Multifocal Intrahepatic Artery Aneurysm with FDG-avid Thrombosis Simulating Metastasis: Report of a Rare Case

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Hepatic artery aneurysm (HAA), although rare, represents a serious diagnostic and therapeutic challenge due to high rupture rate and associated mortality. Early detection and accurate diagnosis are essential for successful management. Here, we present an extremely rare case of multi-focal intrahepatic HAA with confined intrahepatic rupture and hypermetabolic activity at PET imaging, simulating metastasis of melanoma. A retrospective review found only two other HAA at our institution between 2000 and 2015, both of which involved the extrahepatic artery. This report highlights the importance of clinical, radiological, and pathological correlation in the management of this rare condition. (J Clin Exp Hepatol 2016;6:321–325)

Hepatic artery aneurysm (HAA) is a rare condition, with approximately 500 cases reported since it was first described in 1809.1,2 It accounts for only ~0.4% of all arterial aneurysms, but more than ~20% of all visceral artery aneurysms (VAAs), making it the second most common type of VAA, following only splenic artery aneurysm.3 By definition, HAs are true aneurysms, in which hematoma is bound by all three layers of the arterial wall. This is in contradistinction to pseudoaneurysms, which are vessel rupture with the hematoma confined only by tissue outside the arterial wall, with the tunica intima and/or media absent in the aneurysm wall. Unlike pseudoaneurysms, which are mostly secondary to trauma or surgery, HAA is associated with many different conditions. About 80% of HAs occur extrahepatically.4–6 While HAA may be congenital, the vast majority are acquired. Atherosclerosis, as the most common etiologic factor for HAA, is responsible for approximately 30% of HAA cases. Iatrogenic etiologies are the second most common, among which liver transplantation accounts for about 17% of cases. Other relatively less common etiologic factors for HAA include infectious vasculitides, systemic vasculitides, including polyarteritis nodosa, Churg-Strauss vasculitis (eosinophilic granulomatosis with polyangiitis) and those related to systemic autoimmune diseases like lupus and rheumatoid arthritis, fibromuscular dysplasia, segmental arterial medialysis, and inherited connective tissue aberrations, such as Ehlers–Danlos and Marfan syndromes.7,8

True HAs tend to be asymptomatic until rupture, when they present as life-threatening emergencies. The estimated rate of HAA rupture is 65% with a mortality rate of 21%, which is the highest among aneurysms in the splanchic circulation.9 In the last two decades, the implementation of advanced cross-sectional imaging modalities has allowed the early detection of HAs. Moreover, since the first successful treatment of HAA by ligation in 1903, multiple other therapeutic methods have been developed, including endovascular coil embolization and endoaneurysmorrhaphy. Despite these advancements, acute rupture of HAA still has a high mortality and represents a serious diagnostic and therapeutic challenge. Here, we present a rare case of multifocal intrahepatic HAA simulating metastasis.

CASE

Clinical and Laboratory Assessment

The patient was a 76-year-old woman who presented to the Emergency Room (ER) at an outside hospital with fluctuating epigastric pain radiating to the back, nausea, and vomiting. The patient had no history of underlying chronic liver disease. Her medical history was significant for melanoma in the right upper extremity in 1973, status post- excision with wide margins; atrial fibrillation controlled by rivaroxaban; glaucoma; and a remote episode of pancreatitis. She denied any history of previous trauma, surgery, or any other procedures. On the day of transfer to

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Abbreviations: HAA: hepatic artery aneurysm; FDG: fluorodeoxyglucose; PET: positron emission tomography; VAA: visceral artery aneurysm; CT: computed tomography; MRI: magnetic resonance imaging; MRCP: magnetic resonance cholangiopancreatography; SUV: standard uptake value

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our institution, her vital signs were remarkable for hypertension, with blood pressure of 183/115. Her serum transaminase activities were within normal range (alanine transaminase 33 U/L and aspartate transaminase 47 U/L) and alkaline phosphatase was slightly increased (151 U/L, normal range 30–110). All other laboratory values were within normal range.

**Imaging Assessment**

Abdominal computed tomography (CT) was performed during the portal venous phase following the injection of iiodinated contrast. Five discreet hypervascular lesions in the liver were identified: in segment 3 measuring 11 mm, in segment 7 measuring 4 mm, and three in segment 6 measuring 6, 4, and 4 mm. The largest lesion in segment 3 demonstrated homogenous enhancement (Figure 1A). Two low-attenuation areas, each measuring 5 mm in diameter, were also incidentally noted in the uncinate process of the pancreas. None of these changes were present on a CT performed 10 years prior. Contrast-enhanced magnetic resonance imaging (MRI) was performed the following day, which demonstrated heterogeneous intermediate T1 signal within the segment 3 lesion with partial enhancement along the anterior aspect (Figure 1B). On MRCP sequence, no clear communication between the subcentimeter pancreatic lesions and pancreatic duct could be identified. Subsequent hepatic ultrasound showed a 21 mm complex hypoechoic structure with internal echoes and septation in the left lobe of the liver, corresponding to the segment 3 lesion noted on CT and MRI (Figure 1C). Blood flow was noted along the periphery of this lesion (Figure 1D). Two adjacent anechoic structures, up to 12 mm, with surrounding vascularity were noted in the porta hepatis, which demonstrated continuity with surrounding vessels. Multiple smaller anechoic structures, measuring up to 9 mm in diameter, were also noted in the left hepatic lobe, appearing in continuity with the surrounding vessels. A separate anechoic structure containing internal vascularity and measuring 12 mm was noted in the right lobe, consistent with the lesions in segments 6 and 7 identified on CT. Two sub-centimeter splenic artery aneurysms were also noted on the CT and MRI (not shown). The abdominal aorta was normal in course and caliber. Trace abdominal ascites were present on the MRI; however, there was no frank intraperitoneal hemorrhage.

Given the patient’s history of melanoma, positron emission tomography (PET) scan was performed to further characterize the liver lesions. On fused PET-CT imaging, the liver demonstrated mild focal FDG uptake corresponding to the enhancing lesions seen on CT and ultrasound (Figure 2). The maximum standard uptake value (SUV-max) of these lesions was 3.2. Although the lesions were not markedly FDG avid, the consensus was that surgical resection was preferable and to repeat PET/CT scan in two months. The patient underwent elective resection. Two additional masses were identified intraoperatively in the

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**Figure 1** A 75-year-old woman with liver lesions. Contrast-enhanced CT demonstrates a 1.1 cm homogenously and avidly enhancing lesion in segment 3 (arrow). (A) Contrast-enhanced MRI performed the following day demonstrates that only the anterior aspect of the lesion is enhancing (arrow). (B) The posterior portion of the lesion is non-enhancing. Ultrasound performed two days later confirms these findings, demonstrating internal echoes and a fine septation (arrow). (C) With color Doppler, the anterior aspect of the lesion shows vascular flow (arrow). (D) The posterior portion of the lesion containing internal echoes, lack of color flow at US and enhancement on MRI is most consistent with the partial thrombosis and contained rupture of the hepatic artery aneurysm.