THE EVALUATION OF ISOLATED GASTRIC VARICES WITH RESPECT TO LATENCY PERIOD BEFORE CLINICAL APPEARANCE; A CASE REPORT AND REVIEW OF THE LITERATURE

İZOLE GASTRİK VARİSLERİN KLİNİK OLARAK ORTAYA ÇIKIŞLARININ LATENT PERİYODLARI AÇISINDAN DEĞERLENDİRİLMESİ: VAKA SUNUMU VE LİTERATÜRÜN GÖZDEN GEÇİRİLMESİ

Aytekin ALÇELİK*, Burhan YAZICI, Metin AYDIN***, Yusuf AKCAN****, Esin KORKUT*****, Uğur KORKMAZ***

ABSTRACT

We report a case with bleeding isolated gastric varices developed secondarily 21 years after an abdominal gun-shot-wound trauma. Isolated gastric varices may occur as a result of various factors causing splenic vein obstruction in pancreatic and peripancreatic regions. This process may take from 3 years to 21 years to display full-blown bleeding left sided portal hypertension. We reviewed the time sequences of clinical events in such patients from the literature. Splenectomy used to be the therapy of choice. When a patient with a history of blunt abdominal trauma comes to the emergency room with an upper gastrointestinal bleeding, physician should keep in mind that fundal varices could be the cause of this bleeding to manage the patient more rationally.

Key words: Isolated gastric varices, splenic vein occlusion, left-sided portal hypertension.

ÖZET

21 yıl önce geçerilmiş, bir ateşli abdominal silah yaralanmasına sekonder olarak gelişen izole gastrik varis kanamalı bir vaka sunduk. İzole gastrik varisler, splenik ven obstrüksiyonu geliştirebilecek pankreatik ve peripankreatik alanlardaki çeşitli olayların bir sonucu olarak ortaya çıkabilirler. Sol yan portal hipertansiyon kanamasının tam gelişme zamanının süreci 3 ila 21 yıl sürebilir. Buna benzer hastaların klinik olaylarının gelişme zamanları açısından literatürü inceledik. Daha önceleri tedavi olarak splenektomi düşünülmekteydi. Üst gastrointestinal sistem kanaması ile acil servise başvuran bir hastada künt abdominal travma öyküsü varsa; hekim, bu kanamanın fundal bir varise bağlı olabileceğini akılda tutarak hastanın takibini daha akılcı yürütmelidir.

Anahtar kelimeler: İzole gastrik varis, splenik ven oklüzyonu, sol yan portal hipertansiyon.

INTRODUCTION

Gastric varices (GV) are not uncommon in patients with portal hypertension (5,8). Gastric variceal bleeding is often more severe and associated with high mortality (10,14). Frequency and severity of bleeding from GV depend on their location, which forms the basis for their classification.

Isolated gastric varices occur in the absence of esophageal varices or in the presence of only grade I esophageal varices. Isolated gastric varices are rare and are believed to be associated with left-sided portal hypertension (7). Left-sided portal hypertension is a clinical syndrome of splenic vein thrombosis caused by pancreatic pathology and manifests as bleeding gastric varices in patients with a patent portal vein and normal hepatic function. Splanchnic venography is necessary for accurate diagnosis (2).

According to our literature search, abdominal trauma as a causative factor in development of isolated bleeding fundal varices was not reported heretofore. The other reported etiologies are pancreatic pathologies, myelofibrosis, and iatrogenic causes.

CASE

A 50-year old male presented with melena. On administration, he had no complaints of hematemesis, nausea and vomiting, diarrhea, and abdominal pain. He had no history of alcohol abuse or NSAID usage. On examination, he was well with a blood pressure of 130/80 mm Hg, pulse rate: 78/min. He had an abdominal surgical scar on right inguinal area. In previous history, he was treated for pulmonary tuberculosis for a total of six months 30 years ago. In 1983, he was wounded by a gunshot abdominally. He had been undergone an abdominal surgery resulting with a temporary colostomy which was corrected 3 months later.

Laboratory investigations disclosed the following values: Hgb 11.8 g/dL, WBC 7.600/mm^3, platelets 190.000/mm3, creatinine 0.9 mg/dL, PT (prothrombin time) 15.1 sec, total bilirubin 0.57 mg/dL, albumin 4.09 gm/dL, alkaline phosphatase 107 U/L, aspartate aminotransferase (AST) 17 U/L, alanine transaminase (ALT) 18 U/L.

The patient was volume resuscitated and emergency upper endoscopy was performed. There were two colons of grade 1 esophageal varices in the lower 1/3 of esophagus. Meanwhile he had more predominant fundal varices with

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* Düzce Atatürk Devlet Hastanesi, Internal Medicine, Düzce
(İletişim kurulacak yazar: aytekinalcelik@yahoo.com)
** Duzce University, Duzce Faculty of Medicine, Department of Radiology, Düzce
*** Department of Surgery
**** Department of Gastroenterology, Düzce
Kocatepe University, Afyon Kocatepe Faculty of Medicine, Department of Gastroenterology, Afyonkarahisar

many red and blue spots on them. There was no active bleeding. There were some hyperemia and subepithelial petechia and edema, which were all consistent with portal gastropathy. Endoscopic therapy was not attempted. The patient was given octreotide because of the high suspicion of variceal bleeding. IV pantaprazol and propranolol were initiated.

On ultrasonography, the vertical length of liver was 155 mm and its echo pattern was normal. No isolated cystic or solid lesion was observed. Gallbladder was normal. The portal vein diameter, its flow rate and pattern, all were in normal limits. Spleen contours were 150x65 cm and regarded as splenomegalia. Thrombosed and recanalized splenic vein was observed by Doppler USG near the hilum of spleen, and no collaterals could be observed. The pancreas contours were normal and minimally hyperecchoic, regarded as fatty degeneration. These findings suggested isolated gastric varices due to splenic venous occlusion. Percutaneous transsplenic splenoportography was made for definitive diagnosis.

Splenoportography showed an occlusion on the splenic vein flow 2-3 cm away from the hilus and a collateral which is 1.5 cm in diameter, traversing toward the gastric fundus. Portal vein was not seen duo to splenic venous blockage (Figure 1).

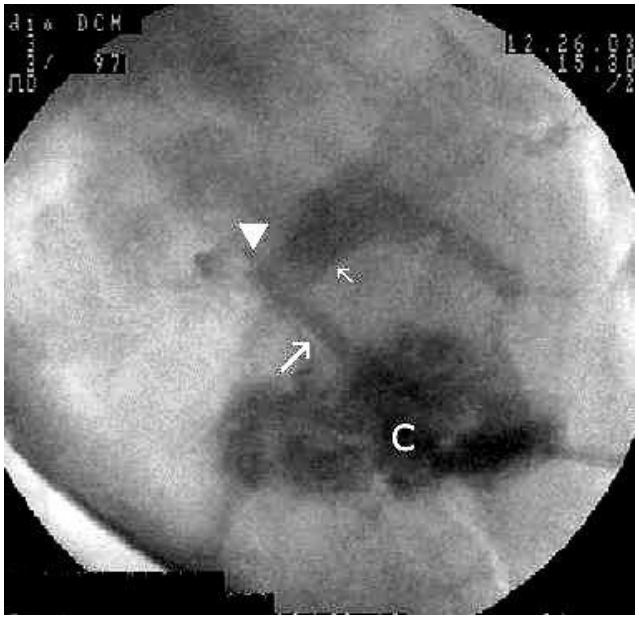

Figure 1. Splenoportography shows an occlusion on splenic vein flow 2-3 cm away from the hilus (arrow heads), recanalized splenic vein before occlusion point (long arrow), and collateral vessel traversing toward the gastric fundus (short arrow), C: contrast media in the splenic parenchyma

We assayed for protein C and S levels and S activity for any possible predilection towards thrombogenesis. Anti-thrombin III level and protein C resistance were measured as well with negative results. Anti-cardiolipin and lupus anticoagulant assays yielded negative results. VDRL was also negative. Hematocrit levels were not high. And there was not any clinical sign indicating any myeloproliferative disease. We decided that the gun-shot wound caused perivenous inflammation and irritation around the splenic vein at that time and caused the blockage of splenic vein. After this observation, we believe that there is a marked time lag needed to develop an isolated fundal varice to appear clinically after a splenic vein blockage in human beings.

Splenectomy is the effective treatment of choice with sinistral (left-sided) portal hypertension patient (2). That is why we preferred the same operation.

DISCUSSION

Isolated gastric varices occur in the absence of esophageal varices or in the presence of only grade I esophageal varices. They could be located in the fundus of the stomach (Isolated GV1) or at ectopic sites such as the body, antrum or duodenum (Isolated GV2). Isolated GV1 or fundal varices are defined as distinct venous columns in the fundus of the stomach, falling short of the cardia by a few centimeters with no esophageal varices (12). Isolated ectopic gastric varices are present either in the body or in antrum of the stomach or upper duodenum. The prevalence, natural history and clinical significance of these varices have not been adequately described (11). The most common cause is pancreatic disease (3). Isolated cluster of varices in the gastric fundus (Isolated GV1) constitute 8% of all gastric varices and 80% isolated GV1 bleed (6). Bleeding gastric varices account for only 10% to 15% of all variceal bleeds, the bleeding is often severe and difficult to manage (4). The most serious complication associated with splenic vein occlusion is massive bleeding from gastric varices. Earlier reports recommended a prophylactic splenectomy for patients with left-sided portal hypertension (3, 4,6,11,13). On the other hand, some authors tried endoscopic methods. Sarin SK et al. reported that bleeding due to isolated ectopic gastric varices was seen only in 3 of 57 (5.7%) patients during a mean follow-up of 36.3 +/- 12.1 months, and could be successfully managed with endoscopic ligation or obliteration (11).

In a case of isolated gastric varices developed secondary to an abdominal surgery performed for a benign pancreatic tumor, the varices were noticed 7 years after the operation and followed for additional 4 years when ultimately operated prophylactically for portal hypertension (15). Sakorafas et al reported that one of the 11 patients with asymptomatic sinistral portal hypertension who underwent pancreatic surgery without splenectomy died of later variceal bleeding 3 years after lateral pancreatojejunostomy (9). Again, in a prospective study by Brandley, only one of the 11 patients with splenic vein occlusion caused by chronic pancreatitis had gastric variceal bleeding during follow-up extending from 2 to 11 years (1).

Regarding the time sequence of clinical events in patients with isolated gastric varices in the literature, it is apparent that they usually occur as a result of various insults to pancreatic and peripancreatic region indicating the development of splenic vein obstruction. This process may take from 3 years to as late as 21 years as in our case to display full-blown bleeding left-sided portal hypertension. Splenoportography is the imaging technique of choice for definitive diagnosis.

Splenectomy used to be the therapy of choice but recently

there are other reports about utilization of various endoscopic techniques like tissue glue injection and ligation (2). As a conclusion, when a patient comes to the emergency room with an upper gastrointestinal bleeding, and if the patient has a history of abdominal blunt trauma even in last 20-25 years, physicians should be very careful about fundal varices to be the cause of this bleeding and manage the patient more carefully. Such patients deserve an early and urgent endoscopic evaluation. If endoscopy is not possible, physician should start an empirical somatostatin infusion therapy considering a variceal bleeding.

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