

Traumatic Portacaval Shunt: A Case Report and Literature Review

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ABSTRACT

Computed tomography (CT) evaluation of the acute polytrauma patient has become well established as a mainstay of ER triage in hemodynamically stable patients. The radiologist plays a pivotal role in directing management by identifying and appropriately categorizing the severity of a patient's injuries. High-grade liver injuries have undergone an increasing trend of nonoperative management over the last several decades, with concurrent decrease in mortality. However, we present a case of a patient with a grade V liver laceration, in whom a rare portacaval shunt was also present. In the setting of this rare injury, the radiologist will likely be the first person to recognize and categorize a severe complication, which may indicate the need for a fundamental change in patient management.

CASE REPORT

CASE REPORT

A 12 year old boy sustained a blunt impact to his right flank during an ATV accident. The impact resulted in fractures of his right 4-10 ribs, with a segmental and displaced fracture of his right lateral 8th rib, which was rotated approximately 180 degrees adjacent to the right lobe of his liver. He sustained a grade V liver laceration, (American Association for the Surgery of Trauma (AAST) liver injury scale [1]), associated with a large portacaval shunt from the right portal vein to the IVC (Fig. 1 a-d). There was no head trauma or loss of consciousness.

Following the trend of increasing conservative management of hemodynamically stable liver injuries, the patient was managed nonoperatively in the pediatric intensive care unit. For the first two days of admission, the patient was alert and conversive. However, on day 3, his first head CT (Fig. 2) was ordered for acute delirium, showing small areas of suspected minimal cerebral edema. Over the next 2 days, he became progressively obtunded and developed pupillary

changes. Lactulose and hypertonic saline were initiated for elevated ammonia levels up to 928 µg/dl (normal: 28.2-80.4 µg/dl), and for cerebral edema. His ammonia levels slightly declined on this aggressive medical regimen on admission days 5 and 6. However, by day 7, continuous hemodialysis was initiated for intractable and worsening hyperammonemia. He also developed a severe coagulopathy during this period. A repeat head CT (Fig. 3), late on day 7, demonstrated massive cerebral edema and early herniation. A nuclear medicine cerebral blood flow study (Fig. 4) was obtained on day 8, demonstrating absence of cerebral Tc 99m-ECD metabolism, consistent with the clinical diagnosis of brain death.

DISCUSSION

Etiology and Demographics

Approximately 6% of blunt abdominal trauma admissions at our institution sustain a liver laceration, of which approximately 32% are high-grade (III-V) injuries [2].

Mortality from blunt hepatic trauma has decreased over the last several decades, with the emphasis on noninvasive techniques and conservative management increasing over this time period. However, liver-related morbidity and mortality remain highest in patients with higher grade hepatic injuries. The most common complications encountered following high grade liver trauma are biliary-tract complications, bleeding, abdominal compartment syndrome, liver abscess, and liver necrosis [2].

While the overall incidence of traumatic intrahepatic shunts is unclear, the majority of these shunts are small arterioportal shunts, often manifesting as a transient hepatic attenuation difference [3]. These shunts are most commonly the result of penetrating trauma or interventional procedures such as liver biopsy, and they typically resolve over the course of several months [4]. We describe a rare complication of a grade V liver laceration: a traumatic portacaval shunt.

Clinical and Imaging Findings

The initial trauma CT showed a large grade V liver laceration, and an abnormal connection between the right portal vein and the IVC. The patient had not sustained head trauma. The causes of the patient's subsequent rapid and progressive mental decline were cerebral edema and herniation secondary to acute hepatic encephalopathy, which resulted from rapidly increasing ammonia levels. While patients with chronic liver disease and elevated ammonia levels can suffer from hepatic encephalopathy, they generally do not incur intracranial hypertension [5]. However, patients with acute liver failure are susceptible to the development of cerebral edema, which develops in 80% of acute liver failure patients, and is fatal in 25% [5]. The pathophysiology of this process is complex, and likely involves a combination of factors resulting both in edema of the cerebral astrocytes and increased cerebral blood flow, resulting in an overall increased brain volume.

Acute liver failure is a rare complication of liver trauma, with liver necrosis occurring as part of multi-organ failure in only 1/230 patients with high grade liver injuries in a multicenter study [2]. However, in our patient, the pathophysiology was more complex. To our knowledge, there has been only one other case reported in the literature of a traumatic portosystemic fistula, which occurred in a 23 year old male who also sustained a grade V liver laceration following a motor vehicle collision. The fistula was successfully managed conservatively in that patient, although the patient did suffer symptoms of hepatic encephalopathy necessitating supportive measures and lactulose therapy. He did not, however, progress to acute liver failure or other cerebral complications. The fistula was followed angiographically over 10 months, and decreased in size over that interval [6].

The effect of therapeutic portosystemic shunting on hepatic encephalopathy has been well described in the literature, with worsening hepatic encephalopathy being a well-described complication of therapeutic TIPS procedures. New or worsened hepatic encephalopathy occurs in 5-35% of TIPS patients, becoming refractory to medical therapy in 3-7% [7].

Treatment and Prognosis

In this patient, a combination of factors was present. His injury resulted in a large, traumatic portosystemic shunt, equivalent to acutely acquiring a high volume TIPS. He also devascularized the right lobe of his liver, resulting in right lobe of liver necrosis and resultant liver dysfunction. The overall effect was massive, progressive, and refractory hyperammonemia. While the trend for conservative management of liver injuries has saved lives, and the only other patient in the literature with a traumatic portosystemic shunt was successfully managed with conservative therapy, the combination of large shunt and right lobe of liver necrosis in this patient proved fatal. Raising awareness of the severity of this injury, along with appropriate reporting of the degree of liver injury and shunting, should be emphasized in radiology reporting, to allow the clinician to appropriately direct management. Early and aggressive management of hyperammonemia should also be emphasized as a mainstay of treatment.

Differential Diagnoses

Differential considerations, in the appropriate clinical setting, may include a Park Type 1 congenital portosystemic shunt (which connects the right portal vein directly to the IVC), or an iatrogenic shunt, such as a transjugular intrahepatic portosystemic shunt - TIPS. However, the findings of adjacent chest wall trauma, a large liver laceration, and obvious acute devascularization of the right lobe of the liver preclude the inclusion of a congenital portosystemic shunt in the differential diagnosis of this patient. In addition, a congenital shunt of this size would have produced symptoms in infancy, and would have necessitated intervention if it did not undergo spontaneous involution [8]. There is no stent to indicate an iatrogenic TIPS procedure in this case.

TEACHING POINT

It is important to recognize the rare occurrence of traumatic portosystemic shunting in the setting of high grade liver injuries, which may result in additive complications, including acute liver failure, acute hepatic encephalopathy, bleeding diathesis and sometimes death. Recognizing this entity as a separate subgroup of high grade liver trauma may allow the radiologist to raise awareness of the potential need for more aggressive management.

REFERENCES

1. Moore EE, Cogbill TH, Jurkovich GJ, Shackford SR, Malangoni MA, Champion HR. Organ Injury Scaling: Spleen and Liver (1994 Revision). *J Trauma* 1995; 38: 323-4. PMID: 7897707
2. Kozar RA, Moore JB, Niles SE, Holcomb JB, Moore EE, Cothren CC, Hartwell E, Moore FA. Complications of Nonoperative Management of High-Grade Blunt Hepatic Injuries. *J Trauma* 2005; 59: 1066-1071. PMID: 16385280

- Ahn JH, Yu JS, Hwang SH, Chung JJ, Kim JH, Kim KW. Nontumorous arteriportal shunts in the liver: CT and MRI findings considering mechanisms and fate. *Eur Radiol* 2010; 20: 385-394. PMID: 19657644
- Guzman EA, McCahill LE, Rogers FB. Arteriportal fistulas: introduction of a novel classification with therapeutic implications. *J Gastrointest Surg* 2006; 10: 543-550. PMID: 16627220
- Bosoi CR, Rose CF. Brain edema in acute liver failure and chronic liver disease: Similarities and differences. *Neurochem Int.* 2013 Mar; 62(4): 446-57. PMID 23376027
- Oppermann TE, Corcos AC, Jones LM, Barrette RR, Varcelotti JR. Traumatic Intrahepatic Portosystemic Venous Shunt: A Rare Complication of Grade V Liver Laceration. *J Trauma* 2007; 63: 1230-1233. PMID: 18212643
- Madoff DC, Wallace MJ, Ahrar K, Saxon RR. TIPS-related Hepatic Encephalopathy: Management Options with Novel Endovascular Techniques. *Radiographics* 2004; 24: 21-36. PMID: 14730033
- Gallego C, Miralles M, Marin C, Muyor, P, Gonzalez G, Garcia-Hidalgo, E. Congenital Hepatic Shunts. *Radiographics* 2004; 24: 755-772. PMID 15143226

FIGURES

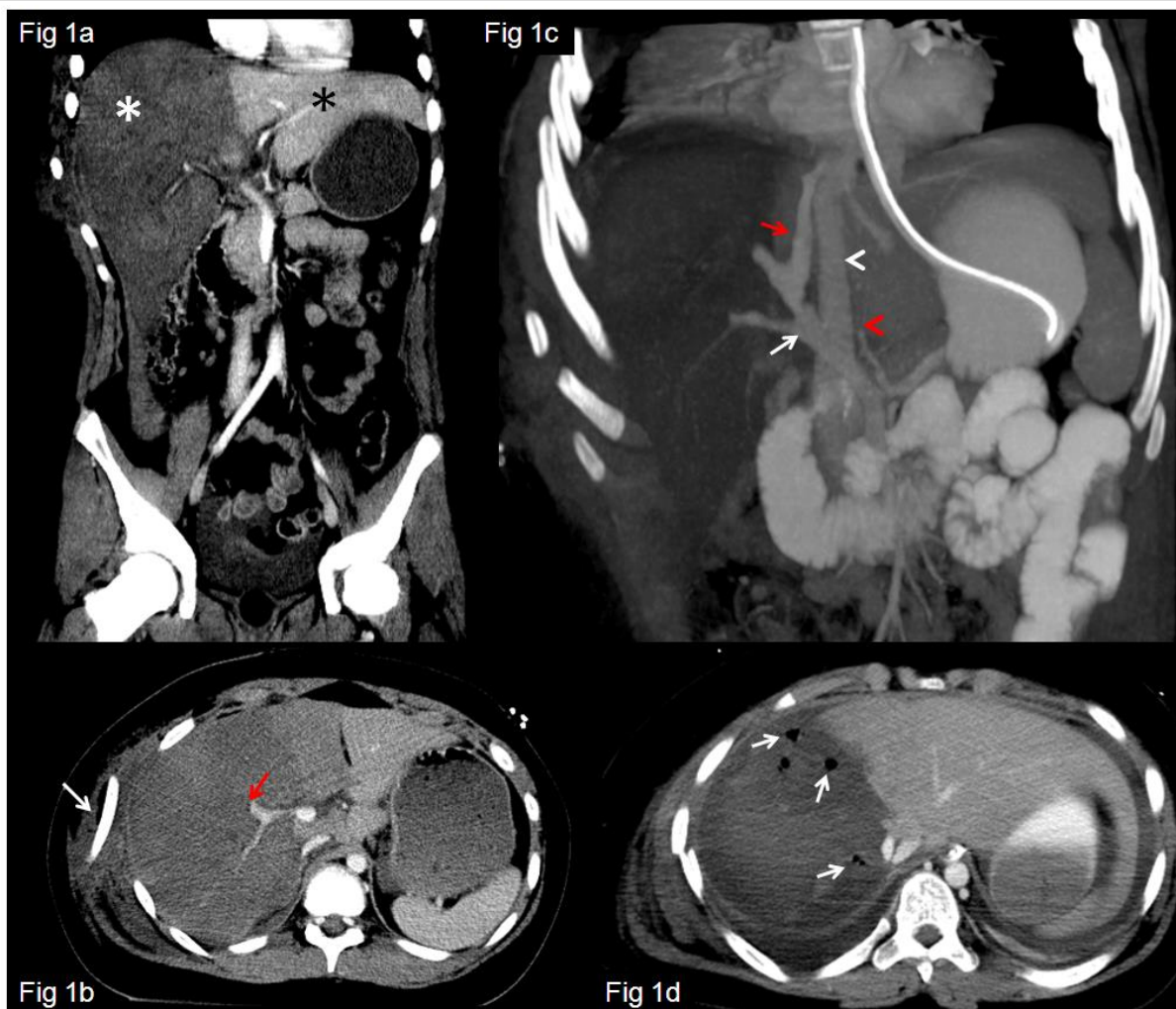


Figure 1: 12 year old male with traumatic portacaval fistula. FINDINGS: Coronal CT image through the liver from the patient's initial trauma CT. Compare the normally perfused and enhancing left lobe of the liver (black asterisk) with the abnormal, devascularized and hypo-enhancing right lobe of the liver (white asterisk). Fig 1b: Axial CT image in liver window from the initial trauma CT shows a displaced right 8th rib fracture, which is rotated approximately 180 and displaced laterally (white arrow). There is abrupt occlusion of central branches of the right portal vein (red arrow). TECHNIQUE: Siemens Sensation 40, 5mm slice thickness, 150cc Omnipaque 300, kVp 120, mA 295, coronal and axial plane. Fig 1c. FINDINGS: 2 day follow up CT. Coronal CT image in portal venous phase, through the level of the liver, where an abnormal fistulous connection (red arrow) between the portal vein (white arrow) and suprahepatic IVC (white arrowhead) is well seen, with occlusion of the right portal vein branches distal to the shunt. Abrupt occlusion of the right hepatic artery (red arrowhead) is also evident. Fig 1d: Foci of gas are now identified within the necrotic right lobe of the liver (white arrows). TECHNIQUE: Siemens Sensation 64, multiphase liver CT, kVp 120, mA 345, 5mm slice thickness, coronal and axial plane. Precise contrast type/amount not available.

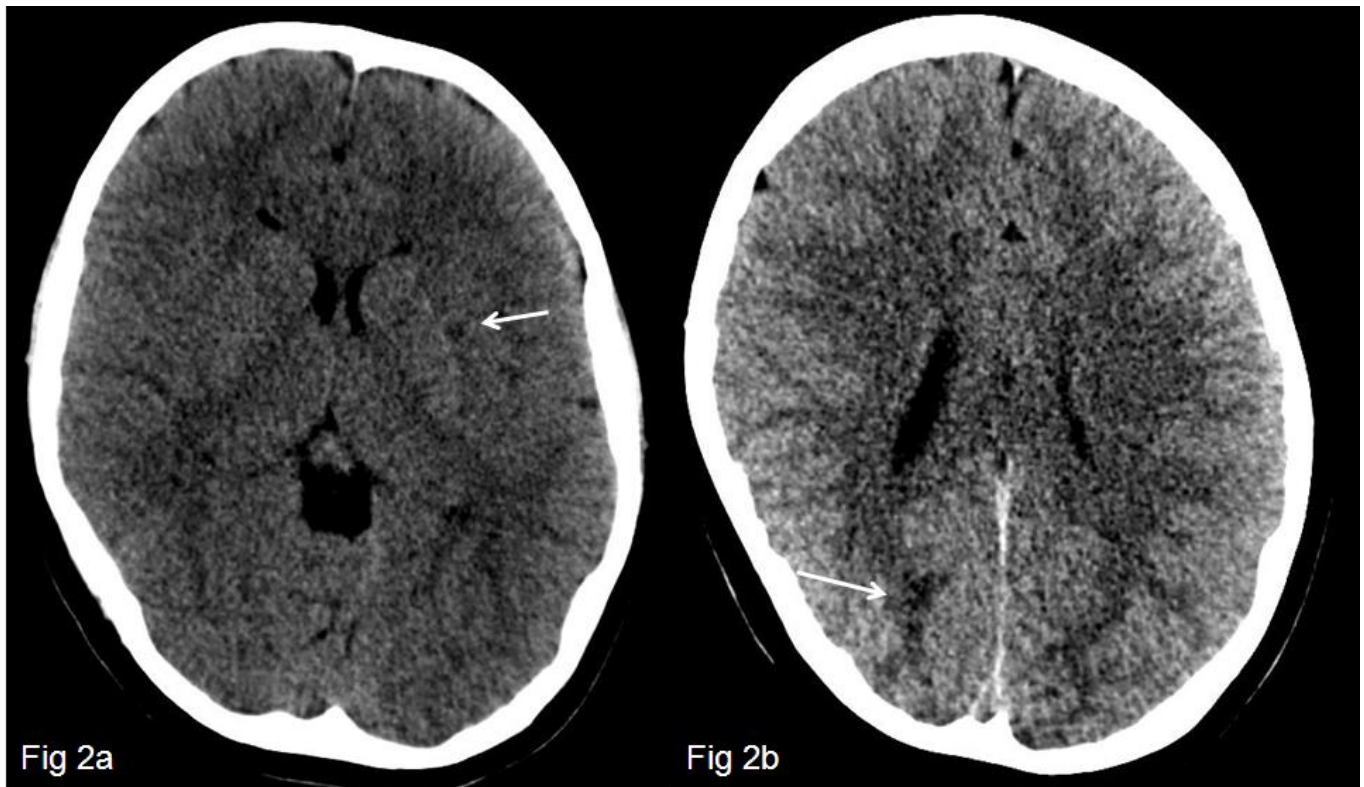


Figure 2: 12 year old male with traumatic portacaval fistula. FINDINGS: 2a and 2b. Multifocal small areas of hypodensity are visualized within the white matter, such as within the left parieto-occipital white matter and left external capsule, suspicious for early edema or ischemic injury. TECHNIQUE: Noncontrast brain CT images, Siemens Sensation 64, 4.8mm slice thickness, kvp 120, mA 450, axial plane.



Figure 3 (left): 12 year old male with traumatic portacaval fistula. FINDINGS: Loss of gray-white differentiation, sulcal and ventricular effacement have developed in the interim, consistent with diffuse cerebral edema (white asterisk). Geographic hypodensity within the left thalamus is consistent with an acute left thalamic infarct (white arrow). TECHNIQUE: Noncontrast brain CT images, Siemens Sensation 64, 4.8mm slice thickness, kvp 120, mA 450, axial plane.

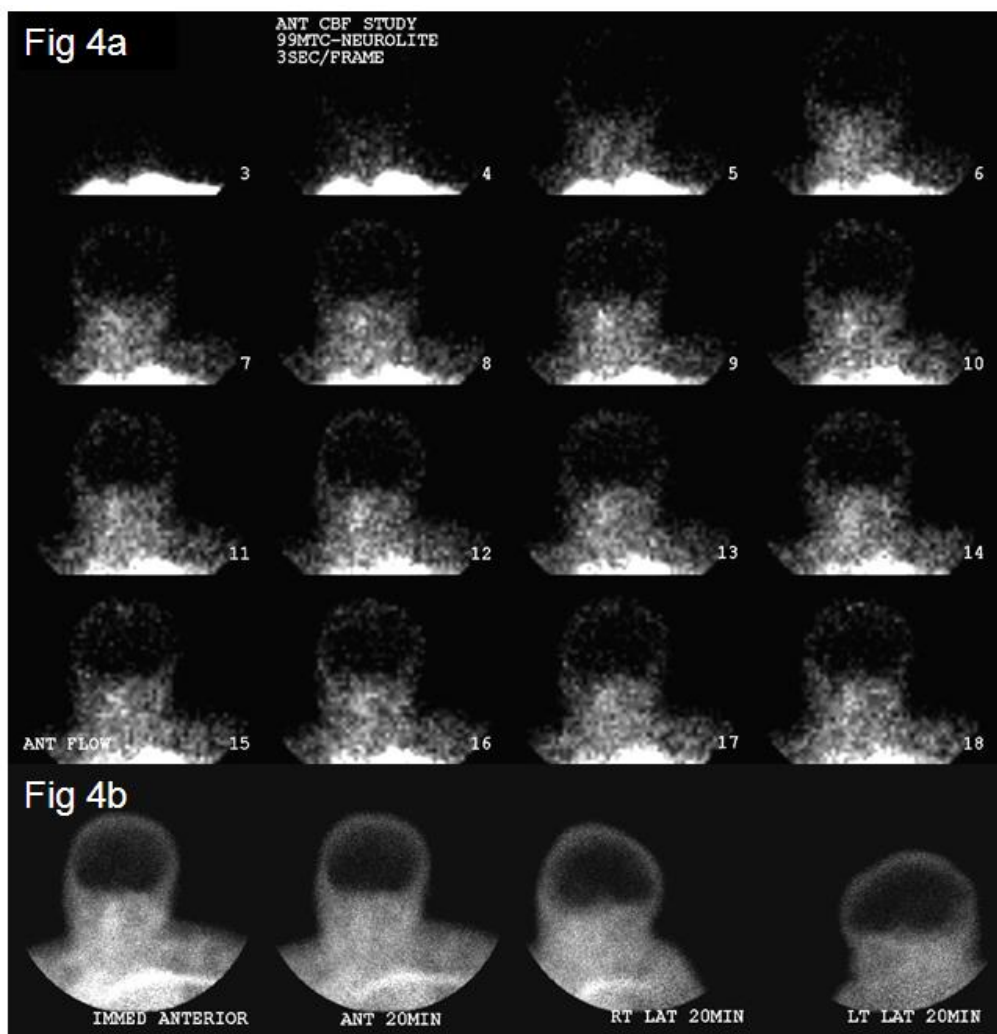


Figure 4: 12 year old male with traumatic portacaval fistula. FINDINGS: Absence of intracranial perfusion on flow images, and absence of Tc99m ECD metabolism on the delayed images, supporting the clinical diagnosis of brain death. TECHNIQUE: Following IV injection of 25.9mCi Tc99m ECD, dynamic flow images were obtained in anterior projection, followed by 20 minute delayed images in the anterior and lateral projections.

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Etiology	High grade liver trauma. Both cases had grade V liver injuries.
Incidence	Rare. To our knowledge, this is the second case reported in the literature
Gender Ratio	Both cases have been male
Age Predilection	Reported cases were age 12 and 23
Risk Factors	High energy trauma to the liver
Treatment	Conservative management was successful for the small fistula previously reported. Larger lesions may require interventional/surgical intervention
Prognosis	Variable: consider both on degree of associated liver injury and size of the fistula, and associated injuries (a right hepatic artery transection in this patient). The other reported patient, with the smaller fistula, demonstrated symptomatic and imaging improvement over 10 months. This patient died of severe hepatic encephalopathy resulting in cerebral edema with herniation.
Findings on Imaging	Abnormal, contrast opacified fistulous connection between the portal vein and intrahepatic or suprahepatic IVC.
Findings on Pathology	Not applicable

Table 1: Summary table for traumatic portacaval fistula

Differential diagnosis	X-Ray	CT
Traumatic portacaval fistula	Non-contributory	Abnormal connection between the portal venous system and IVC. Perfusion abnormality of the devitalized lobe. Evidence of high-grade liver trauma
Park Type 1 congenital portosystemic shunt	Non-contributory	Abnormal connection between the right portal vein and the IVC. Homogeneous hepatic enhancement.
TIPS	Radiopaque stent overlying the right upper quadrant, cranial end near the IVC	Radiopaque stent extending from the right portal vein to either the right or middle hepatic vein

Table 2: Differential diagnosis for traumatic portacaval fistula

ABBREVIATIONS

ATV = all-terrain vehicle
 CT = computed tomography
 ECD = ethyl cysteinyl dimer
 IVC = inferior vena cava
 TIPS = transjugular intrahepatic portosystemic shunt

KEYWORDS

High grade liver injury; grade V liver laceration; traumatic portosystemic fistula; traumatic portacaval fistula, traumatic portosystemic shunt

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