

Case Report

Hepatic Artery Aneurysm: A Rare Presentation as Painless Obstructive Jaundice

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Abstract

Aneurysm of the hepatic artery is a rare and often asymptomatic pathology with a high risk of rupture. Its diagnosis is difficult. They require a preoperative angiographic examination to determine the location and extent of the aneurysm, to assess the circulation of locums and search for other locations. The treatment is mainly surgical, especially in case of acute complication, but arterial embolization is a good therapeutic alternative in some cases and especially in intrahepatic locations.

We report the case of a 54-year-old patient admitted for management of cholestatic jaundice that had appeared for 4 months, leading to the realization of radiological investigations that confirmed the diagnosis of aneurism of the hepatic artery compressing the bile duct main.

The patient was operated on, the procedure consisted of a flattening of the aneurysm with arterial reconstruction by saphenous graft right. The postoperative course was simple. With regression of biological cholestasis, and CT angiography showed good graft patency.

The aneurisms of the hepatic artery, in spite of their low incidence, represent pathology of considerable interest especially with regard to the clinical picture, the diagnostic means and the therapeutic management.

Keywords: Hepatic artery aneurysm; Obstructive jaundice; Mesenteric angiography

Introduction

The Hepatic Artery Aneurysm (HAA) is a rare pathology, its incidence is estimated at 0.25% and thus occupies the second rank in splanchnic aneurysms after that of the splenic artery, in 80% of cases it sits in extrahepatic.

He is often asymptomatic with a high risk of rupture (whose prognosis is fatal), his diagnosis is difficult hence the interest of modern imaging means.

We report the case of a patient admitted for management of cholestatic jaundice secondary to aneurism of the hepatic artery compressing the main bile duct.

Case Report

A 54-year-old patient, with a history of pulmonary tuberculosis treated for 9 months in 1975, bladder stones surgically treated in 2006, dilatation of a stenosis of the urethra in 2009, was referred for a progressive jaundice appeared for 4 months with discolored stools, pruritus and weight loss of 11 kg, without fever or gastrointestinal hemorrhage leading to the realization of a first abdominal CT suggestive of compression of the bile duct by a cystic tumor of the head of the pancreas of 8 cm. Endoscopic ultrasound finds a fluid mass vascular thick wall compatible with aneurysm. Finally, a CT

angiography confirmed the diagnosis of aneurysm of the hepatic artery compressing the main bile duct (VBP) (Figure 1).



Figure 1: Image of the angioscanner that showed the hepatic artery aneurysm compressing the main bile duct.

Lab test have shown

Hb=12.1 g/dL; Blood platelet=391000/mm³; WBC=58200; TP=77%

BT=30 mg/L; BD=21 mg/L; GGT=223U/L

ASAT=53 IU/L; ALAT=78 UI/L

Urea=0.34 g/L; Creatinine=7.7 mg/L

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Glycemia=0.82 g/L; CRP=3.50 mg/L; Albumin=30.13 g/L

Surgical exploration by laparotomy revealed an extensive hepatic artery aneurysm of the celiac trunk at the right branch-left branch bifurcation with an abnormality of the hepatic artery pathway which is retroportal. The latter was compressing the VBP. The intervention consisted of a flattening of the aneurysm which is partially thrombosed (atheroma) with arterial reconstruction by saphenous graft inserted between the splenic artery and the hepatic arterial bifurcation, then a cholecystectomy and cholangiography which showed a good passage of contrast in the duodenum, no biliary gesture was made (Figure 2). The postoperative course was simple. The regression of biological cholestasis was noted, and the CT angiography showed good graft patency.



Figure 2: Perioperative drunkenness of the aneurysm; and the final appearance of saphenous graft reconstruction (arrow).

Discussion

HAA is a rare entity, first reported by Wilson in 1819 [1]. HAA, it is twice as common in humans, and its incidence is maximum between 40 and 60 years [2,3]; he is of extrahepatic seat in 80% whereas he sits in the intrahepatic vessels in 20% and he touches the common hepatic artery (63%) followed by the right hepatic artery (28%) and left (5%) [2-4].

Atherosclerosis and fibromuscular dysplasia represent the main etiologies, secondarily vasculitis, autoimmune diseases and infections that are rare [5,6] (Table 1).

Pseudoaneurysms account for 22% of hepatic aneurysms and are most often intrahepatic [7]. They may appear after closed trauma or a penetrating wound of the abdomen, but they are mainly of iatrogenic origin, after surgery or invasive radiological investigation hepatobiliary-pancreatic [8].

S.N.	Etiologies of hepatic artery aneurysms
1	Atherosclerosis
2	Degeneration of the media
3	Congenital fibrodysplasia
4	Abdominal and surgical trauma
5	Tuberculosis
6	Systemic lupus erythematosus
7	Wegener granulomatosis
8	Intra-arterial chemotherapy infusion
9	Biliary disorders and their percutaneous and endoscopic treatment

10	Pancreatitis Abscess of the liver
11	Orthotopic liver transplantation
12	Terminal hepatopathies
13	Marfan Syndrome
14	Ehlers-Danlos syndrome
15	Hereditary hemorrhagic telangiectasia
16	Disease of Rendu-Osler
17	Kawasaki disease
18	Still's disease
19	Hashimoto Thyroiditis
20	Peripheral arthritis
21	Gestation
22	Portal hypertension

Table 1: Etiologies of hepatic artery aneurysms in order of decreasing frequency [2,9-11].

The aneurysm is often asymptomatic, abdominal pain is the most common clinical sign (right or epigastric hypochondrium), obstructive jaundice is the second sign (as the case of our patient), or by complications such as the condition shock (secondary to rupture in the peritoneum, bile ducts or digestive tract), gastrointestinal bleeding or hemobilia [12,13]. The risk of rupture is very variable in the literature, ranging from 20% to 80% [14]. There is currently no data correlating the size of HAA with the risk of rupture [3]. Mortality associated with rupture ranges from 21% to 35% [15]. The other reported complications are cracking and dissection. Pseudoaneurysms are often

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associated with intrahepatic abscess due to associated biliary trauma, manifested by pain and fever [16].

Jaundice is secondary to extrinsic obstruction of VBP by aneurysm, however HAA is a rare cause of jaundice, the most common causes are cholelith lithiasis, cholangitis, head cancer pancreas and cholagiocarcinoma [4].

Given its poor symptomatology, the diagnosis is based on modern imaging means, the Doppler ultrasound which is an anodyne and sensitive examination shows a well-defined fluid structure in the region of the head of the pancreas with an intraluminal arterial flow [17]. Contrast-enhanced Computed Tomography (CT) is diagnostic and specifies topography, vascular origin, limits, and anatomical relationships, as well as recognition of associated lesions. The coeliomenteric arteriography is the reference examination, it confirms the diagnosis, the exact seat and the impact on the vascularization of the liver, it also allows to search the anatomical variations of the hepatic arterial vascularization (capital concept before any therapeutic gesture), to evaluate alternate routes and to detect other aneurysmal locations. At present, non-invasive angio-MRI is used to dispense arteriography with the same morphological data [5,18,19].

The risk of rupture imposes a therapeutic management.

Possible treatments for HAA include endovascular methods of embolization, stent placement, and surgery either by surgical reconstructions or ligature. The choice of the therapeutic modality depends on the clinical presentation, the morphological characteristics (sacciform or fusiform aneurysm with or without collar) of the aneurysm, the anatomical location, the risk factors and the general state of the patient.

Arterial embolization which consists of filling the aneurysm with coils after neck catheterization in case of sacciform aneurysm or exclusion of the affected artery upstream and often downstream in case of fusiform or collet aneurysm wide can be done if the size of the aneurysm is of medium size, in the absence of infection or a high surgical risk or for a hemorrhagic emergency before a surgical procedure. Stent placement is more difficult because the diameter is smaller with the risk of intra-stent thrombosis.

Therapeutic abstention and regular doppler ultrasound monitoring may be proposed in intra-parenchymal aneurysms of less than 1.5 cm. Some spontaneous resolutions have been reported [9].

Surgery is indicated if the diameter is greater than 2 cm, or if the aneurysm is complicated with rupture, dissection or cracking or a rapid increase in the diameter of the aneurysm, symptoms related to the aneurysm and in women in pregnancy age; treatment is also recommended for the following asymptomatic lesions: aneurysms of the hepatic artery in patients with periarteritis nodosa or fibromuscular dysplasia, intrahepatic pseudoaneurysms [5,20-25].

The techniques include: ligation of the hepatic artery, exclusion or excision of the aneurysm or with reconstructive methods in the case of clean HAA, common HAA encompassing the bifurcation or if clamping causes liver damage, techniques that can be use are resection and flattening of the aneurysm, direct end-to-end anastomosis, or by interposition of a graft that may be an autologous vein graft, allograft, autograft arterial or prosthetic graft [9,26-31].

It is recommended not to ligation the hepatic artery in the presence of cirrhosis and other serious liver diseases. After interruption of the clean hepatic artery or right hepatic artery, necrosis of the gallbladder may occur. It is therefore necessary to simultaneously perform a cholecystectomy [32].

In our case we made a flat aneurysm with arterial reconstruction by a venous graft by the right saphenous vein interposed between the splenic artery and the hepatic arterial bifurcation.

The surgical decision was unavoidable given the size of the aneurism, the seat (aneurysm that extends from the celiac trunk to the bifurcation), the presence of thrombosis, and since the ligation of the common core will cause risks of hepatic hypoxia, and that stenting can be complicated by intraspinal acute thrombosis and focal dissection, embolization requires the exclusion of all the outflow and therefore a sacrifice of the artery concerned.

Conclusion

A variety of options is available for the treatment of HAA. The treatment strategy for HAA must be determined individually in each case; thanks to the development of non-invasive imaging means that allow earlier diagnosis and a better therapeutic approach, the prognosis has become less bleak. Combined surgical and endovascular treatment is likely to reduce the risk of ischemia and liver morbidity.

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None

Conflicts of Interest

The authors have no conflicts of interest to declare.

Informed Consent

Written informed consent was obtained from the patient for publication of this Case Report and any accompanying images.

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