



# Adrenal and renal abscesses following glue embolization of gastric varices: a case description

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## Case report

A 59-year-old man with cirrhosis and portal hypertension presented with melena and was found to have bleeding gastric varices on upper endoscopy. 1.2 mL of Histoacryl glue (B Braun Medical, Bethlehem, PA) was injected into gastric varices at the cardia (*Figure 1*). Haemostasis was achieved and he was discharged. In the subsequent months, he had recurrent episodes of fever partially responsive to antibiotic treatment. During an admission six months after the initial endoscopic treatment, blood cultures yielded *Klebsiella spp.* He was investigated with computed tomography (CT) which revealed multiple multiloculated rim-enhancing lesions in the left adrenal gland and upper pole of the left kidney, with perifocal fat stranding, in keeping with abscesses. The glue cast was seen along the cardia and lesser curvature of the stomach, extending through serpiginous vasculature into the left adrenal gland and adrenal vein. The gastric varices were obliterated.

The patient was treated by CT-guided aspiration of the left adrenal abscess (*Figure 2*) by the interventional radiologist and systemic antibiotics. Culture of the aspirated pus confirmed *Klebsiella spp.* He improved clinically and follow-up CT six months later showed complete resolution of the abscesses. The patient remained well.

## Discussion

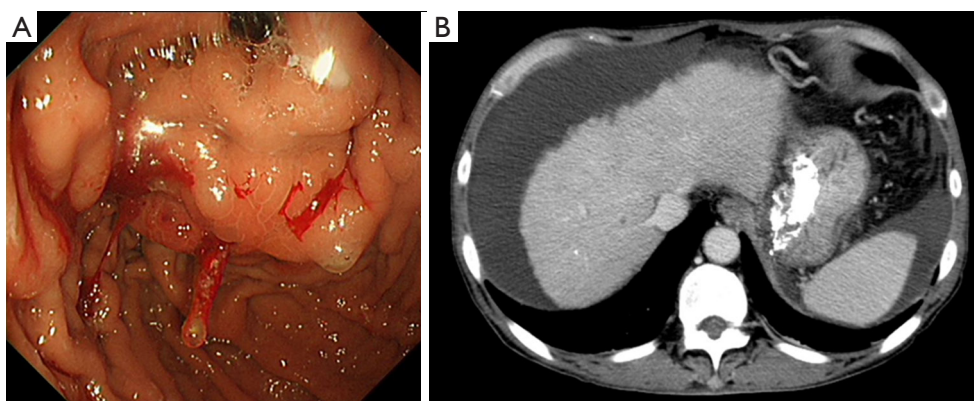
Gastric varices are present in up to 78% of patients with

portal hypertension and though the risk of bleeding is relatively low (up to 36%), it often presents as torrential bleeding and leads to high mortality (1). Conventional treatment options comprise supportive measures including blood transfusion, drugs such as propranolol, endoscopic intervention including sclerotherapy, ligation, or stenting, and radiological intervention such as transjugular intrahepatic portosystemic shunt (TIPS) or balloon-occluded retrograde transvenous obliteration (BRTO).

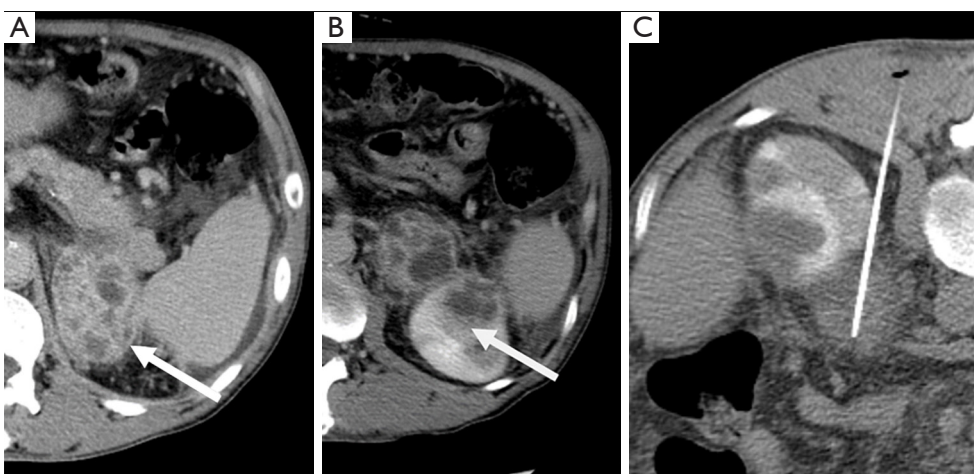
In recent years, cyanoacrylate monomer-based glue such as Histoacryl has emerged as a first line endoscopic treatment of gastric varices. The glue is mixed with lipiodol and polymerizes to form a cast upon contact with an ionic substance such as blood. Histoacryl glue is associated with a higher variceal obliteration rate and lower rebleeding risk as compared to other agents such as alcohol, powders, or band ligation (2), hence it is recommended for first line endoscopic treatment in the Baverno VI Consensus and in the American Association for the Study of Liver Disease practice guidelines (3,4).

Histoacryl glue injection is a generally safe treatment with overall complication rate of approximately 1% and mortality risk of 0.5%. Commonly reported complications include variceal rebleeding, ulceration, or erosion, blockage of injection catheter, local thrombosis of the portal venous system, sepsis, or uncommonly distant embolization if there is a right-to-left shunt (5).

Sepsis secondary to Histoacryl embolization is uncommon and reported on a case basis in the literature.



**Figure 1** Glue embolization of gastric varices. (A) Endoscopic imaging showing a bleeding gastric varix. (B) CT axial cut performed 6 months after embolization of the varices shows a hyperattenuating glue cast (arrow) at the cardia and lesser curvature of the stomach.



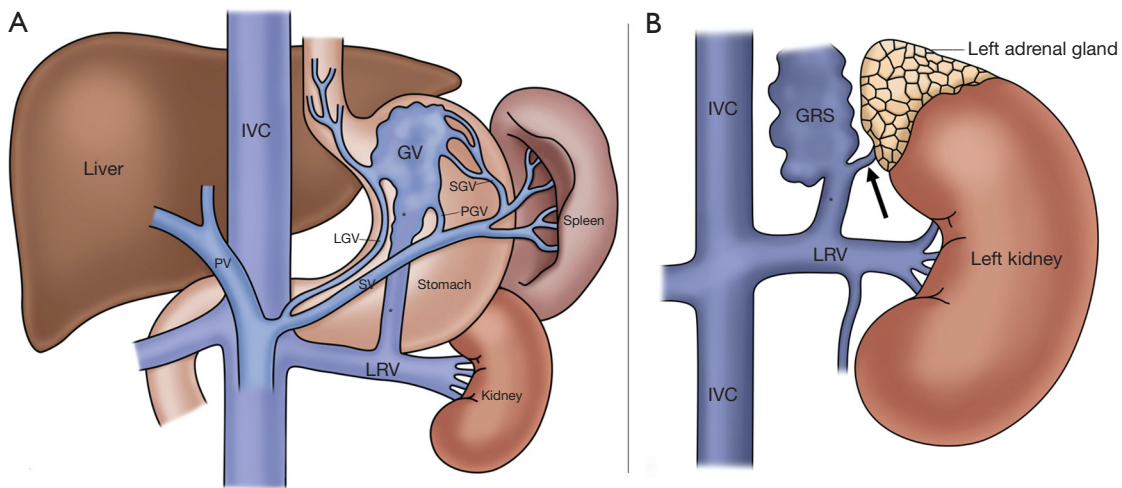
**Figure 2** Left adrenal and renal abscesses six months later. (A,B) Contrast CT performed when patient presented with sepsis six months after glue embolization of gastric varices shows a multiloculated cystic lesion at the left adrenal gland (solid arrow) with contiguous extension into the left kidney (open arrow) with retroperitoneal fat stranding, suggestive of abscess. (C) On the next day, the patient was treated by CT-guided percutaneous drainage of the abscess using a 19-G needle in prone position.

A brief literature review showed scattered reports of post-embolization sepsis involving the splenic vein (6), portal vein (7), pancreas (8), and retrogastric region (9). Lee *et al.* provided the only account of adrenal abscesses as a complication, when the patients presented 4-6 months afterwards (10).

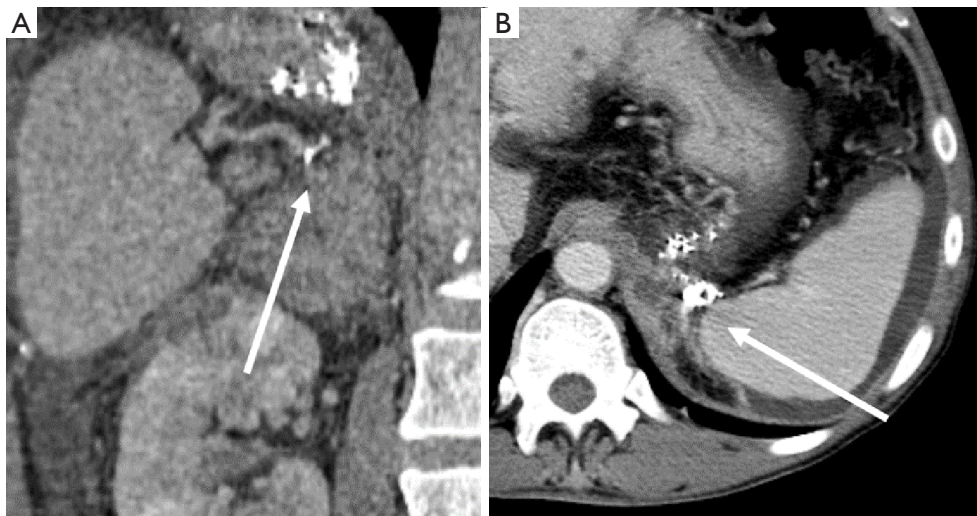
The etiopathogenesis is unknown and we propose a theory based on knowledge accrued by interventional radiologists performing BRTO procedures. Gastric varices are supplied by the left, posterior, as well as short gastric veins, and shunt into the systemic circulation more commonly through a gastroduodenal shunt than a gastrocaval

shunt. The classic gastroduodenal shunt empties into the left renal vein along with the left adrenal vein (*Figure 3*) (11). We propose that the renal and adrenal abscesses in our patient could have occurred due to reflux of glue cast through the gastroduodenal shunt into the adrenal vein, leading to poor clearance of gut microbes received from the portovenous system, thereby creating a septic nidus (12). The direct visualization of the glue cast on CT within the left adrenal gland and adrenal vein provided direct evidence for this theory (*Figure 4*).

The patient was treated by CT guided percutaneous aspiration of the left adrenal abscess in prone position using



**Figure 3** Anatomy of the classic gastorenal shunt. (A) Schematic diagram showing classical anatomy of gastric varices (GV) supplied by the left gastric vein (LGV), posterior gastric vein (PGV), and short gastric vein (SGV), while it empties through a gastorenal shunt (\*) into the left renal vein (LRV). This is more common than the variant of a gastrocaval shunt (not shown) (11). (B) The classic gastorenal shunt (GRS) drains into the left renal vein (LRV) together with the left adrenal vein (arrow) through a common trunk (\*) (11).



**Figure 4** CT performed when patient presented with sepsis six months after glue embolization, reformatted images in coronal (A) and axial (B) planes show glue cast deposited within the left adrenal parenchyma (arrows), presumably propagated through the gastorenal shunt.

a 19-G needle performed the next day, and completed a two-week course of intravenous antibiotics. Culture of the aspirated pus yielded *Klebsiella* species, which was consistent with his persistent *Klebsiella* bacteremia on prior repeat blood cultures. His fever soon subsided, white cell counts normalized, and he was discharged 6 days after the drainage with no further septic complaints afterwards. Gallium whole-body scintigram performed one month later did not

find any gallium-avid focus in the left adrenal bed. Follow-up CT scan six months later showed complete resolution of both the renal and left adrenal abscesses, with only mild stranding remaining in the left retroperitoneal space. There was no evidence of recanalization of gastric varices.

This case highlights that the potential complication, be it thrombosis or sepsis, depends very much on the anatomy of the portosystemic shunts. Awareness of this association

and the relevant anatomy is key to enable appropriate investigations for early diagnosis and treatment in patients who present with sepsis after variceal embolization.

## Conclusions

We report a rare case of delayed development of renal and adrenal abscesses following Histoacryl glue embolization of gastric varices, demonstrating the anatomy of the classic gastrosplenic shunt. Physicians should be aware of this association, including the relevant anatomy, and be ready to investigate appropriately to permit early diagnosis and treatment.

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## Footnote

*Conflicts of Interest:* All authors have completed the ICMJE uniform disclosure form (available at <http://dx.doi.org/10.21037/qims-19-1026>). The authors have no conflicts of interest to declare.

*Ethical Statement:* IRB approval was not required for this article per guidelines of our institution's IRB and no informed consent was required.

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