

A Case of Ruptured Mycotic Hepatic Artery Aneurysm Successfully Treated Using Arterial Embolization

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Mycotic hepatic artery aneurysms (HAAs) have become very rare due to antibiotics. Untreated, they have a high possibility of rupture and mortality. In this paper, on the case of a 67-year-old male who had severe right-upper-quadrant abdominal pain and a history of infective endocarditis is reported. The computed tomography (CT) and arterial angiography findings led to a diagnosis of a ruptured mycotic HAA. The CT showed an HAA and the formation of an intrahepatic hematoma caused by aneurysmal rupture. The arterial angiography showed a mycotic HAA that arose from the right posterior hepatic artery. Percutaneous transcatheter arterial embolization was used to successfully treat the HAA. Since then, the patient has been doing well, without symptoms.

Key Words: Aneurysm, Embolization, Hepatic artery

INTRODUCTION

Hepatic artery aneurysm (HAA) is a rare condition that comprises 0.1% of all arterial aneurysms and 20% of all visceral aneurysms.¹ In the early 20th century, most HAAs were mycotic and associated with bacterial endocarditis.^{2,3} The incidence of mycotic HAA decreased following the introduction of effective antibiotics.^{2,4} HAA has the highest rate of rupture of visceral artery aneurysms, and thus is associated with an extremely high mortality rate.⁴ The present report describes the case of a 67-year-old man who had a ruptured mycotic HAA and a history of infective endocarditis (IE). We successfully treated him using percutaneous transcatheter arterial embolization, and he has since remained asymptomatic.

CASE

A 67-year-old male was admitted with a 5-day history of severe abdominal pain in the right upper quadrant area. He had a history of IE that was diagnosed approximately 5 months prior to the current presentation. At that time, he

was admitted to the infection department. A physical examination revealed a systolic murmur at the cardiac apex. A transesophageal echocardiogram showed multiple vegetations at the mitral valve and perforation of the anterior leaflet with very severe mitral regurgitation. Antibiotics prescribed prior to the admission resulted in negative blood cultures. He underwent an emergency mitral valve replacement. Pathology examination revealed active infective endocarditis, which satisfied the modified Duck's criteria.⁵ He was finally diagnosed with having blood culture-negative IE. He was administered intravenous penicillin and gentamicin for 2 weeks, followed by another 4 weeks of ampicillin medication. Anticoagulation therapy using warfarin was maintained due to the mechanical valve replacement. There was no other specific medical or family history.

At the current presentation, the patient had a body temperature of 36.0°C, a blood pressure of 139/89 mmHg, a pulse rate of 120 beats per minute, and a respiratory rate of 20 beats per minute. The heart sounds were regular with a good metallic click. Abdominal examination revealed normal bowel sounds with no palpable abdominal mass or organomegaly. However, there was severe tenderness to palpation over the right upper quadrant without signs of peritoneal irritation such as rebound tenderness.

Laboratory tests were undertaken. A complete blood count showed: hemoglobin 10.5 g/dL, hematocrit 33.4%, white blood

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cell count 9,300/mm³ and platelet count 260,000/mm³. Other tests showed: serum aspartate transaminase (AST) 25 IU/L, alanine transaminase (ALT) 21 IU/L, alkaline phosphatase 148 IU/L, r-glutamyl transpeptidase 139 IU/L, and total bilirubin 0.7 IU/L. The coagulation profiles showed an international normalized ratio (INR) of 1.90.

A computed tomography (CT) scan revealed a 2.3×1.7 cm-sized lesion arising from the right posterior hepatic artery, which showed contrast enhancement in the arterial phase. Given the history of IE, it was considered to be a mycotic HAA (Fig. 1). A 9.3 cm intrahepatic hematoma was found just distal to the HAA, suggesting aneurysm rupture. No hemoperitoneum or sign of contrast extravasation was seen.

We performed a selective arterial angiography, which showed a mycotic HAA arising from the right posterior hepatic artery. It was decided that the location of the aneurysm was not accessible by surgical treatment, and therefore embolization was attempted. Successful embolization of the feeding branch of proximal right hepatic artery was performed using 8 microcoils (Fig. 2). Angiography performed immediately after the procedure showed obliteration of the aneurysm and good perfusion to the distal part of the right hepatic artery. The patient had no post-procedure fever or abdominal pain, and was discharged 1 week later. He visited the hospital 1 week after discharge and was pain-free with no active bleeding. A CT scan, that was performed at 2 months after procedure, showed that the large hepatic hematoma involving the proximal right hepatic artery remained without change; however, there was newly developed thrombus within the aneurysm (Fig. 3). The patient has remained asymptomatic to date.

DISCUSSION

HAA was first described by James Wilson in 1809.^{6,7} Historically, most aneurysms had already ruptured by the time of presentation, or were incidentally discovered at autopsy.^{6,7} However, recent advances in CT scans have made earlier detection of HAA possible.⁴

Mycotic aneurysm was first described by Osler in 1885. He believed that multiple aneurysms developed from septic emboli in bacterial endocarditis that resembled fungal growth. But since fungal infection is not commonly associated with bacterial endocarditis, the term 'mycotic' is usually a misnomer.^{2,3} The incidence of mycotic aneurysm has decreased dramatically thanks to the introduction of antibiotics.⁴ In 1923, Stengal collected 217 cases of mycotic aneurysm, and 187 (86%) of those cases were associated with endocarditis.⁸ The most

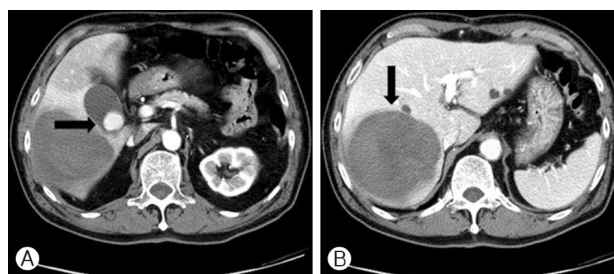


Fig. 1. (A) CT scan shows a 2.3 cm sized intrahepatic aneurysm (arrow) involving the right posterior hepatic artery. (B) A large hematoma (arrow) of the right lobe of the liver is seen.

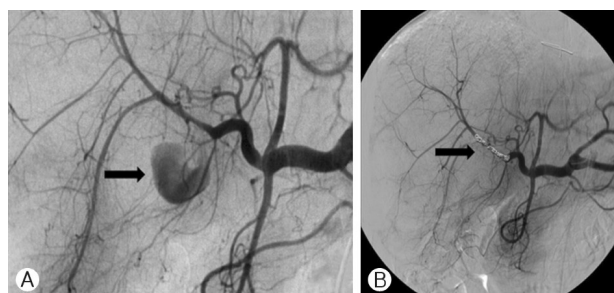


Fig. 2. (A) Arterial angiography shows the right hepatic artery aneurysm (arrow). (B) Successful embolization (arrow) of the proximal right hepatic artery aneurysm using 8 microcoils.



Fig. 3. Follow-up CT scan shows a large hepatic hematoma and thrombus (arrow) within the aneurysm involving the proximal right hepatic artery.

common site of mycotic aneurysm was the aorta, followed by the superior mesenteric, hepatic, and splenic arteries. HAA was observed in 19 cases (8%).² A more recent study showed that mycotic HAA was observed in only 1 of 306 patients with true visceral aneurysms.⁴

The etiologies of HAA are numerous.^{2,9} Prior to the introduction of antibiotics, most HAAs were mycotic and were associated with bacterial endocarditis.⁸ Currently, however, atherosclerosis is the most common cause of hepatic artery aneurysm.^{2,10} Other causes include mediastinal degeneration, iatrogenic, external trauma, radiological intervention, infection, tuberculosis, polyarteritis nodosa, systemic lupus erythematosus, Wegener's granulomatosis, and end-stage liver disease.^{6,10}

Aneurysms that involve the hepatic artery account for 20

% of all visceral artery aneurysms, second to splenic artery.^{1,6} The majority (80%) of HAAs are extrahepatic and solitary. The common hepatic artery is the most frequent site (63%), followed by the right hepatic artery (28%), the left hepatic artery (5%), and both hepatic arteries (4%).^{9,10} The average HAA size is approximately 3.5 cm, with a range of 1.5 to 14 cm, and symptomatic aneurysms are typically 2 cm in size.⁴

Most HAAs are asymptomatic.^{4,9} When symptomatic, the most common complaint is the right upper quadrant abdominal pain which radiates to the back.¹¹ One third of patients show Quincke's symptom triad of abdominal pain, obstructive jaundice and hemobilia.¹¹ A palpable mass and abdominal bruits are occasionally observed in cases of large HAAs.⁹ In our case, the only symptom was right upper quadrant abdominal pain.

Despite the relative low incidence of HAA among visceral aneurysms, HAA has the highest rate of rupture (44%) among such aneurysms.⁷ Risk factors associated with HAA rupture include multiple aneurysms and a nonatherosclerotic origin, in particular polyarteritis nodosa.⁴ In addition, mycotic aneurysm *per se* is also associated with a high risk of rupture, because the pathogenesis is infection of blood vessels that may occur by direct lodgment of infected emboli in the vessel lumen, and the subsequent inflammation may cause necrosis of the vessel wall and eventual aneurysmal dilatation or rupture.^{2,4}

The detection rate of asymptomatic HAA has increased with widespread use of CT and ultrasonography.⁶ Color Doppler ultrasonography is the modality of choice for screening, and CT is a relatively accurate diagnostic tool for detecting even small aneurysms.¹ A multidetector CT scan combined with CT angiography is an excellent diagnostic method, which provides the detailed anatomy of the visceral arteries.¹² However, the 'gold standard' for diagnosis of HAA is conventional angiography since it can provide information on the size, shape, and location of the aneurysm.⁶

Treatment of HAA is recommended in symptomatic patients at high risk of aneurysm rupture.⁴ In general, treatment is recommended when the HAA diameter is greater than 2 cm.⁴ Treatment varies depending on the lesion location, vascular anatomy, etiology and coexisting conditions.^{1,4} Currently, 2 treatment options are recommended for HAA.^{4,6} One option is endovascular treatment including embolization with acrylic glue, coils and endovascular stenting.⁴ Embolization with metallic coils is the most common approach and is preferred for patients with an intrahepatic lesion and for whom surgery is considered high risk.¹⁰ Embolization is also suitable for saccular HAA where the hepatic arterial flow can be preser-

ved.¹³ Consistent with these principles, the current patient was treated using arterial embolization rather than surgery since the aneurysm was saccular and intrahepatic, and there was a high risk of surgical mortality through aneurysm rupture.¹³ The second HAA treatment option is surgery with or without revascularization.⁴ Open surgical treatment options include ligation, excision, venous grafting, and hepatic resection.^{4,6} Surgical treatment is recommended for patients with a lower surgical risk.^{4,7} The HAA in common hepatic artery can be treated using ligation and resection without reconstruction because collateral flow is maintained through gastroduodenal arteries.^{4,7} A distal aneurysm can be treated using surgical excision and vascular repair, and ligation is contraindicated due to the risk of ischemia.^{7,14}

In summary, mycotic HAA has become extremely rare following the introduction of effective antibiotics.^{4,6,8} However, if not treated, this aneurysm is associated with a high possibility of rupture and mortality.^{2,4,6} Successful management depends on early diagnosis in patients with a recent or remote history of sepsis or endocarditis.² In our case, a ruptured mycotic HAA was diagnosed early based on CT and arterial angiography. The patient was successfully treated using percutaneous transcatheter arterial embolization, and is doing well to date. As seen in this case, arterial embolization can be a safe and effective therapy for a ruptured intrahepatic aneurysm when surgery is not advisable. Hence, early diagnosis and prompt arterial embolization in patients with ruptured mycotic HAA can avert the high risk of mortality.

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