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Case Report

Percutaneous microwave ablation for control of massive portal venous bleeding ☆,☆☆,★

*Abdel Aziz Jaffan, MD**, *Steven D. Huffman, MD*, *Sebouh Gueyikian, MD*

Aurora Health Care, St Luke's Medical Center, 2900 W Oklahoma Ave, Milwaukee, WI 53215

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ABSTRACT

We present a case of a 46 years old female with decompensated liver cirrhosis who developed severe intraperitoneal hemorrhage secondary to inadvertent liver puncture during a paracentesis which resulted in a combined hepatic arterial and portal venous injury. The arterial injury was managed with transarterial embolization. The portal venous injury was managed with percutaneous microwave ablation. This article also highlights the importance of evaluating both arterial injury as well as portal venous injury in the setting of hepatic bleeding, particularly in patients with portal hypertension.

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Introduction

Portal venous bleeding usually resolves spontaneously given the relatively lower pressure of the portal venous system. Massive persistent bleeding, however, can be life-threatening and often requires invasive therapy [1]. We present a case of a cirrhotic patient who developed severe intraperitoneal hemorrhage following paracentesis from combined hepatic arterial and portal venous injuries, managed with transarterial embolization and percutaneous microwave ablation respectively.

Case description

A 46 years old female with a history of alcoholic cirrhosis, was admitted with new onset weakness, jaundice, decompensated cirrhosis, acute on chronic anemia, and dark stools. On admission her total bilirubin was 29.4 mg/dL, aspartate aminotransferase (AST) 213 units/L, alkaline phosphatase (ALP) 209 units/L creatinine 0.85mg/dL, international normalized ratio (INR) 3.5, white blood cells (WBC) 17800/mcL, neutrophils 84%, platelets 63,000/mcL, hemoglobin 4.8 g/dL (baseline 7 g/dL), and sodium 122 mmol/L. Her calculated Model for End-Stage

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* Corresponding author.
E-mail address: aajaffan@gmail.com (A.A. Jaffan).
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Liver Disease Sodium (MELD Na) score was 36. Her blood pressure was 105/57 mm Hg, temperature 99.1°F, and heart rate 105 per minute. She denied any abdominal pain, and there was no evidence of abdominal tenderness or distension. Rectal exam showed dark stools with positive guaiac test. The patient was admitted to the intensive care unit and managed medically with Octreotide drip and blood transfusion to maintain her hemoglobin above 7g/dL. A diagnostic esophagogastroduodenoscopy performed the next day showed medium sized esophageal varices, portal hypertensive gastropathy, antral erosions, and no evidence of active variceal bleeding. No intervention was performed.

Given the persistent leukocytosis, an ultrasound-guided paracentesis was performed on day 4 to rule out spontaneous bacterial peritonitis; the puncture was at the mid aspect of the right lateral abdominal wall. Ultrasound showed a small amount of fluid in the right aspect of the abdominal cavity and only 70 mL of serosanguinous fluid were retrieved.

On day 6, the patient developed acute onset of abdominal pain, abdominal distension, and hypotension with acute drop of hemoglobin to 5.4 g/dL (from 7.8 g/dL). Repeat esophagogastroduodenoscopy showed again moderately sized esophageal varices with no evidence of active bleeding; varices were prophylactically banded at this time. She remained hypotensive requiring vasopressors, with continued drop in hemoglobin despite blood transfusion. A bedside left sided paracentesis revealed hemorrhagic ascites; it was believed to be iatrogenic and secondary to the recent right sided paracentesis. Due to the acute renal failure, CT angiogram of the abdomen was not performed, and the patient was brought directly to the interventional radiology suite. The right sided paracentesis puncture site was marked with a metallic marker. An arteriogram with selective injection of the arteries supplying the abdominal wall puncture site showed no contrast extravasation or pseudoaneurysm. The right deep iliac circumflex, an intercostal and two right lumbar arteries were prophylactically embolized using Gelfoam slurry.

On day 7, an arterial phase abdominal CTA, performed for persistent hypotension, showed large hemorrhagic ascites with active contrast extravasation from the right hepatic segment V (Fig. 1). The patient returned to the angiography suite and common hepatic arteriogram showed a pseudoaneurysm at the caudal aspect of hepatic segment V (Fig. 2), corresponding to the CTA finding. Total bilirubin was 16.5 mg/dL, AST 664 units/L, and INR 1.8. The right hepatic arterial branches supplying the pseudoaneurysm were successfully embolized with Gelfoam slurry. Postembolization celiac arteriogram showed no further filling of the arterial pseudoaneurysm (Fig. 3). Delayed portal venous phase however showed a focal round collection of contrast in the same area, suspicious for a portal venous injury (Fig. 4). After a few minutes, common hepatic arteriogram was repeated, and the finding of the suspected portal venous injury was no longer seen.

On day 8, arterial and venous phase CTA of the abdomen, performed for persistent hypotension and continuous blood transfusion requirement, showed no active contrast extravasation on the arterial phase (Fig. 5). The portal venous phase however showed active contrast extravasation from hepatic segment V compatible with portal venous hemorrhage (Fig. 6). This finding correlated with the finding seen on the delayed portal venous phase of the common hepatic angiogram (Fig. 4). At this point, her total bilirubin was 13.6 mg/dL, AST 3,947 units/L, and INR 2.1. Using CT-guidance, the area of the portal venous bleed was localized using anatomical landmarks. Percutaneous microwave ablation of an area measuring approximately 2 centimeters in diameter in hepatic segment V was performed using a single ablation cycle, with a power of

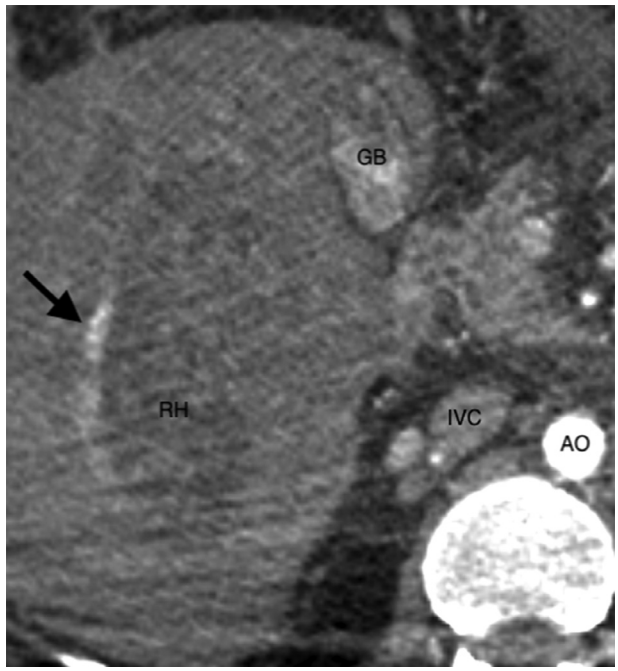

Fig 1 – CTA of the abdomen showing arterial contrast extravasation (black arrow) at the inferior aspect of the right hepatic lobe (RH). AO, aorta; IVC, inferior vena cava; GB, gallbladder.

Fig. 2 – Common hepatic arteriogram showing a pseudoaneurysm (black arrow) at the inferior aspect of the right hepatic lobe. CH, common hepatic artery.

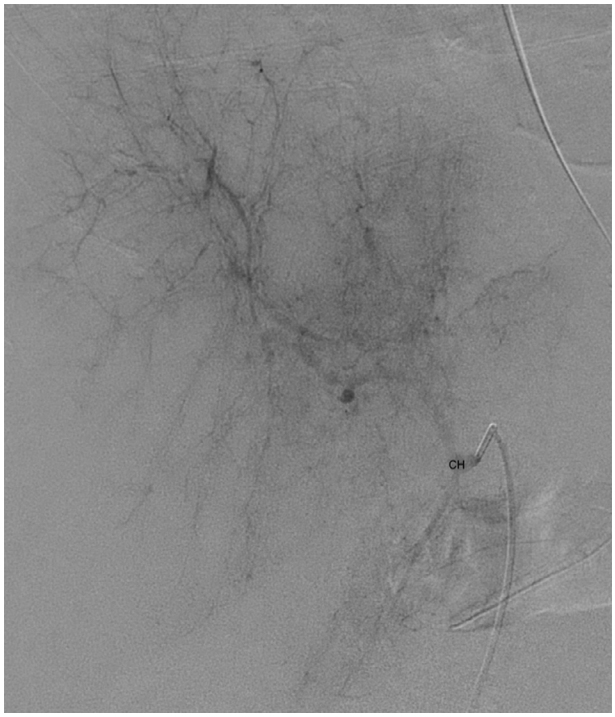

Fig. 3 – Postembolization common hepatic arteriogram shows successful embolization of the arterial pseudoaneurysm. CH, common hepatic artery.

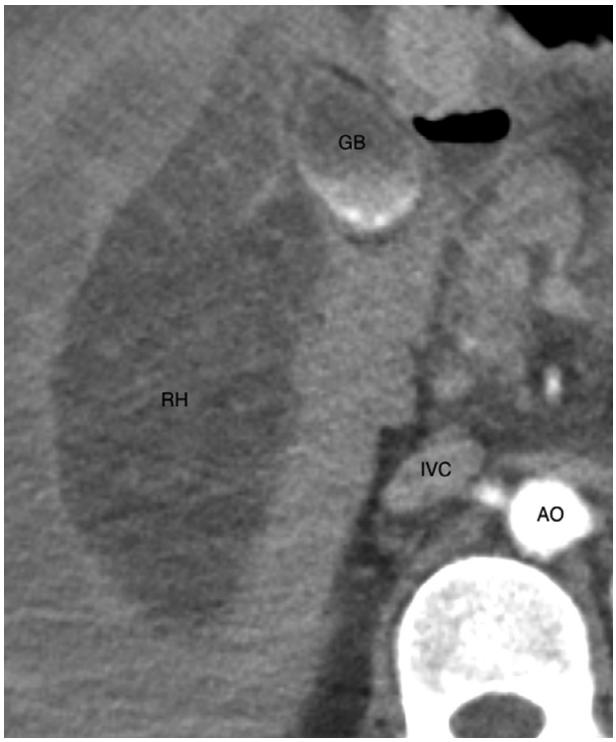

Fig. 5 – CTA of the abdomen showing hemoperitoneum but no active extravasation on the arterial phase. RH, right hepatic lobe; AO, aorta; IVC, inferior vena cava; GB, gallbladder.

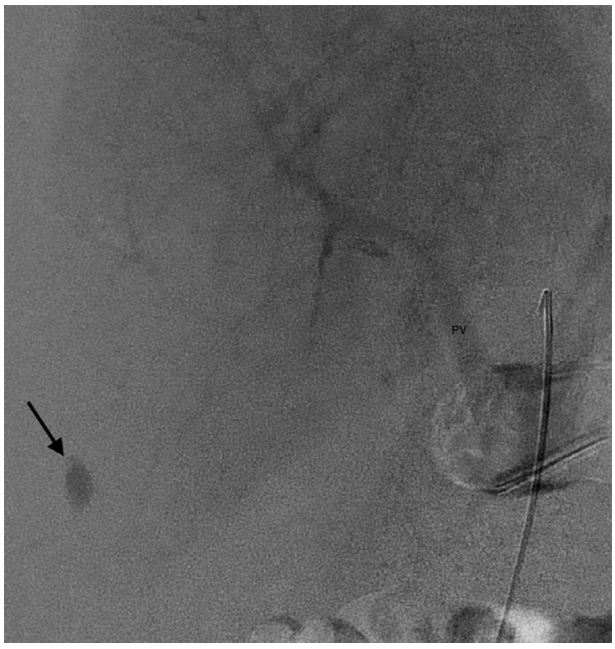

Fig. 4 – Delayed portal venous phase of the celiac artery angiogram showing active extravasation (black arrow) at the inferior right hepatic lobe suspicious for portal venous injury. PV, main portal vein.

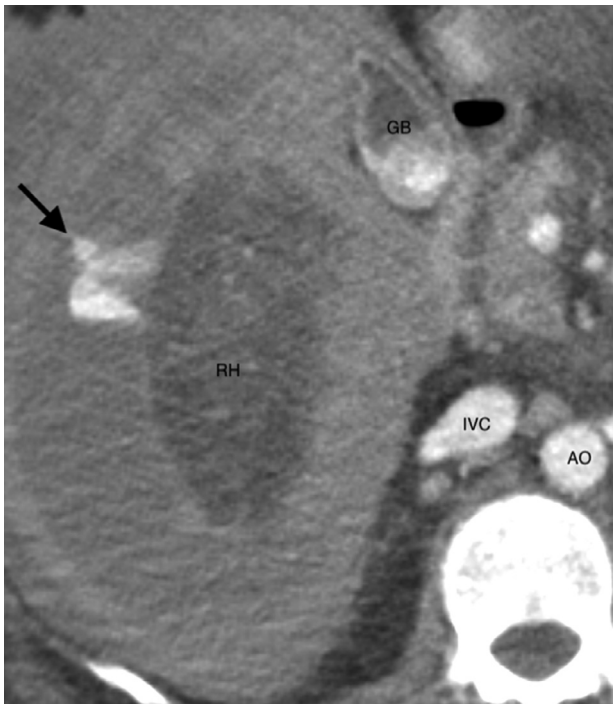

Fig. 6 – CTA of the abdomen showing contrast extravasation on the delayed portal venous phase (black arrow). RH, right hepatic lobe; AO, aorta; IVC, inferior vena cava; GB, gallbladder.

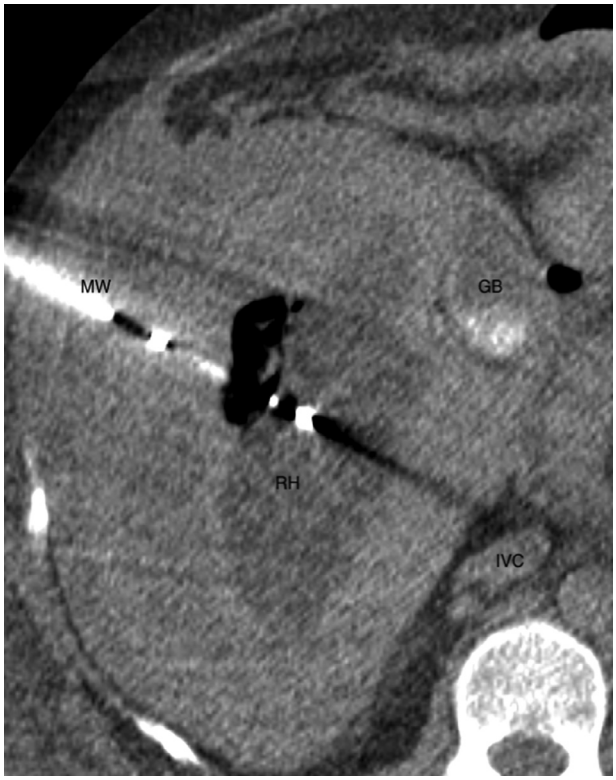

Fig. 7 – CTA of the abdomen showing a percutaneous microwave probe (MW) within hepatic segment V. RH, right hepatic lobe; IVC, inferior vena cava; GB, gallbladder.

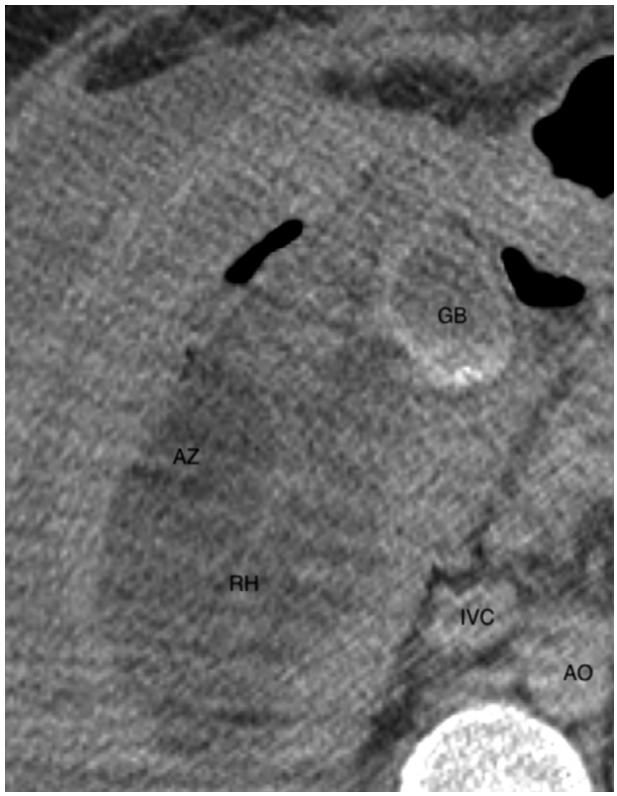

Fig. 8 – Contrast-enhanced CTA of the abdomen post microwave ablation showed no evidence of residual active extravasation on the delayed phase. AZ, ablation zone; RH, right hepatic lobe; AO, aorta; IVC, inferior vena cava; GB, gallbladder.

100 Watts and for a duration of 9.5 minutes (Fig. 7). An immediate postablation CTA of the abdomen showed no evidence of residual arterial or portal venous contrast extravasation (Fig. 8).

Following the ablation procedure, the hemoglobin stabilized, and the patient did not require any additional blood transfusion for the next 3 days. She developed persistent lactic acidosis, refractory hypoxemia, sepsis, and multiorgan failure. Her last set of laboratory tests showed a hemoglobin of 9.2 g/dL, a total bilirubin 17.5 mg/dL, AST 2,713 Units/L, ALP 411 Units/L, and INR 3.9. Given her poor prognosis, the family opted for comfort care only. The patient was extubated on day 11 and expired.

Discussion

Hemodynamically significant portal vein bleeding is uncommon and the literature focusing on iatrogenic portal vein bleeding is scarce [1]. It is usually caused by transhepatic procedures or trauma. It can be isolated or associated with an arterioportal fistulae or pseudoaneurysm. Portal vein bleeding is usually self-limited due to the relatively lower pressure of the portal venous system. Persistent and massive portal vein bleeding can cause hemodynamic instability requiring invasive therapy. When hepatic bleeding is suspected, both hepatic arterial and portal venous bleeding should be considered. Angiographic evaluation should include a delayed phase portogram. If an arterioportal fistula is present, transarterial embolization of the affected hepatic arteries allows healing of the portal venous injury by decreasing the pressurized flow to the portal vein. Active portal vein bleeding without a fistula can be missed on the hepatic angiogram if a delayed portal venous phase is not obtained [1]. In the absence of an arterioportal fistula, embolization requires direct percutaneous access of the portal venous system. Transhepatic (TH) access is the standard technique and involves direct percutaneous puncture of the portal vein and its branches. Access can also be achieved via a transjugular intrahepatic porto-systemic shunt, portosystemic varix, or via a transplenic (TS) approach. Both TH and TS methods are prone to hemorrhage-related complications. In a study comparing the two methods, the TS group's bleeding rate was 12.5%, and the TH group's bleeding complication rate was 8% [2].

Microwave ablation is a technique that generates heating of the surrounding tissue to over 50°C, leading ultimately to coagulation of blood vessels (coagulative necrosis) [3]. It can produce faster heating over a large volume of tissue with less susceptibility to heat sink effect compared to radiofrequency ablation [3]. Microwave ablation also allows cauterization of the needle tract after ablation which has been shown to reduce the risk of hemorrhage [4]. Microwave

ablation is commonly used nowadays for local therapy of primary and secondary hepatic neoplasms [3,4]. Its use for percutaneous control of hepatic bleeding has also been described. In an animal experimental study, Song et al showed that contrast-enhanced ultrasound-guided percutaneous microwave coagulation therapy significantly decreased blood loss in a rabbit model of active liver bleeding [5]. It has been successfully used intraoperatively to control hepatic bleeding [6,7]. Percutaneous microwave ablation use for control of hepatic bleeding from an arterial source has also been reported [8,9]. Its use for control of hepatic bleeding from portal venous source however has not been reported so far.

In this case, the absence of large amount of ascites in the peritoneal cavity increased the probability of inadvertently puncturing the liver parenchyma. This is not the most common etiology of intraperitoneal bleeding following paracentesis, which explains why we evaluated the abdominal wall vasculature looking for a bleeding source first. The injury has resulted in an arterial as well as a portal venous injury. CT angiogram with a delayed phase was a key to the diagnosis, highlighting the importance of cross-sectional imaging in these situations. The arterial injury was successfully treated with transcatheter arterial embolization. The absence of an arterioportal fistula precluded the treatment of the portal venous bleeding via the transarterial approach. Although portal venous bleeding resolves spontaneously in the majority of the cases, we believe that the combination of the patient hypocoagulable status (high INR) with the underlying portal hypertension did not allow spontaneous hemostasis to occur. We elected not to address the portal hypertension with a transjugular intrahepatic porto-systemic shunt in this critically ill patient with a poor prognosis. Percutaneous embolization of the portal venous bleed would have created a new puncture site within the accessed organ (liver or spleen), which itself can become a new source of bleeding; we were concerned that embolization of the tract at the end of the procedure might not be effective in a patient with defective coagulation mechanisms. Compared to percutaneous portal venous embolization, microwave ablation has the advantage of ablating the parenchymal tract at the conclusion of the procedure, which has the potential of reducing bleeding through the tact. The procedure was successful in addressing the acute bleeding as evident by the resolution of active extravasation on the immediate postablation CT, the stabilization of the hemoglobin level for 3 days following the procedure, and no further need for blood transfusion. It is likely that the transarterial embolization and percutaneous microwave ablation were responsible, at least partially, for the worsening of the liver function. Gelfoam slurry was used to achieve a relatively large area of embolization, encompassing all potential sources of the arterial pseudoaneurym, and in a very short period of time in a hemodynamically unstable patient. A more selective embolization would have likely resulted in a less severe insult to the liver. The patient succumbed 3 days later due to her severe underlying medical condition, and therefore we could not determine the intermediate and long-term efficacy of this procedure nor its safety profile.

Conclusion

Hemodynamically significant portal venous bleeding may be acutely controlled with percutaneous microwave ablation. A case series is required however to determine the intermediate and long-term efficacy of such therapy as well as its safety profile. It is important also to consider portal venous source in addition to arterial source in patients presenting with hepatic bleeding, especially in the context of portal hypertension.

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