

Markus Naef^a
Fernando Holzinger^a
Andreas Glättli^a
Beat Gysi^b
Hans Ulrich Baer^a

Massive Gastrointestinal Bleeding from Colonic Varices in a Patient with Portal Hypertension

^a Department of Visceral and
Transplantation Surgery and
^b Gastroenterology Unit, Inselspital,
University of Berne, Switzerland

Key Words

Colonic varices
Gastrointestinal bleeding
Portal hypertension
Portal vein thrombosis

Abstract

Colonic variceal bleeding is a rarity and is most commonly due to portal hypertension. The present report describes a patient with portal hypertension due to portal vein thrombosis who, following esophageal transection and successful sclerotherapy, developed a massive lower gastrointestinal bleeding from colonic varices. The literature is reviewed, and the pathophysiology of this complication is discussed. Possible etiologies of this condition may be esophageal transection and devascularization, successful sclerotherapy, and extensive thrombosis of the portal vein resulting in obliteration of the coronary-azygous anastomotic system. In such a situation other potential sites of portosystemic anastomoses, such as the colon, may be opened up, resulting in the development of colonic varices. Indeed, the incidence of colonic varices in two series after sclerotherapy for esophageal varices was 60-100%. Of 33 candidates evaluated for liver transplantation, colonic varices were found in 1.

Introduction

Upper gastrointestinal bleeding from esophageal or fundal varices is a well-known complication of portal hypertension. Lower gastrointestinal bleeding from colonic varices is less known, with about 50 cases reported in the literature [1-17].

Recently, a new entity, portal hypertensive colonopathy or portal hypertensive intestinal vasculopathy, has been attributed to this condition [12, 18].

In the present report, we describe a patient with portal hypertension presenting with massive rectal bleeding from colonic varices. The literature is reviewed, and possible etiologies for this condition are discussed.

Case Report

A 27-year-old male with a long history of portal hypertension due to portal vein thrombosis (following exchange transfusion for Rhesus factor incompatibility as a neonate) and known former esophageal varices was referred to our hospital with acute lower gastrointestinal bleeding. Since infancy he had repeated episodes of upper gastrointestinal variceal bleeding which were treated by esophageal transection, devascularization, and sclerotherapy. On admission, he was hemodynamically stable. Serum laboratory tests disclosed hemoglobin 11.6 g/dl, serum calcium 1.94 mEq/l, total bilirubin 66.0 µmol/l, albumin 28.0 g/l, and prothrombin time 66%. Upper endoscopy revealed scars from previous sclerotherapy, but no esophageal or fundal varices. Variceal ectasias were present in the first part of the duodenum, but without active bleeding. Selective angiography of the celiac axis and both superior and inferior mesenteric arteries with indirect splenoportography verified the presence of portal vein

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M. Naef, MD
Department of Visceral and Transplantation Surgery
Inselspital
CH-3010 Berne (Switzerland)

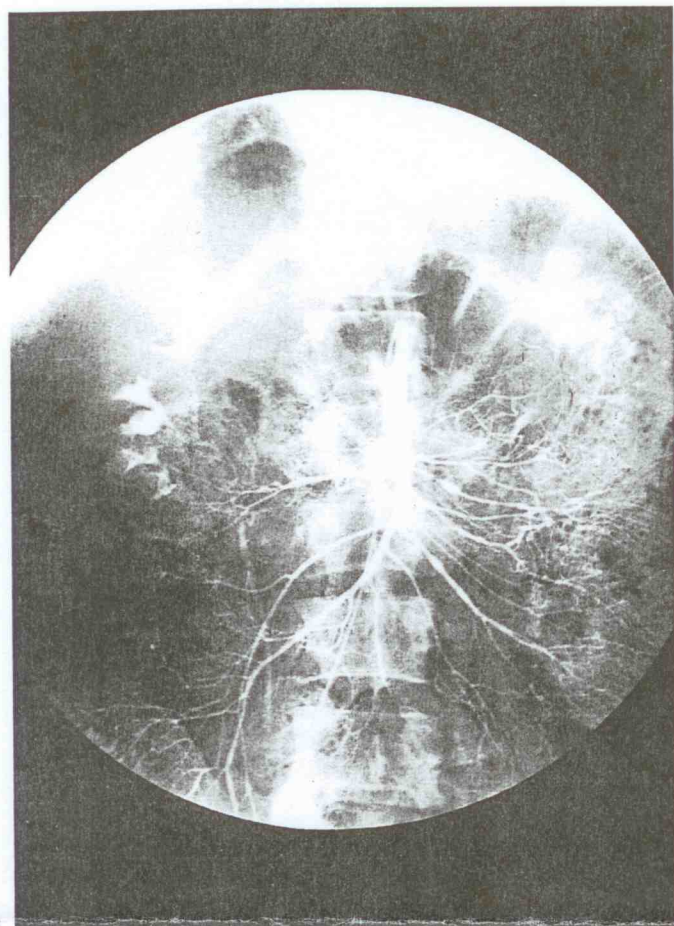


Fig. 1. Selective angiography of the superior mesenteric artery in a patient with massive lower gastrointestinal bleeding and a history of portal hypertension due to portal vein thrombosis. No active bleeding is demonstrated.

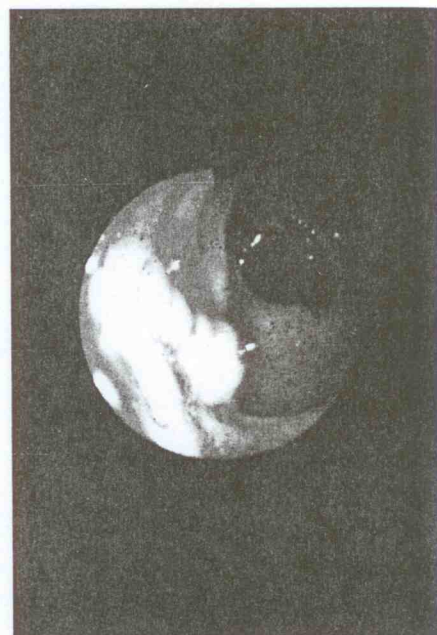


Fig. 2. Emergency colonoscopy demonstrating an active bleeding site in the transverse colon near the hepatic flexure from two colonic varices, one with a cherry spot lesion.

thrombosis with extensive collateral circulation. The splenic vein was also thrombosed. However, no active bleeding was demonstrated (fig. 1). Two days later he developed massive lower gastrointestinal bleeding necessitating transfusion of 13 packed red cells, 8 fresh frozen plasma, and 6 platelet units over 4 h. Emergency colonoscopy demonstrated an active bleeding site in the transverse colon near the hepatic flexure from two varices, one with a cherry spot lesion (fig. 2). An attempt at sclerotherapy was unsuccessful. Since he remained hemodynamically unstable, an emergency laparotomy was performed.

At surgery significant portal hypertension with ascites was found. The liver appeared macroscopically normal. Multiple variceal ectasias of the veins of gallbladder, right colon, and mesentrium of the small bowel were found. The colon was full of blood. Intraoperative colonoscopy revealed no additional active bleeding sources in the remaining colon, although several small varices were seen in the left colon. A right colectomy with end-to-end ileotransversostomy was performed. During the operation the patient received 34 packed red cells, 26 fresh frozen plasma, and 18 platelet units. The postoperative course was uneventful, and he was discharged after 10 days. He was readmitted 3 days later with the clinical picture of peritonitis. At operation he was found to have bacterial peritonitis (*Streptococcus salivarius*), but with no evidence of an anastomotic leak or a perforated ulcer.

Under antibiotic treatment (imipenem/cilastatin; Tienam[®]) he made full recovery and was discharged after 11 days. At follow-up after 6 months, he has had no evidence of recurrent gastrointestinal bleeding.

Discussion

Gastrointestinal bleeding associated with portal hypertension occurs most commonly from esophageal or gastric varices. In addition, congestive gastropathy and peptic ulcer disease are important causes of hemorrhage in cirrhotic patients [19, 20]. While these situations usually do not create diagnostic difficulties, bleeding from colonic varices associated with portal hypertension is a rarity and may be difficult to diagnose.

Varices of the portal system may occur in regions of potential portosystemic anastomoses [6]. These are usually found in the lower esophagus and in the stomach, but may also occur in terminal ileum, ascending colon, and

left colon. With an increased flow and/or a higher pressure, these portosystemic anastomotic veins may get ectatic and varicose [21]. This situation may be present with portal hypertension [22], obstruction of the mesenteric vein, congestive heart failure, and congenital vascular anomaly [12, 23]. Though the small bowel and the transverse colon are usually devoid of these potential shunts [12], recent studies suggest that similar varices may be seen in the small bowel [9, 22].

The most common cause of colonic variceal bleeding is portal hypertension. Rarely, other conditions, such as bowel resection and adhesion-related operations [24–26], congenital vascular anomalies [27], and idiopathic vasculopathy [28–31] are implicated. Although portal hypertension is quite common, colonic varices are rarely found. Available data concerning the prevalence of colonic vascular ectasias in cirrhotic patients are few and contradictory [12]. About 50 cases of bleeding colonic varices are reported in the literature, mostly single case reports and short series [1–17]. This is probably related to the rich coronary-azygous anastomotic system, shunting most of the blood [6]. In a large series of 412 cirrhotic patients evaluated for liver transplantation, no vascular abnormalities were found at colonoscopy [32]. However, in a series of 20 highly selected patients with portal hypertension who underwent endoscopy essentially for gastrointestinal bleeding, 14 patients (70%) were found to have multiple colonic ectasias. Twelve of these patients (60%) have previously had sclerotherapy for esophageal variceal bleeding prior to the diagnosis of colonic ectasias [8]. Once patients are treated with sclerotherapy or esophageal transection and devascularization resulting in obliteration of the coronary-azygous anastomotic system, other potential sites, such as the colon, are opened up. Indeed, in two series the incidence of colonic varices found after sclerotherapy for esophageal varices is 60–100% [8, 15].

In addition, we have examined patients evaluated for liver transplantation for the occurrence of colonic varices. During an 18-month period 33 candidates for liver transplantation underwent colonoscopy; 19 were found to have esophageal varices (58%), of whom 12 underwent sclerotherapy (36%). In 1 patient who has had previous sclerotherapy, multiple colonic varices of the right and transverse colon were found.

The clinical presentation is usually that of a rectal bleeding. Therefore, in a patient with liver cirrhosis and portal hypertension, once an upper gastrointestinal tract bleeding source is ruled out, a lower gastrointestinal tract bleeding site has to be looked for. The first diagnostic investigation is anorectoscopy to rule out the presence of

bleeding hemorrhoids or rectal varices [33]. Once these are ruled out, colonoscopy is performed. It has to be mentioned that the varices may collapse and not be seen, if excessive air insufflation is used. When it is impossible to locate the bleeding source, and the patient is actively bleeding, red blood cell scintigraphy and/or angiography should be performed [1, 6, 8, 25].

The therapeutic approach is controversial. There are three possible modalities: conservative treatment, colonic resection, or portosystemic shunting. Conservative treatment is usually successful in minor bleeding episodes with only hematochezia. Recent studies suggest that beta blockers improve endoscopic grading and decrease subsequent bleeding from gastric ectasias in portal hypertension [8, 34, 35]. The effect of beta-blocking agents on the colonic varices of portal hypertension is unknown.

Massive bleeding from colonic varices in portal hypertension is a grave situation, and once the bleeding has not stopped following correction of coagulopathy and replacement therapy, emergency surgery is indicated. Therefore, every effort has to be made to exactly locate the bleeding site. Once it is located, resection of the involved colonic segment seems to be the procedure of choice. There are several authors stressing the point that in patients with a good liver function and without associated vascular anomalies, selective shunting seems to be the safer procedure concerning short-term outcome [6, 7, 9, 13, 17], but long-term follow-up results are not available. However, mesocaval or atypical cavernocaval shunting is technically possible and may be taken into consideration, since a bowel anastomosis itself is known to be a predominant site for the development of bleeding varices.

It remains controversial if patients who have had their esophageal varices fully eradicated by sclerotherapy should be followed up by colonoscopy for the early occurrence of colonic variceal lesions.

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