Case:
A 65 years old man was admitted to the Gastroenterology department with the complaints of fresh per rectal bleeding for 5 days. The bleeding was painless & intermittent in nature. And for the last 2 days prior admission it was gradually decreasing in amount. He didn’t complain of any abdominal pain, anorexia, weight loss, altered bowel habit or tenesmus. He didn’t give any significant past history. There was no history of surgery. No such complaints were seen running in his family. On examination the patient was mildly anemic. Abdominal examination revealed no abnormality. There were no signs of chronic liver disease or portal hypertension. Other system examinations revealed no abnormality.

Investigation revealed anemia with normal hepatic & renal function. After initial resuscitation an urgent colonoscopy was arranged. Colonoscopy revealed a tuft of dilated tortuous submucosal vessels at the recto-sigmoid junction, suggestive of colonic varix. Rest of the colon including the terminal ileum appeared normal. No evidence of active bleeding was seen. Abdominal USG revealed a normal appearance of liver with uniform echo texture. There was no splenomegaly on USG, neither there were any signs of portal venous thrombosis on doppler examination. Abdominal doppler sonography revealed normal portal flow volume & velocity. There was no evidence of collaterals on USG. For further evaluation and to rule out portal HTN, an upper GI endoscopy and a CT scan of whole abdomen was done, but no abnormality was detected. Echocardiogram was done to exclude Congestive heart failure. The coagulation profile, hepatitis serology, autoantibody profile, pancreatic enzyme levels were within normal limit, virtually excluding all possible causes of colonic varices.

As there was no evidence of active bleeding, a conservative approach was taken. He was managed with oral iron supplements and laxatives. He was discharged with an advice for sclerotherapy, if further episodes of per rectal bleeding recurred.

Discussion:
Ectopic varices are a term used to describe any portosystemic collateral veins that are dilated, circuitous, and located outside of the gastro-esophageal region. These varices are often caused by portal hypertension secondary to liver cirrhosis, but it is estimated that only 3.4% of patients with intrahepatic portal hypertension also have colonic varices. Other less common causes of colonic varices are congestive heart failure, mesenteric vein thrombosis, pancreatitis with splenic vein thrombosis and post-operative adhesions. One study estimated that regardless of etiology, colonic varices have an incidence rate of only 0.07%.

It has been observed that non-idiopathic colonic varices occur equally in males and females and are usually diagnosed after age 50. Idiopathic colonic varices though are seen more often in males at a younger age, with a median age of diagnosis being 41.3 years. Some authors have suggested a possible autosomal recessive mode of inheritance. Idiopathic colonic varices exhibit a much stronger familial association compared to non-idiopathic colonic varices and are more likely to be pan-colonic. Colonic varices should be considered “idiopathic” after other medical conditions either or not.

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Figure 1: Colonoscopy showing a tuft of colonic varix at the rectosigmoid junction.
related to portal hypertension or portal vein thrombosis have been ruled out. The most common segments of varicosities development within the lower gastrointestinal tract are cecum and rectum. It has been estimated that since 1954, there have only been around 100 reports of isolated colonic varices. Patients with colonic varices typically present with hematochezia or blood in the rectum, which may or may not be accompanied by pain. Idiopathic colonic varices, which cannot be explained by an underlying pathology, may also cause recurrent episodes of lower gastrointestinal bleeding.

It is not unusual for patients to first present with a massive gastrointestinal bleed, which is why practitioners must recognize the potential for varices within the colon, even in the absence of portal hypertension or varices elsewhere. Colonic varices may present with serious complications, intermittent hematochezia or severe rectal bleeding and diagnosed accordingly, or discovered incidentally during a colonoscopy performed for gastrointestinal tract symptoms. Colonoscopy is the investigation of choice and varices can be visualized as dilated tortuous venous channels. However, varices may occasionally be mistaken for polyposis or tumor. Colonoscopy during a period of hypotension, along with compression of varices due to insufflation, may cause them to be missed. CT angiography may exhibit the greatest potential because it locates varices in a manner that is both precise and minimally invasive. Mesenteric angiography is a useful diagnostic tool, but diagnosis of colonic varices may be missed on angiography.

Most often in practice, however, a colonoscopy is performed to identify colonic varices. Caution must be taken to ensure that the varices are not mistaken for a tumor or compressed during insufflation and missed. Before concluding that true idiopathic varices are present, underlying cirrhosis and portal venous obstruction should be excluded. Liver function tests, coagulation profile, auto-antibody panel, hepatitis serology, doppler studies for portal venous system should be done to rule out other causes. In our case the patient presented with painless per rectal bleeding & was admitted to hospital for further management. Any other symptoms which could have suggested other organic diseases were absent. Past history & family history was insignificant. Examination revealed no abnormality except for a mild anemia.

There was no evidence of chronic liver disease, nor clinical nor biochemical. USG of whole abdomen showed a normal liver with uniform echo texture. No splenomegaly, ascites or signs of portal venous thrombosis were seen on USG & doppler studies. The serological markers for hepatitis B and C were also negative. Thus, the most common cause of colonic varices, portal hypertension secondary to liver cirrhosis was excluded. Portal hypertension secondary to portal venous obstruction of different etiologies was ruled out step by step. Thrombophilia was excluded by a normal coagulation & autoantibody profile.

The treatment for colonic varices is largely symptomatic management coupled with addressing the underlying pathology that may have caused the varices in the first place. To control episodes of active variceal bleeding, sclerotherapy, band ligation, and surgical resection can be performed. For those with less severe bleeding, adjustments like prescribing a laxative to soften stool in addition to an oral iron supplement have been shown to be effective. Patients who remain hemodynamically stable but are refractory to conservative treatment may respond well to sclerotherapy. A conservative approach is attempted in uncomplicated cases, while when presenting with major bleeding, partial colectomy is required. The importance of a correct diagnosis derives from a serious complication associated with colonic varices, severe lower gastrointestinal bleeding that could endanger the patient’s life and represents a medical emergency.

Conclusion:

Colonic varices may at times present with massive lower GI bleeding. Early diagnosis is the key to control such emergency. A differential of colonic variceal bleeding should therefore always be in the mind, even in the absence of signs of portal HTN. Because the colonic varix could be idiopathic, just like our case.

Reference:


