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Radiology Department

Digestive Haemorrhage Revealing a Pseudoaneurysm of the Right Hepatic Artery: A Case Report

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Abstract

Introduction: Hepatic artery aneurysms (HAA) are rare and account for 20% of all visceral artery aneurysms. Hepatic artery aneurysms are often discovered at autopsy, but their rupture and bleeding cause significant morbidity and may manifest as haemobilia. Here we report a case of idiopathic right hepatic artery pseudoaneurysm revealed by upper GI bleeding. **Presentation:** A 72 year old Moroccan woman presented to the emergency department with an upper GI bleed with haematemesis and melaena without any evidence of jaundice. She was admitted with pre-shock haemorrhage and the initial management required early resuscitation measures he turned out to have a pseudoaneurysm of one of the branches of the right subhepatic artery. The aetiology of the pseudoaneurysm was atherosclerosis. The patient underwent radioembolization, the aftermath of which was uncomplicated. **Clinical Discussion:** Hepatic artery aneurysms (HAA) is a rare cause of gastrointestinal haemorrhage, the management of which requires resuscitative measures Atherosclerosis is the main cause of formation, but it may be associated with connective tissue disorders and arteritis. Most Hepatic artery aneurysms (AAA) are asymptomatic. Aneurysms can be managed by surgical or endovascular interventions. **Conclusion:** Digestive haemorrhage may reveal a pseudoaneurysm of the right hepatic artery by rupture or fissure in the biliary system. Knowledge on this subject is important for emergency doctors, resuscitators, gastroenterologists, vascular surgeons and hepatobiliary surgeons for the diagnosis and early intervention of this rare entity.

Keywords: Digestive arteries, aneurysm - digestive haemorrhage.

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INTRODUCTION

Visceral artery aneurysms (VAA) are rare and are defined as aneurysms involving the celiac artery, superior mesenteric artery (SMA), inferior mesenteric artery and/or their branches. The estimated prevalence is between 0.1 and 2%. Among VAAs, hepatic artery aneurysms (HAAs) and pseudoaneurysms account for about 20%, while splenic artery aneurysms account for about 60%. Rupture and haemorrhage of AAVs cause significant morbidity and mortality, but most aneurysms are discovered incidentally on abdominal imaging for other reasons. The definitive cause of AAH is unclear. In contrast to the beginning of the century, mycotic aneurysms are rarely seen nowadays due to early diagnosis and antibiotic treatment of infections including infective endocarditis. Iatrogenic and traumatic causes, atherosclerosis, vasculitis, as sequelae of peri-inflammation such as cholecystitis and

pancreatitis, which are the most reported causes in recent articles [3-5].

Here we report a case of idiopathic right hepatic artery pseudoaneurysm revealed by upper GI bleeding.

OBSERVATION

A 72-year-old woman was admitted to the emergency department of the Mohammed VI University Hospital in Marrakech, Morocco, with an upper gastrointestinal haemorrhage consisting of melena and moderate haematemesis, which had been evolving for seven days. Her medical and surgical history included a cholecystectomy 3 years ago. On examination, the patient was conscious with GCS 15/15, mucocutaneous pallor without jaundice, tachycardia at 110 beats/minute, blood pressure 90/60mmHg with MAP 62

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bpm, polypneic at 28 cycles/minute, with saturation 99% on room air.

The cardiopulmonary examination was unremarkable, the abdominal examination showed a soft abdomen, no tenderness or tenderness to palpation. A median laparotomy scar was noted; the rectal examination revealed a moderate amount of melena. Biological work-up revealed haemoglobin at 6.7g/dl with haematocrit at 28.7, white blood cells and platelet count was normal. His prothrombin level was 100%, liver and kidney function tests were normal.

Resuscitation measures were undertaken with vascular filling with 20cc/kg/h of saline, transfusion of two packed red blood cells, fresh frozen plasma, and administration of 1g tranexamic acid IV.

After initial resuscitation, the patient improved clinically with a control haemoglobin level of 9.3 g/dl. She underwent upper GI endoscopy which revealed a normal looking duodenal papilla with active bleeding persisting after lavage.



Figure 1: FOGD showing active bleeding from the duodenal papilla

An abdominal angioscan was performed, showing an intensely enhanced spontaneously iso-dense saccular formation of the vesicular bed similar to the aorta measuring 13x8x12mm, On further ultrasound examination, a mixed anechoic saccular formation on colour Doppler was seen, giving a Ying-Yang appearance in relation to a pseudo aneurysm of one of the branches of the hepatic artery. The intra- and extra-hepatic bile ducts were undilated, no intra-peritoneal effusion.

A multidisciplinary discussion was conducted, including vascular surgeons, hepatobiliary surgeons, interventional radiologists. The possibilities of surgical correction, coil embolisation, and angioembolisation were discussed.

Conservative management was indicated. A catheterization of the right hepatic artery showed a pseudo aneurysm fed by the right hepatic and two collaterals, the procedure was completed by the placement of two coils of 3 and 4mm.



Figure 2: Angiography image showing coil embolisation (blue arrow) in one of the branches of the right hepatic artery



Figure 3: Abdominal and pelvic CT scan injected in coronal section: before treatment (a) shows an image of pseudo aneurysm (yellow arrow) of one of the branches of the right hepatic artery. After agiographic treatment (b) disappearance of the aneurysm with evidence of the coil (blue arrow) in the right branch of the hepatic artery

The patient was transferred to the department of visceral and digestive surgery. The post-procedure follow-up was straightforward with a satisfactory fifthday follow-up angioscan.

DISCUSSION

AAH is potentially fatal because the frequency of rupture is higher in AAH than in other AAVs. It is reported to be 20-30%, and the mortality rate after rupture is relatively high, at 35%. AHA can be intrahepatic (20%) or extrahepatic (80%). About 60% of extrahepatic artery aneurysms are common hepatic artery aneurysms. AHAs occur more frequently in men than in women [7]. True aneurysms are much more common than pseudoaneurysms, but in the case of AAH, the two are equal. About half of pseudoaneurysms are located in the liver parenchyma, reflecting the increasing incidence of iatrogenic aetiology.

Atherosclerosis is the main cause of AHA formation, accounting for more than half. Compared to other AAVs, the higher incidence of hepatic artery pseudoaneurysms is due to various complications of invasive and surgical transhepatic procedures. These procedures are the second most common etiology. Less common causes are fibromuscular dysplasia, segmental arterial mediolysis (SAM), polyarteritis nodosa, infection, trauma and biliary disease. Rarely, AAH has been associated with disorders such as Takayasu's arteritis, Kawasaki disease, von Recklinghausen's neurofibromatosis and Wegener's granulomatosis. Congenital causes of AAH include Marfan syndrome, Ehlers-Danlos syndrome, Osler-Weber-Rendu syndrome and hereditary haemorrhagic telangiectasia. Only a few patients are diagnosed with AAV due to infectious causes [8-10].

Most AAH are asymptomatic and are discovered incidentally during screening for other diseases. Large aneurysms can rarely cause mass effects and manifest as obstructive jaundice. If the rupture occurs in the biliary system, patients may usually present with bloody vomit and melena or rectal bleeding and anaemia. Contrast CT angiography, magnetic resonance angiography and abdominal ultrasound help to identify the morphology and characteristics of the aneurysm, which facilitates definitive diagnosis [1].

The relationship between aneurysm size and risk of rupture has been difficult to establish due to the rarity of AAH rupture. However, the risk of rupture is higher in non-atherosclerotic aneurysms [9]. Treatment is generally indicated for all symptomatic AHCs, pseudoaneurysms and aneurysms larger than 20 mm in diameter. Multiple AAHs and non-atherosclerotic aneurysms should also be treated, regardless of size, as these AAHs have a high risk of rupture [11].

As with other AAVs, treatment options for AAHs depend on the anatomy, morphology and location of the aneurysm, as well as the patient's clinical condition and comorbidities. Treatment options may be surgical, such as aneurysmctomy, ligation and bypass with a venous graft or prosthetic vascular graft, laparoscopic surgery (mainly ligation), EVT (embolisation and endograft), treatment of hypertension, or a combination of treatments. Although there are no randomised control trials that have been able to identify EVT as superior to open repair, given the surgical trauma associated with open procedures and the mortality rate.

AHA is a cause of HD that requires multidisciplinary collaboration involving emergency physicians, gastroenterologists, radiologists and surgeons.

It consists of a symptomatic treatment whose essential goal is the restoration of blood volume and a haemostatic treatment which depends on the lesion causing the haemorrhage. The first phase of therapeutic management is to ensure or restore a satisfactory haemodynamic state. The sudden loss of blood is responsible for a drop in tissue oxygen perfusion which must be corrected without delay.

The most urgent action is to place a venous line to allow vascular filling and to restore a systolic blood pressure above 90 mm Hg.

Vascular filling depends on the extent of the haemorrhage. In most cases, crystalloids are used. The use of colloids is only justified in cases of heavy bleeding, pending transfusion.

Oxygen therapy via the nasal route favours tissue oxygenation and is undertaken rapidly in the elderly, in cases of severe haemorrhage or in coronary patients.

Hospitalization in intensive care is recommended in case of decompensated associated disease, active bleeding, high age, with endoscopic signs of recent bleeding.

In our patient, endovascular embolisation was considered to be the best treatment option for this pseudoaneurysm of the right hepatic artery, after the resuscitation measures undertaken had been effective.

CONCLUSION

Digestive haemorrhage may reveal a pseudoaneurysm of the right hepatic artery by rupture or fissure in the biliary system. Life-threatening haemobilia is a notable complication of this rare entity. Early intervention is necessary to prevent mortality. Incidents of hepatic artery aneurysms and pseudoaneurysms due to percutaneous transhepatic procedures and minimally invasive hepatobiliary surgery are increasing.

Knowledge on this topic is important for emergency doctors, resuscitators, gastroenterologists, vascular surgeons and hepatobiliary surgeons for diagnosis and early intervention of this rare entity.

Declaration of interest: The authors declare that they have no conflicts of interest in relation to this article.

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