



Cyanoacrylate Glue Injection for Bleeding Rectal Varices: An Uncommon but Effective Strategy

Fouad Haddad, Aicha Darif*, Zineb Boukhal, Fatima Zahra El rhaoussi, Mohamed Tahiri, Wafaa Hliwa, Ahmed Bellabeh and Wafae Badre

Gastroenterology and Hepatology Department, Ibn Rochd University Hospital Center of Casablanca, Morocco

***Corresponding Author:** Aicha Darif, Gastroenterology and Hepatology Department, Ibn Rochd University Hospital Center of Casablanca, Morocco.

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Abstract

In cirrhosis, hemorrhage from ruptured rectal varices can complicate portal hypertension syndrome. This is a rare complication, but it can be life-threatening. The management of active bleeding from rectal varices is not codified. There are no large series; the literature consists only of clinical cases. Generally, after diagnosis by recto-sigmoidoscopy, treatment is endoscopic (sclerotherapy or elastic ligation). We report the case of a 60-year-old cirrhotic patient admitted for massive hematochezia due to rupture of a rectal varicose vein. Treatment was endoscopic injection of N-Butyl-2-Cyanoacrylate, resulting in effective hemostasis without recurrence of bleeding.

Keywords: Hypertension; portal; varices; rectal bleeding; N-Butyl-2-Cyanoacrylate

Abbreviations

TIPS: Transjugular Intrahepatic Portosystemic Shunt

Introduction

Haemorrhage from ruptured varices secondary to portal hypertension is known to be one of the most frequent causes of death in patients with liver cirrhosis. The management of bleeding from ruptured esophageal or gastric varices is well codified according to the Baveno VII consensus, and treatment is, in the majority of cases, endoscopic by elastic ligation or glue injection [1].

Bleeding due to rupture of a rectal varices is a very rare occurrence, but one which may be life-threatening [2,3]. This is an ectopic localization of varicose veins in portal hypertension, which is well known but whose management remains unclear. The formation of varicose veins in the rectum is secondary to the anatomical presence of a network of collaterals linking the portal system (via the superior rectal vein) to the systemic system (via the middle and inferior rectal veins derived from the iliac network) [4].

The diagnosis should be suspected in the presence of any rectal bleeding in a cirrhotic patient. In published cases, treatment consists of either elastic ligation or sclerotherapy. N-butyl-2-cyanoacrylate injection has been used in only 3 or 4 cases in the literature. We report on a case of lower gastrointestinal haemorrhage due to rupture of a rectal varices in a 60-year-old patient with metabolic cirrhosis, in whom the haemorrhage was successfully managed by endoscopic injection of N-butyl-2-cyanoacrylate into the rectal varices.

Case Presentation

This is a 60-year-old diabetic patient on insulin, followed in our department for metabolic cirrhosis, with hemorrhagic and ascitic decompensation. Therapeutically, the patient is on: Propranolol 40mg/day in two doses and Spironolactone 100mg/day.

The patient was admitted to the hospital with moderate rectal bleeding dating back 3 days before his consultation, with no other symptoms. The patient presented with apyrexia, asthenia, and a poorly tolerated anemic syndrome.

On clinical examination, the patient was conscious but slowed, hypotensive to 07/04 mm Hg, heart rate at 63 bpm, respiratory rate at 16 bpm and SpO₂ at 98% on free air. The patient was icteric, with no flapping. His abdomen was soft with abundant ascites. Examination of the anal margin was unremarkable, and no mass was palpated on rectal examination, but the finger pad came back stained with reddish blood. The rest of the somatic examination was unremarkable.

The laboratory work-up showed anemia at 5.7g/dl, thrombocytopenia at 87 G/L, and a low Prothrombin of 27%. The renal work-up was without anomaly, and the hydro electrolytic work-up showed hyponatremia at 128mmol/L/L. As part of the search for a triggering factor, a cytobacteriological study of the urine revealed no infection, and the ascites fluid was transudate at 10g/L and a sterile culture. The chest X-ray was normal, and the abdominal ultrasound with Doppler study of the portal trunk revealed hepatopathy with signs of portal hypertension.

The therapeutic protocol used in our patient was that of hemorrhagic decompensation: Octreotide IV 50ug bolus then 50ug/hour, combined with antibiotic prophylaxis based on Ceftriaxone 1g/day and Lactulose 1tbs x3/day, and Rifaximin 600mg/day. Beta-blockers and diuretics were suspended. A blood transfusion was also given, with a target hemoglobin of 7-8 g/dl.

An esophagogastric fibroscopy was performed, showing grade 1 esophageal varices with no red signs and mild hypertensive gastropathy; there was no lesion or loss of substance or blood in the stomach.

Following these examinations, and with the aim of seeking the etiology of the bleeding, we completed a complete colonoscopy with ileal intubation, which showed varicose cords in the rectum extending from the middle rectum to the lower rectum. The largest of these varices contained red macular signs (Figure 1).

After multidisciplinary discussion, we hesitated the band ligation because the varices were too large, and the decision was to consider endoscopic gluing with cyanoacrylate. After local preparation with intra-rectal enemas, the procedure was performed un-

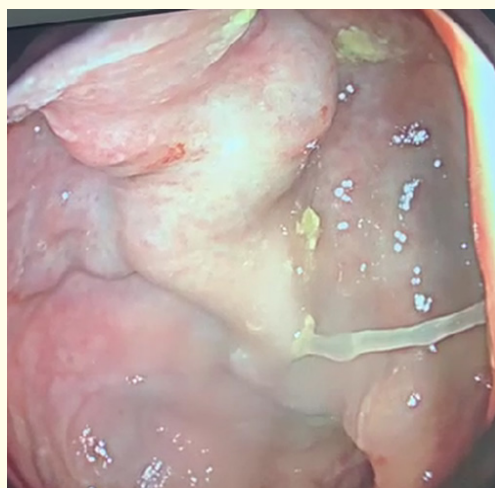


Figure 1: Endoscopic image of rectal varice with red macular signs.

der sedation. The varicose vein was injected with N-butyl-2-cyanoacrylate mixed with lipiodol (1:1) using a 6 mm, 21-gauge injection needle. Every 2 ml at 2 sites of the bleeding varicose vein, a total of 4 ml Histoacryl mixed with lipiodol was injected, without immediate incident. A post-procedure X-ray of the pelvis showed the glue in place (Figure 2).



Figure 2: X-ray of pelvis showing glue in place (D: Right).

Discussion

Rectal varices are a form of ectopic varices that arise as a result of portal hypertension and represent an uncommon but potentially life-threatening source of lower gastrointestinal bleeding. While ectopic varices can occur throughout the digestive tract, rectal varices are among the most frequently identified during endoscopic evaluations in cirrhotic patients, with prevalence ranging from 3.6% to 90% [5,6]. Despite this, active rectal variceal bleeding is rare, occurring in only 1%–8% of all variceal bleeding episodes [7–9].

Diagnosis of rectal varices can be challenging during active bleeding due to clot formation and limited visibility [10], although modern endoscopic and imaging tools (such as endoscopic ultrasound and MR venography) have improved detection [8,11,12]. Once identified, management remains controversial due to the limited number of reported cases and the absence of randomized trials.

Medical treatments such as octreotide and beta-blockers have shown limited efficacy in acute bleeding. Transjugular intrahepatic portosystemic shunt (TIPS) has been proposed as a treatment that reduces portal pressure and prevents rebleeding, with some success reported [8,13]. However, its use in decompensated cirrhosis carries a high risk of hepatic encephalopathy and stent thrombosis. Endoscopic approaches like band ligation [14,15] and sclerotherapy have also been used, though the latter is associated with complications such as post-sclerotherapy ulceration, necrosis, or perforation, especially when large volumes of sclerosant are injected [10,16,17].

N-butyl-2-cyanoacrylate (Histoacryl), a tissue adhesive widely used in the treatment of gastric varices, has shown promising but underreported results in the management of bleeding rectal varices. Its rapid polymerization upon contact with blood leads to plug formation and immediate hemostasis. Although most of the literature consists of isolated case reports, a few studies have described successful outcomes using cyanoacrylate for rectal or other ectopic varices [18–20].

In the seminal study by Soehendra, *et al.*, Histoacryl was successfully used in a large series of bleeding gastric varices, establishing its hemostatic potential [21]. Chen *et al.* later reported

its use in rectosigmoid variceal bleeding with initial hemostasis; however, the patient unfortunately died from rebleeding four days later, highlighting the importance of long-term control of portal hypertension [22]. In contrast, Soo, *et al.* described a case in which a single Histoacryl injection into a large bleeding rectal varices led to effective and sustained hemostasis without complications. Weekly endoscopic follow-up showed a significant reduction in the variceal size and extrusion of the glue cast, with no evidence of ulceration, necrosis, or embolization [23].

Recent case reports have emphasized the advantage of glue injection over traditional endoscopic therapies like sclerotherapy and band ligation, especially in rectal varices where the anatomy and size of the varices may render mechanical ligation less effective [19,20,24]. Moreover, glue injection may serve as a bridge therapy in patients unfit for TIPS due to advanced cirrhosis, hepatic encephalopathy risk, or technical limitations.

In our case, cyanoacrylate injection resulted in immediate cessation of bleeding, radiological confirmation of glue placement, and no evidence of recurrence or complication, supporting the growing evidence that glue injection is a valuable and safe option in the emergency treatment of bleeding rectal varices. This case reinforces the utility of endoscopic cyanoacrylate injection as a feasible and effective alternative for managing bleeding rectal varices, particularly when more invasive treatments like TIPS are contraindicated or high risk due to advanced liver disease.

Conclusion

Hemorrhage from ruptured rectal varices is a rare condition, but one that carries a high risk of complications. Endoscopic treatment is the first option, combined with medical therapeutic measures in line with the Baveno VII consensus. Apart from elastic ligation and sclerotherapy, glue injection with Histoacryl may also be a good option for controlling bleeding.

Acknowledgements

None.

Conflict of Interest

None.

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