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OUR EXPERIENCE WITH ETIOLOGY AND MANAGEMENT OF SINISTRAL PORTAL HYPERTENSION

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ABSTRACT
Objective: Sinistral portal hypertension (SPH), is a rare clinical syndrome of gastric variceal hemorrhage due to splenic vein thrombosis. This retrospective study highlights the varied etiology, clinical features and outcome of surgical treatment. **Subject and Method:** From June 2010 to June 2015 medical records of patients diagnosed as SPH were analysed. Their clinical features, investigations, surgical intervention and follow up were studied. **Results:** 12 patients,5 males and 7 females were included into the study. Etiology of SPH was chronic pancreatitis (CP) in 5 patients, hydatid spleen in 4 patients, mucinous cyst of tail of pancreas in 2 patients and post surgical splenic vein thrombosis in 1 patient. All the patients were operated with splenectomy and devascularisation done in 5, splenectomy with lateral pancreaticojejunostomy (LPJ) with devascularization in 2 cases while splenectomy with distal pancreatectomy and devascularisation done in 4 and distal pancreatectomy with ligation of splenic artery done in 1 patient. After a mean follow up of 38.5 months there was no episode of bleed in any patient. **Conclusions:** SPH has varied etiology and should be suspected in patients with upper gastrointestinal bleed along with spleenomegaly and normal liver functions. In symptomatic cases splenectomy alongwith devascularization provides excellent outcome.

KEYWORDS: Sinistral portal hypertension, Spleenomegaly, Gastric varices.

INTRODUCTION

SPH also known as segmental portal hypertension or left-sided portal hypertension.[1] In 1939 Greenwald and Wasch first described the pathophysiology of sinistral portal hypertension.[2] Sinistral portal hypertension is a rare clinical syndrome of gastric variceal hemorrhage secondary to some diseases in pancreas[3]. The splenic vein being in close proximity with the pancreas is often affected by any disease in pancreas. The important causes include acute and chronic pancreatitis, pancreatic pseudocysts and pancreatic carcinomas.[4,5] Here we report some rare, along with the common causes of sinistral portal hypertension. Combination of various modalities like gastroscopy, liver function tests, ultrasound examination (with Doppler), contrast-enhanced computed tomography(CECT) scan and or MRI of the abdomen helps to diagnose it. The sinistral portal hypertension is distinguished from other forms of portal hypertension in having preserved liver functions and patent extrahepatic portal vein.[6,7] Traditionally, for symptomatic cases management involves surgical removal of the underlying cause if possible, combined with splenectomy.[8] In this report we studied the outcome of various surgical procedures done for varied etiological factors of SPH.

MATERIAL METHOD

We retrieved all the medical records from June 2010 to June 2015 of the patients diagnosed as SPH at our institute, Sheri-Kashmir institute of Medical Sciences. During this period 12 patients were labeled as having sinistral portal hypertension. This retrospective study focused on etiology, presenting features, investigation and surgical outcomes of these patients. (Table 1 & 2). The presenting symptom of most of these patients was upper gastrointestinal bleed for which they were evaluated and found to have some pathology in pancreas or spleen. Pain abdomen was the second most common complaint followed by heaviness or lump in left upper quadrant in few cases. All of these patients had been evaluated thoroughly and underwent biochemical, endoscopic and radiological examinations. SPH was made as diagnosis if (i) Liver functions were normal. ii) History of upper gastrointestinal bleed. iii) Primary

pathology in pancreas or in the vicinity of splenic vein. (iv) Thrombosis or stenosis of splenic vein on Doppler or CECT. And or (v) presence of gastric varices on endoscopy. After confirmation of the diagnosis, patients were vaccinated. Each of the twelve patients was operated and followed upto average of 38.5 months.

RESULTS

Among these 12 patients 5 were males and 7 were females with age range of 20 to 60 years. (Table 1)The biochemistry detected anaemia in 8 patients while liver functions were normal in all the patients. Endoscopy showed gastric varices in 10 patients. CECT abdomen and or MRI with MRCP was done to define the lesions and know status of liver, portal vein and splenic vessels. Liver and extra hepatic potal vein was normal in each case. All the patients had some grade of splenomegaly while splenic vein thrombosis was seen in 9 patients and in 3 patients there was only stenosis of the splenic vein. Based on all these investigations the underlying pathology was detected as CP in 5 patients ,hydatid cyst of spleen in 4 patients (Fig 1), mucinous cyst in tail of pancreas in 2 patients(Fig 2) and post operative splenic vein thrombosis in 1 patient (Fig 3). 3 patients of CP were having pseudocyst in the vicinity of tail of pancreas while 2 were having chronic pancreatitis without any pseudocyst formation. Spleenectomy with distal pancreatectomy with pericardial devascularization was done in all 3 CP patients with pseudocysts. While spleenctomy with LPJ was done in remaining 2 patients of CP. We had one post operative patient in whom lateral pancreaticojejonostomy was done for chronic calcific pancreatitis. After about one year he presented with upper gastrointestinal bleed. On endoscopic examination patient was diagnosed as GIST (Gastrointestinal Stromal Tumour) but on radiological investigations patient was found to have splenic vein thrombosis with pergastric varices. On repeat endoscopy it was confirmed that patient was having gastric varices not GIST.

As this part of the country is endemic for hydatid disease, ours is a high volume centre and contributed a great to the world literature for this diseases. During this period of five years we have found four cases who presented as malena but on further evaluation where found to have hydatid spleen in and around hilum compressing the spleen vein. Spleen is an unusual location for Hydatid disease and thus a very rare cause of sinistral portal hypertension. We also found two cases of cystic lesions of tail of pancreas responsible for upper gastrointestinal bleed. Who after distal pancreatectomy on histopathological examination were diagnosed as mucinous cysts of pancreas.

Follow up

All of these patients were followed for minimum of 6 months and maximum of 60 months with mean follow up of 38.5 months. There was peripancreatic collection in one patient in whom only ligation of splenic artey was done. That collection was drained under ultrasound guidance. None of the patients has developed any upper gastrointestinal bleed and there was marked improvement in hemoglomin levels of each patient. There was no mortality.

Table 1.

Age range	20-60years
Male	5
Females	7
Etiology	
Chronic Pancreatitis	
With Pseudocyst	3
Without pseudocyst	2
Mucinous Cyst tail of pancreas	2
Follow up case of LPJ with splenic vein thrombosis	1
Hydatid cyst of spleen	4

Table 2

Clinical features	9
Malena	7
Pain Abdomen	8
Lump abdomen	12
Spleenomegaly	8
Anemia	10
Gastric varices	
Procedure Performed	5
Spleenectomy alone	2
Spleenectomy with LPJ	4
Splenectomy with distal pancreatectomy	1

Distal Pancreatectomy with ligation of spleen artery (Warshaw procedure)	

Legends

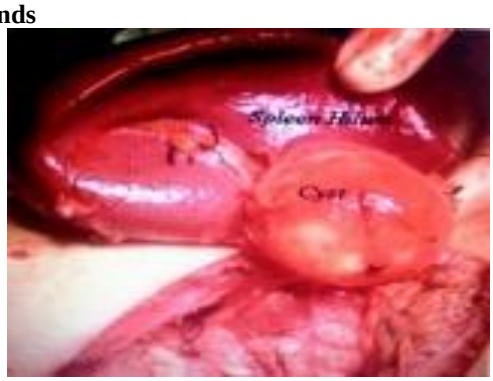

Fig: 1 Operative picture showing hydatid cyst at hilum of spleen compressing splenic vein.

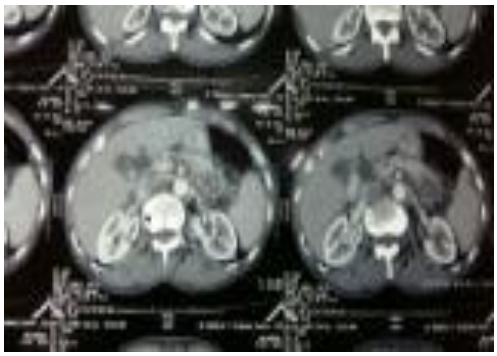

Fig: 2 CECT picture showing cystic lesion at the tail of pancreas.

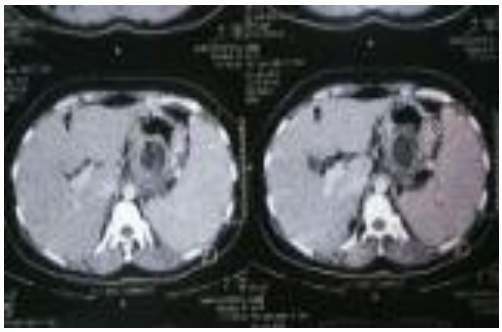

Fig:3 Post operative CECT picture showing varices around splenic hilum.

DISCUSSION

SPH is the clinical manifestation of splenic vein obstruction which is usually an outcome of some disease in the pancreas. Splenic vein thrombosis complicates 7-20% of patients having pancreatitis or a pancreatic pseudocyst however, bleeding occurs in only 5% of patients.[7] Here we have considered only those case which had presented with upper gastrointestinal bleed and were labeled as SPH. In this study we have presented some unusual causes of SPH which have showed that primary pathology needs not to be in pancreas always, contrary to the common belief. Hydatid diseases though rare[9] in spleen can sometimes present as a cause for splenic vein obstruction and thus present as upper gastrointestinal bleed. Hydatid cyst of spleen should be included in the etiological list of SPH, especially in endemic regions. Iatrogenic splenic vein injury has been reported as a cause of SPH[10], but in our case splenic vein thrombosis presented almost after one year of the primary surgery. The reason for this maybe a subtle injury or extension of fibrosis due to ongoing process of chronic pancreatitis.

The combination of various modalities like gastroscopy, liver function tests, ultrasound examination (with Doppler), contrast-enhanced computed tomography (CECT) scan and or MRI of the abdomen helps to make the diagnosis of SPH. In this study endoscopy was done in all patients but gastric varices were recognized in only 10 patients. But during surgery perigastric varices were noticed in all of these patients and for these varices some sort of devascularization was carried out in each of them.

Traditionally, for symptomatic cases management involves surgical removal of the underlying cause if possible, combined with splenectomy. We did not do spleenectomy in a female patient who had cystic leson in the tail of pancreas. She underwent distal pancreatectomy along with pericardial devascularization and only ligation of splenic artey. This patient developed peripancreatic collection which was drained under ultrasound guidance. She has been under our follow up from last six months and there has been no complaint of gastrointestinal bleed. Thus spleen can be preserved if the underlying cause leading to SPH can be treated. But splenic artery should be ligated to decrease the pressures in splenic and short gastric veins while preserving arterial flow to spleen through short gastric arteries.

CONCLUSIONS

SPH should be considered in patients with spleenomegaly, upper gastrointestinal bleed with normal liver functions and patent extra hepatic portal vein. Hydatid cyst in the vicinity should be considered as etiological factor of SPH. Management should be removal of the underlying cause if possible, combined with splenectomy. However, Spleen can be preserved if circumstances are favourable.

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