

Occlusion of an Aberrant Right Hepatic Artery, Originating from the Superior Mesenteric Artery, Secondary to Blunt Trauma

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Hepatic arterial injury secondary to both blunt and penetrating traumas is well documented in the literature by case reports and articles regarding management. Penetrating injuries to the hepatic arteries are more common than injuries from blunt trauma.¹ Hepatic arterial injuries secondary to blunt traumas are often associated with injuries to other vital organs or vascular structures, resulting in higher mortality rates. We describe the case of an individual involved in a motor vehicle crash, who sustained blunt traumatic occlusion of an aberrant right hepatic artery, originating from the superior mesenteric artery. No reports describing such an injury exist in the English language literature.

CASE REPORT

A 57-year-old man was an unrestrained driver of a motor vehicle involved in a rollover crash. He was ejected from the vehicle and sustained a brief loss of consciousness. The patient was transferred to the nearest hospital where his chief complaint was mid-scapular back pain. At arrival, the patient's vital signs were stable and his Glasgow Coma Scale score was 15. Physical examination findings included a left occipital scalp laceration and numerous lacerations and abrasions throughout his lower extremities. He had an area of ecchymosis over his right upper quadrant without physical signs of tenderness or guarding. No neurologic abnormalities were noted. A gross fracture dislocation of his left elbow was present with a palpable distal radial pulse. Radiographic work-up included computed tomographic (CT) scans of his head, C-spine (C7 to T1), chest, abdomen, and pelvis. Reports of the results of these scans were significant for the following: normal head CT scan; fracture of the lamina of C7 with cervical epidural air; right loculated pneumothorax with pneumomediastinum; no evidence suggestive of aortic injury; fluid around the liver, head of the pancreas, and C-loop of the duodenum. A Foley catheter was inserted, the scalp laceration

was closed, and the patient was transferred to Baystate Medical Center.

At arrival to our hospital, the patient was hemodynamically stable, alert, and oriented with a Glasgow Coma Scale score of 15. The findings at physical examination were similar to the findings described earlier. Upright chest x-ray film was normal with no evidence of a pneumothorax. Subsequent CT scan of the C6 to T1 region was significant for epidural air without evidence of fracture. Review of the CT scan of the abdomen and pelvis, received from the referring hospital, suggested the presence of focal intravenous contrast extravasation in the region of the head of the pancreas associated with peripancreatic fluid. Upon repeating this study, no evidence of contrast extravasation was observed. Further work-up to evaluate this problem included an angiogram that revealed occlusion of an aberrant right hepatic artery, originating from the superior mesenteric artery (SMA). A gap of approximately 3 cm exists between the proximal site of occlusion to reconstitution of the more distal portion of the aberrant right hepatic artery (Fig. 1). A celiac axis angiogram (Fig. 2) shows the left hepatic artery originating from the celiac axis, the middle hepatic artery arising from the common hepatic artery at the junction with the gastroduodenal artery, and the right hepatic artery arising from the proximal SMA. The right hepatic artery is occluded, but collateral blood flow comes from the anterior and posterior pancreatic arcade to fill the gastroduodenal artery that gives rise to a branch reconstituting the right hepatic artery (Fig. 2). No extravasation of contrast was noted.

Laboratory data revealed a serum amylase of 123 U/L (0–123 U/L), a serum lipase of 1,115 U/L (23–300 U/L), normal liver function test results, and a normal coagulation profile. A right-sided chest tube was inserted, and the patient was transferred to the Surgical Intensive Care Unit for further management.

DISCUSSION

Isolated injury to the hepatic artery is relatively rare.^{1,2} We report the case of a man involved in a motor vehicle crash, who sustained blunt traumatic occlusion of his aberrant right hepatic artery. This finding has not been described previously.

Jurkovich et al.¹ reported a series of 99 patients with portal

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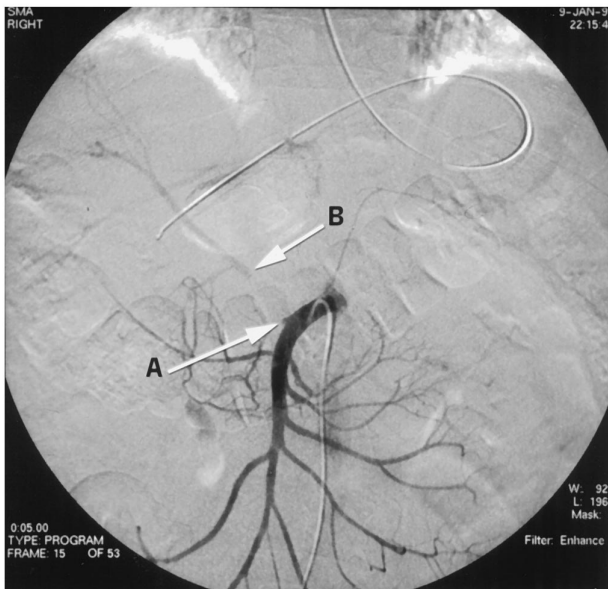


FIG 1. SMA angiogram demonstrating a 3-cm long occlusion of an aberrant right hepatic artery. *A* and *B* arrows delineate the point of interruption and the point of reconstitution of flow in the right hepatic artery, respectively.

triad injuries: 55 had injuries to the portal vein, 28 to the hepatic artery, and 35 to the bile duct. In this review, the hepatic artery was the least common structure injured with equal distribution in frequency between the right, left, proper, and common hepatic arteries. Fifteen hepatic arterial injuries were due to penetrating trauma, and 13 occurred secondary to

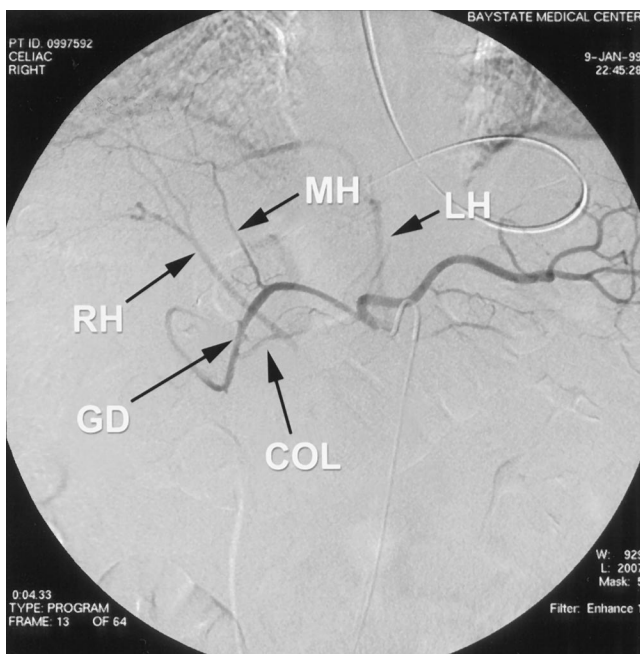


FIG 2. Celiac artery angiogram demonstrating the course of the left hepatic artery (LH) and the collateral pathways to the right hepatic artery (RH). The middle hepatic artery (MH) arises from the common hepatic artery. Reconstitution of the right hepatic artery occurs through collateral vessels (COL) by means of the pancreaticoduodenal arcade and the gastroduodenal artery (GD).

blunt injury. Management consisted of primary repair in 7 patients, with only 1 survivor, and ligation in 19 patients with seven survivors. Two patients died before repair. Among those patients who underwent ligation, one patient who sustained prolonged severe shock eventually developed partial liver necrosis.

The right hepatic artery originates from the SMA in 10 to 12% of individuals.^{3,4} This, aberrant, right hepatic artery courses behind the head of the pancreas and behind the common bile duct. In patients without underlying liver dysfunction, hepatic arterial injuries may be managed by ligation of the offending vessel. This procedure usually can be performed with relative impunity, because approximately 80% of the oxygenated blood received by the liver comes from the portal vein with the remainder provided by the hepatic artery.^{5,6} This distribution allows ligation of either hepatic artery as long as flow is present within the portal vein. In subacute and chronic cases of hepatic arterial occlusion, several physiologic compensatory mechanisms can develop such as the formation of collateral vessels and an increase in hepatocyte oxygen extraction capability.^{2,7,8} However, there are reports of liver necrosis after hepatic artery ligation or transection in patients with prolonged episodes shock or multiple abdominal injuries.^{2,9}

In our case, diagnosis of traumatic occlusion of an aberrant right hepatic artery, originating from the SMA, was made by angiography and management was observation with serial abdominal examination and laboratory testing. No further treatment was necessary for this injury. Our patient did not develop any chemical or radiographic evidence of liver necrosis.

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