doppler ultrasound findings are splenomegaly (100%), hepatomegaly (50%), ascites (50%), coarse liver echotexture (36.6%) and portal vein thrombus (10.0%).

Table VII: Grading of esophageal varices in the study subject (n=30)

Grading of esophageal varices	Extrahepatic (n=17) n(%)	Hepatic (n=13) n(%)	All patients (n=30) n(%)	P value
Grade II with red	6(35.3%)	1(7.7%)	7(23.3%)	0.182ns
Grade III	7(41.2%)	7(53.8%)	14(46.7%)	0.748ns
Grade IV	4(23.5%)	5(38.5%)	9(30.0%)	0.896ns
Total	17	13	30	

Chi-squared Test (χ 2) was done to analyze the data**.** *p≤ 0.05 was considered to be significant.
s= significant, ns = not significant

Table VII showing grading of esophageal varices, maximum patients had Grade III varices 46.7.0%, followed by Grade-IV esophageal varices 30% and Grade II with red sign 23.3%. Severity of grading (grade-III & IV) slightly higher in hepatic cases. No significant difference between groups (p>0.05).

Table VIII: Outcome of band ligation (n=30)

Physical examination	Extrahepatic (n=17) n (%)	Hepatic (n=13) n (%)	All patients (n=13) n (%)	P value
Number of band ligation				
Session 1	5(29.4%)	0(0.0%)	5(16.7%)	0.099ns
Session 2	8(47.1%)	6(46.2%)	14(46.7%)	1.000 ns
Session 3	4(23.5%)	7(53.8%)	11(36.7%)	0.185 ns
Number of bands required in every session				
Band 1	2(11.8%)	0(0.0%)	2(6.7%)	0.588ns
Band 2	10(58.8%)	10(76.9%)	20(66.7%)	0.514 ns
Band 3	5(29.4%)	3(23.1%)	8(26.7%)	1.000 ns
Control of bleeding				
Primary hemostasis	17(100.0%)	13(100.0%)	30(100.0%)	-
Re bleeding				
Early	5(29.4%)	7(53.8%)	12(40.0%)	0.328ns
Late	7(41.2%)	6(46.2%)	13(43.7%)	1.000 ns
No rebleeding	5(29.4%)	0(0.0%)	5(16.7%)	0.099ns

Chi-squared Test (χ 2) was done to analyze the data**.**
*p≤ 0.05 was considered to be significant.
s= significant, ns = not significant

Table VIII shows, maximum children of both groups required multiple sessions of band ligation whereas 5 children of extra-hepatic group required only 1 session. No significant difference found between two group. All children of both groups required 1-3 bands in each session. Primary hemostasis achieved in all children after endoscopic band ligation. Maximum children 25 (83.3%) developed re-bleeding among them early re-bleeding found 40.0%% and late re-bleeding 43.7%. No significant difference observed between extrahepatic and hepatic patients.

Table IX: Complication of band ligation of the study subject (n=30)

Outcome of band ligation	Extrahepatic (n=17) n(%)	Hepatic (n=13) n (%)	P value
Complication			
Nausea + Vomiting	13(76.5%)	11(84.6%)	0.580ns
Esophageal ulceration	0(0.0%)	0(0.0%)	-
Esophageal perforation	0(0.0%)	0(0.0%)	-
Aspiration pneumonia	0(0.0%)	0(0.0%)	-
Sepsis	0(0.0%)	0(0.0%)	-

Chi-squared Test (χ 2) was done to analyze the data**.** *p≤ 0.05 was considered to be significant. s= significant, ns = not significant

Table IX shows, endoscopic band ligation has less complications. Minor complication like nausea and vomiting observed in 76.5% cases in extrahepatic group and 84.0% in hepatic group, while major complications were not found in both groups. No significant difference found in between two groups.

IV. DISCUSSION

Esophageal varices due to portal hypertension is the most common cause of upper GIT bleeding. It is a life-threatening clinical situation in infants and children. Effective management is needed to treat this condition. There are different types of treatment modalities for esophageal varices. Among them endoscopic band ligation showed safer and more effective in adult practice. There was no previous study regarding use of endoscopic band ligation in children. In this study we evaluate the etiology and outcome of endoscopic band ligation in esophageal varices in Bangladeshi children.

In this study total 30 patients with portal hypertension were included. Their age range between 4 to 11 years. The mean (±SD) age of the studied patients was 7.27±2.21 years, among them male 66.7% and female 33.3%. Most 14 (46%) of the patients were in the age group between 6-9 years. Similar results were observed in another study done in Bangladesh by Karim where 31 (56%) were male and 24 (44%) females. [15] A study done by Mahmud in Dhaka Shishu Hospital also found patients age group between 2-16 years and 30 (60%) were male and 16 (40%) were female which is similar to this study. [16] In another study done in BSMMU, patient's age group was found between 2 to 15 years which was also similar to present study. [17]

In this study age of first variceal bleeding were found between 3.5 to 7.5 years. The mean (±SD) age at onset of the first variceal bleeding were 5.33 ± 1.21 years. In extra-hepatic cases it was 5.26 ± 1.45 years and in hepatic cases it was 5.33 ± 1.21 years. So, in extra-hepatic cases first variceal bleeding occurs slightly earlier than hepatic cases. Similar results were found in a study done by Mahmud. [16] They found the mean age at onset of the first variceal bleeding was 5.3 ± 4.5 years. In pre-hepatic cases it was 4.4 ± 3.6 years and in hepatic cases it was 7.6 ± 4.6 years. Similar observation was also reported in North Indian children by Arora. [17]

In this study the most common etiology of portal hypertension was extra-hepatic (56.7%). Among them portal vein thrombosis was 3 (17.6%) and idiopathic cause 14 (82.4%). Mahmud et al. (2017) also found similar results about 32 (80%) due to pre-hepatic causes and 8 (20%) due to hepatic causes. Among them portal vein thrombosis was 20 (62.5%), splenic vein thrombosis 4 (12.5%) and others 8(25%). Arora et al. (1988) also found similar results in their study. [17]

In present study hepatic cause of portal hypertension was found 13 (43.3%). Among them most common cause was wilson disease 6 (46.2%) which is similar to study done by Alam et al. (2018) in BSMMU. [18] But Alam et al. (2010) found infective hepatitis was the most common cause of CLD in a study done in Dhaka Shishu Hospital and Dar et al (2014) in Kashmir found HBV was the most common cause of CLD in children. [19, 20] It was observed that the pattern of etiology is regionally variable. H/O hematemesis and melena found in most of the studied subjects 28 (93%) in this study. Similar results were also found by Mondragón et al (2014) and Shrestha et al. (2017). [21, 22]

Regarding hepatic group of portal hypertension jaundice was the most common presenting feature followed by hepatomegaly, ascites and stigmata of CLD. These parameters were significantly different between hepatic and extrahepatic portal hypertension. Karim and Pargewar found similar results in their study. [23, 24] In this study, regarding liver function test serum bilirubin, serum ALT and serum albumin were significantly altered in cases of hepatic group. Whereas in extra-hepatic portal hypertension liver function test were normal which is similar to a study done by Chaudhary. [25]

Endoscopic band ligation eradicates esophageal varices with less complications. Minor complication like nausea and vomiting were observed in maximum children of both hepatic and extra-hepatic cases and no major complications was observed in present study which is similar to study done by Mahmud et al. (2017). [16] Khattak et al. (2013) found no major complication during the study and follow up period except development of post band ulcer in 3 patients out of 113. [26]

Limitations of the study

The present study was conducted in a very short period due to time constraints and funding limitations. The small sample size was also a limitation of the present study.

V. CONCLUSION

Study result concluded that extra-hepatic portal hypertension was the most common etiology in children and Endoscopic band ligation was effective as well as safer for the treatment of esophageal varices grade-II and above.

VI. RECOMMENDATION

This study can serve as a pilot to much larger research involving multiple centers that can provide a nationwide picture, validate regression models proposed in this study for future use and emphasize points to ensure better management and adherence.

ACKNOWLEDGEMENTS

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REFERENCE

[1]. Al-Busafi SA, McNabb-Baltar J, Farag A, Hilzenrat N. Clinical manifestations of portal hypertension. International journal of hepatology. 2012 Jan 1;2012.
[2]. Ling SC. Portal hypertension in children. Clinical liver disease. 2012 Nov;1(5):139.
[3]. D'Amico G, Morabito A. Noninvasive markers of esophageal varices: another round, not the last. Hepatology. 2004 Jan 1;39(1):30-4.
[4]. Jani PG. Endoscopic variceal band ligation: a local experience. East African medical journal. 2004;81(4):212-4.
[5]. Venon WD, Elia C, Stradella D, Bruno M, Fadda M, DeAngelis C, Rizzetto M, Saracco G, Marzano A. Prospective randomized trial: endoscopic follow up 3 vs 6 months after esophageal variceal eradication by band ligation in cirrhosis. European journal of internal medicine. 2014 Sep 1;25(7):674-9.
[6]. Chaudhary N, Mehrotra S, Srivastava M, Nundy S. Management of bleeding in extrahepatic portal venous obstruction. International Journal of Hepatology. 2013 Jan 1;2013.
[7]. Karim AS, Akter S, Karim MA, Nazir MF. A study of clinical profile of chronic liver disease in children'. Dhaka Shishu Hospital Journal. 1999; 15:15-72.
[8]. Imanieh MH, Dehghani SM, Khoshkhui M, Malekpour A. Etiology of portal hypertension in children: a single center's experiences. Middle East Journal of Digestive Diseases. 2012 Oct;4(4):206.
[9]. Marjan P, Karim AS, Rukunuzzaman M, Das SR, Mondal M, Sarker N, Akther H, Chowdhury AS. Portal Hypertension in Children at the Department of Pediatric Gastroenterology & Nutrition, BSMMU, Dhaka, Bangladesh. Glob Acad J Med Sci. 2023;5.
[10]. Sherlock S, DooleyJ, Anna L, Andrew K, Burroughs, 2012.Sherlock's Diseases of liver and Biliary System,12thed, Blackwell Publishing Ltd, London, pp.152-233.
[11]. Stiegmann GV, Goff JS, Michaletz-Onody PA, Korula J, Lieberman D, Saeed ZA, Reveille RM, Sun JH, Lowenstein SR. Endoscopic sclerotherapy as compared with endoscopic ligation for bleeding esophageal varices. New England Journal of Medicine. 1992 Jun 4;326(23):1527-32.
[12]. Villanueva C, Colomo A, Aracil C, Guarner C. Current endoscopic therapy of variceal bleeding. Best Practice & Research Clinical Gastroenterology. 2008 Apr 1;22(2):261-78.
[13]. Mahmud S, Ahmed SS, Gulshan J, Tasneem F. Etiology of portal hypertension in children-an experience in a tertiary Centre of Bangladesh. Bangladesh Journal of Child Health. 2015;39(1):14-7.
[14]. Butt N, Abbasi A, Khan MA, Butt S, Ahmad SM, Khan MA. Esophageal variceal band ligation interval and number required for the obliteration of varices: a multi-center study from Karachi, Pakistan. Cureus. 2019 Jun 25;11(6).
[15]. Karim AS, Akter S, Karim MA, Nazir MF. A study of clinical profile of chronic liver disease in children'. Dhaka Shishu Hospital Journal. 1999; 15:15-72.
[16]. Mahmud S, Ahmed SS, Gulshan J, Tasneem F, Baidya M. Outcome of Band Ligation in Esophageal Varices of Bangladeshi Children: A Tertiary Centre Experience. Bangladesh Journal of Child Health. 2017 Aug 20;41(1):28-33.
[17]. Arora NK, Lodha R, Gulati S, Gupta AK, Mathur P, Joshi MS, Arora N, Mitra DK. Portai hypertension in North Indian children. The Indian Journal of Pediatrics. 1998 Jul; 65:585-91.
[18]. Alam R, Karim AB, Rukunuzzaman M, Yasmin A, Hossen K, Benzamin M. Non-endoscopic predictors of esophageal varices in children with chronic liver disease and their utility in resource-constrained countries. Indian Journal of Gastroenterology. 2019 Aug; 38:310-6.
[19]. Alam MJ, Ahmed F, Mobarak R, Arefin S, Tayab A, Tahera A, Mahmud S. Pattern of liver diseases in children admitted in Dhaka Shishu Hospital. Int J Hepatol. 2010 Nov 29;1(3):18-24.
[20]. Dar GA, Malik MI, Ganie FA, Jan K. Chronic liver diseases in children: clinical spectrum and etiology. British Biomedical Bulletin. 2013.
[21]. Mondragón FZ, Trujillo JR, Bustamante RC, Tiscareño MM, Barrios EM, León JC, Méndez MC, Monjaraz ET, Mayans JR. Clinical, radiologic, and endoscopic characteristics upon diagnosis of patients with prehepatic portal hypertension at the Instituto Nacional de Pediatría from 2001 to 2011. Revista de Gastroenterología de México (English Edition). 2014 Oct 1;79(4):244-9.
[22]. Shrestha B, Sudhamshu KC, Chaudhary S, Basnet BK, Mandal AK, Poudyal NS. Outcome of endoscopic variceal band ligation. Hemoglobin (gm/dL). 2017 Apr 1; 10:2-07.
[23]. Karim AS, Akter S, Karim MA, Nazir MF. A study of clinical profile of chronic liver disease in children'. Dhaka Shishu Hospital Journal. 1999; 15:15-72.
[24]. Pargewar SS, Desai SN, Rajesh S, Singh VP, Arora A, Mukund A. Imaging and radiological interventions in extra-hepatic portal vein obstruction. World Journal of Radiology. 2016 Jun 6;8(6):556.
[25]. Chaudhary N, Mehrotra S, Srivastava M, Nundy S. Management of bleeding in extrahepatic portal venous obstruction. International Journal of Hepatology. 2013 Jan 1;2013.
[26]. Khattak AK, Manan F, Din RU. Outcome of endoscopic band ligation for oesophageal variceal bleed in patients with chronic liver disease. Gomal Journal of Medical Sciences. 2013 Jul 2;11(1).