Received: January 15, 2024
Accepted: April 8, 2024

Acad Med J 2024;4(2):150-158
UDC: 616.136.41-007.43-089.844
DOI: https://www.doi.org/10.53582/AMJ2442150cr
Case report

RUPTURED HEPATIC ARTERY ANEURYSM SUCCESSFULLY TREATED WITH DUAL-LAYER STENT ENDOVASCULAR TREATMENT: CASE REPORT AND LITERATURE REVIEW

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Abstract

Spontaneous rupture of a hepatic artery aneurysm into the peritoneal cavity is a serious life-threatening complication related to hemodynamic instability and high mortality rate. We present a case of a hepatic artery aneurysm complicated with intraperitoneal bleeding and successfully treated with endovascular stent insertion. A 78-year-old male patient was admitted to a tertiary care center due to an abdominal pain and a large amount of free fluid in the peritoneal cavity. The diagnostic paracentesis confirmed hemoperitoneum. The CT scan revealed an aneurysm of the common hepatic artery that was successfully treated with an insertion of a dual-layered carotid stent. Endovascular stent insertion of a ruptured aneurysm of the common hepatic artery is an effective therapeutic procedure that can provide a definitive curative treatment in some patients.

Keywords: hepatic artery aneurysm, hemoperitoneum, endovascular stent

Introduction

A hepatic artery aneurysm (HAA) is an uncommon vascular abnormality characterized by an abnormal dilatation of the hepatic artery. It is a rare but important entity that was initially described by Wilson in 1809. Seventy-five percent of the HAAs are diagnosed incidentally and most of them remain asymptomatic[1]. On the contrary, pseudoaneurysms and the symptomatic HAA can cause a serious, life-threatening condition related to significant morbidity and mortality. Due to its close relationship with the biliary

tree, rupture of the aneurysm into the biliary tree is the most common presentation of symptomatic HAAs. Hence, the symptomatic HAAs are usually presented with the classic Quincke's triad consisting of gastrointestinal bleeding due to haemobilia, jaundice due to biliary obstruction, and right upper quadrant pain[2,3-6]. However, the classic Quincke's triad is present in only 25%-30% of patients[2]. Other than rupture into the biliary tract, less frequently, the HAAs can also rupture into the peritoneal cavity presenting signs and symptoms of intraperitoneal haemorrhage and hemodynamic instability[7,8-11]. However, due to its close relationship with the biliary ducts, rupture into the biliary tree is more common than in the intraperitoneal cavity[12]. Some patients complain of abdominal pain and a pulsatile mass in the right upper quadrant, and rarely, some patients with HAAs may present only with gastrointestinal bleeding due to arterio-biliary fistula[13,14]. The spontaneous rupture of HAAs into the peritoneal cavity is probably the most serious life-threatening HAA-related complication. It occurs in 25% of the cases and is associated with 70%-100% mortality [15,16]. It occurs more frequently in patients with non-atherosclerotic HAAs, in patients with multiple aneurysms, and aneurysms larger than 2 cm[13,17,18].

Regarding the management of patients with HAAs, the current Society for Vascular Surgeons guidelines recommend performing an intervention in the following settings: (1) all hepatic artery pseudoaneurysms; (2) all symptomatic HAAs regardless of size; (3) asymptomatic patients without a significant comorbidity in HAA >2 cm, if the aneurysms enlarge by 0.5 cm/year; (4) asymptomatic patients with significant comorbidities and an aneurysm larger than 5.0 cm[16]. Some authors also recommend the treatment of multiple aneurysms and those associated with polyarteritis nodosa[19]. There are two main treatment options available: open surgical treatment and endovascular repair. The decision between both options depends on the size, location, morphology of the aneurysm, adequacy of visceral collateral flow, feasibility related to different anatomical variation and the clinical status of the patient[1,20]. Although the open surgical treatment was the modality of choice in the past, nowadays, the endovascular approach is often chosen as a first safer alternative to surgery, or as a bridge to postponed surgical treatment[21].

We present a case of 78-year-old previously healthy male patient who presented with an abdominal pain and significant intraperitoneal bleeding due to a ruptured common hepatic artery aneurysm, successfully treated with endovascular stent insertion.

Case report

A 78-year-old male patient consulted the emergency department of a secondary care hospital complaining on a diffuse abdominal pain. The patient had stabile vital signs (BP 110/70 mm Hg, HR 90/min), he was not febrile, he did not complain on any other symptoms and he did not report any recent trauma. There were no obvious signs of bleeding; the complete blood count showed significant leucocytosis (Hgb 164 g/L, RBC 6.85 x10^{12}, HCT 0.52, WBC 36.3 x10^{9}, PLT 326 x10^{9}), and the results from the biochemical blood analysis and the haemostasis were unremarkable (total protein 65 g/L, albumin 39 g/L, CRP 24.1 mg/L, AP 107, alpha amylase 105 U/L, ALT 22 U/L, AST 23 U/L, LDH 264 U/L, Na 138 mmol/L, K 5.34 mmol/L, Ca 2.58 mmol/L, total bilirubin 17 µmol/L, direct bilirubin 6.7 µmol/L, urea 12.5 mmol/L, creatinine 182 µmol/L, glucose 5.8 mmol/L, PT 11.3 sec, INR 1, aPTT 36.5, TT 19.5, D-dimer concentration 1054 ng/mL). Later, a CT scan was performed that showed a large amount of free fluid in the peritoneal cavity with a density of 50 HU, initially interpreted as pyoperitoneum. Because of the remarkable CT findings and the unclear clinical presentation, the patient was referred to our tertiary care gastroenterology center for further investigation. On physical examination the findings were unremarkable; the abdomen was soft and palpable, with slight diffuse tenderness, without rebound, guarding, palpable

mass, or decreased bowel sounds. We performed an abdominal ultrasound examination which revealed a normal appearance of the parenchymal abdominal organs, unusual oval formation between the duodenum and pancreatic head, a significant amount of anechoic free fluid in the upper and middle sections of the abdominal cavity, and remarkable hyperechoic, almost organized septate formation in the lower abdomen resembling a partially organized hematoma. Considering the ultrasound findings, a diagnostic abdominal paracentesis on two different spots on both sides of the abdomen was performed revealing hemoperitoneum. Afterwards, the patient was immediately transferred to the University Clinic for Digestive Surgery. Within the following hours, the patient was closely clinically and hemodynamically monitored. The next day he remained stable, with stabile vital parameters, with only slight reduction in the blood count and significant PLT count elevation (Hgb 152 g/L, RBC 6.11 $x10^{12}$, HCT 0.47, WBC 27.4 $x10^{9}$, PLT 1235 $x10^{9}$). The biochemical blood analyses were again unremarkable (total protein 61 g/L, albumin 35 g/L, CRP 24.5 mg/L, AP 105, alpha-amylase 74 U/L, ALT 19 U/L, AST 23 U/L, LDH 304 U/L, Na 136 mmol/L, K 5.35 mmol/L, Ca 2.38 mmol/L, total bilirubin 15 µmol/L, direct bilirubin 4.6 µmol/L, urea 12.3 mmol/L, creatinine 152 µmol/L, glucose 3.4 mmol/L). Two days later, a control CT scan was performed that revealed a demarcated dense liquid collection with a density of 62 HU confirming an organized haemorrhagic collection (Figure 1) and a saccular aneurysm of the common hepatic artery on the axial coronary plane of the aortic phase (Figure 2). An indication for angiography was made. After the puncture of the left brachial artery, catheterization of the celiac trunk was performed and the angiography showed a wide neck aneurysm of the common hepatic artery without active contrast extravasation during the examination (Figure 3). The initial strategy was to perform a stent-assisted coiling of the aneurysm with balloon expanding stent and jailing of the microcatheter, but unfortunately, after placement, the stent did not completely cover the whole neck of the aneurysm. Finally, it was decided to place a dual-layer carotid stent across the neck of the aneurysm to act as a flow diverter. The control angiogram showed a flow stagnation in the aneurysm sac (Figure 3). During the intervention, the patient was systemically heparinized with 50,000 IE of heparin. There were no complications during and after the intervention. Two days later, an ultrasound follow-up was performed confirming that the aneurysm was thrombosed and excluded from the circulation. Within the next days, the patient was clinically and hemodynamically stable, and he was discharged a few days later.

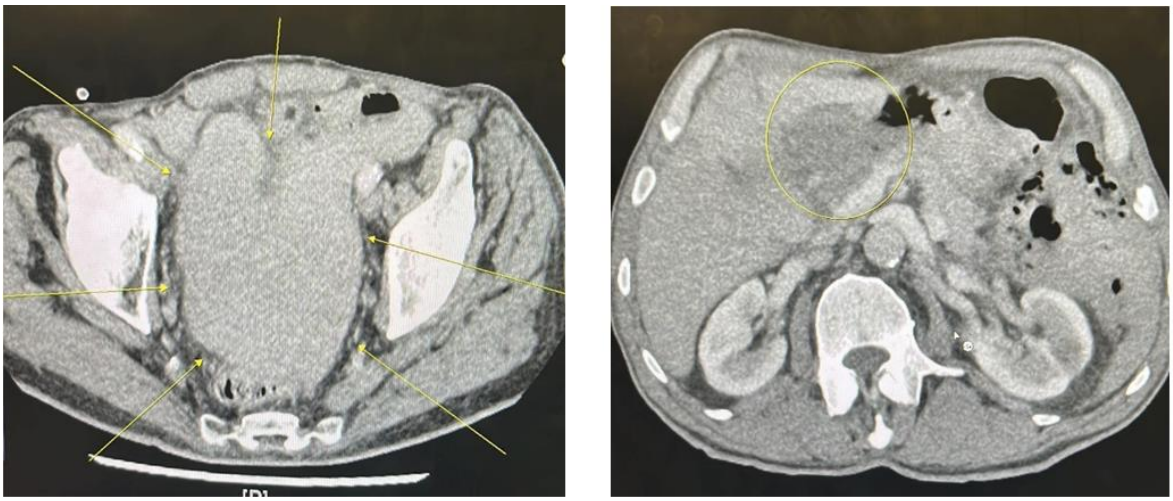

Fig. 1. CT scan revealing a clearly demarcated dense liquid collection with a density of 62 HU confirming an organised haemorrhage collection

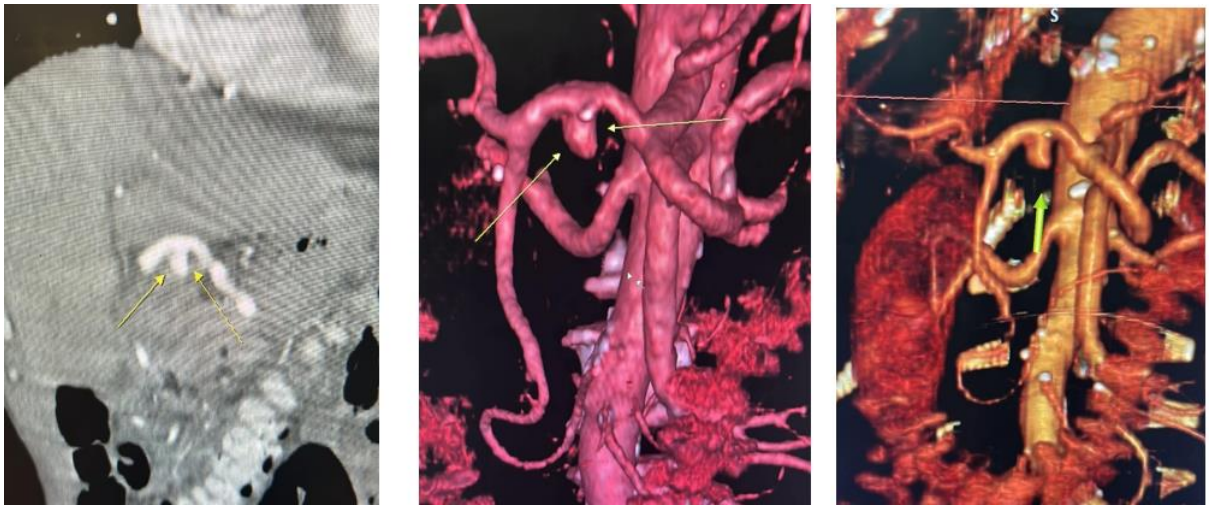

Fig. 2. Saccular aneurysm of the common hepatic artery
(axial coronary plane of the aortic phase and 3D reconstruction)

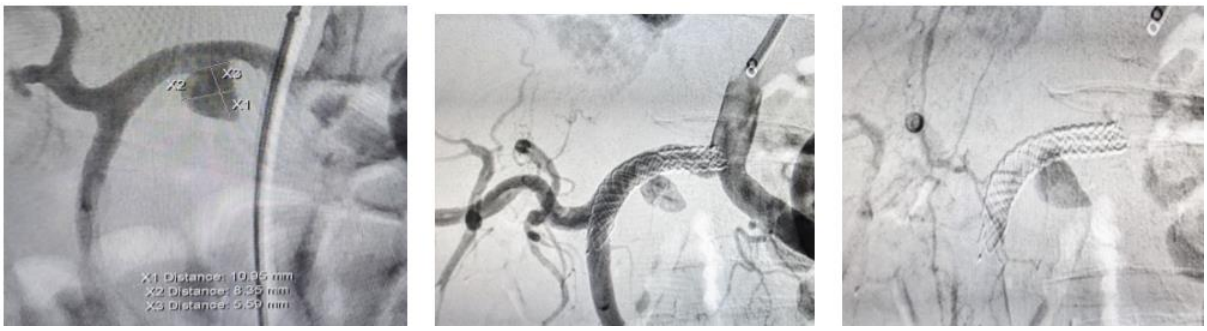

Fig. 3. Angiography showing an aneurysm of the common hepatic artery
(10.95 mm x 8.35 mm) with wide neck (5.59 mm) followed by an insertion
of a dual layered carotid stent into the common hepatic artery

Discussion

HAA is a rare entity that in most patients remains clinically asymptomatic. However, in a significant number of patients, a rupture can be the initial clinical manifestation that is associated with a mortality rate estimated at anywhere between 20% and 100%[22]. Also, depending on the localization, size and type of the complication, there is a large variety in the clinical presentation in patients with symptomatic HAAs. Our case report describes a ruptured aneurysm of the common hepatic artery with an intraperitoneal bleeding in a haemodynamically stable patient without previous vascular or gastrointestinal pathology, successfully treated with endovascular stent insertion. Albeit, there are similar cases previously reported, still, there are few specific aspects in our patient that should be discussed in more details.

Hepatic artery aneurysms (HAAs) account for about 20% of splanchnic artery aneurysms[1,15,23], and after the splenic artery aneurysm, HAAs are the second most frequent visceral artery aneurysms [24] with an incidence ranging from 0.002% to 0.010%[1,15,19]. The HAAs can be true aneurysms, while a significant proportion of them are actually pseudo/mycotic aneurysms, mainly caused by endocarditis or cholecystitis[2]. HAAs are most frequently located in the extrahepatic segment, mainly in the common hepatic artery (60%), followed by the right hepatic artery (30%) and left hepatic artery (5%)[13]. The HAAs are most prevalent in the sixth decade of life, and they are more frequently reported in males,

with 3:2 male predominance[16,25]. The medical conditions that are most commonly related to the presence of HAAs are atherosclerosis, dyslipidemia, hypertension, diabetes, vasculitis (polyarteritis nodosa), connective tissue diseases (systemic lupus erythematosus), fibromuscular dysplasia, trauma, smoking, surrounding inflammation (cholecystitis or pancreatitis) or previous transplantation[1,7,15,21].

Vascular pathology underlying gastrointestinal presentation is not frequently diagnosed; hence, the diagnosis of symptomatic HAAs can be rather difficult and often delayed. Occasionally, HAAs can be incidentally diagnosed on abdominal films as rim calcification[23,26]. Still, abdominal ultrasound is usually the initial diagnostic tool that may locate larger aneurysms and demonstrate blood flow with turbulent arterial waveform and high peak velocity, biliary obstruction, surrounding hematoma, or fluid in the peritoneal cavity[27,28]. However, Doppler ultrasound has a low sensitivity to detect small aneurysms[29]. CT scan is usually more useful in detecting and defining the nature and origin of the lesion, the adjacent structures, and evidence of a rupture[30]. Still, according to the literature, in many cases, the ruptured hepatic artery aneurysm was only diagnosed on exploratory laparotomy [9]. Once hepatic aneurysms are diagnosed, performing arteriography is mandatory since it is still the gold standard diagnostic tool that provides information regarding the size, shape, and location of the aneurysm[30-32]. More importantly, it also provides important information regarding the presence and extensiveness of the collateral circulation which contributes to proper selection of the preferred treatment option[14].

At presentation, our patient was clinically and circulatory stabile and despite the active arterial bleeding and the hemoperitoneum, there was no significant reduction in the complete blood count. The patient had significant leucocytosis and thrombocytosis, and the CT scan was initially indicating pyoperitoneum. Thus, at presentation, there were not many elements that would reasonably rise the clinical suspicion for the presence of this entity. The diagnostic paracentesis proving hemoperitoneum raised the initial suspicion for vascular pathology and the CT angiography detected the aneurysm of the common hepatic artery that helped in establishing the diagnosis. Therefore, even in patients with stigmata for bleeding, hemodynamic instability, or shock, and in patients with previous medical history of arterial aneurysms and/or dissections, high clinical awareness is necessary for the diagnosis of ruptured HAA, especially if CT angiography does not explicitly show the source of bleeding[9].

The surgical and endovascular therapeutic approaches are the two recommended treatment strategies in patients with HAAs. The selected option mainly depends on the type, size, and shape of the aneurysm, but also on the locally available endovascular procedures and expertise. The surgical management options include excision and ligation of the aneurysm, aneurysmorrhaphy, reconstruction through an end-to-end or splenorenal anastomosis, arterial or venous graft interposition or bypass[7,8,13]. The choice mainly depends on the localization of the aneurysm and the surrounding collateral circulation. Hence, whenever adequate collateral vascularization cannot be demonstrated, arterial aneurysm ligation should be followed by some reconstruction technique[33,34]. Aneurysms distal to the common hepatic artery should be treated by revascularization[30-32], however, some authors report that ligation of branch aneurysms was well tolerated[31]. In cases involving the right hepatic artery, there is a possibility of gallbladder ischemia and some authors recommend a simultaneous cholecystectomy[31]. Since surgical treatment is associated with a higher morbidity compared to endovascular treatment, the recently published Society for Vascular Surgery guidelines recommend the endovascular approach [embolization techniques (coil and glue) and endovascular stenting] as the first treatment choice in HAAs[16]. In general, surgery

is the preferred treatment option for extrahepatic aneurysms, while the intrahepatic aneurysm might be treated with some of the endovascular techniques[35].

In patients with bleeding caused by a ruptured HAA, embolization procedures are the preferred treatment option. Embolization requires proper collateral circulation, patency of the portal vein, and separation between the lesion and the gastroduodenal artery[20]. By occluding the inflow and outflow of the HAA, embolization causes interrupting arterial blood flow to the liver, which can lead to liver necrosis or abscess, necrosis of the gallbladder, acute or chronic liver failure, and rarely erosion and migration of the embolic agent into the biliary tree[2,4]. Hence, in the case of embolization of large intrahepatic HAAs, a resection of the involved part of the liver to prevent necrosis is recommended[16]. Coil embolization is also an option in cases with suitable aneurysm morphology and localization and some authors report successful management of patients with HAAs[6,13,36]. In patients with uncomplicated common HAA, endovascular aneurysm repair (EVAR) via covered stents from the coeliac axis into the splenic artery with exclusion of flow into the aneurysm is one of the two main flow-preserving treatment options[8]. However, when the stent is placed in a contaminated area, it may maintain ongoing infection[21]. Also, there is a risk of ischemia, and infarction due to stent stenosis, thrombosis, distal stent migration, or endoleak[2,7,36]. Consequently, the use of covered vascular stents is probably not the best treatment option in patients with complicated HAAs, especially in patients with infection and an arterio-biliary fistula.

In the case of an aneurysm of the common hepatic artery, as in our patient, the preferred treatment option also mainly depends on the development of the collateral circulation. Since HAAs usually develop during a longer period, in most cases a well-organized collateral circulation develops[37]. Aneurysms involving a common hepatic artery may be safely either embolized or ligated in case of the presence of adequate collateral circulation from the gastroduodenal artery or right gastric artery[1,30-32]. If the collateral circulation is not sufficient, the aneurysmal artery needs to be reconstructed or partial hepatectomy might be required[7]. In patients with common HAAs, the proximity of the aneurysm to the coeliac axis excludes coil embolization as a treatment option[8]. Also, if the HAA has a wide neck, it makes this entity more challenging for most endovascular procedures, especially for coiling embolization. In our case, the angiography showed a wide neck aneurysm of the common hepatic artery and sufficient surrounding collateral circulation which was successfully covered with the dual-layered carotid stent.

In conclusion, ruptured HAA with intraperitoneal bleeding is a rare but serious life-threatening emergency that requires a high index of suspicion and clinical awareness to make a proper diagnosis. The diagnostic paracentesis could be a useful diagnostic tool confirming hemoperitoneum, and the proper utilization of the endovascular techniques could contribute to early diagnosis and offer a definitive curative treatment.

Conflict of interest statement. None declared.

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