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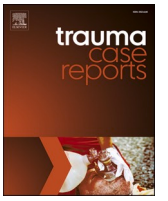

Case Report

Hepatic artery pseudoaneurysm rupture: A case report

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ABSTRACT

Hepatic artery pseudoaneurysms are an uncommon complication of blunt hepatic trauma typically presenting in a delayed fashion. A 40-year-old male presented to a trauma centre after a 6-metre fall from a construction site with multiple injuries including a grade IV liver laceration centred around the porta hepatis. This liver injury was managed non-operatively. On day sixteen of admission, he had a sudden cardiac arrest from haemorrhagic shock with a subsequent CT demonstrating a ruptured extrahepatic proper hepatic artery pseudoaneurysm. Despite laparotomy and vessel repair, he died from pulmonary complications of aspiration pneumonia and acute lung injury associated with massive transfusion. The literature demonstrates conflicting views regarding the utility of repeat CT to detect a pseudoaneurysm in asymptomatic, non-operatively managed patients with blunt hepatic trauma. In particular, the literature does not distinguish the utility of repeat routine CT for intrahepatic and extrahepatic hepatic artery pseudoaneurysm, the latter if which is rarer. Current guidelines recommend against it, but there are observational studies suggesting utility, particularly in high grade (≥IV) liver injury. In patients with a high-grade injury extending to the porta hepatis, repeat imaging should be considered to detect possible pseudoaneurysm.

Introduction

Hepatic artery pseudoaneurysm is caused by spontaneous or traumatic tear through all layers of the hepatic artery with blood flow into space outside the artery contained by surrounding tissue [1]. This is associated with a high risk of rupture and catastrophic haemorrhage. The most common cause is iatrogenic after pancreaticobiliary surgery, however, can also be secondary to infections and trauma (blunt and penetrating) [2]. Blunt hepatic trauma is an uncommon cause, affecting approximately 1–4 % of cases, with extrahepatic pseudoaneurysm being rare, and mostly presenting in a delayed fashion [3]. The following case demonstrates spontaneous delayed extrahepatic pseudoaneurysm rupture from the proper hepatic artery (PHa) as a result of blunt hepatic trauma.

Case presentation

A 40-year-old male presented to a Level 1 trauma centre following a 6-m construction site fall. He was hypoxic and hypotensive prior to arrival, requiring intubation and bilateral finger thoracostomies. Two units fresh frozen plasma (FFP) and two units packed red

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blood cells (PRBC) were administered. In the resuscitation room, a thorough trauma assessment was performed, bilateral intercostal catheters inserted followed by trauma pan CT. He was found to have significant traumatic injuries including multiple facial bone fractures, left ribs 1–4 fractures with flail segments, right pneumothorax, left haemopneumothorax, left lung contusions and pulmonary laceration, American Association for the Surgery of Trauma (AAST) grade I left renal laceration, bilateral forearm fractures, right distal femur fracture, and extensive pelvic fractures with associated left iliopsoas haematoma. He also had an AAST IV liver laceration with extension to the porta hepatis and compression of the portal structures by haematoma but with normal attenuation of the portal vein and hepatic artery (Fig. 1).

The next day he underwent thoracoscopy and surgical fixation of his left rib fractures as well as open reduction internal fixation (ORIF) of his right femur. On day four of admission, he underwent a pelvic ring stabilisation and ORIF of bilateral forearm fractures. He was extubated day six of admission and made steady recovery. On day eight, he had lower limb dopplers demonstrating a 1 cm non-occlusive thrombus in his right posterior tibial vein despite being on prophylactic 40 mg nocte enoxaparin. Therapeutic enoxaparin at

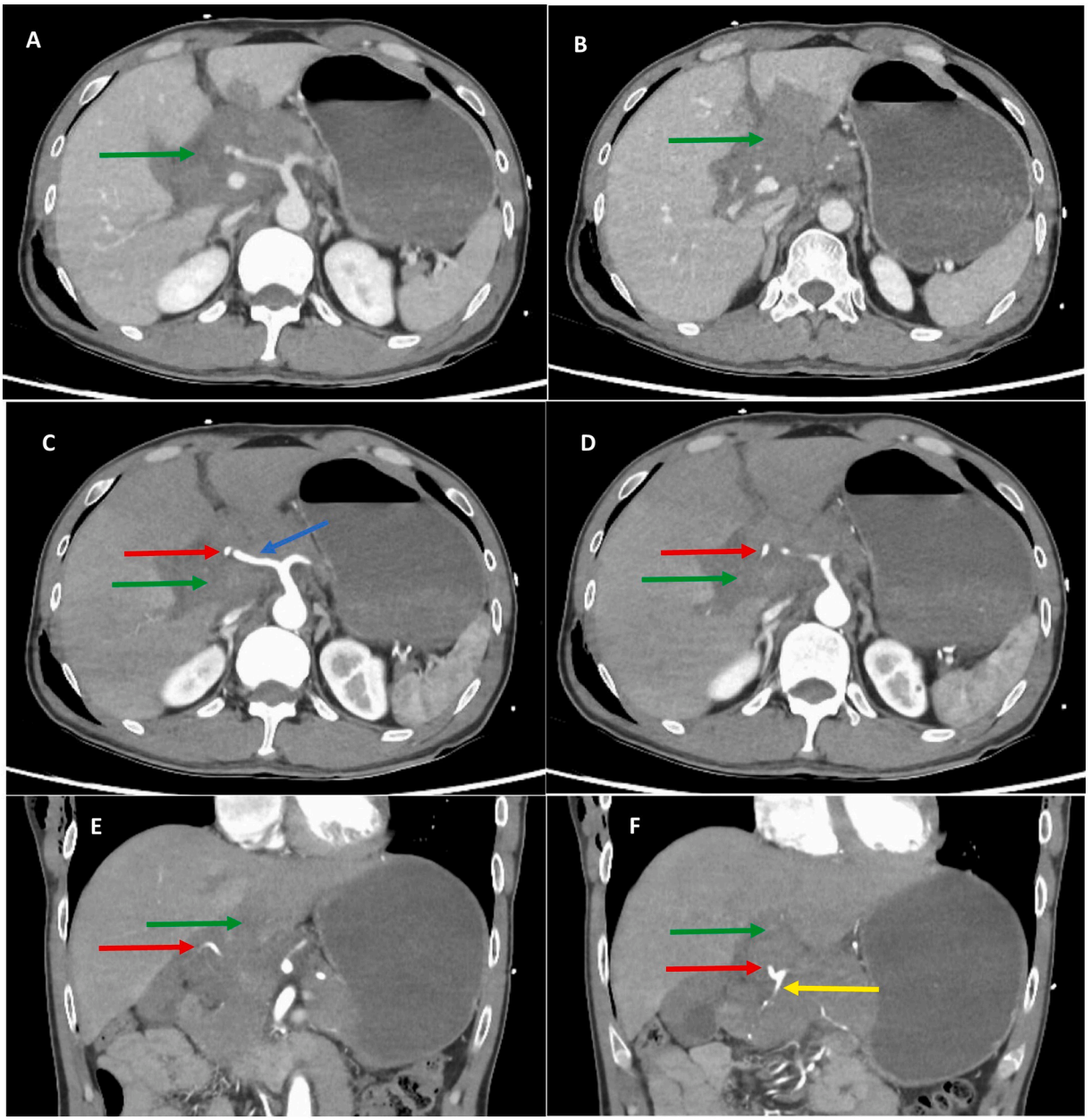

Fig. 1. 1A-1F. Axial (1A-1D) and coronal (1E-1F) slices of initial portal-venous (1A/1B) and arterial (1C-1F) phase CT-abdomen/pelvis demonstrating AAST IV liver laceration (green arrow) centred around the porta hepatis. The normally opacifying common (blue arrow) and proper hepatic artery (red arrow) without pseudoaneurysm can be seen in 1C-1F and the gastroduodenal artery in 1F (yellow arrow).

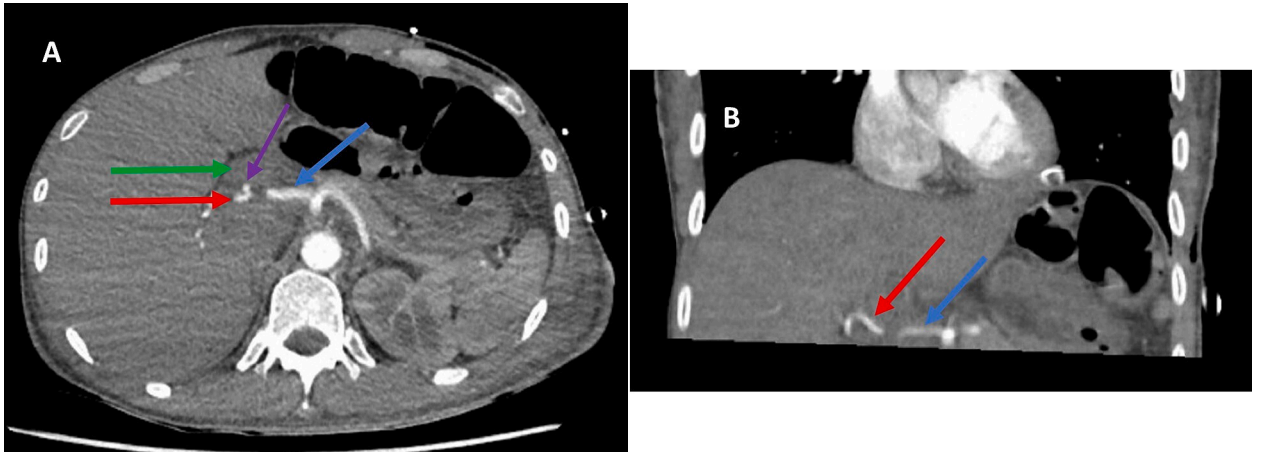

Fig. 2. 2A-2B. Axial (2A) and coronal (2B) slices of CT-pulmonary angiogram showing the partially imaged upper abdomen demonstrating the healing liver laceration (green arrow), common (blue arrow) and proper (red arrow) hepatic artery. Early HPa pseudoaneurysm is seen in 2A (purple arrow).

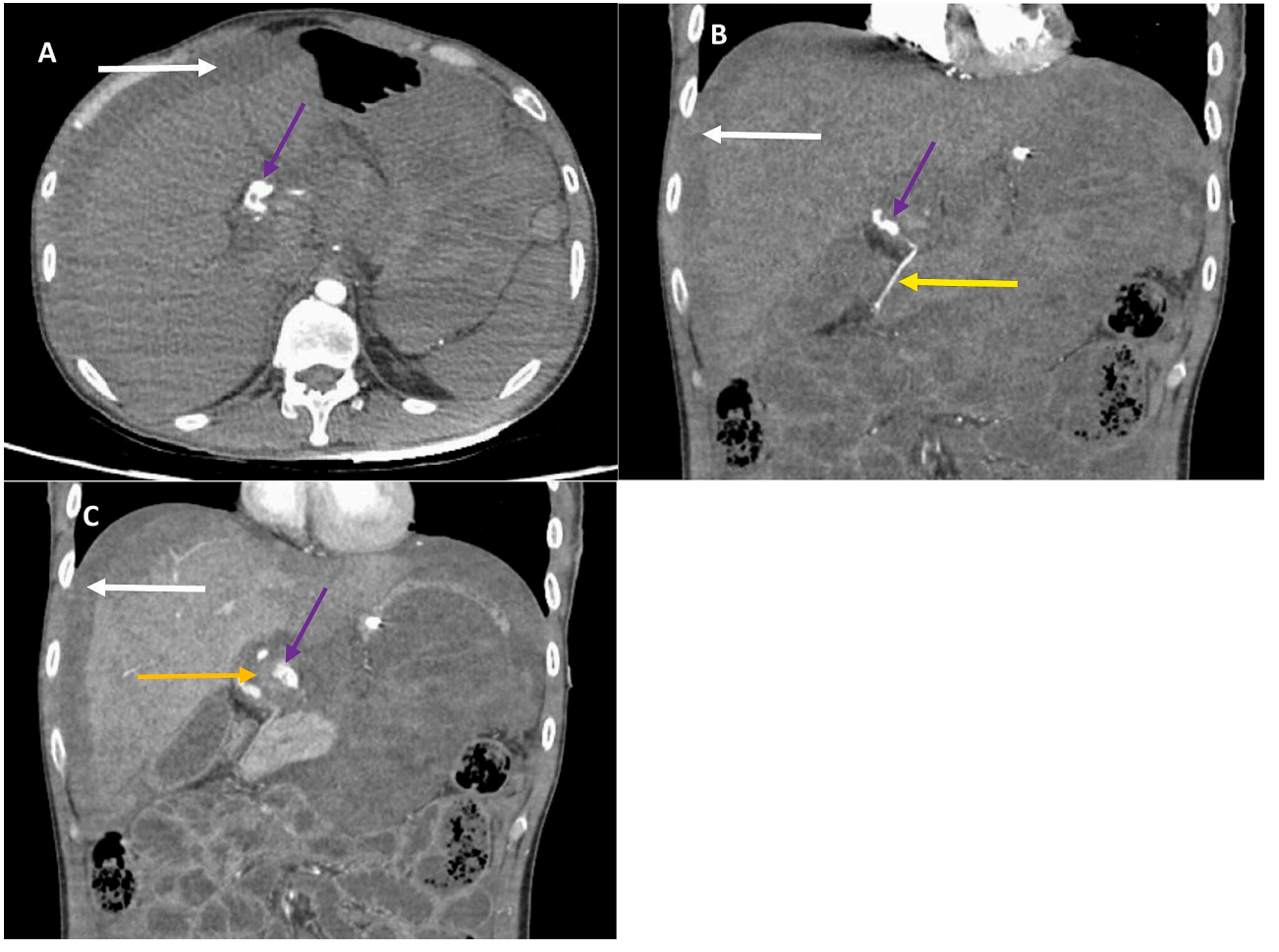

Fig. 3. 3A-3C. Arterial (3A/3B) and portal-venous (3C) phase CT-abdomen/pelvis demonstrating haemoperitoneum (white arrow), ruptured HPa pseudoaneurysm (purple arrow), and haematoma surrounding the portal triad (orange arrow). The gastroduodenal artery is seen in 3B (yellow arrow).

1 mg/kg BD was commenced. The patient developed intermittent oxygen desaturations on day 10, so underwent a CT pulmonary angiogram (CT-PA). This did not demonstrate any evidence of pulmonary embolism (PE) (Fig. 2) on day ten. The CT report made no mention of his partially imaged liver laceration. Haemoglobin (Hb) remained stable between 90 and 100 g/L and he was planned for discharge to rehabilitation.

On day sixteen of admission the patient had an emergency response called for hypotension and sudden unresponsiveness whilst complaining of lower chest pain immediately prior. He was found to be hypotensive to 60s systolic, bradycardic in the 30s, normothermic, with respiratory rate 18 breaths/min and saturating 100 % on 15 L non-rebreather mask. A bolus of 2 L crystalloid fluid was commenced with minimal improvement in blood pressure. His physical examination revealed a slightly distended abdomen and unresponsiveness with GCS 4. He underwent rapid sequence induction and intubation to protect his airway during which vomitus was seen causing aspiration. Subsequently, he became more hypotensive and suffered a pulseless electrical activity arrest requiring 5 cycles of CPR and total 3 mg adrenaline for return of spontaneous circulation. He remained hypotensive and was commenced on a peripheral adrenaline infusion before femoral access was gained. His ECG did not demonstrate ischaemia and his mobile CXR revealed no recurrent haemopneumothorax. His blood gas showed a profound mixed metabolic respiratory acidosis with a Hb of 50. Massive transfusion protocol was commenced, and anticoagulation reversed with protamine sulphate. His haemodynamic parameters improved and so CT was performed to exclude PE and simultaneously scan his brain and abdomen to explain his reduced GCS and abdominal distension.

His CT did not reveal a PE nor a primary cerebral issue, however, demonstrated active contrast extravasation from the PHa likely from a pseudoaneurysm rupture with large volume haemoperitoneum (Fig. 3). The patient subsequently underwent an urgent laparotomy and control of haemorrhage.

Management and outcome

Operatively, 2.5 L of blood was evacuated from the abdominal cavity. After initial packing, the lesser sac was entered with haematoma around the portal structures found and evacuated. The bleeding point was identified in the proximal posterolateral aspect of the PHa. This injury was repaired with prolene sutures. The abdomen was closed, and the patient taken to ICU intubated having received a total 16 units PRBC, 12 units FFP, 6 units cryoprecipitate, and 2 units pooled platelets. Whilst his Hb remained stable, he developed worsening respiratory failure and was unable to be oxygenated despite 100 % FiO2, pulmonary vasodilators and proning manoeuvres. The patient died on day seventeen of admission from respiratory failure likely associated with aspiration and transfusion related acute lung injury.

Discussion

This case illustrates the catastrophic impacts of a PHa pseudoaneurysm spontaneous rupture. It is important to note that up until the moments leading to collapse, this patient was asymptomatic.

There remains uncertainty regarding the role of repeat imaging for patients after blunt hepatic trauma and when this should occur. Current guidelines from the World Society of Emergency Surgery and Eastern Association for the Surgery of Trauma do not recommend routine follow up CT to detect complications associated with blunt hepatic trauma, unless there are symptoms suggestive of them such

Table 1
Summary of studies assessing need for routine repeat CT after blunt liver injury to assess for hepatic pseudoaneurysm as a complication.

Study name	Year	Study type	Study cohort	Findings of note	Recommendation
Status of nonoperative management of blunt hepatic injuries [6]	1996	Case-control	404	Of the 404 non-operatively managed liver injuries, 198 underwent repeat CT – no complicated detected	No routine imaging
Routine follow-up imaging is unnecessary in the management of blunt hepatic injury [7]	2005	Case-control	530	At 1-week routine follow-up scans, only two of the 530 initially non-operatively managed patients required angiography for vascular blush	No routine imaging
Non-operative management of blunt hepatic trauma: does angioembolization have a major impact? [8]	2014	Retrospective cohort-study	396	At routine 24–48 h follow-up CT, only three patients had pseudoaneurysm	No routine imaging for grade I-III liver injury but consider for grades ≥IV
Repeat imaging in blunt hepatic injuries can wait for clinical change [9]	2021	Case-control	122	2/50 patients who had routine follow-up CT underwent intervention compared to 10/72 patients who had repeat CT due to clinical change.	No routine imaging
Hepatic pseudoaneurysm incidence after liver trauma [10]	2020	Case-control	634	Pseudoaneurysm found in 0.06 % of AAST III injury, 4.3 % in AAST IV, and 3.8 % in AAST V	Repeat routine imaging at day 5
Hepatic pseudoaneurysm after traumatic liver injury; is CT follow-up warranted? [3]	2014	Case-control	259	4 % incidence of hepatic pseudoaneurysm at 5 days. 75 % of these patients were asymptomatic	Repeat routine imaging. No timing specified

as abdominal pain, fevers, jaundice, or Hb drop [4,5].

This is supported by evidence from retrospective observational studies such as from Pachter et al. where 198 of 404 non-operatively managed blunt liver injuries underwent repeat CT at varying periods of time and zero complications were found [6]. Similarly, in a 530-patient study by Cox et al., where CT was repeated routinely within 1 week of blunt hepatic injury, only two patients underwent angiography due to presence of vascular blush [7]. As such, they did not recommend routine repeat imaging. In a 2014 retrospective cohort study of 396-patients with blunt hepatic trauma, patients were routinely imaged at 24–48 h to assess for pseudoaneurysm as per local protocol and only 3 demonstrated this, all of whom had AAST IV laceration [8]. As such the study did not routinely recommend follow-up CT for AAST grades I-III but suggested consideration in grades ≥IV. A more recent 122-patient retrospective case control study found that 2/50 of the patients who had routine repeat CT compared to 10/72 who had CT due to clinical change underwent intervention based on the scans. The development of complications on repeat CT was also not significantly different and so it was concluded that repeat imaging should wait for clinical change rather than protocol [9].

From a 2020, 634-patient study of hepatic trauma, the incidence of asymptomatic pseudoaneurysm was 0.06 % in AAST III, 4.3 % in AAST IV, and 3.8 % in AAST V injury [10]. Hence, they suggested routine repeat imaging at day 5 for all liver injury AAST ≥IV. Similarly, Østerballe et al. found the incidence of hepatic pseudoaneurysm to be 4 % at 5 days with 75 % of these patients being asymptomatic and thus recommended routine CT to detect this complication [3].

These studies (summarised in Table 1), however, do not specify location of liver injury. Additionally, there is no differentiation between intra and extrahepatic location of pseudoaneurysm. In the case presented, the injury was centred around the porta hepatis, and no pseudoaneurysm was initially seen. However, on retrospective review, there was a subtle PHa anomaly possibly suggestive of pseudoaneurysm that was not reported on the day ten CT-PA (Fig. 2). A targeted arterial phase abdominal imaging may have defined the anomaly more precisely. AAST grading is typically useful in determining management along with patient clinical condition, however, its flaw is the lack of consideration for damage to extrahepatic portal structures. This patient additionally had an extrahepatic arterial injury, and with a haematoma in the free edge of the lesser omentum on initial CT, should have promoted consideration of this, despite no contrast extravasation at the time.

Conclusion

In this pattern of severe (AAST ≥IV) liver injury centred around the porta hepatis, routine serial repeat imaging may be of value in detecting life threatening extrahepatic pseudoaneurysm.

Declaration of competing interest

The authors declare no conflicts of interest or bias, including financial and personal, in the writing of this article.

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