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PROXIMAL SPLENO RENAL SHUNT AN EXPERIENCE IN TERTIARY TEACHING HOSPITAL

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ABSTRACT

Proximal splenorenal shunt is a well accepted procedure for non cirrhotic portal hypertension. The causes of non cirrhotic portal hypertension were extra hepatic potal venous obstruction and non cirrhotic portal fibrosis. We conducted proximal splenorenal shunt, it is an accepted surgical procedure for portal hypertension in non-cirrhotic patients. It is a retrospective study of shunt patency and symptomatic relief of patients. Analysis of shunt patency and reduction of symptoms is studies by using clinical data, pre and post-operative endoscopy, ultrasound doppler study of shunt and Computed Tomographic Portogram post operatively.

The patients are aged between 5 to 52years. Among them 16 are male and 12 are female. The follow-up period ranges from 6 to 60 months. There was no mortality seen at the time of the last follow-up. Type of shunt is proximal splenorenal shunt and splenectemy done in all the patients. Soon after surgery 80% were found to be patent follow-up. In long term follow-up the patency rate of portorenal shunt decreased. 2 patients shows symptoms like nasal bleed and rectal bleed once treated symptomatically. Remaining all patients shows symptomatic relief in follow-up. Endoscopy shows variceal regression.

We did not encounter any mortality, one patient came with complaints of rectal bleed, and another patient came with complaints of nasal bleed treated conservatively.

KEYWORDS : Extra hepatic Portal hypertension, endoscopic sclerotheraphy, Computed Tomographic Portogram, proximal lieno-renal shunt.

INTRODUCTION:

Extra hepatic Portal hypertension is common cause for portal hypertension in childhood followed by non-cirrhotic portal fibrosis. Patients usually presented Hematemesis, ascites and splenomegaly. Various management protocols are available for management and each has its own merits and demerits, these are endoscopic management of oesophageal varices. But variceal bleed recurrence rate of 12-30% in large series will long follow-up [1, 2]in Endoscopic therapy. Surgery may be only option available to children with uncontrolled bleeding and failure of endoscopic sclerotherapy[1]. Primary shunt procedure have an advantage over sclerotherapyand provides one time solution and take care of hypersplenism.

In 1947, Dr. Robert Linton published his 1st report of the treatment of portal hypertension by splenectomy and splenorenal venous anastomosis with preservation of the left kidney. Over the succeeding years, he performed a large number of splenorenal shunts and, on the basis of his experience, recommended his technique as the procedure of choice for patients with bleeding esophageal varices, with or without associated ascites. Linton's procedured other, supported by convincing data, gained wide recognition and became an accepted method for treating portal hypertension. His shunt attracted even more attention in developing countries where extrahepatic portal vein obstruction is a major cause of portal hypertension. Nevertheless, Linton's shunt is a difficult and technically demanding procedure and has several limitations.

Because splenorenal shunts are performed fairly frequently in India an developing countries, and because Linton's procedure is associated with several inherent difficulties, we developed an alternative technique (Omar's technique) for the surgical treatment of portal hypertension. This simplified Linton's shunt facilitates the construction of a trouble-free and tension-free splenorenal anastomosis. Herein, we describe the advantages of our simplified procedure and recommend its use, especially in those regions of the world where portal hypertension is relatively common.

MATERIAL AND METHODS :

A total number of 32 patients with portal hypertension aged between 5 to 52 years with different causes like Extra hepatic Portal hypertension, non-cirrhotic portal fibrosis. History and clinical data collected. All patients underwent endoscopy, ultrasound, doppler study preoperatively and post-operative endoscopy, doppler and some patients Computed Tomographic Portogram post operatively. Results regarding symptomatic relief hypersplenism, shunt patency studied.

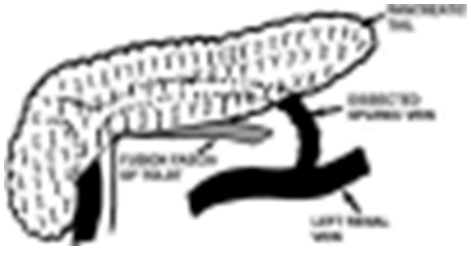
Surgical procedure : splenorenal shunt procedure begins with a left thoracoabdominal incision. before the spleen is removed ,the splenic vein is dissected at the splenic porta so that the maximal length of the vein is available .Next the left renal vein is isolated .The segment of the splenic vein The conventional Linton's splenorenal shunt procedure begins with a left thoracoabdominal incision. Before the spleen is removed, the splenic vein is dissected at the splenic porta so that the maximum length of the vein is available. Next, the left renal vein is lies closest to the splenic vein. The segment of the splenic vein lying on the posterior aspect of the pancreas is then dissected. The small pancreatic branches draining into the splenic vein are isolated and ligated individually. Once an optimal length of splenic vein has been obtained, an end-to-side splenorenal anastomosis is performed.

In our simplified technique for splenorenal shunt, the patient is placed in a supine position on the operating table, with the left flank elevated 15 on a folded sheet. A long, left subcostal incision is carried well into the left flank and is extended across the midline, the right rectus sheath is partially divided, and the rectus muscle is retracted. The splenectomy is performed. Then, holding the pancreas by its tail, along with the bundle of vessels attached to it, we dissect the fusion fascia of Toldt from the tail to the middle of the body of the pancreas. The avascular nature of the fusion fascia permits rapid and easy dissection. Next, the splenic vein is isolated from the pancreas. Obtaining an optimal segment of splenic vein is considerably easier after the fusion fascia has been dissected,

because this dissection exposes a long segment of splenic vein that clearly adheres to the back of the pancreas. The rows of small, tender pancreatic branches of the splenic vein are carefully isolated, ligated, and divided; such care is necessary to avoid bleeding, which can be profuse due to the high pressure in the splenic vein. Because the blood flows away from the liver in patients with portal hypertension, the bleeding resulting from any proximal vascular injury can be controlled easily by applying manual pressure or by using vascular clamps distally. Complete vascular control of the splenic vein is an important advantage of this simplified technique. An optimal segment of the splenic vein is easily isolated and approximated to the left renal vein for the next step, which is to construct a tension-free, end-to-side splenorenal anastomosis. It is advisable to keep the length of splenic vein short; longer segments are predisposed to kinking, which can cause the shunt to fail.

Fig. 1 The dissected fusion fascia of Toldt with the splenic vein is brought into prominence after mobilization of the left side of the pancreas.

Fig. 2 The splenic vein dissection is depicted from the dorsal aspect of the pancreas with distal vascular control maintained by local pressure on the splenic vein.



An optimal length of the dissected splenic vein anastomosis to the left renal vein is shown. Note that the tail of the pancreas is well above the anastomosis.

RESULTS:

It is a prospective study conducted in a tertiary teaching hospital in between 2011 to 2016. A total number of 32 patients underwent Proximalsplenorenal shunt for non-cirrhotic portal hypertension and extra hepatic portal hypertension. 28 patients are available for follow-up. These patients are aged between 5 to 52 years. Among them 16 are male and 12 are female. The follow-up period ranges from 6 to 60 months. There was no mortality seen at the time of the last follow-up. Type of shunt is proximal splenorenal shunt and splenectomy done in all the patients. Soon after surgery 80% were found to be patentat follow-up. In long term follow-up the patency rate of portorenal shunt decreased. 2 patients shows symptoms like nasal bleed and rectal bleed once treated symptomatically. Remaining all patients shows symptomatic relief in follow-up. Endoscopy shows variceal regression.

COMPLICATIONS:

We did not encounter any mortality, one patient came with complaints of rectal bleed, and another patient came with complaints of nasal bleed treated conservatively.

DISCUSSION :

Noncirrhotic portal hypertension (NCPH) is a heterogeneous group of vascular diseases that lead to portal hypertension (PH) with normal or mildly elevated hepatic venous pressure gradient and preserved liver synthetic function. NCPH causes can be classified according to the site of portal flow resistance as prehepatic, hepatic, and posthepatic, with the hepatic causes further subclassified as presinusoidal, sinusoidal, and postsinusoidal. The main diseases producing NCPH are extrahepatic portal vein obstruction, noncirrhotic portal fibrosis (idiopathic PH), hepatic vein outflow tract obstruction, schistosomiasis, and congenital hepatic fibrosis. Manifestations of NCPH include upper gastrointestinal bleeding, ascites, portosystemic encephalopathy, hypersplenism, and portal biliopathy.

The main therapeutic goal in NCPH is to decrease portal pressure and thus prevent and treat PH complications. Surgery in NCPH aims to bypass the portal resistance site by creating, more commonly, portosystemic shunts (PSS); when not feasible, direct treatment of complications of NCPH (eg, ligation of varices) may be undertaken. Surgery is generally reserved for patients with PH complications who do not respond to pharmacologic and endoscopic treatments but can also be indicated in patients with failure to thrive, limited access to healthcare, or desiring a definitive one-time treatment. Surgical PSS can help to treat portal biliopathy resistant to endoscopic treatment, but persistent biliary obstruction may need a biliary bypass. Some patients with symptomatic hypersplenism (such as repeated infections or bleeding, physical discomfort from massive splenomegaly) may require simultaneous PSS and splenectomy.

EHPVO is to be the major cause of portal hypertension and the commonest cause of major upper gastrointestinal bleeding in children. The etiology of EHPVO in the majority is unknown, but the postulated causes include congenital malformation of the portal vein or acquired thrombosis following umbilical sepsis. Most of these cases present with recurrent episodes of massive upper GI bleed, the first usually occurring before the age of 10 years. Although almost all these patients tolerate the bleeding episode well, some children, especially below the age of 5, may develop transient ascites soon after a bleed. Most have a moderate splenomegaly, although in some the spleen may reach the umbilicus. Some children present with repeated epistaxis and on evaluation are found to have hypersplenism. Liver function and histology is invariably normal and the diagnosis easily made by a ultrasonographic examination of the splenoportal axis, which in 90% of children reveals a block at the formation of the portal vein. In the remaining, either the entire splenoportal axis is thrombosed or the block lies in the hilum of the spleen, giving rise to left sided or "segmental" portal hypertension.

The treatment of an acute episode of hematemesis is fairly standardized and consists of resuscitation with blood and endoscopic sclerotherapy, which controls the bleeding in 95%. These episodes are well tolerated in the majority; however mortality rates upto 31% have been reported. It is in the prevention of recurrence of such bleeds that controversy exists and opinion is divided between endoscopic management on one side and surgery with or without splenectomy on the other.

CONCLUSION:

Porto systemic shunt in extra hepatic portal venous obstruction and non-cirrhotic portal fibrosis is variable option. Repeated hospital visits are reduced when compared to other options like sclerotheraphy. Pre-operative and post-operative assessment with endoscopy, doppler study and Computed Tomographic Portogram.

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