GASTROINTESTINAL RADIOLOGY AND HEPATOLOGY

# Acute post traumatic portal venous thrombosis associated with shattered spleen: A case report

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# Abstract

Post-traumatic portal venous thrombosis is a rare event, and is usually seen in association with penetrating injuries. Portal venous thrombosis following blunt abdominal trauma is extremely rare with only few reports in the literature, some associated with underlying coagulation disorders. We report multidetector computed tomography findings in a case of blunt abdominal trauma with otherwise normal coagulation profile, which showed shattered spleen and MDCT evidence of acute thrombosis in the right branch of the portal vein. To the best of our knowledge, this is the first documented report of acute portal venous thrombosis in association with shattered spleen.

Key words: MDCT; portal vein; shattered spleen; thrombosis

# Introduction

Traumatic portal venous thrombosis is extremely rare with only few documented cases in literature. In only few cases, portal venous thrombosis has been attributed to blunt trauma only.<sup>[1-6]</sup> One reported case in the literature was associated with an underlying coagulation disorder,<sup>[7]</sup> and in another case, acute nature of thrombosis was inferred form indirect evidence.<sup>[8]</sup> There are reports of portal venous thrombosis in association with hepatic or pancreatic laceration and ruptured kidney;<sup>[4,6,8]</sup> there is no report of acute traumatic portal venous thrombosis in association with shattered spleen. We report multidetector computed tomography (MDCT) findings in a young patient with a shattered spleen and acute portal venous thrombosis in right branch of portal vein.

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# **Case History**

A 30-year-old male patient presented with 2 hours history of road traffic accident. On clinical examination, patient was unstable with recorded blood pressure of 80/60 mmHg. There were no clinical signs of chronic liver disease or portal hypertension. He was nonalcoholic and nondiabetic. There was no history of jaundice in the past. Serology for hepatitis B and C was negative. His liver functions tests were within normal range. Bleeding and clotting time were within normal limits. Initial screening ultrasound showed splenic injury with significant amount of hemoperitoneum. MDCT was performed for characterization of splenic injury and any other associated injuries. It revealed hyperdense thrombus in the right branch of the portal vein in plain scan. In postcontrast images, hypodense thrombus was

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seen in the right branch of the portal vein with associated hyperenhancing area in the right lobe of the liver. No collateral channels or liver cirrhosis were seen. Splenic and mesenteric veins were normal. MDCT also revealed shattered spleen with grade V injury according to the AAST (American Association for the Surgery of Trauma) splenic injury scale with significant hemoperitoneum [Figures 1 and 2]. No other solid organ injury or fractures were seen. As the patient was hemodynamically unstable, urgent laparotomy was performed. On laparotomy, spleen was shattered with rupture of splenic hilum. Splenectomy was performed. Approximately 2L of hemoperitoneum was drained. Patient made uneventful postoperative recovery. Ultrasound performed after 1 week showed echogenic thrombus in the right branch of the portal vein with no flow on Doppler [Figure 3]. There was no sonographic evidence of bowel ischemic, hepatic infarct, or collateral channels. Patient's liver function test were within normal limits. Oral anticoagulant treatment was instituted after an interval of 10 days from surgery to reduce the chances of postoperative bleeding. Sonography done after 3 weeks from trauma showed partial recanalization of the right portal vein with thickening of its wall. There was no interval development of collaterals or varices. No clinical or sonography evidence of bowel ischemia was seen. Anti-coagulant treatment was continued for 1 month. On follow-up, patient's liver function tests remained normal.

# Discussion

The most common causes of portal venous thrombosis are underlying myeloproliferative disorders and hypercoagulable states, underlying hepatobiliary or



Figure 1 (A-C): Axial NCCT (A) image showing hyperdense (62 HU) thrombus in the right branch of portal vein (white arrow). Also seen in heterogeneous attenuation of spleen (dotted white arrow). Axial (B) and oblique coronal (C) CECT sections showing hypodense filling defect in right branch of portal vein (white arrows in B and C). Also seen wedge-shaped hyperenhancing area in the right lobe of liver secondary to perfusion alteration (dotted white arrows in B and C)

pancreatic inflammatory and malignant process, some drugs such as oral contraceptive pills, cirrhosis and recent surgery.<sup>[7,9,10]</sup> Trauma is a very rare cause of portal vein thrombosis. Most of the cases of posttraumatic portal venous thrombosis are secondary to penetrating trauma with blunt trauma being exceedingly rare cause of portal venous thrombosis.<sup>[3,5,7]</sup> Both local and systemic factors play a role in the development of post-traumatic portal venous thrombosis. It has been suggested that acute trauma can cause portal venous injury by shearing effect causing endothelial injury, which initiates the process of thrombus formation. Underlying hepatobiliary diseases such as cirrhosis, which causes venous stasis will further promote thrombus formation. Moreover, other underlying systemic prothrombotic states such as anti-phospholipid syndrome and oral contraceptive intake can increase the risk of thrombus formation.<sup>[5,7,9]</sup> Acute traumatic state can be associated with hypotension and shock which promotes venous stasis, thereby increasing the risk of venous thrombosis.[11]



Figure 2: Oblique coronal CECT image showing shattered spleen with large intraparenchymal and perisplenic hematomas (white arrow). Also seen in hemoperitoneum (dotted white arrows)



**Figure 3 (A and B):** Axial sonography image (A) showing echogenic thrombus in the right branch of the portal vein (dotted white arrow). Left branch of portal vein is indicated by white arrow. Axial color Doppler (B) image showing normal flow in the left branch of the portal vein (black arrow) with no flow in right branch (dotted white arrow). White arrow indicates prominent right hepatic artery

Formation of portal venous thrombosis secondary to trauma in otherwise normal patients is very rare. It has been reported both as an isolated entity<sup>[1-3,7]</sup> and in association with other organ injuries such as pancreatic and renal injures.<sup>[4,8]</sup>

There are no reports in literature of acute portal venous thrombosis in association with shattered spleen. The pathogenesis of portal venous thrombosis in our case could be embolic phenomenon from smaller vessels from shattered spleen (although there was no evidence of thrombus in splenic vein) or it could have formed *in situ* secondary to shearing endothelial injury.<sup>[6,8]</sup> Our case is also unique in the sense that the acute nature of the portal venous thrombosis can be confidently ascertained due to the hyperdense appearance of thrombus on non-contrast computed tomography, which has not been reported previously.

The most commonly used grading system for splenic injuries is the American Association for the Surgery of Trauma (AAST) grading system which grades splenic injuries from grade I to V, with shattered spleen considered a grade V injury. Higher grades of injury are associated with more chances of surgical management, the decision for which is typically based on the clinical condition of the patient. MDCT is an excellent modality for accurately depicting the site and extent of splenic injury and any other associated injuries.<sup>[12]</sup>

Acute thrombus is anechoic on sonography and can be easily missed unless high index of suspicion is maintained and color Doppler imaging is employed in all cases. Therefore, MDCT is very useful in acute trauma patients because it is not user dependent and can accurately depict associated other injuries. It can also depict the exact extent of thrombosis and any associated signs of mesenteric ischaemic.<sup>[4]</sup>

Management of traumatic portal venous thrombosis is not clearly defined and each case should be assessed on individual basis. Successful intravenous anticoagulation with heparin infusion has been described in a stable patient.<sup>[6]</sup> Associated injuries are also important as a significant solid organ injury, which require surgical intervention could be a relative contraindication to anti-coagulant therapy. Further, there is no clarity regarding the duration of anticoagulation therapy to be given. It is usually administered for longer periods for more widespread thrombosis.<sup>[7,8]</sup> In our case, the thrombus was in the right branch of the portal vein and there were no signs of bowel ischemic or hepatic infarction, therefore, primary treatment was aimed as splenic injury with delayed institution of anticoagulant therapy.<sup>[3,8]</sup> Potential complication of untreated portal venous thrombosis is portal hypertension but may also lead to ischemic bowel stricture if thrombosis extends into mesenteric venous arches.<sup>[5,6]</sup> Moreover, there are isolated reports of post-traumatic portal or inferior mesenteric vein thrombosis with secondary bowel ischaemic and in one of them, it was followed by ischemic stricture, which was managed successfully with resection and anastomosis.<sup>[5,13]</sup>

# Conclusion

In conclusion, acute portal venous thrombosis in setting of acute trauma is extremely rare. It can be easily missed on screening ultrasound performed in the setting of acute trauma. MDCT, whenever performed, is usually diagnostic. A delay in diagnosis has potential for propagation of thrombus with later complications such as portal hypertension and very rarely ischemic bowel stricture.

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#### **Conflicts of interest**

There are no conflicts of interest.

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