Use of gel foam in innumerable hepatic artery pseudoaneurysms

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Introduction

Hepatic artery aneurysms are a rare vascular pathology that can be secondary to trauma or vasculopathies. Management of hepatic artery aneurysms is based in size and etiology. When hepatic artery aneurysms are associated with hemorrhage surgical or endovascular intervention is merited. Present day first line therapy for intrahepatic aneurysms is endovascular therapy. Here we report a case of atraumatic innumerable intrahepatic pseudoaneurysms with subcapsular hematoma and intraperitoneal hemorrhage causing hemodynamic instability. We then discuss diagnostic and management considerations.

Case Presentation

A 70-year-old woman with a history of gastroesophageal reflux, paroxysmal atrial fibrillation on direct oral anticoagulation, and monoclonal gammopathy of undetermined significance presented to an outside hospital with acute onset right upper quadrant abdominal pain with radiation to the right shoulder and a month of diarrhea. A CT at the time of presentation to the emergency room showed new tubular lesions within the liver and a new subcapsular fluid collection concerning for abscess or hematoma. Percutaneous image guided aspiration of the fluid collection demonstrated sterile hematoma. The patient was transferred to a tertiary care center for evaluation by hepatology.

Intervention

Shortly after transfer the patient became hemodynamically unstable requiring massive transfusion protocol. CT demonstrated large volume hemoperitoneum, soft tissue thickening around the celiac trunk, numerous pseudoaneurysms arising from the segmental hepatic artery branches, and extensive heterogeneity of the hepatic parenchyma consistent with a combination of hemorrhage, infarct, and edema (Figure 1). Extensive laboratory work-up for inflammatory vasculitides was negative. Digital subtraction angiography (DSA) of the celiac trunk demonstrated innumerable 3-5 mm pseudoaneurysms arising from the right hepatic artery and segment 3 branches (Figure 2A). Due to the extensive nature of the pseudoaneurysms coiling each lesion was considered infeasible. Instead, the right hepatic and the segment 3 arteries were embolized with Gelfoam (Pfizer) slurry to 5 beat stasis. Repeat DSA of the hepatic artery demonstrated no residual filling of the pseudoaneurysms (Figure 2 B&C). The patient remained hemodynamically stable and did not require further transfusions for the duration of hospitalization.

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Figures

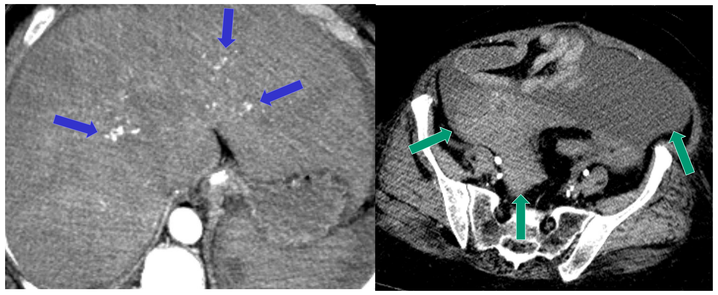

Figure 1: Images from the arterial phase of the CT liver protocol performed when the patient arrived at Henry Ford Hospital. A) Maximal intensity projection image with innumerable arterially filling foci suggestive of pseudoaneuryms (blue arrows label a few of these foci). B) Axial image with large volume hemoperitoneum layering in the pelvis (green arrows)

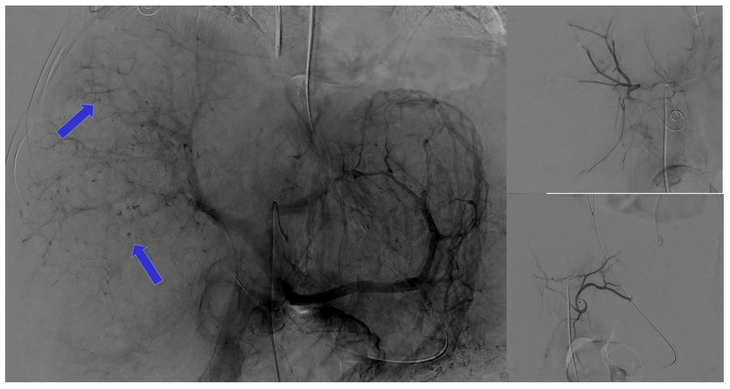

Figure 2: A) Digital subtraction angiography of the celiac trunk showing innumerable tiny psuedoaneurysms (a few are labelled with blue arrows) localized within the proper hepatic artery and its branches. B and C) Selective angiogram of the right hepatic artery (B) and segment 3 hepatic artery (C) showing occlusion of the distal branches and pseudoaneurysms after gel foam delivery.

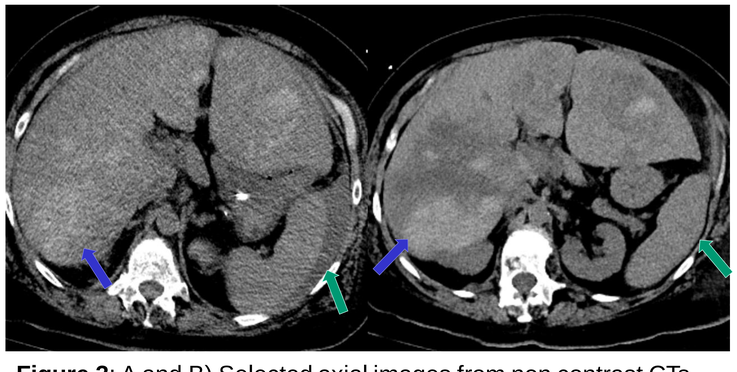

Figure 3: A and B) Selected axial images from non contrast CTs before (A) and 10 days after embolization (B) showing persistent intrahepatic hemorrhages (blue arrows), with resolution of the intraperitoneal hemorrhage (green arrows).

Discussion

Hepatic artery aneurysms (HAA) historically account for approximately 20% of splanchnic artery aneurysms second only to splenic artery aneurysms. Underlying etiologies include trauma/iatrogenic, connective tissue disorders, vasculitidies, and atherosclerotic disease. In some modern case series HAA actually make up ~50% of splanchnic artery aneurysms, which has been suggested to relate to morbidity from increased percutaneous interventions (1). Hepatic artery aneurysms not related to trauma/iatrogenic causes are most commonly single, extrahepatic, and atherosclerotic in etiology. Important vasculitic causes of HAA include polyarteritis nodosa (PAN), segmental arterial mediolysis (SAM) and granulomatosis with (GP).

Once suspected initial evaluation with CTA of the abdomen is appropriate. Clinically distinction between true HAA and hepatic artery pseudoaneurysm (HAP) is important as pseudoaneurysms are more likely to hemorrhage with mortality rate of around 40% once ruptured (2, 3,12). Unfortunately differentiating between HAA and HAP on CTA is not usually possible on imaging, but HAP should be suspected in traumatic/iatrogenic cases. In HAA that are not symptomatic case series have suggested that conservative management with serial CTA to assess for growth is reasonable for atherosclerotic aneurysms <2cm in diameter as there is low risk of rupture (2).

In cases of rupture or high concern for rupture (size >2cm, suspected HAP, vasculitic etiology/multiplicity) intervention is merited. Surgical repair/bypass is the mainstay of extrahepatic HAA. For intrahepatic HAA/HAP endovascular therapy is preferred. It is important to note that need for reintervention is high after endovascular therapy for HAA/HAP at 40%/17% respectively (4).

A variety of endovascular therapies have been described for intrahepatic HAA/HAP including coil embolization, diverting stents, and percutaneous thrombin injection with selection of therapy depending on the location and number of aneurysms (5, 6, 7, 8). Multiple intrahepatic aneurysms such as presented here are most commonly seen with vasculitides. PAN and GP can be responsive to medical treatment and diagnosis should be pursued with laboratory tests and biopsy of the affected organ if needed (9). SAM remains a diagnosis of exclusion (10). Given negative inflammatory workup our case is favored to be related to SAM or PN.

In this case HAP were favored over HAA given hemorrhage. They were large enough in number and peripheral enough in location that coil embolization that has been reported in somewhat similar situations in the liver (5) and kidney (11) was not favored. Similarly, peripheral location in multiple hepatic lobes made stent exclusion nonviable. Use of selective and superselective delivery of gelfoam successfully returned the patient to hemodynamic stability. The patient transitioned to hospice 10 days after the intervention and subsequently expired without a definitive diagnosis or cause of death. A report of a case similar to ours also required embolization of the majority of the hepatic arterial territory to regain hemodynamic stability and the patient had subsequent multisystem failure leading to death several days after the procedure (5). This case and prior reported both point to the high mortality of hemorrhaging multiple HAP despite successful acute management with embolization of the majority of the hepatic artery territory.

Conclusion

In conclusion, we present a case of innumerable HAP presenting with life threatening hemorrhage managed with selective administration of gel foam. Successful management of HAP depends on the following:

1. Appropriate identification utilizing arterial phase imaging and/or traditional angiography. Delineating intra and extrahepatic aneurysms.
2. Appropriate work up of the etiology in non-traumatic cases to identify vasculopathies that are amenable to medical management.
3. Surgical repair, embolization, or flow diverting stent placement in cases where aneurysms are actively hemorrhaging or at high risk of hemorrhage.