Hepatic Artery Aneurysm: A Rare Presentation as Painless Obstructive Jaundice

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Abstract
We present a rare case of a Hepatic Artery Aneurysm (HAA) of the right hepatic artery in a 59-year-old man presenting with acute painless obstructive jaundice. Computed Tomography (CT) and mesenteric angiographic images are presented. HAA, presenting with painless obstructive jaundice often have a poor prognosis and should be considered as a rare cause in unexplained cases of obstructive jaundice.

Introduction
Hepatic artery aneurysms (HAAs) are rare vascular lesions. They account for 16–20% of all visceral artery aneurysms. An HAA was first described by Wilson in 1809.1 We present a case of an HAA of the right hepatic artery (RHA) presenting as painless obstructive jaundice. Computed Tomography (CT), mesenteric angiographic and intra-operative images are presented.

Case report
A 59-year-old man presented with two weeks history generalised pruritis and yellow discolouration of skin and sclera. On questioning he had dark urine colour and pale stool for the same duration. He was previously fit; in particular there was no history of percutaneous intervention or trauma. On examination he was clinically jaundiced with a non-tender abdomen and hepatomegaly. Ultrasound of his biliary tract detected an abnormal mass in right upper quadrant but exact definition of this mass could not be obtained.

A CT scan of the abdomen revealed a large complex HAA (Fig. 1a and b). The aneurysm causes significant mass effect at the porta hepatis with obstruction of the common hepatic duct and compression of the main portal vein (Fig. 1c and d). Selective coeliac trunk angiography confirmed the CT findings of a complex right HAA with aneurysmal dilatation of the common hepatic artery (CHA) (Fig. 2). An accessory left hepatic artery (LHA) was present, arising from the left gastric artery (Fig. 2a). A small right renal artery aneurysm was also noted at angiography (not shown). The remainder of the visceral arteries was normal.

Due to the configuration of the right HAA with intrahepatic branches of the right lobe of liver arising directly from the aneurysm sac there was no endovascular option for aneurysm exclusion whilst maintaining arterial supply to
the right liver. Similarly the small right renal artery aneurysm was not amenable to endovascular treatment due to a wide neck arising at a major bifurcation. Therefore an open repair was planned. Preoperatively a left sided percutaneous transhepatic external biliary drain was placed to decompress the liver.

Intraoperative findings revealed a 9-cm complex HAA involving the RHA with significant distortion of portal anatomy (Fig. 3). Proximal control was achieved with exposure of the CHA and coeliac axis. The gastro-duodenal artery (GDA) was tied and divided. The accessory LHA arising from left gastric artery was also controlled. Distal control could not be achieved because the aneurysm sac was adherent to the subhepatic surface of the right lobe of liver. The common bile duct (CBD) was divided at the time of opening of aneurysm. On opening the aneurysm sac there was minimal backflow bleeding from the liver; therefore the aneurysm and RHA were tied off. A cholecystectomy and a 70-cm Roux loop biliary reconstruction were performed with a choledochojejunostomy and jejunojejunostomy. The right renal artery aneurysm was found to be very small (<6 mm size) and therefore no surgical repair was performed. The patient made good postoperative recovery.

Discussion

HAA is not initially diagnosed in many cases because of its rarity and lack of unique symptomatology. The average age at presentation is 40 (10–83) years, and the male to female ratio is 2:1. The natural history of HAAs is unclear. 60% of the aneurysms are true and 40% are false. Of the HAAs, extrahepatic aneurysms occur 4 times more frequently than intrahepatic lesions, with two thirds of the extrahepatic lesions arising from the CHA. An increase in incidence has been noted in the last 20 years, reflecting the increasing use of modern imaging. The aetiopathogenic factors include atherosclerosis, medial degeneration, vasculitides, fibromuscular dysplasia, secondary periarterial inflammation and iatrogenic trauma. HAAs have a high frequency of rupture that ranges from 20 to 80%. Their clinical presentation is often non-specific prior to rupture, with most
patients being asymptomatic, detected incidentally whilst imaging other symptoms or exhibiting upper abdominal pain. Unlike aortic aneurysms, the risk of rupture relative to size is unknown, and it is therefore stated that all lesions warrant treatment.² Rupture can be either intraperitoneal, into the biliary tree or into the gastrointestinal tract. Obstructive jaundice can occur, through either extrinsic compression of the CBD by the aneurysm or intraductal occlusion by blood clot.

The diagnosis of HAA can be difficult, even at surgery, in many instances. As many as 30–50% of cases are completely unsuspected and are discovered only at autopsy.⁴ On ultrasonography, HAA appear as an anechoic or complex mass with an anechoic centre located at the porta hepatis or rarely at the intrahepatic level. With advances in available investigative modalities like CT, MRI and mesenteric angiography pre-operative planning can be much more precise. Angiography is not only the gold standard in the diagnosis of HAA, but also serves as a guide for therapeutic embolization in suitable cases.¹,⁵

There are a few recent reports in the literature regarding HAAs causing obstructive jaundice with abdominal pain but HAAs presenting with painless jaundice are very rare.³ Treatment of a specific aneurysm depends on its location and regional vascular anatomy. The therapeutic options include embolization of the aneurysm, stenting across the parent vessel and embolization of the common hepatic artery or open surgical repair with or without reconstruction. The incidence of hepatic necrosis following interruption of the common hepatic artery is low owing to the rich hepatic collateral supply.⁶,⁷ Embolization is the accepted treatment of choice for the intrahepatic aneurysms.⁴ Aneurysms of the hepatic artery proper (distal to GDA) require excision and reconstruction with an autologous conduit. CHA (pregastroduodenal) aneurysms can safely be treated by ligation and exclusion, aneurysmectomy without arterial reconstruction or embolisation.⁸ In our case the liver had a good collateral supply from an accessory left hepatic artery via the left gastric artery and an accessory segment 4 artery. As a result the liver did not suffer ischaemic damage despite ligation of RHA. Postoperatively the patients liver function normalised.

In conclusion, HAAs are an extremely rare cause of painless jaundice. HAAs are associated with a poor prognosis if the diagnosis is missed. HAAs should be considered in unexplained cases of obstructive jaundice.

Conflict of Interest

None.

Figure 2  (a) Early phase of selective coeliac trunk angiogram demonstrating the large Right HAA (double asterisk) with early filling of the complex sac (single asterisk) arising from the aneurysmal Right Hepatic Artery. Accessory left hepatic artery supplied by the Left Gastric Artery (arrow head). (b) Late phase of selective coeliac trunk angiogram demonstrating the full extent of the complex aneurysm sac.

Figure 3 Intra-operative findings at laparotomy. a – complex hepatic artery aneurysm involving right hepatic artery, b – liver, c – gall bladder. Sling control of common hepatic artery (red), splenic artery (yellow) and gastroduodenal artery (blue). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)
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References