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CASE REPORT

Gastrointestinal Hemorrhage Due to Duodenal Varix: A Case Report

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Abstract

We report a case of a 72-year-old female with a past medical condition of non-alcoholic steatohepatitis who presented in the emergency department with altered mentation. An Esophagogastroduodenoscopy was performed which showed a normal esophagus and stomach, but revealed grade III varices which were appreciable in the second portion of the duodenum. Her colonoscopy report revealed multiple small and large mouthed diverticula in the sigmoid colon along with hematin throughout the colon, yet no evidence of active bleeding, mass or inflammation. We discuss the patient's rarity of the clinical entity, clinical development, and elements used for diagnosis along with the treatment modalities involved.

Keywords: Duodenal varices, Endoscopy, Gastrointestinal bleeding

1. Introduction

Non-alcoholic fatty liver disease (NAFLD) progresses to NASH which is associated with liver damage (fibrosis) caused by inflammation and over time the extensive scarring of the liver (cirrhosis). PTH, a known complication of liver cirrhosis in turn results in several other clinically notable complications such as the development of ascites, esophageal and gastric varices, which carry a significant morbidity and mortality risk. Porto-systemic shunting is a phenomenon that causes diversion of portal blood into systemic circulation due to PTH and this results in variceal formation. These varices are located mainly in the esophagus or stomach. EcV are defined as dilated splanchnic veins or dilated portosystemic collaterals that occur in unusual sites along the gastrointestinal (GI) tract. Although they have four times the risk of bleeding of esophageal varices, they constitute approximately 2%−5% of all GI variceal bleeds.[1] These lesions present a clinical challenge as they are difficult to locate, can occur at distal sites, and the management guidelines are unclear if they are identified. The consequences of missing or misdiagnosing these lesions can be fatal, with a mortality rate of up to 40%.[1]

2. Case presentation

This is a case of a 72-year-old female with a known history of diabetes mellitus, hypothyroidism, chronic kidney disease III, hypertension, morbid obesity (NASH cirrhosis who presented in the

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emergency department with altered mental status and confusion. On arrival, the patient was agitated, striking out at the emergency medical services staff. On physical examination, the patient was lethargic, disoriented and was unable to participate in an interview. The body temperature was 97.7 °F, blood pressure was 110/60 mmHg, heart rate was 60 beats/min and O_2 saturation was 97%. The patient's eyes were anicteric. No abnormalities were seen in the abdominal, cardiovascular, pulmonary, and skin examinations, however, 1+ pitting edema was found in the lower extremities bilaterally.

Initial laboratory data at the time of presentation showed White blood cells (WBC): 6.4 × 109/L, hemoglobin (Hb): 9.1 gm/dL, mean corpuscular volume (MCV): 106 fl and platelets: 87x103/μL. Serum aspartate aminotransferase (AST), total bilirubin, blood urea nitrogen (BUN) and ammonia levels were raised to 64 U/L, 3.5 mg/dl, 50 mg/dl and 218 mcg/dL, respectively (Table 1). Toxicology was negative. On imaging, Computerized tomography (CT) of the head revealed no evidence of any acute intracranial abnormality and CT abdomen/pelvis without contrast, showed a cirrhotic liver with no discrete mass, splenomegaly, diffuse body wall anasarca without abdominal ascites and normal pancreas and bile ducts.

Table 1. Laboratory data on initial presentation.

WBC	$6.4x10^9$/L
Hb	9.1 gm/dL
MCV	106 fl
Platelets	$87xl0^3$/nL
BUN	50 mg/dL
Cr	1.2 mg/dL
AST	64 U/L
ALT	35 U/L
ALP	116 U/L
Total Bilirubin	3.5 md/dL
Albumin	3.0 g/dL
Ammonia	218 mcg/d L

WBC: White Blood Cells, Hb: Hemoglobin, MCV: Mean Corpuscular Volume, BUN: Blood Urea Nitrogen, Cr: Creatinine, AST: Aspartate Aminotransferase, ALT: Alanine Transaminase, ALP: Alkaline phosphatase.

The patient was admitted for grade II hepatic encephalopathy. She was treated with lactulose and rifaximin with significant improvement in her mental status but had notable acute on chronic anemia without reports of melena, hematochezia, or hematemesis. Fecal occult blood test was positive. On repeat lab investigations, the hemoglobin had dropped to 5.5 g/dl, Hct to 27.1%, ammonia to 42 mcg/dL while BUN and Cr had risen to 55 mg/dl and 1.3 mg/dl. She was transfused 2 Units of packed red blood cells and transferred to a higher level of care facility for GI intervention. Upon transfer, the GI team was consulted, and the patient was noted to have dark tarry stools. She was started on a pantoprazole drip, octreotide drip and recommended for a restrictive transfusion strategy.

An Esophagogastroduodenoscopy (EGD) was performed (Fig. 1). No evidence of varices, bleeding or ulceration was found on the esophagus or the stomach, however, grade III varices were present in the second portion of the duodenum which were tortuous, 8 mm in diameter with several dimpled areas but no source of bleeding was found. The patient continued to have drops in her blood counts and required daily blood transfusions. An EGD was repeated which had similar findings. She was then recommended for a colonoscopy (Fig. 2) which revealed multiple small and large mouthed diverticula in the sigmoid colon and hematin throughout the colon, yet no evidence of active bleeding or colonic mass or colitis was found. After her colonoscopy findings, she was recommended for an outpatient video capsule endoscopy, however, her blood counts continued to drop until a rapid response was called for hypotension with blood pressures in the 40–60 mmHg range. The patient reported abdominal pain and was started on pressors. A CT abdomen/pelvis without contrast was repeated which revealed diffuse air-fluid levels in

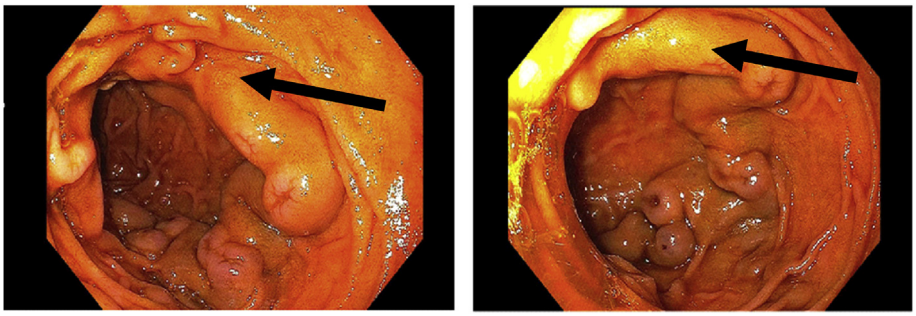

Fig. 1. Note the (black arrows), Endoscopic photograph of a duodenal varix involving the second portion of the duodenum, several tortuous dimpled areas.

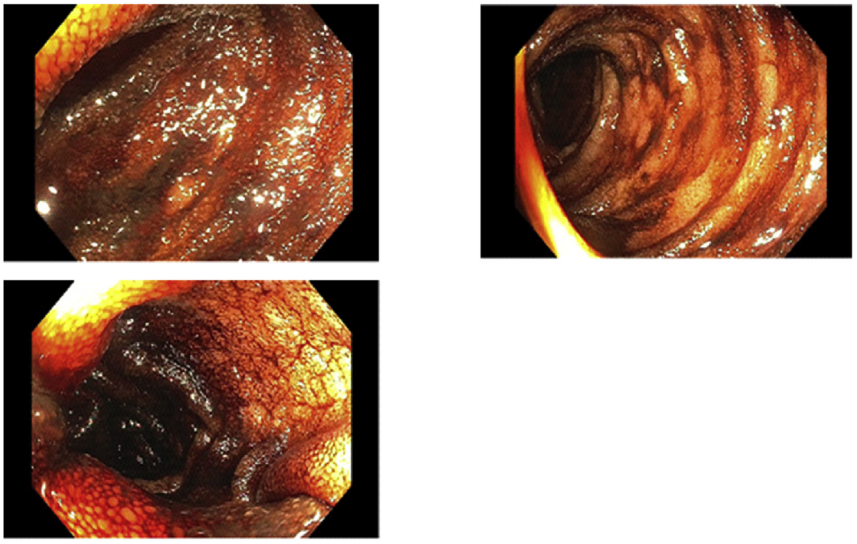

Fig. 2. Multiple small and large mouthed diverticula in the sigmoid colon and hematin throughout the colon, yet no evidence of active bleeding or colonic mass or colitis.

the small and large bowel, maybe representative of ileus. No evidence of obstruction and a similar cirrhosis appearance, and diverticulosis was noted. Unfortunately, after the rapid response the patient continued to deteriorate, became lethargic, was placed on non-rebreather and subsequently intubated. Despite maximal medical therapy she developed multi-organ failure and the family consented to comfort care.

3. Discussion

Factors which may hint towards a potential diagnosis of EcV in patients with PTH include acute bleeding and negative findings on upper endoscopy. Examination and assessment of the vascular integrity of the small intestine becomes essential in such patients. Different sites of EcV can include the duodenum, jejunum, ileum, colon, rectum, peristomal, biliary tree, peritoneum, umbilicus, falciform ligament, bare area of the liver, splenic ligament, right diaphragm and genitourinary.[2] A nationwide survey in Japan reported the rectum (44.5%) as the most frequent site of EcVs, followed by the duodenum (32.9%).[3] Norton et al. looked at 169 patients with variceal bleeding due to EcV and found 17% to occur in the duodenum, 17% in the jejunum, ileum, 14% in the colon and 8% in the rectum concluding that duodenal varices were the most common[4]

The etiology of EcV can be divided into two categories, generalized PTH and splanchnic venous occlusion. These occlusions can be due to thrombosis of the splenic vein, of a spontaneous gastro renal shunt, mesenteric veins, or the main portal vein. In the west, the most common etiology of PTH is cirrhosis due to alcoholic liver disease, NASH, and hepatitis C infection. Surgical procedures such as gastrectomy, ileostomy, and post–balloon-occluded retrograde transvenous obliteration (BRTO) of gastric varices are also associated with EcV[1] Rarely, varices may be present in the colon in the absence of PTH due to anomalies of portal venous outflow, such as congenital anomalies of normal portosystemic anastomoses, abnormal vessel structure, or arteriovenous fistulae.[4]

Duodenal varices are most commonly associated with the cirrhosis of the liver but can also develop due to portal vein thrombosis and obstruction of the splenic vein and inferior vena cava. Our patient presented with varices in the second portion of the duodenum which is the second most common site after the duodenal bulb. Rupture can lead to severe hemorrhage. Varices in the duodenal bulb, which occur most frequently in the United States and Europe, are caused by extrahepatic portal obstruction though they may occur in intrahepatic PTH as well.

Clinical presentation of ectopic variceal bleed depends upon the location of the varices and can include GI bleeding of obscure origin, occult GI bleeding, hematemesis, hematochezia, or it can even be an accidental finding. In a study of 37 patients with liver cirrhosis who underwent capsule endoscopy, 3 (8.1%) were found to have small-bowel varices. Some cases unfortunately are diagnosed at autopsy.[6,7] Presence of EcV should be suspected in patients with GI bleeding with PTH, if both upper and lower endoscopies fail to show a definite

source.[6] Because of the low prevalence of extrahepatic PHT in the western countries, most cases of bleeding from EcV reported in the West are usually associated with intrahepatic PHT[5] Diagnosis in a timely manner is important and endoscopic and angiographic techniques and CT scans of the abdomen and pelvis are most frequently used.[7]

Furthermore, clinical evidence suggests that bleeding from duodenal varices specifically and EcV in general have a poor clinical outcome with a 40% mortality at initial bleed from duodenal varices was reported in one study.[2] The presence of varices is more commonly noted in patients with intrahepatic PTH. When no bleeding source is identified in such patients who have undergone upper GI endoscopy with clinically diagnosed PTH, bleeding from EcV is highly suspected.

In availability of adequate treatment guidelines in the presence of diverse bleeding sites, varying clinical presentations, different causes of PTH have to a certain degree hampered the way for effective treatment which may also be influenced by factors such as the availability of local resources and patient's condition.

In our patient, liver cirrhosis eventually led to the development of EcV which was associated with intrahepatic PTH along with NASH. Health risk-benefit ratio has contributed significantly towards the selection of preferred treatment option and surgical options such as variceal suture ligation or resection, and portocaval shunt are no longer the first choice of action as high mortality rate of up to 30%–40% and rebleeding in up to 40% has been reported[4,9] therefore surgery is now rarely performed. Surgical interventions are only considered as a potential treatment option if endoscopic or radiologic interventions, have failed to achieve hemostasis.

In case of ectopic variceal bleeding, adverse outcome of endoscopic treatment modalities is insignificant and can be easily controlled. Endoscopic modalities for intervention include endoscopic variceal band ligation (EVBL), endoscopic cyanoacrylate injection, and endoscopic sclerotherapy. Endoscopic variceal obturation (EVO) involves injecting a tissue adhesive agent, such as cyanoacrylate, which leads to solidification and thrombosis in the varices should therefore be considered in controlling ectopic variceal bleeding.[10] Cyanoacrylate injection is highly effective but carries a risk of embolization, whereas EVBL and endoscopic sclerotherapy carry risks of recurrent bleeding from procedural-related ulceration.

Despite the risks involved, cyanoacrylate injection has been used to stop active bleeding or high-risk stigmata in endoscopic management of patients suffering from duodenal varices and the evidence was reported in a case series where 4 of 14 patients underwent treatment, 100% initial hemostasis was achieved with no re-bleeding on 7–30 month follow up.[14] Akazawa et al.[11] showed successful management of duodenal variceal bleeding by EVL and subsequent BRTO. Malik et al.[12] and Mora-Soler et al.[13] reported 1 and 5 patients with duodenal variceal bleeding treated successfully with cyanoacrylate injection, respectively.

We administered pantoprazole drip, octreotide drip (to reduce splanchnic blood flow and variceal pressure may be beneficial in patients bleeding from esophagogastric varices and recommended for a restrictive transfusion strategy. Similarly, terlipressin, which has been beneficial in the management of bleeding esophageal varices may be tried, but this drug is not currently available in the United States. Even though there is no comprehensive data available that validates the role of beta blockers in the long-term management of EcV, but some case reports have raised concerns as they have conflicted with the information about their effectiveness.

Colonic (Fig. 1) and rectal varices are identified as serpiginous vessels projecting into the lumen. If the colon appears normal and bleeding continues, angiography may be useful in identifying varices or in localizing a nonvariceal source of hemorrhage. Existing literature advocates that band ligation is not a safe and reliable treatment technique for large EcV as after the band sloughs off, the defect in the varix is widened. Band ligation however can be considered in situations where the varix is less than a diameter. Embolization of the varices can be achieved by integrating different techniques which includes steel coils, thrombin, gel foam, collagen, and autologous blood clot either alone or in combination with a variety of sclerosants. Almost a 94% success rate has been reported for bleeding control in such patients.[16,17] The drainage of varices into the low-pressure systemic vein is essential and it needs to be protected from the high-pressure portal system and this is carried out by occluding the feeding vein that is, the vein on the portal venous side to the EcV.

One of the most relevant procedure known to essentially reduce PTH and the risk of its associated complications that is EcV is Trans jugular intrahepatic portosystemic shunt (TIPS). Our patient however expired before being operated for TIPS. It is emphasized in the published literature that cirrhotic patients in whom embolization of the EcV have failed to produce the desired outcome with continued bleeding should immediately be managed under TIPS procedure. However eligible candidates for this procedure are patients on the waiting list of

liver transplantation.[7] Case reports of TIPS alone and TIPS with embolization have shown good results in patients with Duodenal Varices and the rebleed rates after TIPS decompression are 20%–40%. In one study of bleeding EcV, TIPS with embolization was superior with only two out of seven patients (28%) presenting with re-bleeding in comparison to 5 out of 12 patients (42%) that had TIPS only without initial embolization.[18] Duodenal varices may bleed at lower portosystemic pressure gradients; thus, TIPS alone may be unlikely to be a sufficient therapy. Other treatment modalities include Balloon-occluded retrograde transvenous obliteration (B-RTO) which is a shunt occlusion procedure as TIPS alone may not be appropriate and adequate given how duodenal varices may bleed at a lower portosystemic pressure gradient. Additional IR interventions include: Percutaneous embolization or Antegrade obliteration techniques. Surgery may be the last. Endoscopic and interventional radiological procedures are not always successful and, in such patients, surgery is performed as a salvage option which involves de-arterialization of bleeding duodenal varices, enterectomy for bleeding ileal varices, resection and anastomosis for colorectal varices, and stapled anoplasty for anorectal varices.

In conclusion it is fair to say that minimal visualization, delayed diagnosis and insufficient definite treatment plans have made the management of EcV a real challenge which is further leading to an increased incidence in the fatality rate. Therefore, more evidence-based data, comprehensive and extensive literature is essential in order to curb the adverse effects of this condition.

Conflicts of interest

There is no conflict of interest.

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