Ruptured aneurysm of replaced left hepatic artery as a cause of haemorrhagic shock: a challenge of diagnosis and treatment

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Abstract

An isolated, spontaneous, ruptured aneurysm of the replaced left hepatic artery (LHA) arising from the left gastric artery, in a 72-year-old female, leading to haemorrhagic shock treated by surgical ligation is reported. To our best knowledge, this is the second case report of a ruptured hepatic artery aneurysm in this location. A thorough knowledge of hepatic arterial anatomy and variations, and prompt diagnosis and urgent surgical intervention are necessary in such a potentially lethal condition.

Keywords: Hepatic artery aneurysm • Computed tomography • Angiography • Haemorrhagic shock

INTRODUCTION

An isolated, spontaneous, ruptured aneurysm of the replaced left hepatic artery (LHA) arising from the left gastric artery treated by surgical ligation is reported. To our best knowledge, this is the second case report of a ruptured replaced LHA aneurysm [1, 2]. A thorough knowledge of hepatic arterial anatomy and variations, prompt diagnosis and urgent surgical intervention are necessary in such a potentially lethal condition.

CASE REPORT

A 72-year-old female patient presented to the emergency room with severe epigastric pain and subsequent haemorrhagic shock with an haemoglobin and haematocrit level of 6.49 g/dl and 18.3%, respectively. She had been discharged from the hospital the same day after undergoing a left total knee prosthesis 4 days ago. She had hypertension under drug control, and was under low molecular weight heparin treatment during her hospital stay. She had no previous abdominal surgery. Moderated tenderness in the epigastrium at presentation generalized to all four quadrants with rebound tenderness and severe distension. No gastrointestinal bleeding was demonstrated. Abdominal ultrasound demonstrated intra-abdominal fluid, which was bloody on abdominal tap. Abdominal computed tomography angiogram (CTA) revealed intra-abdominal bleeding from the LHA (Fig. 1). The patient was operated on by the general surgery and transplantation team together with a vascular surgeon. Urgent laparotomy revealed ~1000 ml of blood in the peritoneal cavity. Upon exploration of a haematoma noted under the hepatogastric ligament, bleeding from a ruptured aneurysmatic dilatation of the replaced LHA branching off the left gastric artery was discovered; the rupture was just at the junction of the left gastric artery and LHA (Fig. 2). There was fresh blood clot in the artery. It was not suitable for excision and anastomosis because of intimal degeneration comprising the whole extra-hepatic portion of the artery. The artery was ligated since sufficient back bleeding was seen upon perfusion. Vascular wall injury with total loss of endothelial cells and irregularity of the internal elastic membrane were found at the histopathological examination of the artery. Her postoperative course was uneventful. She is doing well with no symptoms, no tumour formation or new aneurysm described on CTA after 14 months.

DISCUSSION

Hepatic artery aneurysm (HAA), a very rare disease with an estimated incidence of 0.002–0.4%, is most commonly seen in the fifth to sixth decade of life with a male/female ratio of 3/2 [2]. HAA, recently, has supplanted splenic artery aneurysm as the most frequently reported visceral artery aneurysm (30% and more). The most common site of the HAAs has also changed in the last decade; 40% are seen in the common or the proper hepatic artery and half in the right hepatic artery, while they are infrequent in the left hepatic and more peripheral branches [3].

A true aneurysm is a permanent, localized dilatation (>1.5 times expected diameter) of an artery, that involves all three layers of the vessel. The mean diameter of the LHA in the general population is reported to be around 0.3 cm; variant arteries are even smaller [4, 5]. The incidence of a replaced or accessory LHA off the left gastric artery (Michels type II) is reported to be up to 34% [6]. There is only one case report of a ruptured HAA in this location in the literature, which was also diagnosed and treated upon rupture [1]. None of the factors
blamed in the aetiology of HAAs such as trauma, infection, arteriosclerosis and medial degeneration was present in our case [3, 7]. Most of the HAAs are asymptomatic; large aneurysms may be associated with a pulsatile mass or an abdominal bruit [1–3]. The rate of rupture of HAAs is reported to be up to 20–30%. Rupture into the hepatobiliary tree or gastrointestinal system is a little more common than rupture into the peritoneal cavity. Gastrointestinal bleeding and obstructive jaundice, and severe abdominal pain together with haemorrhagic shock are seen, accordingly [2, 3, 7].

Abdominal CTA or magnetic resonance angiography yields the diagnosis of HAAs in most cases; however, selective catheter angiography is the gold standard for diagnosis as well as for planning and performing therapeutic interventions [2, 7]. Although some authors limit the indications of treatment of HAAs to symptomatic cases and aneurysms in pregnant patients, aneurysms greater than 2 cm in diameter or aneurysms with demonstrated growth warrant treatment; another highly accepted approach is to treat even the asymptomatic extra-hepatic aneurysms in good-risk patients once discovered, since their propensity to rupture is high and there are no prodromal symptoms before rupture, and the mortality with ruptured HAAs is still around 35% [3, 4, 8].

Some ruptured HAAs, especially in high-risk patients and the intra-hepatic ones, can be treated by angiographic interventions, such as embolization and stent application in haemodynamically stable cases; nevertheless, follow-up is essential in these cases since re-interventions might be necessary [7].

Surgical treatment of ruptured HAAs such as ligation and aneurysm exclusion, excision or revascularization can be considered when (i) the patient is in shock or in an unstable condition, (ii) the aneurysm is extra-hepatic, (iii) endovascular intervention fails, and (iv) the aneurysm or rupture recurs in spite of multiple interventions [3, 7, 9]. Aneurysms in common hepatic artery may be ligated without revascularization in most cases; however, haemorrhagic shock as a result of a ruptured HAA might increase the likelihood of liver necrosis upon ligation of the aneurysm without revascularization. Proper hepatic and right and left hepatic arteries generally require revascularization [3, 9]. Although right and left hepatic arteries are shown to be end-arteries in cadavers, it has been known for a long time that intra-hepatic anastomoses exist in vivo [10]. This, together with the observation of good liver perfusion upon ligation, was the basis for ligation of the aneurysmatic replaced LHA without revascularization in this case.

Urgent or emergent surgery should be the first choice in patients with a ruptured HAA with active intra-abdominal bleeding causing haemorrhagic shock. Management of an HAA in a bloody and obscured field can be extremely hard; therefore, a quick preoperative CT angiography, if the patient's condition permits, should be performed to orient the surgeon directly to the lesion. Even an aneurysm in a replaced LHA can be ligated without revascularization after a test occlusion demonstrating good liver perfusion.

Conflict of interest: none declared.

REFERENCES


