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CASE REPORT | ENDOSCOPY

Post-Treatment Ulceration and Bleeding After Cyanoacrylate Injection of Duodenal Varices

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ABSTRACT

We report a case of recurrent gastrointestinal bleeding in the setting of diffuse duodenal and colorectal varices. These varices were secondary to either congenital absence of the portal vein or chronic occlusion of the portal vein leading to cavernous transformation of a collateral network of varices. He was acutely managed with injection of N-butyl-2-cyanoacrylate into a large complex of duodenal varices. His hospital course was complicated by a postprocedural gastrointestinal bleed within the first 24 hours after the procedure arising from a new duodenal ulcer at the site of injection, likely secondary to ischemia after obliteration of the varices.

INTRODUCTION

Duodenal varices (DV) represent a subset of ectopic varices (ECV), which consist of anastomoses between mesenteric venous branches and the systemic circulation and/or the portal system. Although the inherent bleeding risk of DV is unknown, ECV bleeding accounts for 1%–5% of all variceal hemorrhage.¹ Management of DV is highly dependent on the underlying vascular anatomy. Although endovascular therapies such as transjugular intrahepatic portosystemic shunt placement are sometimes plausible, our case will focus on endoscopic approaches such as variceal band ligation, sclerosant injection (EIS), and as in our case, cyanoacrylate (CA) injection. The risks and benefits vary with all of these endoscopic therapies, but postprocedural ulceration is a unifying concern. Although primarily associated with EIS, ulceration has been noted in rare cases with band ligation and CA injection.^{2,3} Although areas of CA glue extrusion can ulcerate, to date, there have not been any documented cases of CA-related ulceration leading to significant postprocedural bleeding.^{1,4} We report a case of a patient who experienced severe ulceration within 24 hours of CA injection.

CASE REPORT

A 63-year-old man with noncirrhotic portal hypertension due to chronic occlusion of his portal and mesenteric venous system with subsequent collateral vascular formation presented with hematochezia. Esophagogastroduodenoscopy (EGD) revealed numerous large (>5 mm) nonbleeding DV. A mesenteric venogram revealed tortuous varices arising from the superior mesenteric vein and penetrating the submucosa of the second portion of the duodenum (D2) without an obvious outflow. Endovascular management by interventional radiology was deemed impossible because of his abnormal vascular anatomy. He underwent CA injection in 2 separate areas, with 2 mL aliquots totaling 4 mL (Figure 1). Endoscopic ultrasound was performed preinjection and postinjection with complete obliteration of the D2 variceal complex and retained papillary patency. A day later, the patient developed hematochezia requiring 1 unit packed red blood cells transfusion. Repeat EGD demonstrated a large nonbleeding duodenal ulcer overlying the sites of before CA injection (Figure 2). High-dose proton-pump inhibitor therapy was initiated, and the follow-up EGD revealed healing of the ulceration.

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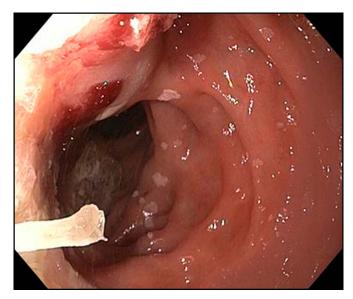


Figure 1. Duodenal varix at the time of cyanoacrylate injection.

DISCUSSION

We report a case of a patient who experienced severe ulceration after CA injection. Overall, a paucity of data exists for CA therapy in the setting of DV. Of the data available, no bleeding episodes were reported during the follow-up period. Cases of postband ligation mucosal ulceration have been noted after esophageal variceal (EV) therapy and DV therapy. The presumption in these cases is that mucosal ischemia related to the band ligation leads to shallow ulcerations that may lead to mucosal oozing or erosion into submucosal vessels that cause significant arterial bleeding. EIS therapy has been associated with more significant mucosal ulceration in both EV and DV cases and because of this

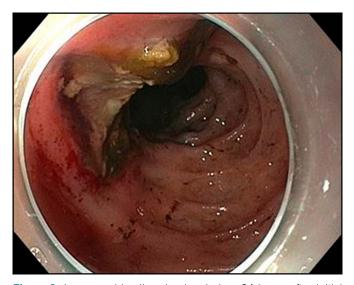


Figure 2. Large nonbleeding duodenal ulcer 24 hours after initial procedure, the sites of previous cyanoacrylate injection.

has been removed from the current guidelines as a reasonable option for treatment of EV.^{3,5-7} CA is not an alcohol-based compound and therefore cannot be viewed similarly to EIS. Previous evidence of CA injection for EV, graft volume, and ECV has not noted acute ulceration such as in our case report. When ulcerations have occurred, they are late and often associated with other risk factors such as nonsteroidal anti-inflammatory drug use, suggesting it is not a compound-specific problem.

In our case, the unique finding is not only the development of a mucosal ulceration in the absence of a glue cast but also how rapidly it occurred—within 24 hours. Given the underlying severity of venous thrombosis within the mesenteric veins, it is possible that occlusion of our patient's submucosal shunt led to venous ischemia locally. However, the timing seems rather acute and severe for an ischemic ulcer related to venous outflow obstruction. Alternatively, filling the submucosal varices with CA may have led to arteriole compression in the submucosal bed or capillary compression within the mucosal bed, causing arterial ischemia to this area, but if that were the case, then we would expect to see this complication more often. It is possible that the total volume of CA injected (4 mL), typical for injection of gastric varices, was too much for the duodenum. Perhaps this should have tempered to 0.5 or 1.0 mL aliquots instead. Overall, our case illustrates the varied underlying pathophysiology that contributes to the development of DV and a unique complication to consider when choosing your therapeutic modality.

DISCLOSURES

Author contributions: M. Radlinski, T. Fie, and R. Richhart wrote the manuscript. B. Wentworth revised the manuscript. S. Caldwell and Z. Henry approved the final manuscript. Z. Henry is the article guarantor.

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Informed consent could not be obtained from the patient or the family of the patient despite several attempts. All identifying information has been removed from this case report to protect patient privacy.

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REFERENCES

- Henry ZH, Caldwell SH. Management of bleeding ectopic varices. Techniques in Gastrointestinal. *Endoscopy*. 2017;19(2):101–7.
- Sato T, Yamazaki K, Toyota J, et al. The value of the endoscopic therapies in the treatment of rectal varices: A retrospective comparison between injection sclerotherapy and band ligation. *Hepatol Res.* 2006; 34:250-5.
- Seo Y, Kwon Y, Park S, et al. Complete eradication of duodenal varices after endoscopic injection sclerotherapy with ethanolamine oleate: A case report. Gastrointest Endosc. 2008;67:759–62.

- Ryu S, Moon J, Kim I, et al. Endoscopic injection sclerotherapy with Nbutyl-2-cyanoacrylate in a patient with massive rectal variceal bleeding: A case report. Gastrointest Endosc. 2005;62:632–5.
- Schmitz RJ, Sharma P, Badr AS, Qamar MT, Weston AP. Incidence and management of esophageal stricture formation, ulcer bleeding, perforation, and massive hematoma formation from sclerotherapy versus band ligation. Am J Gastroenterol. 2001;96(2): 437-41
- Garcia-Tsao G, Sanyal AJ, Grace ND, et al. Prevention and management of gastroesophageal varices and variceal hemorrhage in cirrhosis. *Hepatology*. 2007;46:922–38.
- Singal A, Sarin SK, Sood GK, Broor SL. Ulcers after intravariceal sclerotherapy: Correlation of symptoms and factors affecting healing. *J Clin Gastroenterol.* 1990;12(3):250–4.

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