Section: Miscellaneous

Case Series

DECOMPRESSIVE SHUNT SURGERY IN PORTAL CAVERNOMA CHOLANGIOPATHY: A CASE SERIES

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ABSTRACT

Background: Portal Cavernoma Cholangiopathy (PCC) is a rare complication of portal hypertension, primarily associated with extrahepatic portal vein obstruction (EHPVO). This study aims to evaluate the efficacy and outcomes of decompressive shunt surgery in symptomatic cases of PCC.

Materials and Methods: This retrospective analysis involved six patients with symptomatic PCC who underwent Proximal Splenorenal Shunt (PSRS) surgery at a tertiary hospital from 2020 to 2024. Preoperative and postoperative assessments included imaging, routine haematological parameters, liver function tests (LFTs), and evaluations of shunt patency. Statistical analysis was performed using SPSS Version 20 software. Fisher's exact test and Student's t-test were used, with p-value of <0.05 as significant.

Results: All six patients underwent PSRS for symptomatic PCC. The mean pre-shunt serum bilirubin level was 2.57 ± SD mg/dl, and the mean pre-shunt serum alkaline phosphatase (ALP) was 391.33 ± SD IU/ml. Post-surgery, the mean bilirubin was 1.06 ± SD mg/dl, and the mean ALP was 133.67 ± SD IU/ml, with both showing statistically significant improvements ($P < 0.001$) at six weeks postoperatively. Doppler USG assessments consistently indicated that 5 patients (83.3%) had patent shunts. one patient experienced shunt thrombosis, continued to exhibit abnormal LFTs and showed a persistent dominant biliary stricture on imaging, requiring endoscopic intervention.

Conclusion: Patients with symptomatic PCC experience significant benefits from decompressive shunt surgery, which often reverses PCC and facilitates safer subsequent endoscopic and biliary drainage procedures if required. These findings highlight the effectiveness of PSRS in alleviating biliary obstruction, decompressing collaterals, and improving long-term outcomes for symptomatic PCC.

Keywords: Portal Cavernoma Cholangiopathy, Decompressive shunt, Portal Biliopathy.

INTRODUCTION

The term "portal biliopathy" refers to abnormalities in both intrahepatic and extrahepatic bile ducts, as well as the gallbladder wall, that are associated with portal hypertension. The term "Portal Cavernoma Cholangiopathy (PCC)" has been established by the Working Party as the consensus nomenclature.[1] PCC is defined as abnormalities in the extrahepatic biliary system, including the cystic duct and gallbladder, with or without changes in the first- and second-generation biliary ducts. This condition occurs in patients with a portal cavernoma. For a diagnosis of PCC to be confirmed, all three of the following criteria must be met:

1. The presence of a portal cavernoma.
2. Cholangiographic changes observed on endoscopic retrograde cholangiography (ERC) or magnetic resonance cholangiography (MRC) that are consistent with typical changes associated with this entity.
3. The absence of other conditions that could explain these biliary changes, such as bile duct

injury, primary sclerosing cholangitis, or cholangiocarcinoma.

PCC is a rare complication of portal hypertension, most commonly associated with extrahepatic portal vein obstruction (EHPVO) in 80-100% of cases, but it can also occur in patients with non-cirrhotic portal fibrosis (NCPF) and cirrhosis.[2-4] In most patients, PCC remains asymptomatic, but it can present with obstructive jaundice, sometimes accompanied by cholangitis in symptomatic cases.[5-12] Early intervention for biliary obstruction is crucial, as ongoing obstruction can lead to pressure changes in the liver and result in secondary biliary cirrhosis6. Symptomatic patients with PCC may require surgical decompression of the portal venous system to relieve symptoms and reversal of biliary obstruction.[5-9] In this article, the term PCC is used in place of portal biliopathy.

MATERIALS AND METHODS

This study was conducted at a Tertiary Referral hospital. We collected prospectively maintained data from patients who underwent Proximal Splenorenal Shunt (PSRS) surgery for symptomatic PCC between 2020 and 2024, and performed a retrospective analysis. We evaluated their data regarding demographics, clinical presentations, routine haematologic investigations, liver function tests (LFTs), and prothrombin time with INR. Preoperative Doppler Ultrasonography (USG) of the abdomen was performed for evaluation, and postoperative assessments were also conducted to evaluate shunt patency. If results were inconclusive, contrast-enhanced computed tomography (CT) or magnetic resonance (MR) Portography was carried out. Magnetic resonance cholangiography (MRC) was performed specifically for patients with PCC.

Surgical Procedure

All 6 patients underwent PSRS. The procedure was carried out through an L-shaped incision with the patient in a supine position. An anastomosis was created using 6-0 Prolene suture material in a continuous fashion, with an appropriate growth factor used unless the vein size exceeded 15mm. Intraoperative portal pressure was measured before and after shunt completion in the omental vessel. Heparin was administered intraoperatively at the time of anastomosis. An intraoperative liver biopsy was obtained where indicated.

Postoperatively:

Post-surgery, patients were anticoagulated with heparin for five days. Antiplatelet treatment was initiated when platelet counts exceeded 450,000 cells/mm³.

1. Shunt patency was assessed postoperatively at 5 days, 6 weeks, and 6 months.
2. A complete blood count (CBC) was performed on day 7 postoperatively for patients presenting with hypersplenism.
3. In patients where PCC indicated surgery, LFT and radiological imaging were conducted after 6 weeks to evaluate regression.

Outcome Measures: Patient outcomes were evaluated based on:

a) Improvement in CBC on postoperative day 7.

b) Improvement in PCC on imaging and LFT.

c) Shunt patency at postoperative days 5 and 6 weeks, and then every 6 months.

Statistical Analysis

Statistical analysis was performed using SPSS Version 20 software. We applied Fisher's exact test and Student's t-test for various factors, considering a p-value of <0.05 as significant.

RESULTS

Demographics: Our study included 6 patients with EHPVO who underwent PSRS surgery. The demographic data and aetiology are summarized in Table 1. Among the patients, 4 were male and 2 were female, with ages ranging from 16 to 38 years (mean age: 27 years).

Clinical Presentation: All patients presented with jaundice. Their clinical presentations included a combination of symptoms like recurrent hematemesis, cholangitis, massive splenomegaly, and symptomatic hypersplenism, as detailed in Table 1.

Investigations: All patients exhibited features of hypersplenism on CBC. Elevated serum bilirubin and alkaline phosphatase (ALP) levels were noted in all patients, with a mean serum bilirubin of 2.57 ± SD mg/dl and mean serum ALP of 391.33 ± SD IU/l. However, serum AST and ALT levels remained within normal ranges. Prothrombin time was normal across all patients. Post-surgery, there was a noted improvement in all haematological parameters (Table 1). Specifically, in patients with PCC, the pre-shunt mean serum bilirubin was 2.57 ± SD mg/dl and pre-shunt serum ALP was 391.33 ± SD IU/ml. Post-shunt surgery, the mean serum bilirubin decreased to 1.06 ± SD mg/dl, while the mean serum ALP was 133.67 ± SD IU/ml, with both parameters showing statistically significant improvement ($P < 0.001$) at 6 weeks postoperatively (Table 2). In the follow-up Doppler USG performed to assess shunt patency, 5 out of 6 patients had patent shunts, while 1 patient experienced shunt thrombosis. The analysis could not demonstrate a significant association between shunt patency and postoperative improvement in PCC due to the small sample size (Table 3).

Follow-up after PSRS:

Doppler USG assessments consistently indicated that 5 patients (approximately 83.3%) had patent shunts, while 1 patient exhibited shunt thrombosis. One patient continued to have deranged LFTs and showed persistent dominant biliary stricture on imaging, necessitating endoscopic intervention for management.

Table 1: Characteristics and Outcome of Decompressive Shunt in Portal Cavernoma Cholangiopathy (PCC)

SL. No	Age	Sex	Clinical Presentation	Hb in gm/dl	TLC cells/ mm3	Platlets cells/mm3	INR	Bilirubin mg/dl	ALP IU/L	Diagnosis	Shunt Patency at day 5	Shunt Patency at 6weeks	Shunt Patency at 6months	Hb in gm/dl day 7	TLC cells/ mm3 at day7	Platelet (count/ mm3)at day 7	Bilirubin (mg/dl) at 6 weeks	ALP (IU/L)at 6 weeks	Improvement in PCC at 6 weeks	Intervention Required
1	24	M	Jaundice with Symptomatic Hypersplenism	7.6	1400	1E+05	1.2	2.6	348	EHPVO	Patent	Patent	Patent	9.8	8500	2E+05	0.8	102	Yes	No
2	16	F	Fever with Chills and Jaundice with Massive Splenomegaly	7.5	1500	80000	1.02	1.9	373	EHPVO	Patent	Patent	Patent	9.3	9100	4E+05	1.1	98	Yes	No
3	22	M	Jaundice with Massive Splenomegaly	12	3500	2E+05	1.33	2.8	401	EHPVO	Patent	Patent	Patent	10	8400	2E+05	1	110	Yes	No
4	38	M	Fever with chills and Jaundice	8.6	3500	2E+05	1	2.6	378	EHPVO	Patent	Patent	Patent	10	7600	2E+05	1.1	124	Yes	No
5	32	M	Jaundice with Recurrent Hematemesis	8.9	4600	2E+05	1.2	2.1	554	EHPVO	Patent	Patent	Patent	12	10100	2E+05	1	246	Yes	No
6	30	F	Hematemesis with Jaundice	8.4	3600	2E+05	1.9	3.4	294	EHPVO	Patent	Patent	Thrombosed	8.7	8900	1E+05	1.4	122	No	ERC Stent

M- Male, F- Female, Hb – Hemoglobin, TLC – Total Leucocyte Count, ALP – Alkaline Phosphatase, ERC – Endoscopic Retrograde Cholangiography.

Table 2: Association of Biochemical Parameters Pre-shunt and Post-shunt Surgery in PCC at 6-weeks

Parameters N=6	Pre-Shunt Values		Post-Shunt values		P value*
	Mean(Range)	SD	Mean(Range)	SD	
Bilirubin (mg/dl)	2.57 (1.9-3.4)	0.53	1.06 (0.8-1.4)	0.19	<0.0001
ALP (IU/ml)	391.33(294-554)	87.66	133.67 (98-246)	56.01	<0.0001

***Unpaired t-test applied, ALP- Serum Alkaline Phosphate, P<0.001, Significant.**

Table 3: Association Between Shunt Patency and Improvement in PCC

Shunt patency	Improvement in PB (N=6)		P value*
	Yes	No	
Patent	5	0	0.1667
Thrombosed	0	1	

***Fisher's exact-test applied P = 0.1667, Not Significant**

DISCUSSION

PCC is a rare complication of portal hypertension, most commonly linked to EHPVO in 80–100% of cases.[1-3] Patients with PCC are typically classified as symptomatic or asymptomatic.[13] Asymptomatic patients often exhibit biliary abnormalities identified through imaging studies or endoscopic retrograde cholangiography (ERC), despite lacking any biliary symptoms. Reports indicate that asymptomatic PCC is observed in approximately 78–100% of patients, while symptomatic cases occur in around 5–38% of individuals.[7-17] Symptomatic patients may present with jaundice, sometimes accompanied by biliary colic and cholangitis, characterized by jaundice and fever with chills, primarily due to biliary strictures or stones.[18]

PCC features both reversible and irreversible components.[18] The reversible component typically resolves upon decompression of collaterals, while the irreversible component persists despite such intervention. In cases of EHPVO, prolonged obstruction of the portal vein results in the formation of large collateral veins accompanying the common bile duct (CBD) and potentially intra-choledochal varices—referred to as cavernomatous transformation of the portal vein. These large collaterals can exert pressure on the CBD, leading to the characteristic changes observed on ERC.[7,8,11] A study by Dilawari et al8. noted that out of 20 patients,[18] exhibited indentations indicative of external compression on ERC. Evidence supporting the reversibility of biliary changes following portal decompressive surgery,[1,19-21,24-26] or transjugular intrahepatic portosystemic shunts (TIPS),[24,25] underlines this mechanism. The persistence of biliary tract changes can be attributed to several factors: (i) ischemia at the time of portal vein thrombosis, (ii)localized ischemia resulting from prolonged collateral compression, or (iii) encasement by a fibrous "solid tumor-like cavernoma" surrounding the bile duct18. Dhiman et al,[26] examined bile duct changes post-shunt surgery

in five patients, observing complete reversal in one patient, partial in three, and none in one, suggesting that ischemia or scarring might play a role in the persistence of bile duct alterations.

The first report of surgical intervention for PCC was published in 1965 by Hunt,[27] who described separating collaterals from the bile duct wall, successfully relieving jaundice postoperatively; however, this method carries a significant risk of intraoperative haemorrhage. Choudhuri et al28. proposed portal decompression for the management of PCC in 1988, and subsequent studies have reinforced the effectiveness of portosystemic shunt surgery as the preferred approach for symptomatic patients. Chaudhary et al,[17] then published a series of 9 patients, in which 7 patient underwent PSRS for symptomatic PCC. Of these 7 patients, 5 patients had a reversal of PCC with two patients requiring a biliary drainage procedure (Roux-en-Y hepaticojejunostomy) for refractory bile duct strictures. Among patients with EHPVO and PCC, the reasons for favouring portosystemic shunt surgery include:

1. Successful portosystemic shunting prevents variceal bleeding, decompressing both peri-choledochal and intra-choledochal varices, thereby alleviating symptomatic PCC.[19,21,24,28-30]
2. Performing upfront biliary drainage for PCC poses a high risk of torrential bleeding from large peri-choledochal varices during the procedure.[11,19,21,24,31,32]
3. Biliary obstruction may not resolve completely, with a partial response being observed in a subset of cases post-shunting; a patent shunt reduces pressure on both periportal and peri-choledochal collaterals, rendering subsequent biliary bypass or endoscopic interventions less hazardous.[19,21]

Portosystemic shunt surgery is also recommended for asymptomatic patients exhibiting PCC features on imaging, especially those undergoing surgery for other complications of portal hypertension, such as recurrent hematemesis or symptomatic hypersplenism, since these patients could develop significant bile duct obstruction later, even after splenectomy and devascularisation.[11,33]

Advancements in Endo-therapy have allowed for the management of PCC patients with multiple stents over extended periods. However, performing interventions like sphincterotomy, stone extraction, or stricture dilation in patients with collaterals raises the risk of complications. The rationale for surgical intervention lies in the fact that it is a one-time procedure that minimizes the need for repeated hospital visits, particularly relevant for EHPVO patients who may lack access to advanced endo-therapy and specialized medical facilities.[25]

Several studies evaluating the outcomes of shunt surgery in patients with portal biliopathy illustrate the efficacy of this approach. Khare et al,[20] analysed 13 symptomatic PCC patients with EHPVO, categorizing them into three groups based on their conditions. Group A, consisting of five patients with biliary strictures, showed jaundice relief in three following splenorenal shunts, while two required endoscopic management for persistent strictures. In Group B, three patients with bile duct stones experienced a staged procedure, and Group C included five patients facing both strictures and stones, all of whom necessitated surgical intervention due to failed endo-therapy. Further studies have corroborated these findings, indicating that while shunt surgery offers substantial benefits, additional biliary interventions may still be required postoperatively, underscoring the complex nature of managing PCC. A retrospective study conducted by Vibert et al,[24] examined 64 patients, out of which 19 had symptomatic portal cavernoma cholangiopathy (PCC). Among the 10 patients who underwent splenorenal shunt surgery, 7 experienced relief from jaundice in the early follow-up, but 5 of these patients later required a biliary bypass.

Similarly, Agrawal et al,[21] studied 39 symptomatic PCC patients from a larger cohort of 177 EHPVO patients. In this study, 37 patients underwent proximal splenorenal shunt (PSRS) as the initial procedure, with 13 ultimately needing a second-stage bilio-enteric bypass. In a separate analysis by Chaudhary A et al,[19] direct biliary surgery without a prior shunt was performed on 2 patients; however, both cases resulted in severe intraoperative complications, including one postoperative death and another suffering from an anastomotic stricture at the choledocho-jejunostomy site. Chattopadhyay S. et al,[25] reported on 56 PCC patients with NCPH, noting that the majority underwent portal-systemic shunt (PSS) procedures. The authors emphasized that a portosystemic shunt should be prioritized wherever feasible, while splenectomy with devascularization may be beneficial in patients lacking a shuntable vein.

The elevation of serum bilirubin and alkaline phosphatase (ALP) levels are notable indicators in PCC patients. Following shunt surgery, the decompression of collateral circulation leads to a reduction in pressure on the biliary system, resulting in improved laboratory parameters. Resolution of PCC is typically defined as the alleviation of biliary obstruction symptoms in symptomatic patients, a decrease in bilirubin and ALP levels, or improvement in radiological findings in asymptomatic individuals. In Agrawal et al.'s study,[21] between June 1996 and December 2007,[39] surgical interventions were performed on patients with portal biliopathy due to EHPVO; 37 of these underwent PSRS as the primary procedure. In follow-up, 13 patients proceeded to second-stage surgery, with a patent shunt rate of 92.3%. Chattopadhyay's et al,[25] long-term evaluation of 56 patients indicated that follow-up interventions, such as endoscopic retrograde cholangiopancreatography (ERCP) and hepaticojejunostomy, were necessary in some cases. Other literature on the surgical

management of symptomatic PCC reports a second-stage surgery requirement in about 50% of patients.[18-21,25]

In our own study, involving 6 patients with symptomatic PCC due to EHPVO who underwent shunt surgery, there was a notable improvement in serum bilirubin and ALP levels postoperatively. Of these patients, 5 had patent shunts while 1 experienced shunt thrombosis, leading to the necessity for endoscopic intervention, specifically ERCP stenting, for persistent biliary stricture during follow-up. Although 5 patients with patent shunts showed improvement in PCC, the results were not statistically significant due to the small cohort size.

It is critical for patients with EHPVO to be routinely assessed for PCC since derangements in liver function tests can manifest without evident jaundice or cholangitis. Both symptomatic and asymptomatic patients with PCC should be closely monitored, and timely surgical interventions should be pursued to mitigate the effects of chronic subclinical biliary obstruction. Initial management for symptomatic PCC should focus on PSRS; however, those with ongoing symptoms due to dominant biliary strictures are at a higher risk for unsatisfactory outcomes and may require further endoscopic or surgical interventions.

CONCLUSION

Patients with symptomatic PCC benefit significantly from decompressive shunt surgery, which not only reverses PCC in most instances but also renders subsequent endoscopic and biliary drainage safer when faced with no or partial improvement. Routine monitoring and timely surgical intervention are advisable for both symptomatic and asymptomatic PCC patients to thwart disease progression.

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