A Rare Case of Gastric Perforation Secondary to Endoscopic Glue Injection for Gastric Varices

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Abstract
Gastric perforation is very rare. Gastric perforation following endoscopic glue injection for gastric varices (GVs) in patients with portal hypertension (PHT) has not been documented till date. They are associated with significant morbidity and mortality. We present a glue injection-induced gastric perforation in patients with PHT that has been treated with primary closure at our institution. To the best of our knowledge, this is the first case report of such complication following endoscopic glue injection for GVs. Such site of glue-induced perforation has not been reported in literature till date. A 38-year-old male patient having cirrhosis of liver with PHT underwent repeated upper gastrointestinal (GI) endoscopic procedures for recurrent upper GI bleed. After 20 days of third endoscopic procedure in which he had undergone glue injection for GVs, patient developed abdominal pain and abdominal radiograph was suggestive of bowel perforation. Emergency laparotomy was performed that showed gastric perforation near to glue injection site which was repaired with primary closure at our institution. Complication such as glue injection-induced gastric perforation has not yet been documented. It is potentially life-threatening. Early recognition may lead to a better prognosis through earlier intervention.

Keywords
► perforation
► gastric varices
► glue injection

Introduction
The natural history of gastric varices (GVs) is quite less understood than that of esophageal varices (EVs). GVs may be seen in 18 to 70% of the patients with portal hypertension (PHT) and are a probable source of bleeding in 10 to 36% of patients with acute variceal bleeding.1–4 The risk of bleeding from GVs seems to be lower than that of EVs; however, when GVs bleed, it is often severe and bleeding-related mortality can be as high as 45%. Endoscopic treatment of bleeding GVs with N-butyl-2-cyanoacrylate glue (cyanoacrylate) is considered the best hemostasis with a lower risk of rebleeding compared with other endoscopic methods. The reported rate of rebleeding varies significantly from 0 to 50% in studies.5,6 There is also a potential risk of embolism especially in patients with underlying gastrorenal or gastrocaval shunts. Other serious complications reported in literature are sepsis, fistula, and adherence of the needle to the varix.7,8 We present a case with glue injection-induced gastric perforation, which was successfully managed by primary closure of perforation.

Case Report
A 38-year-old male, who was recently diagnosed with ethanol-induced cirrhosis of the liver with PHT (Child C MELD
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24), was first admitted to our hospital in intensive care unit (ICU) with grade III hepatic encephalopathy (HE) and ascites. He had a history of hematemesis 2 days before admission. Gastroscopy showed large EVs with red-color signs and severe portal hypertensive gastropathy (PHG). He underwent endoscopic variceal band ligation (EVL) procedure. Other issues such as grade III HE, coagulopathy, and spontaneous bacterial peritonitis were managed accordingly. The patient was discharged in stable condition.

Patient presented again ~20 days later with hematemesis. Upper gastrointestinal endoscopy showed residual varices with post-EVL ulcers, small gastroesophageal varix type 1 (GOV1) with stigmata of bleeding. About 1 mL of undiluted cyanoacrylate glue was injected into GV using a 21G sclerotherapy needle (Fig. 1A and B). On observing for few minutes as there was mild ooze, additional 0.5 mL of glue was injected using a separate 21G sclerotherapy needle. He was discharged in stable condition.

About 20 days following this discharge, he presented with complaints of severe abdominal pain. The pain was diffuse over abdomen, moderate intensity, nonradiating pain which was not associated with vomiting. He also complained of abdominal distension for past 3 days along with decreased urine output. On examination, the patient had tachypnea, tachycardia, hypotension, deep icterus, and bilateral pedal edema. Abdomen was distended with diffuse tenderness and sluggish bowel sounds. The patient was admitted to ICU. Labs showed severe anemia, thrombocytopenia, coagulopathy, and acute kidney injury. Ultrasonography-guided diagnostic ascitic tapping showed hemorrhagic fluid with high white blood cell count (30,000 cells per mm³). Abdominal radiograph was suggestive of pneumoperitoneum.

After stabilization of general condition, decision for emergency laparotomy was taken after explaining the potential risks. With the availability of fresh frozen plasma, platelet, and cryoprecipitate for transfusion during surgery, the patient was taken for emergency laparotomy.

Intraoperatively, we found blood in peritoneal cavity. The examination of bowel initially did not reveal any perforation; however, thorough examination showed perforation site near gastroesophageal junction (Fig. 2). Site of perforation was sutured, and two drains were kept in peritoneal cavity: one in the left subdiaphragmatic space and other in pelvis.

In postoperative period, the patient was managed in ICU on vasopressors and adequate transfusion of platelets, fresh frozen plasma, cryoprecipitate along with other supportive medications. He had a difficult postoperative course but finally discharged in stable condition after 10 days. Repeat gastroscopy after 1 month showed a glue cast but no active gastric varix.

Discussion

Although glue injection is one of the proven treatment modalities for GVs, glue-related perforation is a dreaded complication for both patient and the physician after an episode of bleeding from GOV.

Various complications which can occur after glue injection of GVs are embolization, local venous thrombosis, fistulization, ulceration, erosion, extrusion, and sepsis. Histoacryl injection site. Literature search did not reveal any case of active gastric perforation. Battaglia et al report two cases of visceral fistulae after Histoacryl injection, a previously unreported complication. One patient underwent treatment of GVs, which was complicated by an empyema of the left pleural cavity 6 months later. A fistulous connection between the gastric fundus and pleural cavity was documented. Authors suggested that misguided injection of Histoacryl into the stomach wall may have caused this
complication, as Histoacryl is known to be ulcerogenic to tissue. The management of gastric perforation in a patient with decompensated cirrhosis is a challenge considering various issues such as ascites, jaundice, hypoalbuminemia, coagulopathy, and sepsis. Moreover, surgical repair of gastric wall at perforation site in patient having PHG is tough job for an operating surgeon. Correction of coagulopathy and thrombocytopenia is required during intra- and postoperative period.

The prevention of occurrence of this kind of complication can be achieved by careful injection of glue into the vascular lumen after confirming needle position by aspirating blood, or by adopting image-guided glue injection technique (fluoroscopy-guided or endoscopic ultrasound-guided injection).

Conclusion

Glue-induced gastric perforation has not yet been documented. It is potentially life-threatening and difficult to manage in the presence of decompensated cirrhosis. Careful injection of the glue into the vascular lumen preventing extravascular extrusion into gastric wall can prevent this complication.

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References