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Traumatic Injuries of the Portal Vein

The Role of Acute Ligation

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Injuries to the portal vein are rare but have a high risk with a mortality of 50–70% secondary to exsanguinating hemorrhage. When managing injuries to the portal vein, lateral venorrhaphy, end to end anastomosis, or an interposition graft should be attempted whenever possible. However, in a hemodynamically unstable patient or when confronted with a nonreconstructable injury, acute portal vein ligation may be the procedure of choice as it is safely tolerated in some 80% of patients. Of eleven reported patients in whom the portal vein was ligated acutely for traumatic injury, six survived. Four of the nonsurvivors died of massive associated injuries. Of the six surviving patients, five tolerated acute ligation of the portal vein without complication. Should portal vein ligation be performed a "second look" operation is essential in 24 hours to examine the bowel for viability. A portosystemic shunt with its inherent complications should not be done as a primary procedure when attempts at reconstruction of the portal vein have failed. Shunting should be reserved for those few patients who develop stigmata of portal hypertension or impending infarction of the bowel.

PATIENTS WITH INJURIES to the portal vein and periportal structures often die of hemorrhage before reaching the hospital, but in those surviving long enough to reach the operating room, the mortality is still 50–70%. This high mortality is due to the difficulty in controlling hemorrhage and secondarily to associated enteric and pancreatic injuries. Current methods of dealing with injuries to the portal vein have been described by Fish[9] as follows. 1) End-to-end anastomosis. 2) Lateral venorrhaphy. 3) Graft interposition (autologous vein or synthetic material). 4) Superior mesenteric vein to splenic vein anastomosis. 5) Ligation of the portal vein with portosystemic shunt. 6) Ligation of the portal vein alone. This paper describes a case of acute portal vein ligation for trauma with three year follow-up and review of the literature.

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Case Report

A 26-year-old man entered the emergency room with a stab wound of the abdomen and a blood pressure of 50mmHg. Despite resuscitation with 3 l of Ringer's lactate and 2 units of whole blood, the patient was still hypotensive. At operation a large expanding retroperitoneal hematoma on the right was noted. This was entered, and a 4cm laceration of the lateral wall of the inferior vena cava was found and repaired. The lesser sac was then explored as blood was noted to accumulating there. A through and through injury of the body of the pancreas with a virtually complete transection of the portal vein was found. Distal pancreatectomy and splenectomy were performed, because of the patient's unstable condition, the portal vein was ligated and reconstruction was not attempted. Immediately after ligation, the superior mesenteric vein distended markedly and pressure within it rose to 56cm H_2O. The patient was returned to the operating room 24 hours later for a "second look." At this time, ascites was found and the pressure within the portal vein was 50cm H_2O. All of the small bowel appeared viable. Postoperatively, he developed a large left upper quadrant abscess in continuity with the splenic flexure. The abscess was drained with resection of the splenic flexure and an end colostomy and mucous fistula was established.

A superior mesenteric arteriogram performed one month postoperatively, showed reconstitution of portal flow with filling of the

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TABLE 1. *Portal Vein Ligation for Traumatic Injuries*

Source	Number of Patients	Post-Operative Portal Hypertension
Mattox	4 (0)*	—
Chisholm	2 (1)	0
Peterson and Sheldon	3 (3)	0
Pachter	1 (1)	0
Patton and Johnson	1 (1)	1
Total	11 (6)	1

( ) Survivors.
* All patients died of massive associated injuries.

intrahepatic portal vein by collateral vessels. Six months later the colostomy was closed and at that time, pressure in the superior mesenteric vein was 14cm H_2O. Followed closely in the three years since operation, the patient is asymptomatic and without evidence of ascites.

Discussion

Although reports of portal venous injuries usually refer to single cases,[2,6,10] large series have been reported by Graham[11] and Peterson.[17] It is generally recognized that lateral venorrhaphy with primary repair of the portal vein may be the procedure of choice. The effects of acute portal ligation have been studied in experimental animals since the late 1800's. Bernard (1859)[1] and Schiff (1861),[18] showed in dogs that acute portal vein ligation was uniformly fatal. Subsequently, studies by Solowieff[19] and by Neuhof,[13] demonstrated that death in this animal could be prevented if the portal vein were occluded gradually rather than ligated acutely. Their experiments seemed to suggest that development of collateral circulation might prevent death from portal vein interruption. In 1877, Eck[8] showed that the portal vein could be ligated with impunity if decompression were accomplished by end to side superior mesenteric vein to inferior vena caval shunt.

Brewer[3] successfully ligated the portal vein in a patient with a hyatid cyst of the liver in 1908, but the concept persisted that acute portal vein ligation was uniformly fatal. In 1950, Child, et al.[4] demonstrated that acute portal vein ligation could be tolerated in approximately 80% of monkeys. In this species, acute portal vein ligation resulted in the rapid fall of systemic arterial blood pressure. Although the arterial pressure returned to normal levels within one to four hours, the elevated portal pressure persisted. Three weeks later, portal pressure was less, but still higher than preligation levels. Armed with these observations, Child, et al.[5] successfully ligated the portal vein in six patients for malignancy. Eleven case reports of ligation of the portal vein for traumatic injuries have been described (Table 1) with six patients surviving. The four patients reported by Mattox[12] who underwent portal vein ligation, did not survive. It should be recognized however, that these patients died of massive associated injuries. Of the remaining six patients who did not succumb to concomitant injuries, portal vein ligation was successful in five.

Current objections to portal vein ligation arise from the possibility that it could result: 1) in portal hypertension, varices, and upper GI bleeding, 2) fatal hypotension as a result of sequestration of large volumes in the splanchnic circulation, 3) thrombosis and subsequent infarction of the bowel. Patton and Johnson,[16] described two patients who actually developed portal hypertension—one after acute ligation and the other after lateral venorrhaphy, which subsequently thrombosed. On this basis, Graham and Mattox[11] stated that portosystemic decompression was the procedure of choice for portal vein injuries that resulted in severe narrowing or for injuries to the portal vein which otherwise require ligation.

On the other hand, portosystemic diversion at the initial surgical procedure may be unnecessary and carries its own inherent complications. Encephalopathy after shunting procedures* in the cirrhotic patient, for example, varies in incidence from 11 to 25%.[7,14,15,20] Reporting on the encephalopathic complications in noncirrhotic patients, Voorhees[21] evaluated 16 patients who underwent portosystemic shunts in childhood. Eight patients had pathologically normal livers and eight patients had pathologically abnormal livers at the time of surgery. Of the eight patients with normal livers, five had severe psychological problems and three developed organic brain syndromes. Moreover, four of the five patients described by Fish,[9] who underwent immediate portosystemic shunt after portal vein ligation, became encephalopathic. Viewed in light of this experience, immediate portosystemic shunt for unreconstructable portal vein injuries may in fact be more hazardous than acute ligation. Initial rise in portal pressure followed by ligation usually either declines significantly[4] or disappears completely as in the reported case. Should a significant degree of portal hypertension persist (greater than 40cm H_2O) with variceal bleeding, an elective portosystemic shunt can then be considered.

Volume depletion secondary to splanchnic bed sequestration with resulting hypotension and a decrease in cardiac output can be avoided with the use of a Swan-Ganz thermodilution catheter. The left heart filling pressure as well as cardiac output can then easily be managed by adjustments of appropriate amounts of fluid.

* Excluding the Warren Shunt.

With regard to thrombosis of the superior mesenteric vein and subsequent infarction of the bowel, a "second look" operation is mandatory 24 hours later. Should the bowel appear on the verge of compromise a venous bypass graft from the superior mesenteric vein to the hepatic end of the portal vein could then be constructed with either autologous vein or synthetic material. The 24 hour period after the initial procedure should allow ample time to correct deficiencies in volume, acidosis, respiratory dysfunction and cardiac instability.

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