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Case Report

Duodenal varices diagnosed by endoscopic ultrasound: A case report

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Abstract

Portal hypertension and associated complications cause significant morbidity and mortality in cirrhotic patients. Variceal development is the most important portal hypertension related complication. Varices most commonly occur around the gastroesophageal junction, but ectopic varices may develop in many gastrointestinal and extra-gastrointestinal localizations. Duodenum is one of the most common localizations for ectopic varices. Diagnosis of duodenal varices is usually made by upper gastrointestinal endoscopy, but endoscopic appearance is not diagnostic and usually further investigations are required in order to make accurate diagnosis. Endoscopic ultrasound is the gold standard method for the examination of gastrointestinal submucosal lesions therefore it is also useful in the work up of suspected duodenal varices. Here we present a patient with cryptogenic liver cirrhosis followed in our clinic, whom duodenal lesions suspected of duodenal varices were noticed during upper gastrointestinal endoscopic examination and endoscopic ultrasound was used to confirm the presence of duodenal varices.

Keywords: Portal Hypertension; Endoscopic Ultrasound; Varices.

INTRODUCTION

Variceal development is one of the most important complications associated with portal hypertension and variceal bleedings cause significant morbidity and mortality in cirrhotic patients. Gastrointestinal (GI) varices most commonly develop around the gastroesophageal junction but ectopic varices occurring in many gastrointestinal (duodenum, small intestine, colon, rectum, etc.) and extra-gastrointestinal (abdominal wall, retroperitoneum, bile duct, etc.) localizations have been reported in the literature (1,2). The most important complication associated with ectopic varices is bleeding. Ectopic variceal bleeding is difficult to treat with high morbidity and mortality rates (3,4). Duodenum is one of the most common localizations for ectopic varices. The reported prevalence of duodenal varices (DV) in cirrhotic patients varies in different studies in relation to the method used for diagnosis of varices but varicose changes in duodenal vasculature have been reported to be present in up to 40% of cirrhotic patients who had undergone visceral angiography (5). Unlike esophageal varices, DV are located in the serosal layer of the duodenum which makes endoscopic diagnosis difficult. In most instances, DV appear as mucosal raised nodular lesions in the duodenum, and since the appearance is not characteristic, further studies are usually needed for definitive diagnosis. Transfemoral or transhepatic angiography, video capsule endoscopy, computed tomography (CT) angiography, and contrast-enhanced 3D magnetic resonance angiography have been reported to be used for the diagnosis of ectopic varices (6).

Endoscopic ultrasound (EUS) is a minimally invasive technique which combines endoscopy and ultrasound and it is widely used in the field of gastroenterology. Along with many other diagnostic and therapeutic applications, EUS is accepted as the golden standard method for the assessment of submucosal lesions of the GI tract. EUS can also be safely used for the diagnosis and treatment of DV (7,8). In this manuscript, we described a patient with portal hypertension and esophageal varices who was also suspected to have DV during upper GI endoscopic examination. EUS was used to confirm the presence of DV. Written informed consent for was obtained from the patient for this case presentation.

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CASE REPORT

A 65-year-old woman admitted to our clinic with the complaints of fatigue. She stated that, she was diagnosed with cirrhosis a couple of years ago and was on regular follow up since then in another clinic. On admission, physical examination findings were as follows; she seemed to be in good health with clear consciousness and good orientation of time, place and person. Her vital signs were stable (body temperature: 36.8 °C, pulse: 78 / bpm, respiration: 12 / min and arterial blood pressure: 90/60 mm Hg). Abdominal examination revealed dullness to percussion consistent with the presence of ascites, the liver and spleen were not palpable but dullness to percussion was again noticed in Traube's space. The rest of the physical examination findings were assessed to be unremarkable. Laboratory tests results on admission were as follows; Hb: 12.2 g / dl, Hct: 40.7%, WBC: 5000 / µl, Platelet: 130000 / µl, erythrocyte sedimentation rate: 6 mm / hr, AST: 24 U / L, ALT: 24 U / L, ALP: 95 U / L, GGT: 27 U / L, Albumin: 4 g / dl, Total bilirubin: 1,5 mg / dl, Direct bilirubin: 0.56 mg / dl, Urea: 30.1 mg / dl, Cre: 0.66 mg / dl, and prothrombin time (INR): 1.2. Trans-abdominal ultrasonograph showed that liver contours were irregular and the echo pattern of the liver parenchyma was coarse granular and heterogeneous. Moderate amount of ascites and splenomegaly (longest axis of spleen measured was 15 cm) were also noted during abdominal ultrasound examination.

The patient was diagnosed with cirrhosis and further tests were prompted to evaluate the etiology of cirrhosis. The results of the etiological tests were as follows; viral markers for hepatitis B and C (hepatitis B surface antigen, anti-hepatitis B core antibody, anti hepatitis C antibody) and autoimmune markers (antinuclear antibody, anti-smooth muscle antibody, anti liver kidney microsomal antibody, anti-mithochondrial antibody) were all negative. Laboratory studies for hemochromatosis (serum iron concentration, serum iron binding capacity, transferrin saturation and ferritin concentration) and Wilson's disease (serum copper and ceruloplasmin concentrations, 24 hours excretion of copper) were within normal limits and serum alpha-1 antitrypsin concentration was also found to be normal. The calculated Child-Pugh Score was 6 (Child Class A) and Model for End-Stage Liver Disease (MELD) score was 10. An upper GI system endoscopy was performed which showed the presence of grade 3 esophageal varices. The gastric mucosa was assesed to be hyperemic and edematous with characteristic mosaic-like pattern typical of portal hypertensive gastropathy during the endoscopic examination. The mucosa of the duodenal bulb was in congested appearance and multiple, 0.3-0.4 cm sized, nodular lesions were noted to be present in the duodenal bulb (figure 1).

Considering the presence of cirrhosis and associated portal hypertension, ectopic DV were suspected and endoscopic forceps biopsy was not performed. EUS examination of the duodenum was planned. EUS examination showed the presence of submucosal tortoise, anechoic tubular structures which were assumed to be vascular in nature in grayscale mode. Color Doppler examination demonstrated the presence of blood flow within these vascular structures confirming the diagnosis of DV (figure 2).

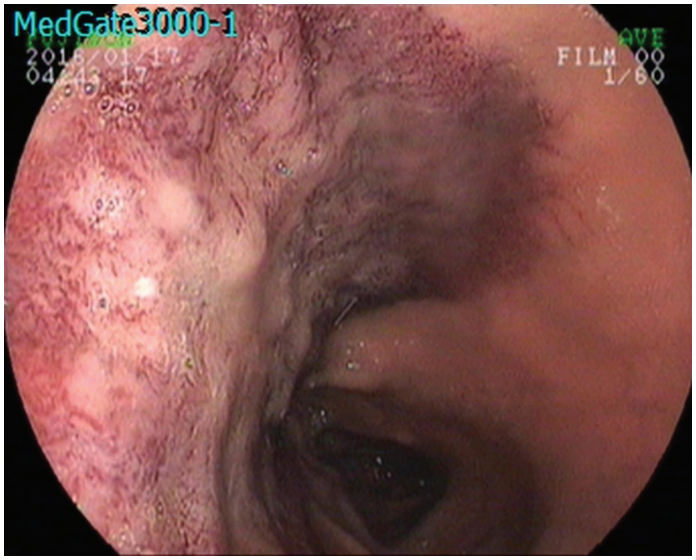

Figure 1. Endoscopic view of the duodenal bulb. Duodenal mucosa has congested appearance with multiple, 0.3-0.4 cm sized nodular structures

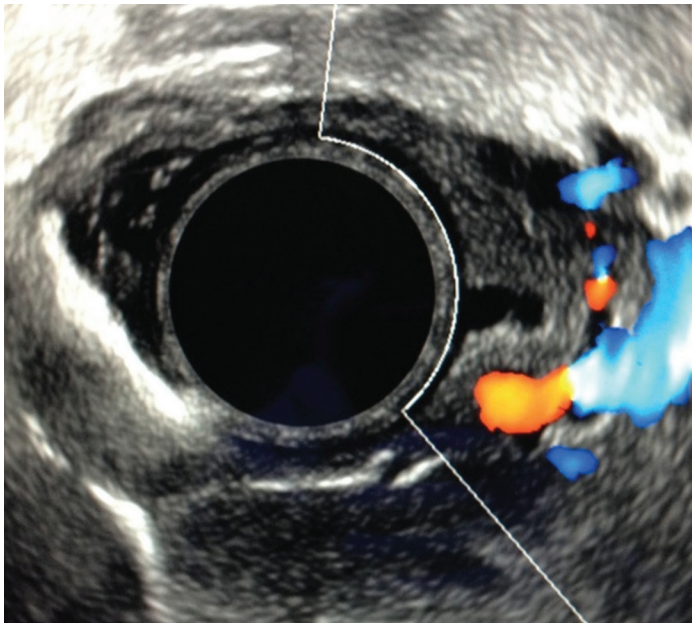

Figure 2. Endoscopic ultrasound image of the duodenal varices. Submucosal tortoise, anechoic tubular vascular structures filled with color are seen in Doppler ultrasound

DISCUSSION

Portal hypertension and associated complications cause significant morbidity and mortality in cirrhotic patients. Variceal development and bleeding from these varices are the most important portal hypertension related complications. Varices usually develop in the esophagus and stomach but variceal development can also occur in

other regions of the GI tract as well as in various extra-gastrointestinal localizations. Duodenum is one of the most common sites for ectopic varices occurrence (9). The mechanism of development of DV is associated with portosystemic shunts as in the case of varices of esophagogastric junction. Collaterals developing between the pyloric vein, gastroduodenal vein and inferior and superior pancreaticoduodenal veins contribute to variceal development by causing increased hepatofugal blood flow (10). As in our patient, cirrhosis is the most common cause of DV but DV may also develop in patients with splenic or portal vein thrombosis.

DV most commonly develop in the first part of the duodenum and they are usually detected during routine upper GI endoscopic examinations. The first presentation of DV may also be with an active bleeding episode. Since endoscopic appearance of DV is not characteristic, additional investigations are usually required for definitive diagnosis. In most cases, DV appear as nonspecific submucosal nodular lesions during upper GI endoscopic examination therefore endoscopic appearance is not easy to differentiate from other submucosal lesions of the GI tract. In addition, endoscopic biopsy of such lesions is not appropriate prior to definitive diagnosis as it may result in serious bleeding. Differential diagnosis should include hemangiomas and lymphangiomas, adenomatous and inflammatory polyps, Brunner gland hyperplasia, duplication cysts, ectopic pancreas and other submucosal lesions of the duodenum (lipoma, leiomyoma and leiomyosarcoma, gastrointestinal stromal tumor, neuroendocrine tumors etc.). Portal hypertensive duodenopathy should also be considered in differential diagnosis as it may cause diverse endoscopic findings such as erythema, patchy or diffuse congestion of the duodenal mucosa, erosions, telangiectasia, exaggerated villous pattern, noduler and polypoid lesions in the duodenum which may mimic the endoscopic appearance described in our patient. EUS has made great progress in the past 10 years and it has gained numerous diagnostic and therapeutic applications in gastroenterology. EUS is considered as the gold standard for evaluation of submucosal lesions of the GI tract. It determines with great accuracy whether a GI submucosal lesion is solid or cystic in nature and which layer of the gut wall it originates from. In addition, during EUS examination, color Doppler ultrasound can be used to assess the blood flow in lesions suspected of vascular structures. Therefore EUS is a great tool to differentiate DV from other lesions with similar endoscopic appearances. In our experience with this patient, EUS was found be extremely useful for the diagnosis of DV. EUS clearly showed the presence of submucosal tortoise vascular structures in the duodenum and color Doppler examination was used to demonstrate the blood flow within these variceal vessels.

EUS has also been reported to be used with success for the treatment of DV. Currently well established guidelines for the treatment of DV does not exist and endoscopic hemostatic methods such as cyanoacrylate injection, band ligation and hemostatic hemoclipping are considered as first line treatment options in acutely bleeding DV (11-13). Interventional angiographic methods such as endosvascular embolization and transjugular intrahepatic portosystemic shunt (TIPSS) have also been reported with success in actively bleeding DV where initial endoscopic treatment attempts fail (14). In recent years, there are increasing numbers of studies reporting treatment of DV using EUS guided techniques such as coil embolization of varices with or without concomitant cyanoacrylate injection (15).

In conclusion, the possibility of ectopic varices development should not be overlooked in patients with portal hypertension. Surveillance endoscopic examinations should be scheduled and in addition to assessment for gastroesophageal varices, duodenum as well as other GI locations should be carefully evaluated for the presence of ectopic varices. As described in the current report, we believe that EUS is a reliable, inexpensive and minimally invasive method for the evaluation of ectopic varices and it should be the study of choice in the evaluation of GI lesions suspected to be ectopic varices including DV.

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