

## Case Report

# Patient with jaundice & melaena: A rare case report of haemobilia due to idiopathic hepatic artery pseudoaneurysm

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## Introduction

Haemobilia is defined as the presence of blood in the biliary tree characterized by the triad of jaundice, right hypochondrial pain and upper gastrointestinal bleeding. Aetiologies include liver trauma, inflammatory causes (acute and chronic cholecystitis), infections (cholangitis, liver abscesses, parasitic infections) and vascular diseases (aneurysms, vasculitis, arteriovenous malformations) [1].

Pseudoaneurysms occur when there is a breach in the vessel wall with blood leaking through the wall but contained by the adventitia or surrounding soft tissue. The incidence of hepatic artery aneurysm is estimated at 0.002% and approximately 50% of them are pseudoaneurysms [2]. Hepatic artery aneurysms (true aneurysms and pseudoaneurysms) account for nearly 10% of haemobilia cases [3]. The most common cause of hepatic artery pseudoaneurysm (HAP) is iatrogenic trauma such as liver biopsy, transhepatic biliary drainage, cholecystectomy and hepatectomy. They may also occur with infections associated septic emboli. HAP has also been reported, rarely, in patients with acute pancreatitis [4].

## Case presentation

A 60-year-old man with a past history of glioblastoma with recurrence, for which he had undergone excision of tumour and follow up chemotherapy, was admitted to the ward with complaints of melaena, generalized abdominal pain and lethargy of 05 days duration. He also had noticed progressive yellow discolouration of sclera. There was no history of pruritus. He had symptoms of anaemia such as lethargy, shortness of breath and mild chest pain. He did not have symptoms of dyspepsia or complain of dysphagia. There was no history of abdominal trauma.

On examination, he was not emaciated but was pale and deeply icteric. He was mildly tachypnoeic. Hyperpigmentation of the buccal mucosa or skin creases was not seen. He was tachycardic with a pulse rate of 120 beats per minute with a stable blood pressure reading of 110/70 mmHg on admission. His oxygen saturation was 98% on room air. Abdominal

examination showed tenderness in the right hypochondrium without any organomegaly or palpable masses. There was no clinical evidence of free fluid. Digital per rectal examination showed melaena. All other system examinations were unremarkable.

Blood was taken for investigations and he was stabilized with a cross-matched blood transfusion and fluids. Intravenous (IV) tranexamic acid 1g and IV Vitamin K 10mg stat doses were given to arrest any bleeding. IV ceftriaxone 1g was given empirically to prevent infective complications. He was kept nil by mouth. The results of his blood and urine investigations are shown in Table 1.

**Table 1: Result of investigations**

| Investigations                       | Results   |
|--------------------------------------|---|
| Full Blood Count (FBC)               | White Blood Cells (WBC): 14.21/ $\mu$ L (4-10)<br>Neutrophils: 83.4%    Lymphocytes: 7.5%<br>Monocytes: 8.9%    Eosinophils: 0.1%<br>Red Blood Cells (RBC) $1.56 \times 10^3$ / $\mu$ L (4.3-5.3)<br>Haemoglobin (Hb): 4.3 g/dL (13.0-16.0)<br>Haematocrit (Hct): 12.4%<br>Mean Corpuscular Volume (MCV): 79.5 fL (80-100)<br>Mean Corpuscular Hb (MCH): 27.6 pg (27-32)<br>Platelets (Plt): 390/ $\mu$ L (150-450) |
| Liver Function Test (LFT)            | Alanine Aminotransferase (ALT): 116 U/L (16-63)<br>Aspartate Aminotransferase (AST): 185 U/L (15-37)<br>Alkaline Phosphatase (ALP): 948 U/L (46-116)<br>Total Protein (TP): 40g/L (64-82)<br>Albumin: 15g/L (34-50)<br>Globulin: 24g/L (22-48)<br>Total Bilirubin: 204 $\mu$ mol/L (0-17)<br>Direct Bilirubin: 170 $\mu$ mol/L (0-3)  |
| Serum Amylase                        | 346 U/L (25-115)  |
| Serum electrolytes                   | Sodium (Na): 132 mmol/L (136-145)<br>Potassium (K): 3.6 mmol/L (3.5-5.1)  |
| Blood Urea                           | 14.6 mmol/L (2.5-6.4)   |
| Serum Creatinine                     | 105 $\mu$ mol/L (62-115)  |
| Erythrocyte Sedimentation Rate (ESR) | 43mm/1 <sup>st</sup> hour   |
| C-Reactive Protein (CRP)             | 34 mg/L (0-3)   |
| Lactate Dehydrogenase (LDH)          | 219 U/L (120-246)   |
| Troponin I                           | < 0.012 ng/ml (0-0.15)  |
| International Normalized Ratio (INR) | 1.1 (0.8-1.2)   |
| Urine Full Report (UFR)              | Protein: +                      White Cells: Field full<br>Red Cells: Nil                Crystals: Nil<br>Casts: Nil                      Urine Bile: +   |
| Random Blood Sugar (RBS)             | 128 mg/dL   |

His 12 lead ECG showed sinus tachycardia with no evidence of acute ischaemic changes. Chest X-Ray was normal. Ultrasound scan (USS) of the abdomen was performed and it showed a cystic structure with bi-directional colour flow near the pancreatic head suggestive of a hepatic artery aneurysm. The common bile duct and the intrahepatic ducts were found to be dilated due to compression by the aneurysm. Both kidneys showed an increased cortical echogenic pattern. Pancreas, spleen, bladder and prostate appeared normal. There was no free fluid in the abdomen.

An urgent oesophagogastroduodenoscopy (OGD) was done but it did not reveal any bleeding point up to the duodenum. There was mildly blood-stained fluid seen in the duodenum and there was a bulge in the gastric body and first part of the duodenum suggestive of an external compression. (Figure 1)

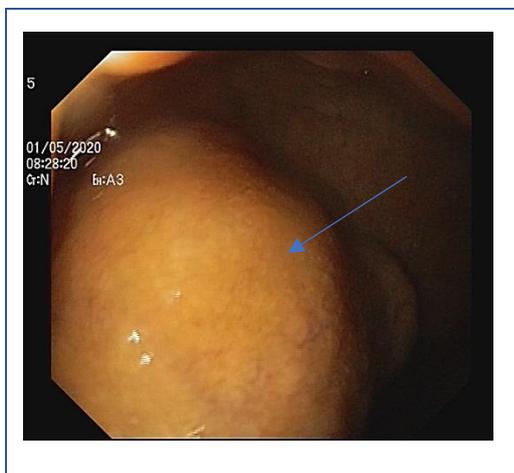


Figure 1: OGD image showing the bulge due to external compression

A non-contrast computed tomography (NCCT) of the abdomen and CT angiogram were performed and it showed an enhancing lesion in the abdomen arising from the distal main hepatic artery, favouring a false aneurysm. (Figures 2,3 & 4)



Figure 2: NCCT Abdomen (Axial view) showing hepatic artery pseudoaneurysm

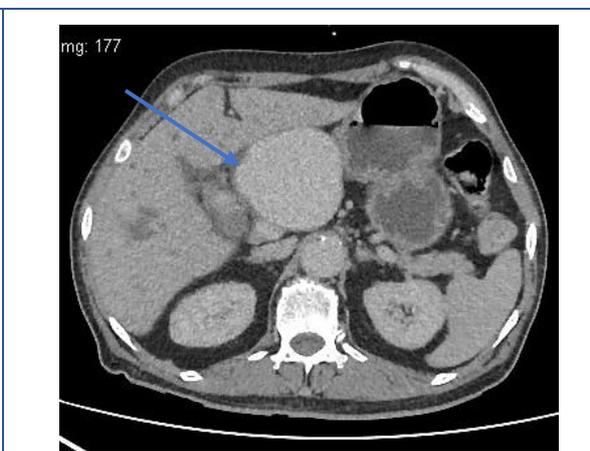
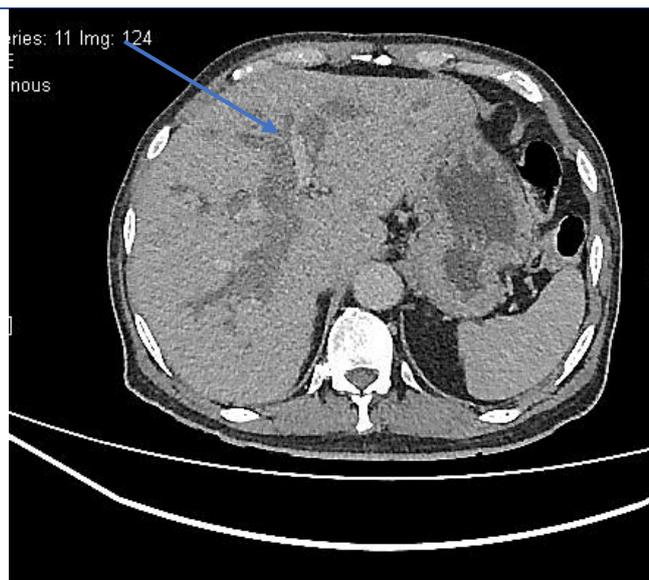


Figure 3: NCCT Abdomen (Coronal reconstruction) showing hepatic artery pseudoaneurysm

The gall bladder showed high intensity material with a density of 61 Hu, raising the suspicion of bleeding into the gall bladder due to an existing connection between the gall bladder and the hepatic artery pseudoaneurysm. Liver showed intra hepatic bile duct dilatation due to compression at the porta hepatis by the pseudoaneurysm. (Figure 5)



**Figure 4:** 3D Volume rendered image of aorta and its branches showing pseudoaneurysm in right hepatic artery



**Figure 5:** NCCT Abdomen (Axial view) shows intra hepatic bile duct dilation due to compression at porta hepatis.

After initial stabilization, the patient was further transfused with 04 pints of crossmatched blood, 06 units of platelets and 10 units of cryoprecipitate. Oral metoprolol 40mg 8 hourly was started to control his tachycardia that persisted even after correction of anaemia and dehydration. He improved symptomatically and there was no melaena by Day 3.

Haemoglobin level was found to be static at 10.1 g/dL. He was transferred to the surgical ward for further interventions on Day 3.

Inputs regarding the management were obtained from a multidisciplinary team of physician, gastrointestinal surgeon, interventional radiologist, clinical oncologist and neurosurgeon. Due to the large size of the aneurysm and considering the likely mortality rate of the procedure, the possibilities of surgical resection, coil embolization and angioembolization were ruled out. Percutaneous thrombin injection to obliterate the aneurysm coupled with stenting of the right hepatic duct to relieve obstruction due to external compression was considered the better option given the patient's background, clinical conditions and the procedure related complications.

Unfortunately, the patient developed melaena and fresh bleeding per rectum indicative of massive bleeding from the aneurysm and succumbed. The cause of death was concluded to be rupture of a HAP resulting in massive blood loss.

## Discussion

HAP is potentially fatal, as it can lead to sudden life-threatening haemorrhage as seen in this case. The incidence of hepatic artery aneurysm rupture has been reported to be around 21%–80% with a mortality of 21%–43% which is quite high [2]. HAP can be intrahepatic or extrahepatic. Ultrasound demonstrates a cystic structure along the course of hepatic artery

with brisk internal colour flow, to-and-fro pattern and Ying-Yang sign. CT Angiogram of the abdomen and pelvis can be very helpful in cases of visceral aneurysms and pseudoaneurysms.

HAP is mainly caused by acute or chronic artery injuries such as blunt or penetrating injuries and interventional radiological procedures. A minority may occur as a result of bile duct damage, usually associated with stone impaction or procedure-related infection. The cause of HAP in this patient was not established [4]. Acute pancreatitis due to an unknown cause may have led to pseudoaneurysm formation due to autodigestion of pancreatic enzymes [4] but the laboratory investigations did not suggest the possibility of acute pancreatitis and CT findings did not favour it as well.

Idiopathic HAP is also reported occasionally. As there were no apparent causes for the development of HAP in our patient, a diagnosis of idiopathic HAP was made.

Not all HAP results in haemobilia. There should be a connection between the hepatic artery and the biliary tree, like in this case where the HAP opened into the cystic duct. Most complications from haemobilia are attributable to acute blood loss; other complications are secondary to thrombus formation in the biliary tree [5].

Management of HAP depends on the severity, location and underlying aetiology. In mycotic pseudoaneurysms, surgical resection is the routine treatment, because endovascular material may serve as an infectious nidus. Intravascular occlusion can be beneficial to stabilize patients with active bleeding prior to surgery.

Percutaneous thrombin injection is widely used to treat peripheral pseudoaneurysms. Percutaneous thrombin injection has also been described for the management of visceral pseudoaneurysms. Coil embolization is one of the cornerstones in treating the intrahepatic pseudoaneurysms. Angioembolization results in rapid bleeding control, lower transfusion requirement and shorter hospital stay. Currently, angioembolization is replacing surgery as the initial modality of choice [2].

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