

Duodenal and jejunal varices due to superior mesenteric vein thrombosis presenting as a massive gastrointestinal tract bleeding: a case report

Case Report

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Abstract: Duodenal and jejunal varices usually occur due to portal hypertension and are rare causes of gastrointestinal tract bleeding. We report the case of a patient with no clinically significant history of a coagulopathy disorder, cirrhosis, or portal hypertension who presented with duodenal and jejunal varices that resulted in lower gastrointestinal bleeding with superior mesenteric vein thrombosis. Mesenteric angiography revealed superior mesenteric vein thrombosis and varices at the fourth part of the duodenum and the proximal jejunum draining into the main portal vein, but no active bleeding source was recognized. The patient was hemodynamically unstable. Emergency laparotomy was performed and revealed duodenal and jejunal varices, with occlusion of the superior mesenteric vein. Partial resection of the duodenum and jejunum and end-to-end anastomosis were performed. After surgery, the patient was hemodynamically stable with no recurrence of bleeding.

Keywords: Duodenal varices • Jejunal varices • Superior mesenteric vein thrombosis • Lower gastrointestinal tract bleeding

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1. Introduction

Duodenal and jejunal varices usually result from portal hypertension and are rare causes of gastrointestinal tract bleeding (GITB) [1,2]. There are a few cases in the literature of patients without portal hypertension who had duodenal or jejunal varices that resulted in GITB [3,4].

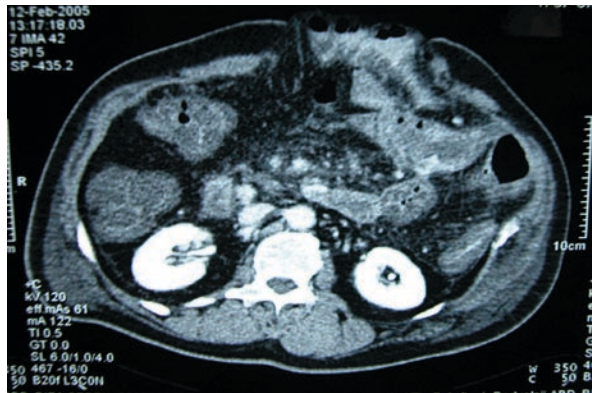
We report the case of a patient with no significant history of a coagulopathy disorder, cirrhosis, or portal hypertension who presented with duodenal and jejunal varices that resulted in massive GITB with superior mesenteric vein thrombosis.

2. Case Report

A 51-year-old man presented to our emergency service with hematochezia. His medical history included deep vein thrombosis of the right lower extremity, type 2 diabetes mellitus, and hypertension. He had a surgical history of partial resection of the small bowel with end-to-end anastomosis due to strangulation of an umbilical hernia 3 years prior to admission. He was taking coumarin. His blood pressure was 110/70 mm Hg, and his heart rate was 120 beats per minute. A rectal examination did show only hematochezia. The physical examination did not reveal jaundice, ascites, or other

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Figure 1. Duodenal varices and jejunal collaterals seen on abdominal CT.



signs of hepatic disease; a tumor mass; or a bruit from an abdominal vascular lesion.

The initial serum hemoglobin level was 8.4 g per deciliter, and the hematocrit was 23.4%. The control hemoglobin level was 6.5 g per deciliter, and the control hematocrit was 19%. The International Normalized Ratio (INR) was 1.4.

He was admitted for further work-up without placement of a nasogastric tube. Initial gastroduodenoscopy showed a nonbleeding peptic ulcer on a clean base on the anterior surface of the second part of the duodenum. Colonoscopy demonstrated black clots of blood throughout the colon. He had a serum hemoglobin level of 6 g per deciliter and a hematocrit of 18% after 4 units of packed red cells and 2 units of fresh frozen plasma. Abdominal computed tomography (CT) was normal except for the presence of duodenal varices

and jejunal collateral vessels (Figure 1). Endoscopic examinations did not yield a definitive diagnosis for an ongoing bleeding source. Mesenteric angiography was then performed; it revealed superior mesenteric vein thrombosis and varices at the fourth part of the duodenum and the proximal jejunum that drained into the main portal vein, but no active bleeding source was recognized (Figure 2a-2b). In the follow-up period, the patient continued to have massive gastrointestinal bleeding, despite medical therapy, and he was hemodynamically unstable, with a blood pressure of 80/60 mm Hg and heart rate of 130 beats per minute. Emergency laparotomy revealed duodenal and jejunal varices and occlusion of the superior mesenteric vein (Figure 3). Partial resection of the duodenum and jejunum with an end-to-end anastomosis was performed. The patient required 6 units of packed red cells and 2 units of fresh frozen plasma during surgery. After surgery, he was hemodynamically stable, with a blood pressure of 110/70 mm Hg and heart rate of 86 beats per minute. Partial resection of the duodenum and jejunum resulted in complete resolution of the bleeding. On the first postoperative day, the rheumatology department was consulted, and prednisolone was started in case of the antiphospholipid syndrome. He had protein C, protein S, and antithrombin III deficiency [the protein C level was 27% (normal range, 70-250%), the protein S level was 19% (65-250%), and the antithrombin III level was 26% (80-120%)]. Other coagulation laboratory results were normal [tests for antinuclear antibody, anti-double-stranded DNA, and factor V Leiden mutation were negative; the level of anticardiolipin IgM antibody was < 6.25 U/ml (normal range, <15 U/ml),

Figure 2. Angiography with superior mesenteric artery injection (a) revealed duodenal varices and proximal jejunal collaterals, with a patent portal vein. The superior mesenteric vein is occluded; no extravasation is seen (b).

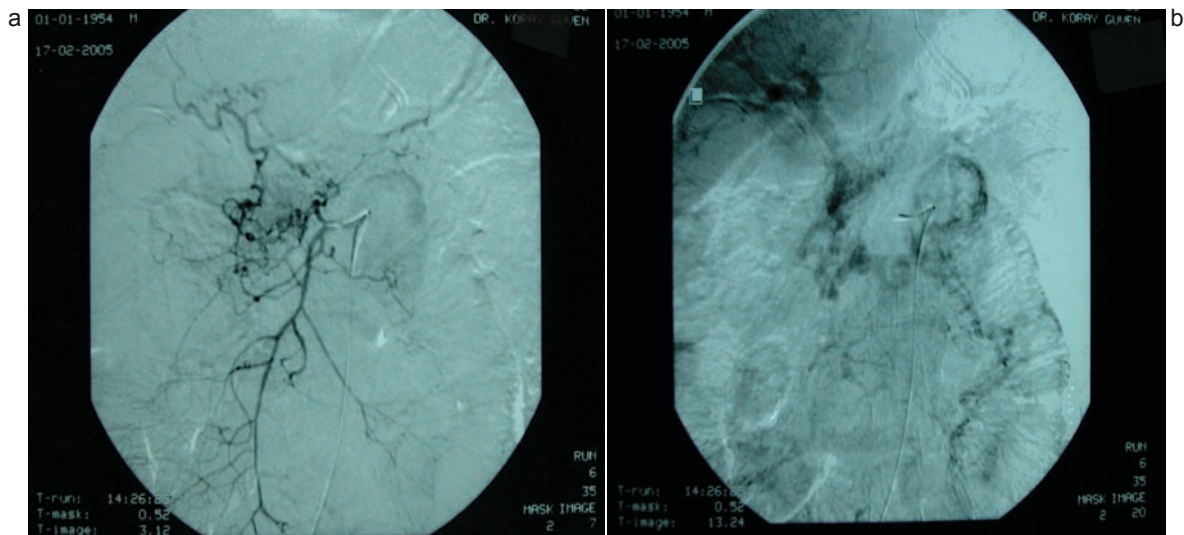
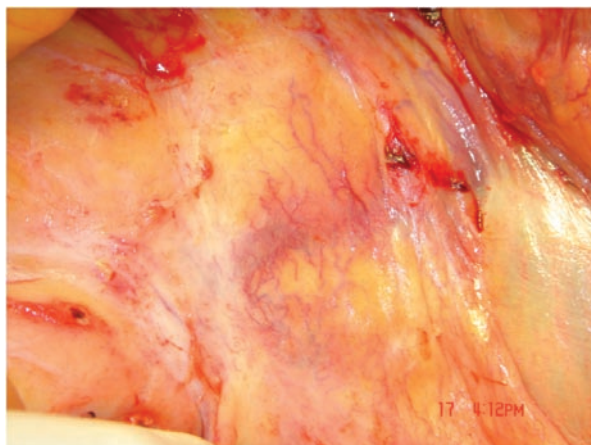


Figure 3. Varices on the mesenteric side of the jejunum.

and the anticardiolipin IgG antibody was <6.25 U/ml (<15 U/ml). Histological examination of specimens of the resected duodenum and jejunum demonstrated ruptured and unruptured submucosal varices. He remained hemodynamically stable, and no recurrence of bleeding was detected.

3. Discussion

Esophagogastric varices are a common manifestation of portal hypertension and are the most common site of variceal bleeding. Other than ectopic varices seen in esophageal and gastric lesions, they are commonly found in the duodenum and can also be present in the jejunum, ileum, colon, and rectum, as well as at the stoma of an enterostomy and at the site of intestinal anastomosis [5]. Duodenal and jejunal varices typically are associated with both extrahepatic portal vein obstruction and cirrhosis, without an existing hepatic disease they are a very rare cause of GITB. In our patient, duodenal and proximal jejunal varices were probably related to the formation of collateral vessels around the thrombosed superior mesenteric vein that connected the first jejunal and pancreaticoduodenal veins and drained into the patent portal vein (Figure 2b).

Harward *et al.* [6] reported 16 patients with mesenteric venous thrombosis, 3 of whom presented with GITB and 7 of whom had a previous history of deep vein thrombosis. Coagulopathy was identified in nine patients. Six patients had protein C deficiency, two protein S deficiency, and one factor IX deficiency. Portal vein thrombosis was detected in three patients. In another study Berney *et al.* [7] reported 45 patients with portal and superior mesenteric vein thrombosis. They classified these cases according to cause and found

that 47% of the patients had cirrhosis and 33% had pancreatitis. Our patient had protein C, protein S, and antithrombin III deficiencies and superior mesenteric vein thrombosis, but no cirrhosis or pancreatitis. His history of deep vein thrombosis required continuous treatment with coumarin.

Evaluation of gastrointestinal bleeding in patients who have had previous abdominal surgery and mesenteric venous hypertension should include small-bowel anastomotic and adhesion-related varices, despite their infrequency. These types of varices are associated with intraabdominal adhesions, especially post-operative ones [8,9]. In one case series, four patients with GITB due to trans-anastomotic porto-portal varices were reported; all of these patients had a previous history of abdominal surgery, and all four anastomotic varices resulted in lower gastrointestinal bleeding [10]. Tang *et al.* reported a case of recurrent GITB caused by jejunal anastomotic varices that were secondary to superior mesenteric vein occlusion after an abdominal gunshot wound [11]. Although our patient had had an intestinal anastomosis 3 years previously, at the time of the emergency laparotomy on inspection of the anastomosis, no varices were detected.

A key first step in the evaluation of gastrointestinal bleeding is typically to localize the bleeding site to either the upper or lower tract. Patients who have hematemesis usually have a lesion proximal to the ligament of Treitz. Although melena is typically associated with upper gastrointestinal bleeding and hematochezia with lower GITB, it should be emphasized that patients with slow oozing from the distal small bowel or cecum may have melena and patients with aggressive bleeding from an upper gastrointestinal source may occasionally present with hematochezia.

If clinically evident bleeding persists or recurs after negative endoscopic examinations, it is termed obscure overt gastrointestinal bleeding [12]. When upper and lower endoscopic examinations do not yield a definitive diagnosis, further evaluation depends on the severity of bleeding. In patients with subacute or intermittent bleeding, the focus of investigation should rapidly move to the small bowel. Small-bowel examination can be performed with a number of radiological modalities (enteroclysis, CT, magnetic resonance imaging, angiography and technetium scanning), as well as endoscopic (double-balloon enteroscopy and capsule endoscopy) and surgical (exploratory laparotomy) techniques [13]. In patients with ongoing bleeding, angiography or technetium-99 radionuclide scanning should be performed to confirm the source of bleeding. Although, angiography is invasive and less sensitive than technetium-99 radionuclide scanning, it may

allow for treatment by coiling the bleeding lesion [14]. Angiography can also identify nonbleeding lesions by the vascular pattern, such as vascular ectasias [15]. In our case, we performed angiography because it was likely to reveal a source of the aggressive bleeding, and specific therapy might then be possible. For patients such as ours, angiography, with particular attention to both the arterial and venous phases, is the procedure of choice [16].

There is currently no definitive conservative therapy for duodenal varices. Vasoactive drugs may be administered initially to control the bleeding. If bleeding duodenal ectopic varices are visualized at endoscopy, endoscopic therapy (sclerotherapy or band ligation) can be carried out [17,18]. Interventional radiological treatment methods such as percutaneous transcatheter embolisation (PTCE) and transjugular intrahepatic porto-systemic shunts (TIPS) have also been used to treat duodenal varices [19]. The rate of rebleeding after

PTCE is high because the portal hypertension persists. In patients in whom embolization fails to prevent rebleeding, TIPS placement may be considered. In our case with massive bleeding, PTCE and TIPS were not the treatment of choice because of the patent portal vein; partial resection of the duodenum and jejunum with end-to-end anastomosis resulted in complete resolution of the bleeding.

In conclusion, duodenal and jejunal variceal bleeding due to superior mesenteric vein thrombosis is a very rare condition, and ectopic variceal bleeding should be considered in all patients with obscure overt gastrointestinal bleeding, even if it occurs in patients without portal hypertension or cirrhosis. Angiography, with attention to both arterial and venous phases, is the preferred diagnostic procedure. The management of individual cases varies greatly depending on the specific features of the case and local expertise.

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