CASE REPORT

Traumatic pancreatic fistula with sinistral portal hypertension: Surgical management

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Combined ductal and vascular injuries are awesome complications of pancreatic injury. We report on a 29-year-old male unrestrained driver who sustained a blunt abdominal injury from the steering wheel in a high velocity head-on car collision. He developed a pancreatic fistula, portosplenic venous thrombosis and sinistral portal hypertension as a result of complete duct disruption at the pancreatic neck. We describe a safe surgical strategy of spleen-preserving distal pancreatectomy after failed medical and endoscopic management.

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Key words: Spleen-preserving distal pancreatectomy; Pancreatic fistula; Portosplenic venous thrombosis; Sinistral portal hypertension

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INTRODUCTION
Pancreatic injury is uncommon but can be lethal when associated with adjacent organ injury[1,2]. The victim can develop awesome sequelae such as pseudocyst or fistula when the pancreatic ductal injury escapes initial diagnosis[3-5]. Portosplenic venous thrombosis can also develop from direct injury or the inflammatory process and can cause sinistral (left-sided) portal hypertension[6]. We describe a formidable pancreatic injury with ductal and vascular complications and our successful multimodality management.

CASE REPORT
A 29-year-old male unrestrained driver sustained a blunt abdominal injury from the steering wheel in a high velocity head-on car collision 12 mo ago. He had a trauma laparotomy in a community hospital and a retroperitoneal hematoma was discovered but not explored as it was stable and not pulsating. He developed a persistent serous discharge at the tube drain wound site in the right upper abdomen after removal of the drain 10 d after the operation. This was treated as a fistula but was not resolved by total parental nutrition and octreotide. By the time of referral (8 mo after injury), there was a high output (400 mL/d) serous discharge rich in amylase (13647 U/L) and lipase (119348 U/L). He had intractable pain and had lost 24 kg. Magnetic resonance (MR) and endoscopic retrograde cholangiopancreatography showed a complete duct disruption at the neck of the pancreas. We describe a safe surgical strategy of spleen-preserving distal pancreatectomy after failed medical and endoscopic management.
with sinistral portal hypertension because of the presence of short gastric varices in MR (Figure 2) although he had no clinical or endoscopic evidence of bleeding varices. Endoscopic stenting was not successful as a guide wire could not pass through the disruption. The patient was explored using a bilateral subcostal incision including the cutaneous fistula orifice and the fistula tract was excised down to the pancreatic neck in the lesser sac after incising the gastrocolic ligament (Figure 3). The self-truncated distal pancreas was found tethered to the neck by fibrous tissue. The distal pancreas was resected en bloc with the densely adherent and obliterated splenic artery and vein. The splenic artery was ligated near the celiac origin and the vein at the confluence with the portal vein. The inferior mesenteric vein was identified and ligated. The other end of the splenic vessel was transected with linear stapler just at the tip of the pancreatic tail, preserving the splenic hilum. The pancreatic head stump was oversewn with continuous polypropylene suture. The patient recovered uneventfully, gained 5 kg and resumed work 1 mo later. He did not have steatorrhoea or diabetes. His blood smear showed normal platelets and absence of Howell-Jolly bodies and Doppler ultrasound of the spleen showed normal size and intact perfusion of all zones at 4 mo follow up.

DISCUSSION

Steering wheel injury in high velocity traffic accidents is a common mechanism for a blunt pancreatic injury when the abdominal organs compress the soft gland on to the rigid and fixed spine. Initial pancreatic injury is difficult to diagnose as the gland is embedded by a retroperitoneal hematoma. Low output fistula from a partial duct transection can be successfully managed by conservative treatment (total parental nutrition and octreotide) and endoscopic stenting. However, these lengthy and costly conservative treatments proved unsuccessful in our patient with complete duct abruption and high output fistula. The injury also resulted in inflammatory thrombosis of the portosplenic venous confluence and sinistral portal hypertension. We described our rationale of a safe and successful surgical strategy to deal with this complex injury in an inflammatory peritoneum.

The first controversy was the mandatory splenectomy-
my for uncomplicated sinistral portal hypertension in a difficult inflammatory peritoneum. Preservation of the spleen is worthwhile as it avoids the 3.2% sepsis, 1.3% sepsis-related mortality and the bleeding complication in re-operation for chronic inflammatory peritoneum. While splenectomy is the treatment of choice for variceal bleeding, there is no consensus about the treatment of asymptomatic patients. Previous studies may have overestimated the incidence of variceal bleeding in splenic vein thrombosis and have suggested prophylactic splenectomy. However, two recent studies with advanced imaging identified more asymptomatic sinistral portal hypertension and found that the true variceal bleeding risk was as low as 4%. Heider et al. could incidentally identify 40 splenic vein thrombosis and varices among chronic pancreatitis patients by computed tomography (CT) and they did not have prior bleeding. By adopting an expectant approach, only 4% eventually bled and required splenectomy in a mean follow up of 40 mo. This expectant approach had better survival rates than the historical control of routine splenectomy which carried more complications. Similarly, Koklu et al. reported 4% life-time risk of variceal bleeding in a mean follow up of 20 mo among the 24 patients who had concomitant diagnosis of sinistral portal hypertension by CT. Likewise, a low bleeding incident was confirmed by two prospective longitudinal studies of chronic pancreatitis. Bernades et al. reported 2 bleeding incidents among 35 patients with portal hypertension in a median follow up of 22 mo. Izbicki et al. reported no bleeding incidents in 36 patients with extrahepatic portal hypertension in median follow up of 51 mo. Lofrus et al., in his retrospective comparative study, justified an expectant treatment for 12 sinistral portal hypertension patients without prior history of bleeding as their 3 year survival and bleeding rates were the same as the 25 patients requiring splenectomy for variceal bleeding. These 5 studies also found higher morbidity for the difficult operation for portal hypertension and concluded that varical bleeding was rare in extrahepatic portal hypertension and did not justify prophylactic surgery. These studies justify our spleen preservation surgery in order to avoid potential bleeding and sepsis complication.

The second controversy following spleen preservation was the splenic viability when the main splenic vessels could not be dissected from the pancreas due to chronic inflammation and scar tissue. There were studies showing that distal pancreatectomy with en bloc resection of splenic vessels can be safely done. In these cases, the splenic vessels were ligated just at the tip of the pancreatic tail. The splenic hilar pedicle is preserved to allow circulation of the splenic hilar vasculature can be preserved. Thrombosed splenic vessel are sacrificed as long as the splenic hilar vasculature can be preserved. In summary, high output pancreatic fistula, portosplenic thrombosis and sinistral portal hypertension can be the sequelae of severe traumatic complete pancreatic duct disruption. In the presence of chronic inflammation and adhesion, these complex injuries can be safely managed by distal pancreatectomy after failure of conservative management. Without prior history of variceal bleeding, the normal-sized spleen can be preserved even when the thrombosed splenic vessel are sacrificed as long as the splenic hilar vasculature can be preserved.

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Bojal SA et al. Traumatic pancreatic fistula with sinistral portal hypertension

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