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

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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

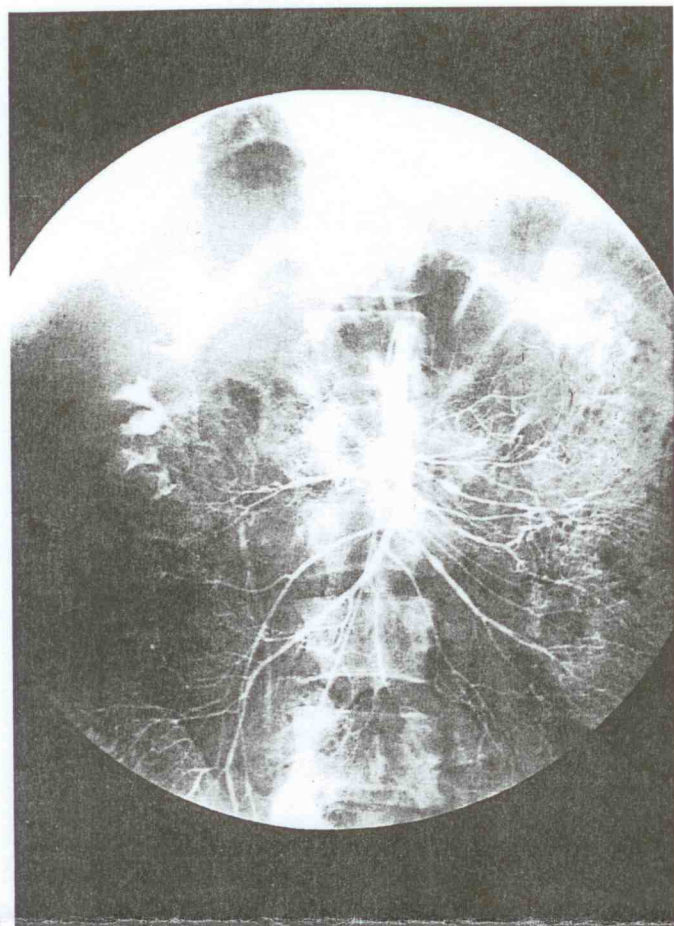
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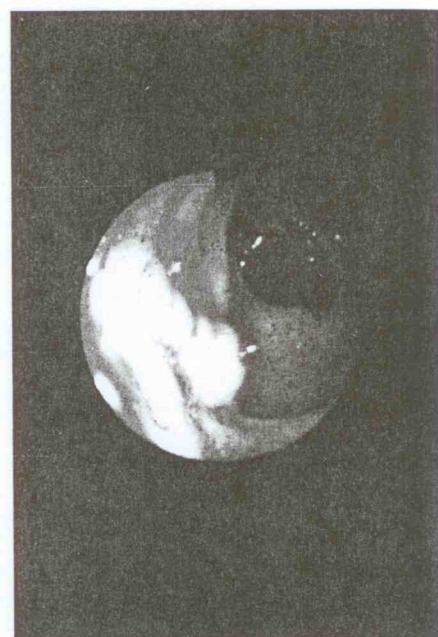
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Fig. 1. Selective angiography of the superior mesenteric artery in a patient with massive lower gastrointestinal bleeding and a history of portal hypertension due to portal vein thrombosis. No active bleeding is demonstrated.

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

