Blunt abdominal trauma –
An important cause of portal venous pseudoaneurysm

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ABSTRACT

Aneurysms and pseudoaneurysms of the portal venous system are rarely seen following abdominal trauma but clinicians need to be aware of them as possible vascular complications following blunt trauma. This case report of a 10 year old boy following a handlebar injury demonstrates a clear causal relationship between trauma and portal venous pseudoaneurysm. Portal venous aneurysms have a prevalence of less than 0.4% and most are found in patients with underlying hepatocellular disease. Many are asymptomatic in which case surveillance is an accepted management strategy, with Doppler ultrasound proving useful. Complications including thrombosis, distal embolism, compression of the biliary tree and haemorrhage are usually indications for surgical management. Portal venous pseudoaneurysms may be managed conservatively but transcatheter embolisation can be used if there are ongoing complications or haemorrhage.

CASE REPORT

A 10 year old boy was admitted after falling from his bike. The handlebars had impacted against his lower chest and upper abdomen. He complained of abdominal tenderness with pain referred to his shoulder tip and on examination had bruising to his right lateral rib margin and an abdomen that was particularly tender at the right upper quadrant. He had no head injury or haematuria and was not clinically shocked (BP 106/62 mmHg, pulse 88bpm). A chest radiograph demonstrated no rib fracture or pneumothorax. Contrast enhanced multislice CT imaging was undertaken to investigate the possibility of liver injury (Figure 1).

This demonstrated a grade 3 liver laceration with subcapsular haematoma and intraperitoneal blood. There was no contrast blush to indicate active extravasation. All other solid viscera were normal. The boy was managed conservatively with a plan for surgical treatment if transfusion requirements exceeded 40ml/kg. Hb was 11.2 g/dL (normal 11.5 - 15.8) and LFTs were deranged with an ALT of 331IU/L (normal 5-40) though at this stage ALP was normal. Repeat Hb a few hours later showed a Hb of 10.4 g/dL and so the boy was transferred to PICU and ultrasound was performed (Figure 2).

There was free fluid around the liver, spleen and within the pelvis where there were septations. The area of liver laceration was demonstrated as an echogenic region within the right lobe of the liver. Appearances were thought to be stable with those on the previous CT. A persistent ileus and progressive abdominal distension raised the concern for a
biliary leak. The boy’s LFTs continued to deteriorate with ALP rising to >550 IU/L (normal 150-530 IU/L) and ALT to >400 IU/L. CT was repeated (Figure 3) which demonstrated an increased volume of intraperitoneal fluid and an avidly enhancing 12mm focus (‘contrast blush’) in close proximity to the intrahepatic portal vein. Maximum intensity projections demonstrated that the cause was a pseudoaneurysm of the intrahepatic right portal vein (Figure 4).

In this case the right anterior portal vein arises from the left portal vein, one of the four commonly described branching patterns of the intrahepatic portal vein (1). The gallbladder and bile duct were dilated with high attenuation within the gallbladder consistent with recent haemorrhage (Figure 5).

A diagnosis of bile peritonitis was made and it was decided that he should undergo surgical drainage with a view to subsequent angiography of the hepatic arterial tree in order to exclude hepatic artery pseudoaneurysm. Initially a laparoscopy was performed. There was purulent bile stained fluid with multiple fibrinous adhesions. The abdomen could not be adequately assessed or lavaged and so the procedure was converted to open laparotomy. 1.5L of bile stained fluid and old clot was removed and multiple adhesions were noted. Two intra-abdominal drains were placed and three units of blood transfused. The boy was sent for angiography to exclude concurrent injury to hepatic arteries (Figure 6).

The hepatic vessels were distorted by the intrahepatic haematoma but there was no hepatic artery aneurysm and the diagnosis of traumatic infrahepatic right portal venous pseudoaneurysm was confirmed. In the post-operative period the biliary leak steadily diminished from 700ml/day and finally stopped spontaneously after 2 weeks. He has made a good recovery and a follow-up ultrasound has demonstrated no free fluid.

TEACHING POINT

Both congenital and acquired causes of portal venous aneurysms have been suggested. Congenital aneurysms may be diagnosed in utero (7) or may be identified incidentally in patients with otherwise histologically normal livers and stable follow-up appearances (8). They arise because of inherent weakness in the wall of the portal vein and are usually diagnosed in children and young adults (9).

Many acquired aneurysms are found in patients with portal hypertension or underlying hepatocellular disease. The development of portal hypertension leads to altered haemodynamics which predisposes to the formation of aneurysms and so is often considered the most important aetiological factor. Others include pancreatitis, surgery, and interventional procedures such as sclerotherapy (10).

Following trauma, portal venous pseudoaneurysms have been rarely described (11). Rather than being a focal dilatation of a vessel, a pseudoaneurysm is a pulsatile haematoma that results when blood leaks beyond a tear or disruption in the vessel wall and forms a haematoma. The blood is contained by surrounding parenchyma and surrounding haematoma.

The most common location for portal venous aneurysms and pseudoaneurysms is the main portal vein and branching sites of the infrahepatic portal vein (12) though extrahepatic portal vein aneurysms have been reported (9). Most are asymptomatic though they may cause non-specific abdominal pain. Complications such as thrombosis, distal embolism, compression of the biliary tree causing jaundice and rupture causing bleeding may occur.

Treatment depends on clinical presentation (5). If the aneurysm is an incidental finding in patients with no associated liver disease or portal hypertension, surveillance is regarded as safe as the aneurysm is not expected to enlarge. Doppler sonography is particularly helpful in following up the haemodynamics of the vascular anomaly (13). If aneurysms are enlarging or there are complications then decompressive surgeries may prevent progression of the aneurysmal dilatation (13). Blunt liver injury can usually be safely managed conservatively. In the case of hepatic arterial pseudoaneurysms complications such as rupture and associated haemorrhage or fistulisation mean transarterial catheter embolisation is a useful adjunct to non-operative management (14). The portal venous system is a lower pressure system and so often conservative management is successful. However, transcatheter embolisation via selective portography may be used in the case of ongoing haemorrhage (15).

DISCUSSION

Whilst arterial aneurysms are relatively common, in the context of trauma, hepatic artery aneurysms have a prevalence of less than 1% (2). Hepatic artery aneurysms are most common following penetrating trauma though some have been reported following blunt trauma (3).

Primary venous aneurysms and pseudoaneurysms are rare. Most arise in the popliteal, saphenous and jugular veins. Portal venous aneurysms are focal dilatations of the portal venous system. When a dilated segment of vein measures over the accepted upper limit of normal for diameter it is classified as aneurysmal - in the case of the extrahepatic portal vein this is 15mm (4). The vein may show fusiform or saccular configurations and the aneurysm may have a short or wide neck. Portal venous aneurysms represent less than 3% of all venous aneurysms (5). A recent study of over 4,000 consecutive patients suggested a prevalence of less than 0.4% (6).
References


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Figure 2 (top): 10 year old male with portal venous pseudoaneurysm. Ultrasound (Toshiba, curvilinear array, 4MHz) demonstrates a) hyperechoic region within the right lobe of the liver consistent with the known liver laceration (arrow), b) free fluid around the liver (arrow) and c) free fluid and septations (arrow) within the pelvis.

Figure 4 (right): Maximum Intensity Projection (MIP) images of contrast enhanced CT (Siemens Somatom Sensation 4 slice scanner, 120 kV, 380mA, 65mls omnipaque contrast media) demonstrating a) pseudoaneurysm (arrow) originating from the portal venous system and b) pseudoaneurysm originating from the right anterior branch of the portal vein (narrow arrow) arising from the left portal vein (wide arrow). Again the intraperitoneal fluid and liver laceration are demonstrated.
Blunt abdominal trauma - An important cause of portal venous pseudoaneurysm

**Figure 5:** 10 year old male with portal venous pseudoaneurysm. 2 weeks following blunt abdominal trauma axial portal venous contrast enhanced CT (Siemens Somatom Sensation 4 slice scanner, 120 kV, 380mA, 65mls omnipaque contrast media) demonstrates high attenuation material within a distended gallbladder (arrow) consistent with recent haemorrhage. Intraperitoneal fluid is seen within the abdomen surrounding the liver capsule and spleen.

**Figure 6:** 10 year old male with portal venous pseudoaneurysm. Angiographic appearances. An SOS Omni selective catheter was introduced into the celiac axis. Contrast run demonstrates normal hepatic arterial system distorted by clot.

**ABBREVIATIONS**

- ALP = Alkaline phosphatase
- ALT = Alanine aminotransferase
- BP = Blood pressure
- Bpm = Beats per minute
- CT = Computed tomography
- g/dL = Grams per deci-litre
- Hb = Haemoglobin
- IU/L = International units per litre
- L = Litre
- LFTs = Liver function tests
- PICU = Paediatric intensive care unit

**KEYWORDS**

Portal venous aneurysm, Trauma

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