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OUTCOME OF PROXIMAL SPLENORENAL SHUNT IN EXTRA HEPATIC PORTAL VEIN OBSTRUCTION

Hepatobiliary Surgery

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ABSTRACT

Introduction : EHPVO and NCPF are common cause of non cirrhotic portal hypertensionwhich accounts for 40% of Portal hypertension in developing countries.The aim of this study is to analyze the clinical presentation, management of patients with EHPVO treated with surgical shunt, devascular arisation and endotherapy and outcome of these treatment modalities at our institute.

Materials and methods : This study is a retrospective analysis of patients with EHPVO admitted at our institute during the period from January 2009 to december 2018. Clinical presentation, etiology of EHPVO, laboratory and radiological workup, endoscopic findings and management,outcome and follow up details were analyzed.

Results : During the study period, among 52 EHPVO patients diagnosed, 70% of patients belong to 15 – 30 years of age. 90% presented with variceal bleeding and 35% with portal cavernomacholangiopathy of which 44% being symptomatic. In our study, 50 % (26/52) patients underwent proximal splenorenal shunt, 26%(14/52) patients managed with endotherapy and 23% (12/52) patients underwent Splenectomy with esophagogastricdevascularisation procedure because of unavailability of shuntable vein. Mean splenic vein diameter and shunt diameter was 9.8 mm and median fall in portal pressure was 13 mm of Hgin patients who underwent PSRS. Variceal regression rate was 92% in PSRS patients and regression to obliterated varices from grade III is 80% with PSRS group whereas 67% in devascular arisation group. Rebleeding rate was 17% in devascular arisation group versus no rebleeding in PSRS group.Portal biliopathy could be reversed with PSRS in 94% of patients.

Conclusion : In EHPVO, Proximal splenorenal shunt is the good treatment option to prevent rebleeding , to treat hypersplenism and to reverse biliopathy.

KEYWORDS

INTRODUCTION

Extra hepatic portal vein obstruction and non cirrhotic portal fibrosis arecommon cause of non cirrhotic portal hypertension which accounts for 40% of Portal hypertension in developing countries especially in india[1].EHPVO accounts for 54% of Portal hypertension and 85%of upper gastrointestinal bleeding in children from the developing world. EHPVO is defined as "a vascular disorder of liver, characterized by obstruction of the extrahepaticportal vein with or without involvement of intrahepatic PV radicles or splenic or superior mesenteric veins"[2]. Chronic portal vein thrombosis leads to hepatopetalcollaterals to bypass the obstruction followed by hepatofugal collaterals.Thus the portal vein is replaced by numerous tortuous vascular channels called portal cavernoma.As these collateral channels are insuffcient to bypass the entire blood flow from the spleno-mesenteric axis, the clinical signs of portal hypertension develops.

Varicealbleeding, hypersplenismand portal biliopathy, growth retardation are important manifestations.Proximal splenorenal shunt (PSRS) is the most commonly performed shunt for EHPVO[3]. It reduces portal venous pressure thereby control varicealbleeding and relieves symptoms of enlarged spleen and hypersplenism.The aim of this study is to analyze the clinical presentation,etiological factors, management and outcome of patients with EHPVO at our institute

MATERIALS AND METHODS

This study is a retrospective analysis of patients with EHPVO admitted at our institute during the period from January 2009 to december 2018. The hospital data were scrutinized and all EHPVOpatients above 12 years of age were included in the study. These patients were managed with PSRS or splenectomy and devascularisationor conservatively. Patients with portal hypertension due to cirrhosis, patients with acute portal vein thrombosis and cirrhotics with portal vein thrombosis were excluded from the study.Data on patient demographics, clinical presentation, etiology of EHPVO, laboratory and radiological workup, endoscopic findings and management,outcomeand follow up details were analyzed from hospital records.

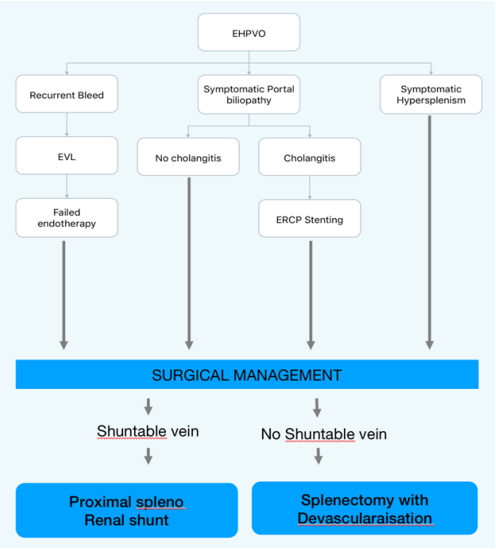
Management algorithm

Patients with recurrent, symptomatichy persplenism, growth retardation, symptomatic biliopathy were treated with surgery. Our institute management algorithm for EHPVO patients is given in flow chart.

Splenectomy was done after ligation of splenic artery and splenic vein was meticulously dissected and mobilized.Left Renal vein was dissected at renal hilum. After applying satinsky clamps, endto side splenorenal shunt was performed with 6-0prolenecontinuous suture leavinggrowth factor.Splenic vein pressure was recorded before and

30 min after completion of shunt. Esophagogatric devascularaisation was done in cases for which shuntable vein was not available. Doppler USG was done to check shunt patency at 1 week, 3 month,6 month and at 12 months after PSRS.Patients with underlying prothrombotic states were continued on anticoagulant drugs. Endoscopic findings were recorded every three months in first year and as required thereafter.

Management algorithm for EHPVO patients at our institute

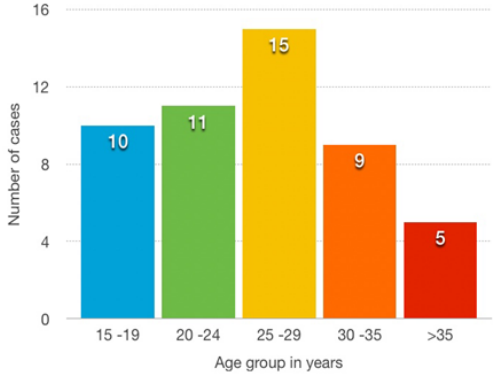


Results

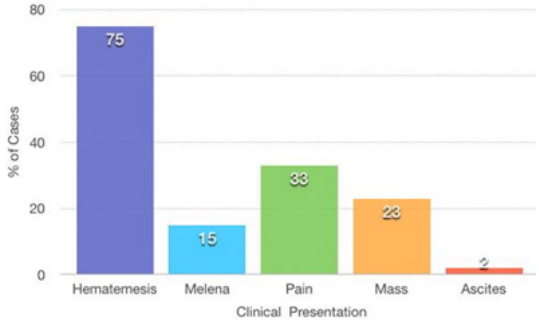
Clinical characteristics

During the study period, total number of 52 patients were diagnosed as EHPVO and managed at our institute of which 28were males and 24 were females.Patient clinical characteristics and endoscopic findings given in table 1. Approximately 70% of patients belong to 15 – 30 years of age. 90% of patients presented with variceal bleeding of which 82% presented with hemetemesis and 13% patients with melena and 3% presented with bleeding per rectum.Other symptoms like abdominal pain (33%), abdominal mass(23%), Ascites (2%),jaundice (2%) were less common complaints.On examination all patients had splenomegaly of which approximately 40 % had massive splenomegaly and 60% had moderate splenomegaly.

Age distribution



Clinical presentation



Prothrombotic tendency present in 21% of cases of which 63% had protein C deficiency, 27% had protein S deficiency , 9% had antithrombin III deficiency. Approximately 7% of cases had history of umblical sepsis at childhood and there is no identifiable etiological factor for 54% of patients. Prothrombotic work up could not be done for 18% of cases due to financial reasons. Approximately esophageal varices and fundal varices present in 85% and 32% of patients respectively. Ectopic varices present in 11.5% of cases(Duodenal, jejunal and rectal) and portal hypertensive gastropathy present 17% of cases. Endoscopy couldn't be done in one patient due to inadequate mouth opening because of mandibular hypoplasia.Among 52 patients, 35% of cases had portal cavernomacholangiopathy at presentation of which 44% were symptomatic biliopathy.

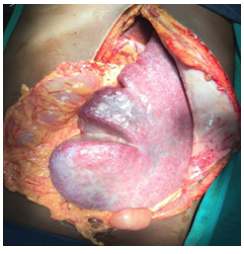
Table 1 : Clinical characteristics of patients with EHPVO

Clinical characteristics	EHPVO (n = 52)
Male/female	28/24
Age distribution, decade wise (%)	
• 15 - 19years	10
• 20-24 years	11
• 25-29years	15
• 30-35 years	9
• > 35 years	5
Clinical presentation	
• Hematemesis	39(75%)
• Melena	8(15%)
• Abdominal pain	17(33%)
• Abdominal mass	12(23%)
• Ascites	1(2%)
• Jaundice	1(2%)
• Splenomegaly	52
• Massive	20
• Moderate	32
Pre operative Investigations (mean ± SD)	
• Hemoglobin (g/dl),	9.91 ± 2.02
• Total count	4950 ± 2838
• Platelet count	142000 ± 99927
• Total bilirubin (mg/dl)	1.03 ± 0.36
• Alkaline phosphate (U/L)	230 ± 138
Bleeder/non bleeder	50/52
Portal biliopathy	18
• Symptomatic / asymptomatic	8/10
Hypersplenism	22
• Symptomatic / asymptomatic	10/12
Grade of oesophagealvarices	
• Grade III	47
• Grade I and II	4

Treatment

In our study, 50 % (26/52) patients underwent proximal splenorenal shunt, 26%(14/52) patients managed with endotherapy and 23% (12/52) patients underwent Splenectomy with esophagogastric devascu larisation procedure because of unavailability of shuntable vein.

Mean splenic vein diameter and shunt diameter was 9.8 mm in all patients who underwent PSRS. Median fall in portal pressure was 13 mm of Hg. The median operative time was 5 (4 - 6) hours in both PSRS and devascularaisation group. Median intraoperative blood loss was 150 (100–200) ml in PSRS group and 270 ml in devascularaisation group.



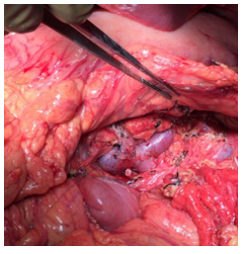


Table2 :Intra Operative details

	PSRS	Devascularisation
Splenic vein diameter (in mm)	9.8 ± 3.03	Unshuntable
Shunt diameter (in mm)	9.8 ± 3.03	-
Splenic vein pressure		-
• Pre shunt	29 ± 5mmHg	
• Pressure drop	13± 2 mmHg	
Operative time (in minute)	310	480
Operative blood loss (in ml)	160	440

Outcome

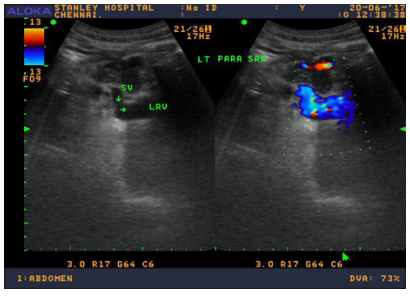
Cytopenia improved in all patients after splenectomy in both group of patients. During follow-up of 12 months to 10 years, 2 patients developed shunt thrombosis and both these two patients showed persistence of grade III varices necessitating endotherapy.

Table. 3 : Post operative Outcome after PSRS vs Devas cularaisation

Outcomeparameters (%)	PSRS (n = 26)	Devascularisation (n =12)
Splenic vein diameter <10 mm	12	-
Splenic vein diameter10 mm and >10 mm	14	-
Shunt thrombosis In <10 mm SV	2/12	-
Shunt thrombosis in >10mm SV	0/14	-
Shunt patency at 3 months	92%(24)	-
Change in variceal status		-
• Obliteratedoesophagealvarices	80%(21)	67(8)
• Varicealgrade I	12%(3)	33(4)
Rebleeding rate	0	17(2)
Ectopic varices	0	25(3)
Need for endotherapy on follow-up	8(2)	33(4)
Reversal of biliopathy	94(17/18)	-
Post opcomplications (gradeIII/ IV)	12(3)	33(4)
Post op medianhospital stay(days)	11	15
OPSI	0	0
Mortality	0	17(2)

(Values in parentheses are actual number ofpatients)

Doppler study showing patent PSRS



Variceal eradication and rebleeding

In PSRS group, 80% of patients had regression of esophageal varices from Grade III/IV to obliterated varices and 12% regressed to grade I and 8% with shunt thrombosis had persistence of Grade III varices. None of the patients had any ectopic varices. No rebleeding reported in PSRS group.In devascularisation group, 67% patients had regression of varices from grade III to obliterated varices and remaining 33% regressed to grade I varices and 25% patients developed ectopic varices. Rebleeding was reported in 17% patients .

Portal biliopathy could be reversed with PSRS alone in 94% of patients except one patient required PSRS followed by CBD exploration andhepaticojejunostomy for ischemic stricture with proximal calculus.

Postoperative hepatic encephalopathy was not observed in any of the patients.

ClavienDindo Grade III/IV postoperative complications occurred in 12% patients in PSRS group and 33%patients in devascularaisation group. Two patients in who underwent devascularaisation were reexplored for reactionary hemorrhage and both of them expired due to Coagulopathy.No mortality in PSRS group and 17%mortality for those who underwent devascularisation.

DISCUSSION

Portosystemicshunt,Mesorex shunt and esophagogastric devascularisationare Surgical options for EHPVO. Portosystemic shunts includeproximal splenorenal shunt (PSRS), side to side splenorenal shunt, distal splenorenal shunt (DSRS) and mesocaval shunt.In EHPVO, Most commonly performed shuntis PSRS which divert blood flow from portal system to systemic circulation and decrease portal pressure. Splenectomy component relieves symptoms of splenomegaly and effects of hypersplenism.

Outcome of PSRS is good with shunt patency rate of 90% to 95%, rebleeding rate of 2% to 11% and 15 years survival of >96%.

Splenic vein diameter less than 10mm is significant predictor of shunt thrombosis in many studies[4]. Shunt thrombosis in our study is 8% which occurred in cases with splenic vein diameter less than 10 mm.In contrast to other studies, there is no statistically significant correlation between shunt thrombosis and splenic vein diameter(p value 0.41). This might be due to small sample size of our study. Prasadet al.[5]reported 11% rebleeding rate in 160 patients of EHPVO treated with PSRS with 15 yr survival of 95%. Wani et al compared endoscopic sclerotherapy and shunt surgery in RCT and reported significantly lower rebleeding rates in the shunt surgery group (3.3% versus 22.6%).

If shuntable vein is unavailable, Splenectomy with esophagogas tricdevascularisation (Hassab's procedure) isthe salvage procedure to control variceal bleeding in failed endotherapy. Splenectomy with esophagogastricdevascularisation has re-bleeding rate of 11%, control of bleeding in 96%, without encephalopathy, and overall survival was 95%.

In our study, rebleeding rate of 17% for devascularisation and no rebleeding after PSRS which reflects the better variceal eradication rate after shunt compared to devascularisation and endotherapy.Variceal regression noted in PSRS patients was 92% and regression to obliterated varices from grade III is 80% after PSRS whereas 67% in patients who underwentdevascularisation.

Portal biliopathy or portal cavernomacholangiopathy is defined as anatomical and functional alterations of the intra hepatic or extrahepatic bile ducts in patients with portal hypertension due to EHPVO.[6] Compression by collaterals around the bile duct originating from paracholedochalvenous plexus of Saint and pericholedochalvenous plexus of Petren or ischaemia of bile duct lead to portal cavernomacholangiopathy.[7] Indentation in bile duct due to collaterals, strictures, angulations, focal narrowing, stones, and irregular walls may be seen on imaging. Portal biliopathy is recognized in 90% to 100% of the cases, however only few patients are symptomatic[8,9,10,11].Incidence of Portal biliopathy is 35% of which 44% symptomatic in our study in contrast to 80- 100% incidence in EHPVO in many other studies.In our study, Portal biliopathy could be reversed with PSRS alone in 94% of patients which is the primary modality of treating this subset of patients.

Shunt procedures may also result in an improved quality of life (QOL). Krishna et al.[12] reported improvement in physical, psychosocial, and total QOL scores after surgery compared to endotherapy even though the differences were not statistically significant.Improved QOL is indirectly evident in our patients with relief symptoms related to splenomegaly, decreased rebleeding, requirement for endotherapy and return to normal work.Many studies from India and Mexico have shown low rates overwhelming postsplenectomy infectionas as there is no OPSI in our study.

Clavien– Dindo Grade 3 and above complications are more with devascularisation compared with PSRS in our study. There is no encephalopathy after shunt or devascularisation reported in our study.

CONCLUSION

Considering low rate of rebleeding, good shunt patency rate, reversal of biliopathy and improved quality of life Proximal splenorenal shunt is the good treatment option for patients with extra hepatic portalvein obstruction. Splenectomy with devascularisation may be useful as salvage therapy in patients with unshuntable vein.

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