Hemoperitoneum from Gallstone Erosion

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Introduction

Hemoperitoneum and hemobilia are exceedingly rare, life-threatening conditions that require rapid diagnosis and treatment. The main sources of intraabdominal bleeding are visceral organs, especially the liver (hepatomas) and spleen, and vascular injuries such as aneurysms or pseudoaneurysms.[1] There are few documented reports of spontaneous hemoperitoneum from cystic artery rupture[2], especially without underlying disease.

Case Presentation

A 58-year-old man with hepatitis C and alcohol-induced cirrhosis post-TIPS was brought to the ED by EMS after being found down. He was responsive upon arrival and undergoing treatment for diabetic ketoacidosis.

He deteriorated to agonal breathing with associated altered mental status and coffee ground emesis and was intubated. His hemoglobin dropped from 11.8 to 7.3 and massive transfusion protocol was initiated. CT abdomen and pelvis was concerning for hemorrhagic gallbladder perforation with gallstones seen in the right paracolic gutter. No pseudoaneurysm or aneurysm was identified.

Interventional radiology was consulted for emergent angiography and potential intervention. DSA performed in the common hepatic artery through a Progreat microcatheter showed active extravasation from a distal branch of the cystic artery and filling defects were noted in the abdominal cavity, suspected to be gallstones. Gelfoam and three microcoils were deployed in the cystic artery; final DSA runs showed complete stasis and occlusion of the cystic artery.

Following the procedure, he continued to have multiple metabolic derangements and shock requiring multiple pressors. His family transitioned him to DNR, and he passed away that night.

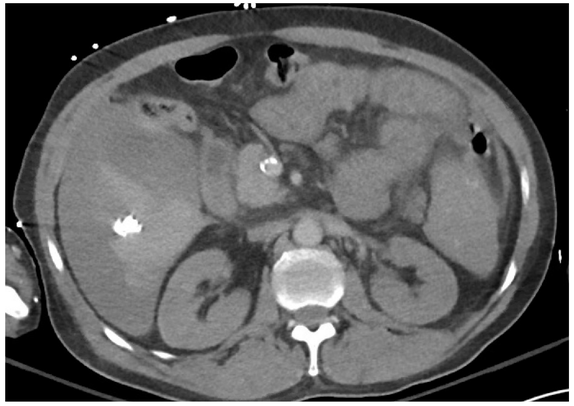

Figure 1- CT findings: perihepatic fluid consistent with blood.

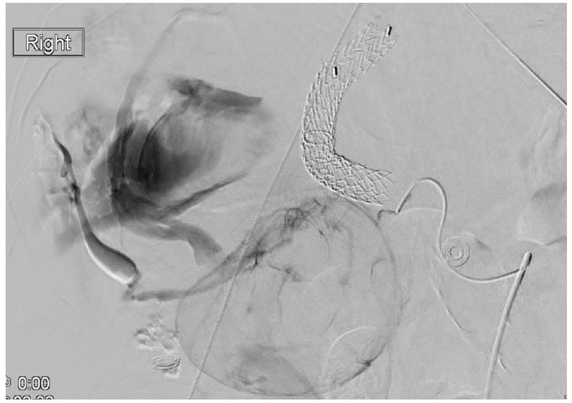

Figure 2 - Angiogram demonstrating extravasation from cystic artery

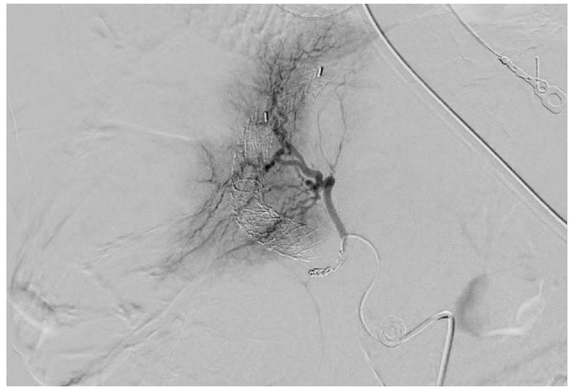

Figure 3 - Post embolization angiogram shows resolution of active extravasation.

Discussion

Spontaneous hemoperitoneum is nontraumatic hemorrhage into the peritoneal cavity, with the most common sources being gynecological, hepatic, splenic, and vascular.[1] CT scans are the mainstay in the work-up as it helps localize the source, determine presence of active bleeding, and can distinguish between new and old hemorrhage.[1]

The most common cause of hemorrhage from the cystic artery is iatrogenic or traumatic.[3] Nontraumatic causes of bleeding are often due to gallstones, which typically causes only microscopic bleeding.[4] Other causes of bleeding from the cystic artery include inflammatory lesions, hepatitis, tumors, or vascular abnormalities.[3] To our knowledge, there has only been one other case of hemoperitoneum from a ruptured cystic artery without underlying cholecystitis.[2]

Angiography is the gold standard for visceral arterial bleeds causing hemobilia or hemoperitoneum, especially in the hemodynamically unstable patient.[2,4] With a false negative rate of 10%,[4] it allows for reliable identification of active bleeding with the ability to therapeutically embolize the source. In this case, both the actively bleeding branch and the main cystic artery were embolized with Gelfoam and coils to achieve complete stasis. The chief concern with embolizing the cystic artery is gallbladder ischemia and necrosis.[3] The rate of these complications with cystic artery embolization are reported to be less than 2%, with an increased risk associated with micro-particles.[3] This low rate can be explained by the abundant collateral supply between the right hepatic artery and cystic artery.[2] While a low rate, the morbidity and mortality associated with ischemia is enough to avoid embolizing the cystic artery if possible.

Data on complications is lacking, as cystic artery embolization is typically performed as a temporizing measure before cholecystectomy[4], preventing follow up of gallbladder damage.

Results

In this case, there was active extravasation from an otherwise normal appearing cystic artery and gallbladder.

Massive hemobilia and hemoperitoneum from a normal cystic artery is an exceedingly rare entity with few reports in the literature. The case was a success with complete embolization of the cystic artery following Gelfoam and coil deployment.

The patient unfortunately passed away from his multiple metabolic derangements before we could follow-up with long-term outcomes and complications from this procedure.

Conclusion

Our case demonstrates that although not without risk, the cystic artery can be embolized if identified as the source of bleeding.

Further studies are needed to help determine rates of gallbladder necrosis in patients that do not undergo cholecystectomy after celiac artery embolization.

References

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