Isolated Splenic Vein Occlusion: A Report of Two Cases

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Abstract

In two patients presenting with haemetemesis a diagnosis of isolated splenic vein occlusion (SVO) was established at laparotomy. SVO was caused by a pseudocyst of the pancreas in one case and a hydatid cyst of the left kidney in the other. A splenectomy plus distal pancreatectomy was done in the first case and cyst excision with ligation of the gastric varices in the second case.

Case 1

An 18-year old male presented with recurrent bouts of haemetemesis over a 10-month period following a road accident. Physical examination revealed moderate anaemia and splenomegaly. The haemoglobin was 9.6 g/dl. The liver function tests and serum proteins were within normal limits. No abnormality was detected on an upper gastrointestinal barium study. On gastroscopy a tumour was suspected arising from the posterior wall of the stomach near the fundus; the oesophagus was normal. At laparotomy varices were seen involving the gastroepiploic, short gastric and gastric fundic veins. The spleen was moderately enlarged but the liver appeared normal. Exploration of the lesser sac revealed a cystic swelling arising from the pancreatic tail. A diagnosis of splenic vein occlusion (SVO) was made and a splenectorny and distal pancreatectomy were performed.

On section the cyst was thick walled and filled with an opalescent fluid. Absence of an epethelial lining on histology confirmed the diagnosis of pseudocyst of the pancrease. The accompanying figure shows the splenic vein being obliterated by the cyst.

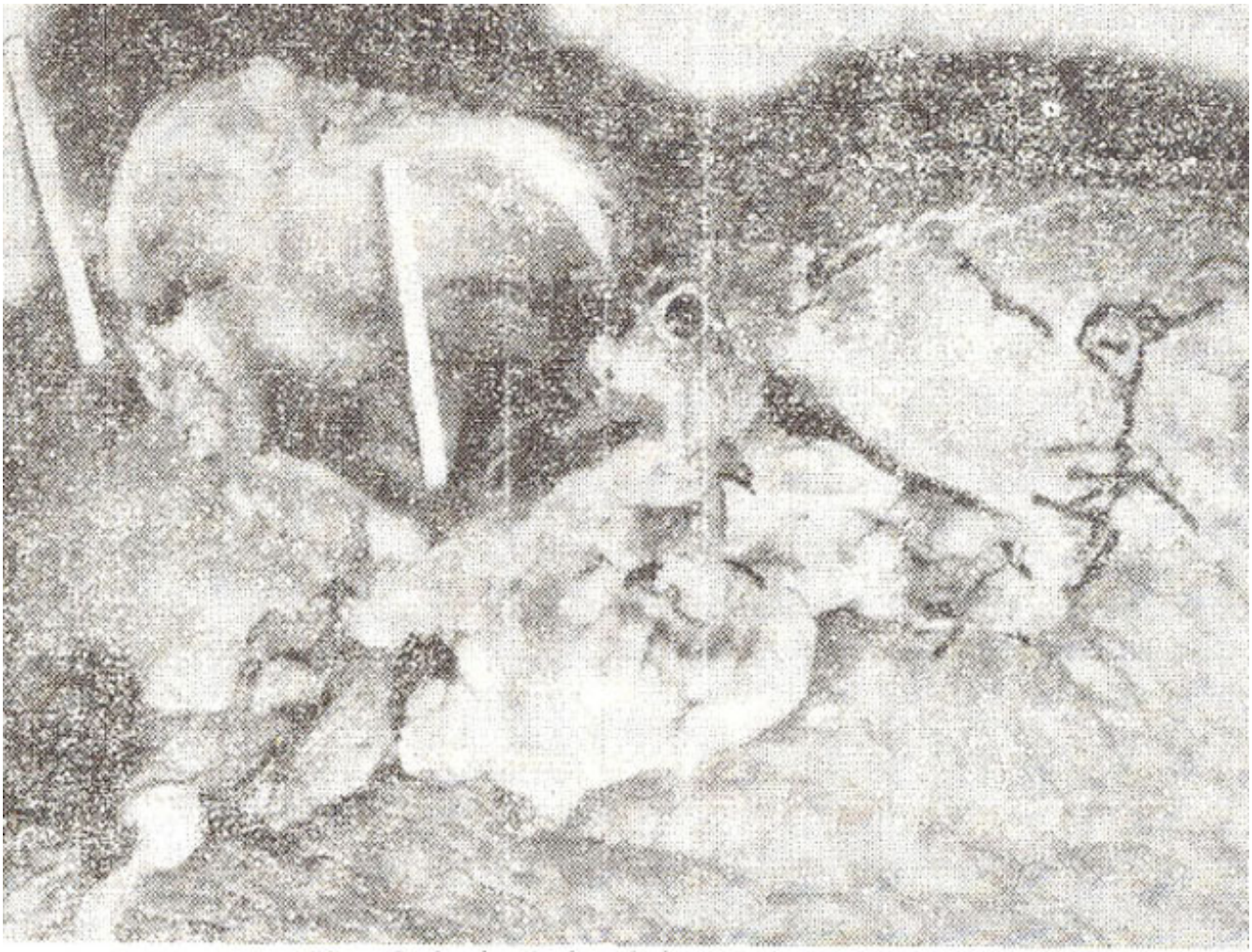

Fig: Splenic Vein obliterated by the cyst.

The post-operative recovery of the patient was uneventful and there has been no recurrence of symptoms during a 1-year follow up period.

Case 2:

A 40 year old male was admitted to Liaquat National Hospital in a state of shock following profuse upper gastrointestinal bleeding. Haemoglobin was 3.5 gfdl and blood urea 28.2 mmol/L. Following resuscitation an upper gastrointestinal barium study was attempted but was inconclusive. A massive rebleed within 48 hours of admission necessitated a laparotomy. Gastric fundic varices were found, but the liver and spleen appeared normal.

Exploration of the lesser sac revealed a cyst arising from the upper pole of the left kidney. Aspiration of the cyst resulted in obvious decongestion of the varices. The cyst was excised and in addition the gastric varices were over-run with chromic catgut sutures through a gastrotorny. Scolices were found in the cyst and confirmed it's hydatid nature. The post-operative recovery was unremarkable, the blood urea quickly returning to normal values. An intravenous pyelogram showed normal function in the left kidney with slight distortion of the upper calyx. The patient has remained well for last one year.

Discussion

Left-sided portal hypertension resulting from isolated SVO is now an established clinicopathological entity. The splenic vein is intimately related to the posterior surface of the pancreas and may be readily occluded (Bevin and Pickett, 1967) in traumatic, inflammatory (Shaldon and Sherlock, 1961) and

neoplastic lesions of that organ (Hurwitt et al, 1954). Pseudocysts of the pancreas are the commonest cause of SVO. Neoplastic invasion from the stomach, colon and left kidney have also been incriminated. The rarer causes of SVO on record are: retroperitoneal fibrosis, aneurysm of the splenic artery, benign gastric ulcer, reti-. culosis and primary thrombosis of the vein in polycythaemia.
Following complete SVO, blood flow is redirected to the portal vein via: (i) the gastro-epiploic vein and (ii) the short gastric, fundic and coronary veins. The size of the collateral circulation determines whether or not there is going to be lelt-sided portal hypertension. In fact, gastric varices and splenomegaly develop in 65% cases (Sulton et al, 1970). Occasionally the coronary vein drains into the splenic vein instead of the portal vein (Little and Moosa, 1981). In this event oesophageal varices may also occur as a result of SVO.
The patient may present with haemetemesis and malena or with anaemia as a result of chronic occult blood loss. In a patient with suspected SVO a history of abdominal trauma or acute panceratitis should be sought. In addition to the splenomegaly,an upper abdominal mass may be palpable when a neoplasm or pseudo-pancreatic cyst is the cause of SVO. Liver function tests and serum proteins are as a rule within normal limits. Gastric varices are notoriously difficult to diagnose on barium examination and on endoscopy (Kar's Whol, 1960 and Hershfield and Morrow, 1968) but prominent folds in the fundic area have occasionally been observed (Little and Moosa, 1981).
SVO may be diagnosed by means of a splenoportogram, although this procedure has its own hazards. The venous phase of a coeliac axis angiogram is safer and is diagnostically accurate. However, angiographic facilities are not always available and often the diagnosis is only established at laparotomy. Variceal dilatation of the gastro-epiploic, short gastric and fundic veins, splenomegaly and a normal liver should direct attention to the lesser sac for a possible cause of SVO (Little and Moosa, 198!). Occasionally the lesser sac may be inaccessable as a result of adhesions.
The treatment of left sided portal hypertension is splenectomy. In addition a pancreatic pseudocyst may be anastomosed to the bowel or resected. Anterior gastrotomy and over running of the gastric varices has usually been under taken if the bleeding continued after splenectomy. In the second case presented here, simple excision of the renal cyst along with under-running of the varices seems to have sufficed. Prognosis following splenectomy in non-malignant cases, is excellent with a reported recurrence of bleeding in only 3% cases.

Acknowledgement

We would like to thank Dr. Sarwar Zuberi for referring the first Case and performing cudoscopy.

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