Rare disease

Thrombosis of the portal venous system following blunt abdominal trauma

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Summary

A 60-year-old man presented to the accident and emergency department with a 4-day history of abdominal pain following blunt abdominal trauma. An initial CT scan showed thickened walls of the proximal jejunum and thromboses in the portal, splenic and superior mesenteric veins. He was given warfarin and the abdominal pain resolved. A repeat CT scan 1 week later revealed significant resolution of the mural thickening and the portal vein thrombosis. A subsequent thrombophilia screen was negative and he continued taking oral anticoagulants for a total of 6 months. A repeat CT scan 3 months after presentation revealed complete recanalisation the portal venous system.

BACKGROUND

Thrombosis of the portal venous system resulting from blunt abdominal trauma in otherwise healthy individuals is rare, with few cases reported in the literature. This case report relates to a patient who developed thrombosis of the portal, splenic and superior mesenteric veins following minor injury to his abdomen and demonstrates the clinical presentation, the typical radiological findings and the response to treatment.

CASE PRESENTATION

A 60-year-old man presented to the accident and emergency department with a 4-day history of abdominal pain following blunt abdominal trauma. While working on the engine of his car, he had fallen from a stool onto the wing of the car; 4 h after the incident, he began to experience abdominal pain. This was constant, sharp in character and located in the right iliac fossa; there was also a dull pain felt in the left iliac fossa. These symptoms were not relieved by paracetamol and prevented him from sleeping at night. The pain persisted and, on the day of admission, was accompanied by two episodes of coffee-ground vomiting.

His medical history comprised an appendectomy in childhood and thromboembolic disease. At the age of 52 years, he had developed a pulmonary embolism following a plane journey and received oral anticoagulants for 6 months. There was no family history of thromboembolic disease.

He was married, lived with his wife and worked as a lorry driver. He was an ex-smoker with a 40 pack-year history, but had now stopped, and had never consumed alcohol to excess. There were no risk factors for chronic liver disease.

On examination, he was afebrile, with normal oxygen saturations. The cardiovascular system was normal and the chest was clear. He was not icteric and there were no stigmata of chronic liver disease. Abdominal examination revealed tenderness in the epigastrium and periumbilical

area; there was no hepatomegaly or splenomegaly, no rebound tenderness and bowel sounds were present; rectal examination was normal.

INVESTIGATIONS

Full blood count, urea and electrolytes, and amylase were within normal limits. Table 1 documents the trends in those blood tests found to be abnormal, most of which returned towards normal after 10 months of follow-up. The bilirubin remained raised, mostly due to an increase in unconjugated bilirubin at 23 mol/litre.

A screen for underlying causes of liver disease, including viral hepatitis serology and autoantibodies, was entirely negative.

A chest x-ray was normal and there was no free air under the diaphragm. An abdominal CT scan reported thickened walls of the proximal jejunum, a small amount of free fluid in the pelvis and thromboses in the portal vein, superior mesenteric vein and splenic vein (figures 1 and 2). No free intraperitoneal air was observed and there was no evidence of portal hypertension.

Table 1 Results of the liver function tests and C reactive protein values at admission and, subsequently, at 10 month follow-up.

Test (normal range)	Admission	Discharge	10 Month follow-up
Bilirubin (3–20 mol/litre)	58	Normal	37
Alanine transaminase (0–37 U/litre)	55	75	46
Alkaline phosphatase (38–149 U/litre)	96	194	Normal
γ-Glutamyl transferase (12–64 U/litre)	150	842	117
C reactive protein	58	Normal	NA

NA, not applicable.

Figure 1 Abdominal CT scan showing splenic vein thrombosis (L, liver; S, spleen; SVT, splenic vein thrombosis).

Figure 2 Abdominal CT scan showing portal vein thrombosis (L, liver; S, spleen; PV, portal vein thrombosis).

An oesophagogastroduodenoscopy revealed a Schatzki ring and a small hiatus hernia but no features of portal hypertension. A thrombophilia screen during admission was negative.

DIFFERENTIAL DIAGNOSIS

- ▶ Inherited thrombophilic disorders: factor V Leiden mutation, protein S and C deficiency.
- ▶ Acquired disorders of coagulation: myeloproliferative diseases, antiphospholipid syndrome, malignancy and paroxysmal nocturnal haemoglobinuria.
- ▶ Chronic liver disease.
- ▶ Intra-abdominal inflammatory conditions: sepsis (eg, diverticular abscess, appendicitis) pancreatitis and inflammatory bowel disease.
- ▶ Malignant infiltration of portal veins.
- ▶ Blunt abdominal trauma.

TREATMENT

A diagnosis of thrombosis of the portal venous system precipitated by blunt abdominal trauma was made. The patient received 6 months of treatment with warfarin. In view of his history of pulmonary embolus, the aim was to achieve an international normalised ratio (INR) of 3–4.

OUTCOME AND FOLLOW-UP

Most of the patient's symptoms resolved within 1 week of starting treatment and, apart from mild non-specific abdominal pain, he had no other issues over the following 8 months.

A week after the patient began taking anticoagulants, a repeat abdominal CT scan was performed that showed resolution of the mural thickening of the jejunum and significant reduction in size of the portal vein thrombus (figure 3). A further CT scan 3 months after initial presentation revealed a complete recanalisation of the portal,

superior mesenteric and splenic veins, although multiple collateral vessels were noted at the splenic hilum (figure 4). The patient underwent anticoagulation treatment. After 6 months, a joint decision was made by the patient and the haematologist to withdraw warfarin and only consider long-term anticoagulation if he experienced further thrombosis.

DISCUSSION

Thrombosis of the portal venous system may be due to intra-abdominal disease or thrombophilic disorders. Portal venous thrombosis is not uncommon in patients with established chronic liver disease, occurring in 11% to 21% of those with cirrhosis^{1 2} Other local predisposing factors include malignant invasion of the portal vein, pancreatitis and intra-abdominal sepsis. In the absence of gastrointestinal pathology, 40% to 60% of patients with thrombosis of the portal venous system are found to have thrombophilic disorders^{3 4} and the proportion of patients classified as having idiopathic thrombosis has fallen with advances in genetic typing.⁵

While blunt abdominal trauma has been linked with thrombosis of the portal vein and its tributaries, this clinical entity is very rare. A literature review identified three cases where an exhaustive search for a cause had failed to reveal an alternative aetiology.^{6–8}

Because thrombosis of the portal venous system is uncommon, especially in the absence of cirrhosis, the data on the benefits of treatment are restricted to case reports and retrospective studies. Intravenous or intra-arterial catheter-directed thrombolytic treatment has been shown to be effective in the resolution of acute portal vein thrombosis.^{9–14} However, the risks of haemorrhage from varices with thrombolytic treatment must be considered. Anticoagulation, however, appears to be safe in portal venous system thrombosis. A large study involving 136 patients without cirrhosis or malignancy showed no increased risk

Figure 3 Abdominal CT scan showing patent blood flow through the portal vein (L, liver; S, spleen; PV, portal vein).

Figure 4 Abdominal CT scan showing collaterals around the splenic hilum (C, collaterals; L, liver; S, spleen)

of bleeding or death from haemorrhage in the anticoagulated group.¹⁵ A retrospective study reported that in patients without cirrhosis or cancer, 45% had improved portal flow with anticoagulants compared with only 15% of those left without treatment.¹⁶

There are no guidelines with regards the duration of anti-coagulants or the target INR range, but it seems appropriate to consider patients on an individual basis. A longer course of anticoagulants will be needed in patients with widespread thrombus extending into the splanchnic veins and with underlying thrombophilic disorders.¹⁷

The possible relevance of pre-existing liver disease in this case report must be addressed. There was no clinical evidence of chronic liver disease, nor were there any predisposing factors. The investigations showed a negative screen for the causes of liver disease and there was no evidence, on CT scan or at gastroscopy, of features suggestive of portal hypertension. Although the liver function tests were abnormal during admission, they returned towards normal. The cause of this disturbance remains uncertain but may be a consequence of the thrombosis itself. The intermittent rise in unconjugated bilirubin may be accounted for by underlying Gilbert's syndrome.

The close temporal relationship between the abdominal injury and the onset of pain strongly supports the aetiology of this patient's portal venous system thrombosis being blunt abdominal trauma. This may well have been aggravated by an underlying thrombophilic state although the screen for this was negative. However, the patient's history of thromboembolic disease would support a currently undiagnosed abnormality in coagulation. There is little evidence from the patient to support alternative causes for portal venous system thrombosis.

Learning points

- ▶ To consider abdominal venous thrombosis in patients with blunt abdominal trauma
- ▶ To consider abdominal venous thrombosis as a cause of abdominal pain
- ▶ Treatment of abdominal venous thrombosis is with anticoagulation.

Competing interests None.

Patient consent Obtained.

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