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Unique de novo presentation of blunt liver trauma: Multiple small intra-hepatic arterioportal fistulas

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Abstract

Post-traumatic arterioportal fistulas are usually large and single to few in number. Mechanism of arterioportal shunt in traumatic settings is primarily due to direct hepatic artery to portal vein communication. A unique case of multiple and small arterioportal fistulas related to blunt abdominal trauma without capsular or biliary radical involvement is presented. The mechanism of this finding is probably due to microscopic mechanical disruption of the liver parenchyma. Shunting was successfully treated with selective gelfoam embolization of the hepatic artery branch. Post-traumatic arterioportal fistulas could be the only manifestation to blunt liver trauma representing an early stage of liver trauma ahead of CT-based Injury Severity of Blunt Hepatic Trauma stages. Minimal intervention has a role in safely managing this condition and averting long-term sequelae.

Keywords: Blunt hepatic trauma; Arterioportal fistula; Embolization; Multiple; Small

1. Introduction

Blunt hepatic trauma is a spectrum of injury ranging from minor capsular injury to massive bilobar tissue destruction (Table 1). The incidence of hepatic vascular injuries after blunt trauma is much lower than that after penetrating injury and the incidence is still not established [1]. Arterioportal shunting can happen with blunt hepatic trauma. Arterioportal fistulas APF-abnormal flow between branches of hepatic artery and portal vein-occur in multiple clinical scenarios, including congenital and acquired causes. Arterioportal fistulas in post-traumatic setting are thought to be due to direct arterioportal connections, however also transsinusoidal and transvasal routes are possible in setting of shock or post-traumatic occlusion of branches of hepatic artery, hepatic and portal vein [2]. Angiogram and angiographic embolization are standard management of post-traumatic arterioportal fistulas.

2. Case report

A 23-year-old male patient, who had no remarkable past illness, was a victim in slow speed motor vehicle accident. The patient was riding a motorcycle with speed of approximately 25 mph and was hit by a car. On admission he complained of the left knee, B/L upper extremity and left sided chest pain. On physical examination he had open wound of the left knee and abrasions of both upper extremities, which were appropriately managed. Pulses were present in all four extremities and physical examination of his abdomen and chest was benign. Vital signs on the admission were stable. The patient did not lose consciousness nor had amnesia. There were no signs of significant blood loss. There was no gross blood per rectum. Laboratory values on admission were normal except for mildly elevated WBC count (11,400). The patient was not on any medications, including steroids. He was allergic to Penicillin and Amoxicillin. The patient was a heavy smoker—2 packs/day (unspecified for how long) and a social drinker. To his knowledge he was not HBV or HCV positive.

A contrast-enhanced dynamic CT examination of the abdomen was performed as per trauma protocol with a multi-detector CT (Toshiba Aquilion 16-slice). This revealed a circumscribed area of contrast enhancement within the right

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Table 1
CT-based injury severity of blunt hepatic trauma

CT-based grade	Criteria
1	Capsular avulsion, superficial laceration(s) less than 1 cm deep, subcapsular hematoma less than 1 cm in maximum thickness, periportal blood tracking only
2	Laceration(s) 1–3 cm deep, central-subcapsular hematoma(s) 1–3 cm in diameter
3	Laceration greater than 3 cm deep, central-subcapsular hematoma(s) greater than 3 cm in diameter
4	Massive central-subcapsular hematoma greater than 10 cm, lobar tissue destruction (maceration) or devascularization
5	Bilobar tissue destruction (maceration) or devascularization

Source: Ref. [6].

hepatic lobe, measuring 3.5 cm in coronal dimension, 5 cm in sagittal diameter and 7.5 cm in cranio-caudal dimension (Fig. 1A–C). Diagnosis of hepatic contusion was made and angiographic study was recommended for further evaluation. There was no rib fracture. There was mild fat stranding of the soft tissues within the right aspect of the abdomen compatible with contusion.

Subsequently, hepatic arteriogram was performed revealing a delayed blush in an area corresponding to the CT finding and early filling of multiple small branches of the portal vein, consistent with multiple small arterioportal fistulas (Figs. 2–4). Subsequently, selective angiogram of the right hepatic artery was performed and the branch of the right hepatic artery supplying the area of the blush was embolized with gelfoam. Follow-up angiogram did not demonstrate any further blush or early filling of the portal vein radicals suggesting occlusion of the arterioportal fistulas (Fig. 5).

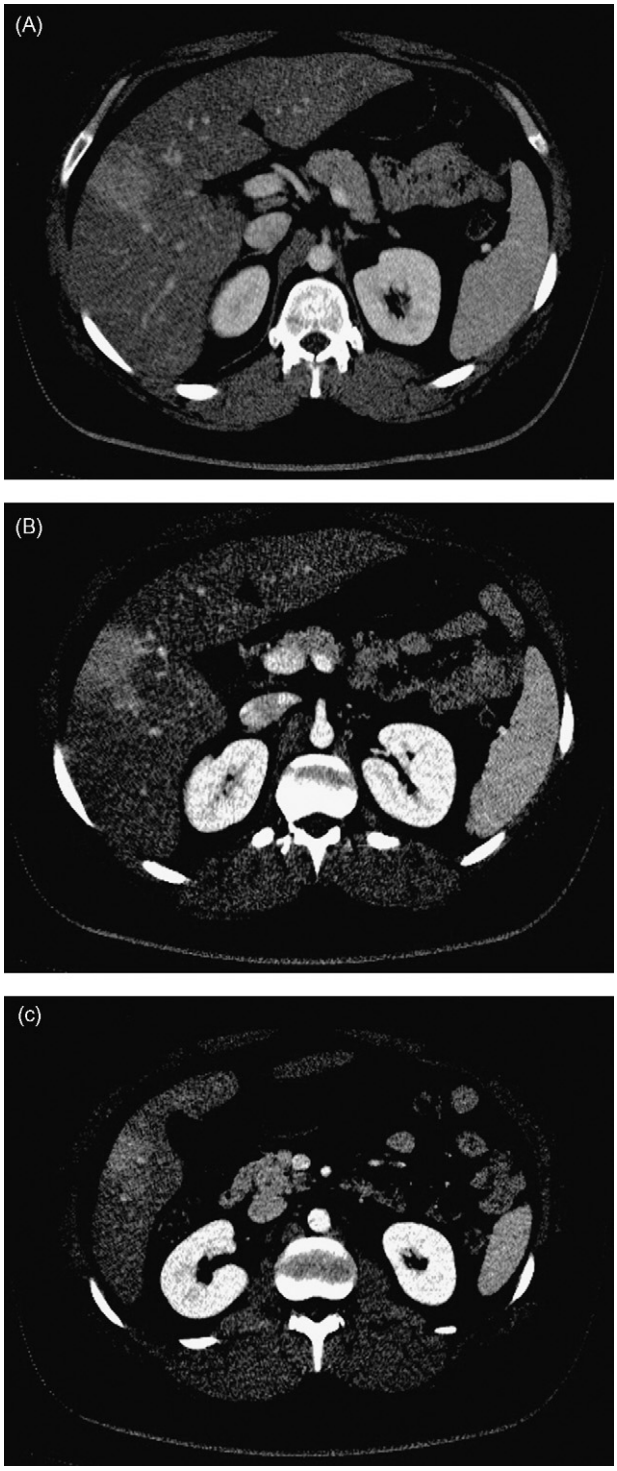

Fig. 1. Enhanced CT arterial phase liver window: (A) the celiac artery level, (B) the level of the superior mesenteric artery SMA, and (C) below the level of the SMA origin showing a region of blush within the right lobe.

3. Discussion

The most common cause of liver injury is blunt abdominal trauma, which is secondary to motor vehicle accidents in most instances. The incidence of hepatic vascular injuries after blunt trauma is much lower than that after penetrating injury and the incidence is still not established [1].

Boijsen et al. [3] suggest that in over half of the cases with the angiographic diagnosis of hepatic trauma, arterioportal communication is evident. Most post-traumatic arterioportal flow presumably reflects disruption of the adjacent hepatic artery and portal vein. However, it must be emphasized that in some cases the shunt may occur via transvasal or transsinusoidal routes, secondary to shock, or incident to traumatic occlusion of an arterial, portal, or hepatic venous branch [2].

Although Hepatic APFs can be asymptomatic, they can cause hemobilia, abdominal pain, and diarrhea, and, if they become large enough, they can be associated with complications of portal hypertension (variceal bleeding and ascites) and life-threatening heart failure [4].

Post-traumatic APF may take months or even years to become clinically manifest; presentation even 23 years after formation of an APF has been reported [5]. The low resistance and high capacity of the portal system can compensate for the APF-related increased flow without a significant increase in pressure. The latter depends on the shunt volume and sinusoidal resistance and, indirectly, on the capacity of the portal vascular bed, which may explain late onset of symptoms such as variceal bleeding and recurrent ascites [5].