Case report

Traumatic bruising of the hepatoduodenal ligament can conceal a catastrophic injury to the hepatic artery

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SUMMARY

We present the case of a 22-year-old man transferred to the regional major trauma centre following a fall of ~15 m. He remained consistently haemodynamically stable for over 10 hours of observation until he deteriorated suddenly with major haemorrhagic shock requiring immediate trauma laparotomy. At laparotomy, 2 L of blood was drained from the abdomen but no source of active bleeding identified. 30 minutes after closure of the abdomen, 500 mL of fresh blood was noted in the drain so he was returned to the theatre where the bleeding source was found to be—after manual compression of a mildly bruised hepatoduodenal ligament—the proper hepatic artery (PHA). This case describes an unusual finding at relaparotomy and shows that even when there is no active bleeding from abdominal organs or classified vessels, it is possible to have isolated injury to PHA.

BACKGROUND

Isolated injury to 3 cm long proper hepatic artery (PHA) following blunt trauma is an infrequent finding in the clinical practice of acute care surgeons.^{1,2} Therefore, it is important to note that blunt injury to PHA has a distinct pattern, which is based on anatomical and histological features of the liver arteries. The distal portion of the artery that ramifies into right and left (and middle) hepatic arteries is more likely to be damaged at the moment of impact of a localised external force as the muscular layer of the common hepatic artery and proximal PHA is more profound and, therefore, stronger.³ Although most patients with injury to the main hepatic artery are brought into the hospital with signs and symptoms of severe exsanguination and haemorrhagic shock, alternative initial clinical presentations are possible. Examples include thrombosis and false aneurysm of the artery.^{4,5} Not surprisingly, reports of damage caused by PHA injury are sporadic. We aim to disseminate three messages. First, to the operating surgeon: be vigilant and more aggressive having in hand and palpating bruised but not bleeding hepatoduodenal ligament. Second, to the trauma radiologist and trauma medical director: precision radiological assessment and closely monitored non-invasive cardiac output monitoring in the trauma ward are of paramount importance. Third, to provide a case-based mini-handbook to all.

CASE PRESENTATION

A 22-year-old male patient was transferred to the regional major trauma centre from a district trauma hospital having sustained a fall of 15 meters. At the local hospital, he had been stable, in terms of A, B, C (airway, breathing, circulation). A trauma CT scan was performed. The trauma radiologist reported a right-sided haemopneumothorax associated with displaced fractures of the proximal portions of five lower ribs. A 6×2 cm liver laceration involving branches of the right hepatic vein of segment 7 with perihepatic haemoperitoneum was reported. An intercostal chest drain was placed to the right thorax cavity.

On arrival to the major trauma centre, he remained alert at Glasgow Coma Score 15 and physiologically stable with blood pressure 122/61 mm Hg and heart rate 97 beats/min. Examination of the abdomen was unremarkable. Blood tests showed haemoglobin of 124 g/L 8 hours after the fall. A review from critical care was undertaken, and a decision was made to observe the patient on the major trauma ward as clinical observations and blood tests did not show cause for concern.

He remained on the major trauma ward undergoing regular observation, where all recordings remained unchanged. Suddenly, 5 hours after admission to the trauma ward (13 hours from the traumatic event), he deteriorated. Blood pressure and oxygen saturation were not recordable due to severe hypovolemic shock and cardiac periarrest. The abdomen became acutely distended. An emergency call was put out, and the major haemorrhage protocol triggered. The anaesthetic team promptly intubated the patient. He was taken for an immediate trauma laparotomy to control the intra-abdominal bleeding.

DIFFERENTIAL DIAGNOSIS

The first consideration for the cause of the bleeding was continuous haemorrhage from hepatic veins of segment 7 or intrahepatic portion of the right hepatic vein. Second, injury to juxta-hepatic veins. Third, bleeding from mesenteric vessels. Fourth, exsanguination from any parenchymal organ or classified vessel of the abdominal cavity. In retrospect, bleeding-specific differentials should include the right, medial, left, proper, common hepatic arteries along with the injured coeliac axis.

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Figure 1 Contrast-enhanced liver CT scan, performed 10 hours after relaparotomy, and blood serum alanine aminotransferase (ALT) levels between postoperative day 1 and 14: (A) right and left branches of the portal vein (long arrow on the right side of the image), demarcation line at the midline of the liver (short arrow) and hypoperfused (ischaemic) left hemiliver; (B) changes in elevated ALT levels in line chart; dashed line is the upper limit of the normal ALT level (55 units/L). Alkaline phosphatase, prothrombin time and the international normalised ratio changes were unremarkable.

TREATMENT

At trauma laparotomy, 2 litres of blood and clots was evacuated from the upper abdomen, which was subsequently packed for 30 minutes allowing haemostatic resuscitation to continue. On inspection, after removal of the packs and blood clots, no active source of bleeding was found in the abdominal cavity. Retroperitoneal regions and mesentery were intact. Grade 1 liver laceration to segment 4b, mildly bruised hepatoduodenal ligament and a grade 1 contusion to the pancreatic head were noted, neither of which were bleeding. Hepatogastric and gastroduodenal ligaments were opened. Pancreas, left gastric and common hepatic vessels were inspected and palpated. Drains were placed, and the abdomen closed.

Thirty minutes after closure of the abdominal wall, 500 mL of fresh blood was suddenly seen in the subhepatic drain. The patient was, therefore, immediately returned to the operating table for a relaparotomy. Two hundred millilitres of blood were evacuated, and the upper part of the abdominal cavity was again packed. The original midline incision was extended transversely to the right to create a reverse L letter type incision. During manual compression of the vessels of hepatoduodenal ligament, sudden arterial bleeding began. It was found originating from a 3 mm sized defect of the medial wall of the PHA within the middle portion of the hepatoduodenal ligament. This

Figure 2 Contrast-enhanced trauma CT scan on admission: (A) ill-defined hypodense area within segment 7 of the liver, extending to the right hepatic vein—a liver laceration; (B) a terminal portion of the right hepatic artery and its ramification into anterior and posterior sectional arteries; (C) the structure arrowed is the right hepatic artery; (D) the arrow targets the proper hepatic artery.

was repaired with one 4/0 prolene suture. No further surgical attempts made to identify the precise injury anatomy and its relationship with lobar liver arteries. On inspection, the colour of the right and left lobes of the liver remained the same and unchanged during surgery.

OUTCOME AND FOLLOW-UP

A CT scan was undertaken to assess the blood supply of the liver on postoperative day 1 (figure 1). It showed hypoperfusion of the left hemiliver. Portal vein and its branches were patent. Liver function tests had become deranged with alanine aminotransferase of 4086 units/L. The patient developed multiple organ dysfunction including acute kidney injury. Over the next 2 weeks spent in the critical care unit, the biochemical levels of blood serum returned to normal. Duplex ultrasonography of liver vessels was carried out. It demonstrated that PHA was patent with flow velocity measuring 32 cm/s. Right hepatic artery was identified well. No flow was appreciated in the left hepatic artery. Heterogeneous changes within the left lobe of liver with partial

Table 1 Summary of data from published literature on traumatic injury to the hepatic arteries in the last 30 years

Characteristics	CHA, n=2	PHA, n=1	RHA, n=13	LHA, n=5	RHA and LHA, n=2	Not-specified, n=92
Cause-of-injury	Shooting	Fall>2 m	RTA mainly Violence	RTA, mainly Fall>2 m	Not-specified	Not-specified
Type-of-injury	Penetrating	Blunt	Blunt, chiefly Penetrating	Blunt	Not-specified	Not-specified
Portal vein injury	50%	None	None	20%	50%	None
Liver injury: grade 4 or 5	50%	100%	20%	20%	Not-specified	Not-specified
Clinical syndrome	Haemorrhage	Haemorrhage	80%: haemorrhage 20%: pseudoaneurysm	Haemorrhage	Haemorrhage	Haemorrhage Pseudoaneurysm
Management	50%: ligation 50%: angioembolisation	Angioembolisation	70%: angioembolisation 30%: ligation, repair, packing	Angioembolisation Ligation	Angioembolisation	Angioembolisation
Sequelae	None Hepatic failure if PV injured	None	70%: necrosis/abscess of right hemiliver with/without necrosis of the gallbladder	20%: liver necrosis/abscess	Liver necrosis/abscess Gallbladder necrosis	25%: necrosis/abscess of the liver with/without necrosis of the gallbladder
Survival	50%	100%	100%	80%	100%	100%

CHA, common hepatic artery; PHA, proper hepatic artery; RHA, right hepatic artery; LHA, left hepatic artery; n, number of patients.

atrophy secondary to changes of recent infarction were reported. The patient remained clinically well and was discharged from the major trauma centre to continue his recovery at a rehabilitation centre. A CT scan performed 12 weeks after trauma showed signs of atrophy of left hemiliver. He is making an excellent recovery in all other respects and successfully continues his studies at university.

DISCUSSION

Following polytrauma, the liver is the most common organ injured.⁶ Uncontrollable haemorrhage following complex hepatic injury is the leading cause of mortality in these patients.⁷ The American Association for the Surgery of Trauma published the Organ Injury Scaling 2018 update where liver injuries were graded from level 1 through to 6.⁸ Injury to the hepatic arteries automatically puts the patient at a grade 3 and when that bleeding enters the peritoneal cavity such as in the reported case it becomes a grade 4.

The mechanisms of injury of hepatic arteries in blunt abdominal trauma can be categorised into three patterns—acceleration, deceleration and compression type.⁹ However, blunt abdominal trauma-related injuries often are the result of combined interactions of kinetic energies from all of these mechanisms. Acceleration injuries result from a force compressing the chest wall, resulting in the propulsion of the liver and its vessels. On the right hemiliver the triangular ligament, and the falciform ligament for the left hemiliver, act as stable points of fixation of the liver creating areas of high-force impact and subsequent laceration. Similarly in deceleration injuries, the liver continues to move and collides with the chest or posterior abdominal wall depending on the direction of the force. The hepatoduodenal ligament can also act as a fixed point during these injuries, and so the high-energy force is stressed through it and its structures resulting in shearing and occasionally evulsion of the hepatic arteries.

An increasing evidence pool has led to the use of non-operative management for blunt liver injuries and, when selection of patients is appropriate, is associated with a <10% need for delayed laparotomy.^{10–14} The decision to proceed with non-operative management and either embolisation or stenting of a potentially injured hepatic artery is dependent on both the patient condition and the report from the trauma CT scan. Initially in this case, it was reported that there was no visible source of active bleeding and no injuries to hepatic arteries were suspected. However, on a retrospective review of the images (figure 2), it is possible to suspect absence of arterial supply to the left hemiliver. Had this been noted at the time and with the patient initially being haemodynamically stable the next step would have been for the angiogram.

At initial trauma laparotomy, the hepatoduodenal ligament was found to be bruised but with no overt ongoing bleeding. Unlike the gastroduodenal and hepatogastric ligaments, it was not opened and vascular components of it—hepatic arteries and portal vein—were not inspected. There has been a previous report of bruising to the hepatoduodenal ligament found at laparotomy due to blunt abdominal trauma.¹⁵ Here, the ligament was not explored as no active bleeding could be found; this resulted in a missed hepatic artery injury that represented months later with complications of pseudoaneurysm formation distal to the origin of the gastroduodenal branch. In both cases, the bruised hepatoduodenal ligament was concealing a hepatic artery injury. It should be considered, therefore, when encountered by the trauma surgeon that this finding is a sign

Patient's perspective

I am a university student. After a night out on Christmas Eve, I suffered a fall from height. I do not recall anything of the incident but, piecing the events together, I was heard shouting for help in the bottom of a quarry by the owner of an adjacent property early on Christmas morning. An ambulance was called and, some time later, I was found and taken by wheelchair conscious, into the ambulance. I was treated in the ambulance for hypothermia before being transferred to General District Hospital A&E department. My injuries at the time were not considered to be life-threatening. I believe that I had a CT scan and was treated for a collapsed lung. I was then transferred to the Aintree Hospital Major Trauma Ward.

After some time in the ward in a condition assumed to be stable, I passed out and emergency procedures were initiated. I understand that during the next 4 hours surgeons performed two consecutive emergency operations to stem bleeding to my liver. One of these was to the main artery feeding the liver, which had been damaged by the fall. As a result of the time taken to reach the operating theatre, I suffered from oxygen starvation to vital organs including my brain and kidneys. After the operations, I was transferred to the Critical Care Unit of Aintree Hospital where I spent the next 3 weeks. Of these, 2 weeks were in an induced coma. I was then transferred back to the Major Trauma Ward until my discharge on 19 January.

Since my discharge I have had a further CT scan on my liver and am awaiting a consultation to understand the long-term implications of my injuries. Nevertheless, I have made significant progress in my general recovery since leaving hospital and recommenced my studies in mid-February.

Learning points

- Mortality attributed to traumatic injury of hepatic arteries is very high (56%); consideration of such vascular bleeding and prompt control saves lives.
- Radiological assessment of the hepatic arteries and arterial blood supply to the liver via protocolled trauma CT scan is the key selecting a most appropriate option of further treatment—emergency radiological angioembolisation or trauma laparotomy.
- A bruised hepatoduodenal ligament should be considered for surgical exploration as it can conceal substantiate injury to the hepatic artery; the alternative of it—selective angiography on the operating table—is possible in a well-equipped Trauma Centre.
- To control haemorrhage, simple ligation of the proper hepatic artery (PHA) is the correct management of this injury.
- PHA suture, especially when it is situated at the level of the ramification of this artery, can cause surgical occlusion or thrombosis of the left hepatic artery.

of significance and further exploration should be undertaken to identify the aetiology of the bruising, as the undiagnosed injury to the hepatic artery can be a cause of further sudden haemorrhage and, in combination with severe hypovolemic shock-related low portal flow, induce liver infarction or deteriorate it.

A review of the literature for cases or series of traumatic hepatic artery injuries in the last 30 years was performed.^{16–28}

The results are summarised in [table 1](#). Due to the variable blood supply to the liver and the ability to form multiple prompt collateral vessels, ligation or embolisation of hepatic arteries does not necessarily result in catastrophic hepatic damage. It has long been acknowledged that the liver can survive on portal blood flow alone which supplies 80% of the total hepatic flow.²⁹ The hepatic arteries are responsible for 50% of the oxygen delivery to the liver.³⁰ The 'standard' anatomical position of the common hepatic artery arising from celiac trunk leading to proper, left, and right hepatic arteries is found in only 55% of people.³¹ Angiographic studies have demonstrated the right hepatic artery arising from the superior mesenteric artery in 18% of the population, the left hepatic from the left gastric in 13% and the common hepatic from the superior mesenteric artery in 6%.³²

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