



Letter to the Editor: Current approaches to the management of colonic variceal bleeding

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Abstract

In a case report published in the *World Journal of Gastroenterology*, Tomita *et al* presented a patient without liver cirrhosis and portal hypertension with recurrent colonic variceal bleeding at the site of transverse colon anastomosis who was successfully treated by percutaneous transhepatic embolization using ethanolic oleate and N-butyl-2-cyanoacrylate. Colonic varices are a very rare cause of lower gastrointestinal bleeding. Various methods including pharmacotherapy, endoscopic modalities, interventional radiology procedures, and partial colectomy have been described as treatment options for colonic variceal bleeding. Nevertheless, the optimal approach has not yet been established due to the lack of randomized controlled trials involving a large cohort of patients necessary for its development.

Key Words: Colonic variceal bleeding; Management; Pharmacotherapy; Endoscopic modalities; Interventional radiology procedures; Partial colectomy

Core Tip: Various methods including pharmacotherapy, endoscopic modalities, interventional radiology procedures, and partial colectomy have been described as treatment options for colonic variceal bleeding. Nevertheless, the optimal approach has not yet been established, due to the lack of randomized controlled trials involving a large cohort of patients necessary for its development.

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TO THE EDITOR

I read with interest a case report published in the *World Journal of Gastroenterology*, where Tomita *et al*[1] presented a patient without liver cirrhosis and portal hypertension with recurrent colonic variceal bleeding at the site of transverse colon anastomosis who was successfully treated by percutaneous transhepatic embolization using ethanolamine oleate and N-butyl-2-cyanoacrylate. He had previously undergone a laparoscopic transverse colectomy for the mid-transverse colon cancer. Reconstruction was performed using a functional end-to-end anastomosis technique.

Gastrointestinal varices in patients with portal hypertension are formed as a result of the development of portosystemic collateral circulation. Since Gilbert and Carnot[2], it has been believed that the formation of portosystemic shunts is associated with the opening and expansion of pre-existing vessels in the areas of embryonic communications between the portal and systemic circulatory channels due to increased portal pressure. Currently, the important role of active hypoxia-induced angiogenesis in this process has been proven[3,4]. At the same time, the cause of the formation of gastrointestinal varices in the absence of portal hypertension is sometimes unclear.

Colonic varices are a very rare cause of lower gastrointestinal bleeding. According to autopsy data, their prevalence may be 0.07%[5]. As a rule, colonic varices is formed as a result of portal hypertension in liver cirrhosis or extrahepatic portal vein obstruction[6]. There are cases of colonic varices in consequence of splenic vein thrombosis[7], portomesenteric venous thrombosis[8], external compression of the splenic vein in tumor invasion[9], and chronic superior mesenteric vein occlusion after pancreaticoduodenectomy[10] in case of development of collateral veins adjacent to the colon, as well as due to inferior mesenteric arteriovenous fistula[11]. They can also be a manifestation of Klippel-Trenne-Weber syndrome[12]. Sometimes the reason of colonic varices remains unknown[13]. A case of massive colonic variceal bleeding after endoscopic injection sclerotherapy for esophageal varices, transection and devascularization of the esophagus in a patient with extensive thrombosis of the portal and splenic veins is described. This occurred as a result of obliteration of the coronary-azygos collateral canal system and the opening of the retrograde portal flow through the mesenteric vein[14]. Colonic varices at the site of anastomosis are extremely rare. Only a few clinical cases are described in the available literature[15-17].

Currently, there are no randomized controlled trials or specific evidence-based guidelines for the management of ectopic variceal bleeding (including colonic varices). Therapeutic strategy depends on the location of the bleeding, its severity, the presence of portal hypertension and its cause, the institutional experience[18]. Besides, a thorough knowledge of the vascular supply to the ectopic varices is very necessary, in particular, the pathways of portosystemic collateral circulation. Afferent vessels carrying blood to the colonic varices include the ileocolic vein, the right, middle, left, and sigmoid colon veins. The efferents are the right gonadal vein, the right renal vein, and the systemic lumbar veins, which drain into the retroperitoneal veins of Retzius, as well as the veins of the ascending colon, which drain blood through the capsular renal veins into the inferior vena cava[19].

Various methods including pharmacotherapy (octreotide and β -blocker)[20], endoscopic modalities, such as ethanolamine oleate injection[21], N-butyl-2-cyanoacrylate (histoacryl) injection[22,23], clipping[24], band ligation[25], and argon plasma coagulation[26], as well as coil embolization and histoacryl injection[27], percutaneous transhepatic embolization[28], transileocolic vein obliteration[29], balloon-occluded retrograde transvenous obliteration[30,31], coil-assisted retrograde transvenous obliteration[32,33], transjugular intrahepatic portosystemic shunt (TIPS) with collateral embolization[34,35], and partial colectomy[11] have been described, usually in case reports, as treatment options for colonic variceal bleeding. Besides, cases of colonic variceal bleeding have been reported in patients without liver cirrhosis or portal hypertension caused by thrombosis of the splenic or superior mesenteric veins, which were successfully cured by interventional transhepatic recanalization and subsequent stenting of the corresponding veins[7,8]. However, owing to the rarity of these lesions, no standardized treatment strategy has been established. It is usually determined by the experience and technical capability of the clinic, as was described in case report by Tomita *et al*[1].

According to the current guidelines, pharmacotherapy and endoscopic modality should be the first line of therapy if bleeding ectopic varices are available for endoscopic imaging. However, in inaccessible cases, TIPS or percutaneous transhepatic varices embolization should be performed in patients with a patent portal vein and cirrhotic and non-cirrhotic portal hypertension[36]. Balloon-occluded retrograde transvenous obliteration could be considered as an alternative to endoscopic treatment or TIPS, provided it is feasible (type and diameter of shunt) and local expertise is available. TIPS may be combined with collateral embolization to control bleeding or to reduce the risk of recurrent ectopic variceal bleeding, particularly in cases when, despite a decrease in portosystemic pressure gradient, portal flow remains diverted to collaterals[37].

Conclusion

A case report by Tomita *et al*[1] has shown that treatment strategy for colonic variceal bleeding can consist in a multidisciplinary approach and the definition of individual therapeutic option for each patient. Due to the ineffectiveness of endoscopic clipping, which was performed during the first and second bleeding episodes as the first line of therapy, and the absence of portal hypertension, percutaneous transhepatic embolization of colonic varices was chosen as the second line of therapy. Given the diffuse submucosal variceal network and multifocal drainage, the combined ethanolamine oleate + N-butyl-2-cyanoacrylate, together with flow control (temporary balloon occlusion of the inflow channel and selective embolization of the efferent veins), in this clinical situation was optimal to achieve reliable hemostasis while minimizing non-target embolization and bowel ischemia. The long-term results confirmed the correctness of the approach chosen by the authors. Nevertheless, the preferred management strategy for colonic variceal bleeding has not yet been established. Its development with the introduction of an optimal clinical algorithm will be possible in the presence of randomized controlled trials involving a large cohort of patients.

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