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Duodenal varices as an unusual cause of gastrointestinal bleeding due to portal hypertension: A case report

Portal hipertansiyona bağlı nadir bir gastrointestinal kanama olgusu: Olgu sunumu

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Duodenal varices that develop in patients with portal hypertension rarely cause hemorrhage, but varix rupture is a serious and often fatal event. We report the case of a 38-year-old man with hepatic vein occlusion who was referred to our hospital for gastrointestinal hemorrhage of unknown origin. Upper gastrointestinal endoscopy revealed varices in the distal third of the duodenum. These varices were identified as the source of the bleeding. The patient was treated with endoscopic band ligation, and with coil embolization of a shunt between the superior mesenteric vein and the left renal vein.

Keywords: Upper gastrointestinal bleeding, duodenal varix

Portal hipertansiyonu olan olgularda gelişen duodenal varislerden kanama nadir görülen fakat ciddi ve sıklıkla ölümcül seyredebilen bir durumdur. Bu makalede merkezimize nedeni bilinmeyen üst gastrointestinal sistem kanaması ile gönderilen 38 yaşında hepatik ven oklüzyonu olan bir hastayı sunuyoruz. Kanamanın nedeni olarak üst gastraointestinal sistem endoskopisinde duodenum 3. kıtasında varisler tesbit edildi. Tedavide varisler endoskopik band ligasyonu ile bağlanıp, superior mezenterik ven ile sol renal ven arasındaki şanta da koil embolizasyonu yapıldı.

Anahtar kelimeler: Üst gastrointestinal kanama, duodenal varis

INTRODUCTION

Variceal bleeding is one of the main causes of death in patients with portal hypertension. Although bleeding from gastroesophageal varices occurs commonly in patients with portal hypertension, bleeding from varices located at other sites is unusual. Duodenal varices are rare and a high index of suspicion is required to detect them. The treatment of duodenal varices depends on the physician's experience, and must be decided on a case-by-case basis. Here we report a case of recurrent gastrointestinal bleeding caused by duodenal varices formed secondary to hepatic vein occlusion.

CASE PRESENTATION

A 38-year-old man was referred to our hospital for investigation of recurrent upper gastrointestinal bleeding of unknown origin. One month earlier, he had presented to another center vomiting fresh blood. Endoscopic studies and a laboratory work-up at that time had revealed esophageal varices but no obvious site of bleeding. Ultrasonography of the liver performed at this center showed splenomegaly and ascites indicative of chronic liver disease, and laboratory investigation revealed HBsAg (hepatitis B surface antigen) positivity. The patient was transfused with 12 units of packed red blood cells, but experienced recurrent bleeding episodes during his hospital stay. He was referred to our center for further investigation.

On admission to our hospital, physical examination showed normal vital signs, mild jaundice, and a palpable spleen below the costal margin. There was no history of alcohol abuse, intravenous-drug abuse, or use of alternative medicine.

Routine laboratory investigations revealed the following: hemoglobin 11.5 g/dl; white blood cell count 8.1×10^3 cells/mm^3; platelet count 83.9×10^3 cells/mm^3; total protein 4.5 g/dl; albumin 2.5 g/dl;

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total bilirubin 4.2 g/dl; direct bilirubin 2.5 g/dl; alkaline phosphatase 193 U/L; alanine aminotransferase 261 U/L; aspartate aminotransferase 183 U/L; prothrombin time 20.6 s; INR: 1.8; positive serology for HBsAg and anti HBe; negative serology for anti-HBs, anti-hepatitis C virus antibodies, and anti-hepatitis D antibodies; and no detection of hepatitis B virus DNA with polymerase chain reaction methods.

In light of the patient's ongoing hematochezia, emergency upper gastrointestinal system (UGI) endoscopy was performed. This revealed grade II esophageal varices and a duodenal ulcer that was not actively bleeding. The patient also underwent colonoscopy for investigation of possible colon lesions; however, since complete colon preparation had not been done, we were unable to examine the colonic mucosa beyond the sigmoid flexure. The patient was admitted to the intensive care unit to follow.

After this bleeding episode resolved the patient did a full colonic preparation and colonoscopy was repeated. This revealed nothing that could explain the bleeding. At this stage, we repeated UGI endoscopy using a pediatric colonoscope to allow assessment of the more distal part of the duodenum. This demonstrated varices in the distal third of the duodenum. The lesions were not actively bleeding at the time of detection; however, they started to bleed during the course of the endoscopic examination. The bleeding varix was identified and successfully band ligated, and this resolved the problem (Figure la, lb).

The patient's hemoglobin level was still stable at this stage, and he experienced no other bleeding episodes in the following 20 days. In this time, we investigated the etiology of liver disease. Ultrasonography of the upper abdomen showed granularity in the liver parenchyma and an enlarged spleen. Doppler ultrasonography did not demonstrate the normal hepatic venous connections to the vena cava inferior, thus Budd-Chiari syndrome was diagnosed. We investigated further by catheterizing the inferior vena cava, but were unable to demonstrate the left hepatic vein on injection of inferior vena cava. We also performed liver biopsy in the same session for determining the severity of liver pathology. Histopathological examination of the tissue showed portal-portal bridging fibrosis and mild parenchymal damage. Transhepatic hepatovenography was also done, and this confirmed stenosis of the hepatic vein and collateral vascula-

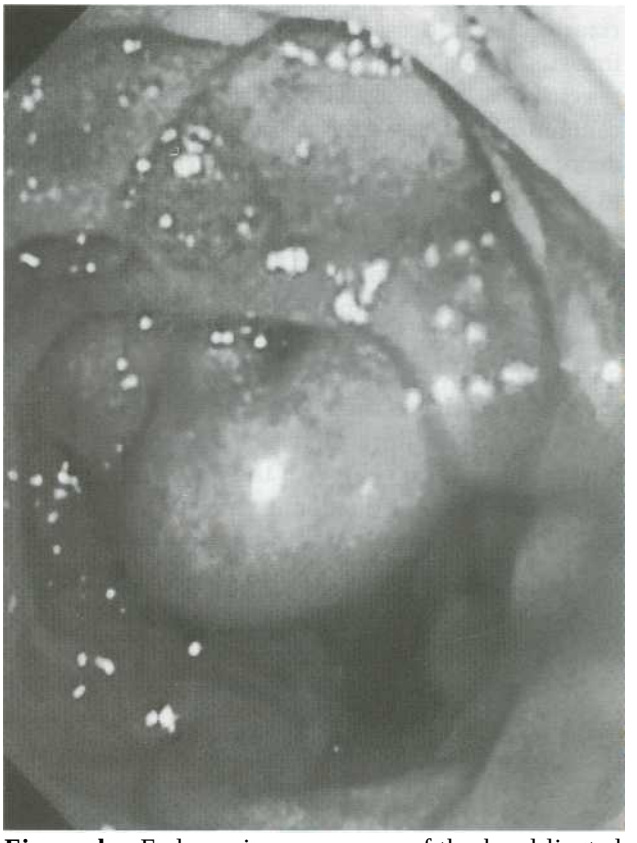

Figure la. Endoscopic appearance of the band-ligated duodenal varix

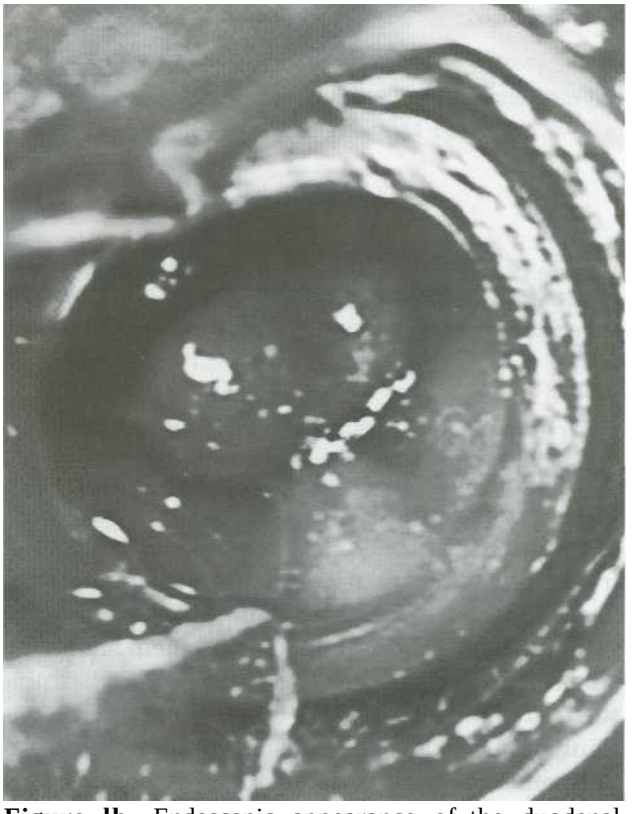

Figure lb. Endoscopic appearance of the duodenal varices

rization. To address the hepatic vein stenosis, we inserted three expandable metallic stents during percutaneous transluminal angioplasty. After placement of these stents free hepatic vein pressure decreased from 15mmHg to lmmHg. In order to prevent thrombosis, warfarin anticoagulation therapy was initiated.

Although the patient's clinical status and biochemical parameters improved, the gastrointestinal bleeding started again 20 days later, after the ligation procedure. Splenoportography was performed to investigate the splenoportal anatomy, and this showed a collateral vessel connecting the superior mesenteric vein with the left renal vein, causing formation of duodenal varices. We placed a coil in this collateral, and the patient had no further bleeding problems (Figure 2a, 2b).

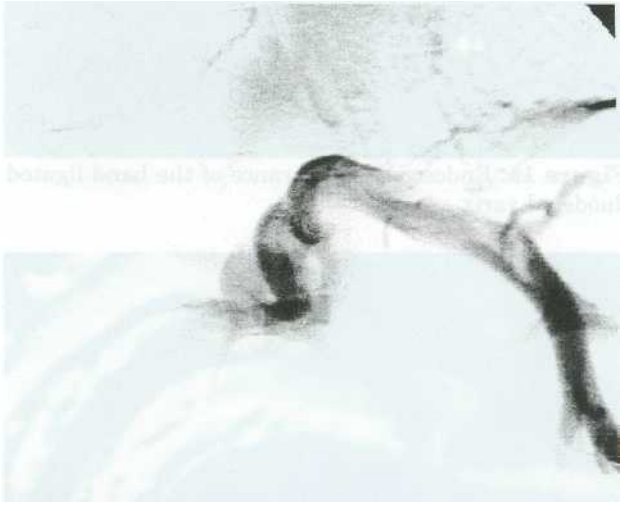

Figure 2a. Percutaneous transluminal angioplasty shows the collateral vessel that had formed between the superior mesenteric vein and the left renal vein

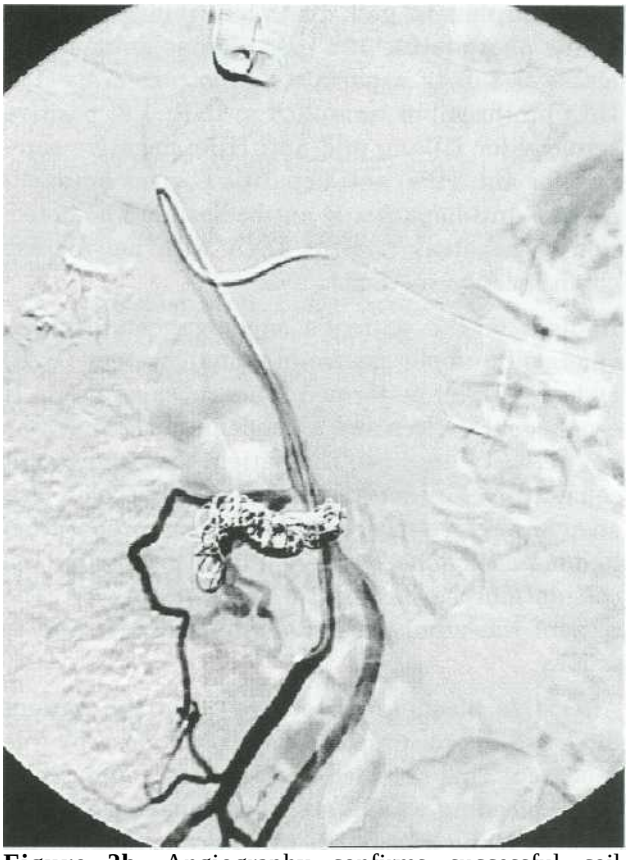

Figure 2b. Angiography confirms successful coil embolization of the collateral vessel

DISCUSSION

Although gastroesophageal varices occur commonly in patients with portal hypertension, varices located at other sites are unusual. Bleeding from these ectopic varices is an uncommon complication of portal hypertension, and the mortality rate in such cases is high. This problem should be suspected when neither upper nor lower gastrointestinal endoscopy reveals a bleeding site in a patient with portal hypertension and gastrointestinal hemorrhage. In most of these cases, the varices are in the small or large intestine; however, they may also be found in the biliary system, vagina, bladder and peritoneum. The majority of patients with ectopic varices exhibit portal hypertension associated with either cirrhosis or portal vein thrombosis (1-3).

Hemorrhage from ectopic varices, including those in the duodenum, is often massive and life-threatening. Therefore, it is imperative that this condition be diagnosed promptly and treated with early intervention. Duodenal varices resulting from retroperitoneal porto-systemic shunts are caused by increased hepatofugal blood flow through the cystic branch of the superior mesenteric vein, the superior and inferior pancreaticoduodenal veins, and the gastroduodenal and pyloric veins (4). Patients with Budd-Chiari syndrome exhibit obstruction of hepatic vein outflow at any level, from the small hepatic veins to the junction between the inferior vena cava and the right atrium (5). If obstruction continues, portal hypertension and cirrhosis eventually develop. Formation of venous collaterals is an important compensatory mechanism in these cases.

The treatment possibilities for Budd-Chiari syndrome include attempts to reperfuse the obstructed hepatic veins with thrombolytic therapy, percutaneous angioplasty or surgery. Liver transplantation is considered a major form of restoration of hepatic outflow. For patients with short-segment stenosis of the hepatic veins, percutaneous angioplasty is the treatment of choice. Most authors have documented immediate relief of obstruction with this method, and recent reports on the use of wall stents have noted long-term patency rates of 80% to 90% (6,7). Percutaneous angioplasty with stent placement may be used in select patients to defer and perhaps avoid shunt surgery or liver transplantation. We opted to insert wall stents in our case; however, though our patient's hepatic vein outflow improved, his gastrointestinal bleeding problems continued. The presence of a collateral vessel connecting the superior mesenteric vein with the left renal vein permitted our treatment of this collateral with coil replacement. We expect that our patient's portal pressure will decrease in time with improvement of chronic changes in liver.

The therapeutic alternatives for bleeding varices include sclerotherapy of the varices, ligation of varices, transjugular intrahepatic portosystemic stent-shunt, portosystemic shunting, resection of a segment of bleeding site, balloon-occluded retrograde transvenous obliteration, beta-blocker therapy, and vasopressin infusion via the superior mesenteric artery (8-11). However, because ectopic varices are uncommon, none of these treatment modalities has been investigated in large series of patients, and it is not clear which method or methods are superior. Thus, the management of ectopic varices depends on the experience of the physician, and must be planned on a case-by-case basis. In our patient, we used band ligation to treat a bleeding duodenal varix that we detected on extended UGI endoscopy. This did not prevent more bleeding from recurring, but it stabilized the patient's condition long enough for us to implant stents. Although stent placement improved the hepatic vein outflow, coil embolization was also needed to stop the variceal bleeding.

This report describes a patient who presented with upper gastrointestinal bleeding, which is a common sign of ectopic varices. However, this case is of particular interest because his duodenal varices, a rare cause of hemorrhage, were beyond the reach of a standard UGI endoscope. We recommend that any patient with portal hypertension who presents with gastrointestinal bleeding of unknown origin should be very thoroughly investigated for ectopic varices. Although variceal band ligation may be the treatment of choice according to the physician's level of experience, coil embolization seems to be more effective in selected cases.

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