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Case Report

A rare case of thrombocytosis presentation in patient alcoholic liver disease with portal hypertension gastropathy: a stepwise evaluation

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ABSTRACT

Thrombocytosis is rarely found in patient with chronic liver disease (CLD). The possibility of reactive thrombocytosis could be due to sustained process such as iron deficiency anemia (IDA) because of occult bleeding. Occult bleeding can happen in CLD patient because of portal hypertension gastropathy (PHG) as complication of portal hypertension. A carefully evaluation of anemia can lead to underlying cause of disease, even in limited of supportive evaluation and some other confounding presentation that is thrombocytosis. We report a case of 54 years-old male patient with severe anemia. He had same symptom previously and got transfusion. Peripheral blood smear showed microcytic hypochromic anemia, anisocytosis, and poikilocytosis even pencil cells (pencil cells or cigar cells) with thrombocytosis. No symptom of acute inflammation setting and no clear blood loss was founded. As patient admitted to smoking and heavy alcohol consumption in the past, Ultrasound was performed for screening of underlying disease that cause occult bleeding. Ultrasound of the liver showed generally increased echogenicity suggestive of liver cirrhosis, splenomegaly and minimal ascites. Thus, our patient clinically be suggestive of CLD with portal hypertension that cause PHG.

Keywords: Iron deficiency anemia, Thrombocytosis, Alcoholic liver disease, Portal hypertension gastropathy

INTRODUCTION

Thrombocytopenia seen in up to 78% of cirrhotic patients. Severe thrombocytopenia is a poor prognostic factor associated with significant morbidity, indicating an advanced liver disease with established portal hypertension.[1] Portal hypertension is responsible for its most severe complications, including ascites, bleeding from gastro-esophageal varices and encephalopathy.[2,3]

The presence of esophageal varices and a Child-Pugh class B or C at enrollment were found to predict the incidence of PHG, which might range between 30 and 45%.[4,5]

PHG typically presents in patients with symptoms related to chronic GI bleeding and chronic blood loss, often manifest as IDA.[2,6]

Here we reported a rare case of thrombocytosis presentation in patient alcoholic liver disease with portal hypertension gastropathy.

CASE REPORT

A 54-year-old male patient was brought to the ER with complaints of fainting, worsen chronic general weakness, nausea, vomiting and decrease appetite. Hematemesis and melena were denied. He had same symptoms 5 month ago and got transfusion. He underwent a colonoscopy with normal results (Figure 1). He admitted to smoking and heavy alcohol consumption in the past. No history of routine NSAID tablet consumption.

On physical examination, his blood pressure was found to be 95/60 mmHg, pulse 102 times/minute, respiratory rate

20 times/min, temperature 36.9°C. He looks pale with anemic conjunctiva. The liver was not palpable, but epigastric tenderness was found and there was splenomegaly. Ascites is absent. There is mild pitting edema in all four extremities. No other liver stigmata.

Laboratory results showed a slight leukocytosis (10.08×103/µl), severe anemia (5.6 mg/dl) with microcytic hypochromic features, RDW 18.2% and thrombocytosis (645×103/µl). Liver and renal function, blood glucose and electrolyte were normal. Total protein decrease, albumin was very low (1.9 g/dl) and globulin was normal. Chest x-ray was normal (Figure 2). The results of the stool and urine examination were normal. Peripheral blood smear showed microcytic hypochromic anemia, anisocytosis, and poikilocytosis even pencil cells (pencil cells or cigar cells). An ultrasound of the liver showed generally increased echogenicity suggestive of liver cirrhosis, splenomegaly and minimal ascites (Figure 3).

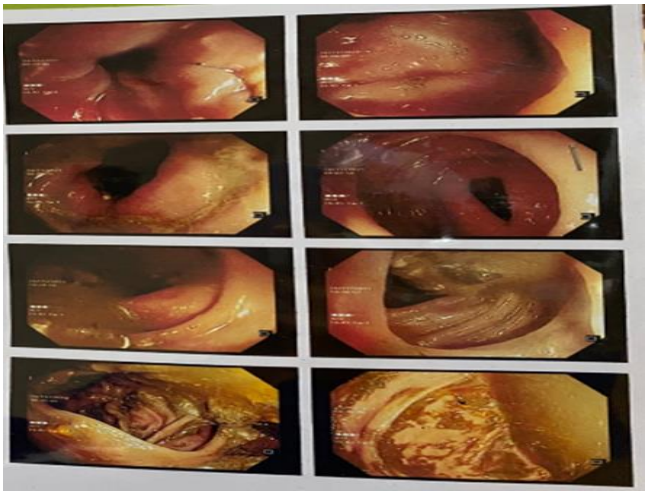

Figure 1: Normal colonoscopy result.

He was diagnosed with CLD ec alcohol with PHG, severe anemia, severe hypoalbuminemia, mild ascites. He was given infusion of NaCl 0.9% and dextrose 5%, ceftriaxone 1 g every 12 hours, esomeprazole 40 mg every 24 hours, ondansetron 4 mg 3 times a day, packed red cell transfusion and 20% albumin infusion. Lactulose liquid 3 times as much as 15 cc.

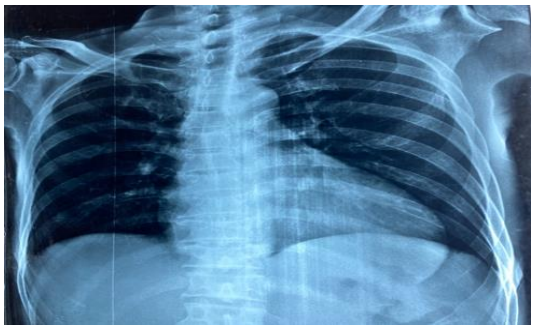

Figure 2: Normal chest X-ray.

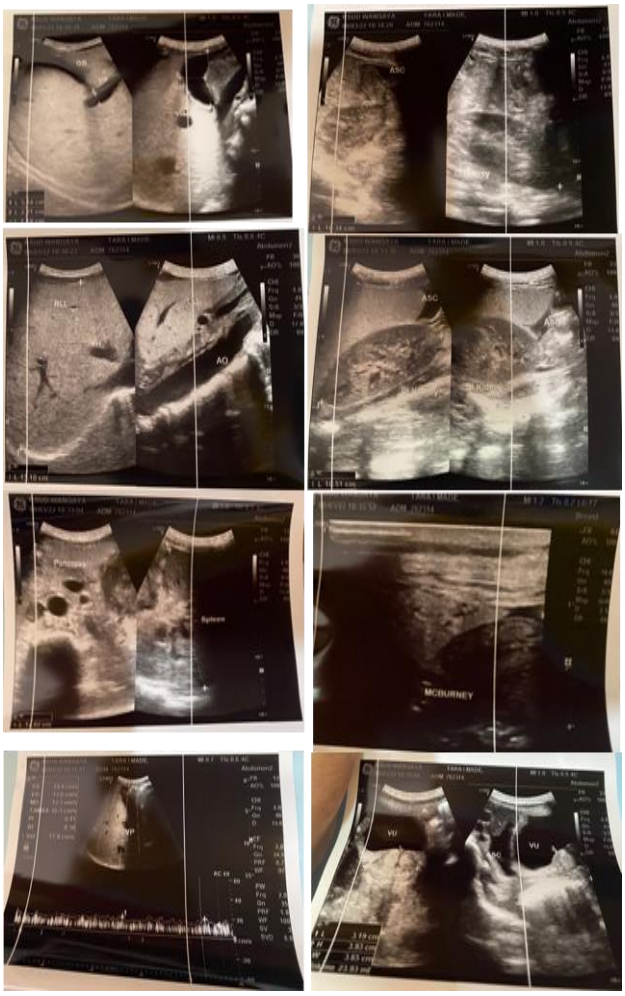

Figure 3: Ultrasound of the liver showed generally increased echogenicity suggestive of liver cirrhosis, splenomegaly and minimal ascites.

DISCUSSION

Iron deficiency anemia (IDA) is leading cause of anemia worldwide.[7-9] In this study, patient came with severe anemia, with CBC and peripheral blood smear support diagnose of IDA. Bone marrow aspiration for diagnosis of IDA is invasive and rarely done routinely.[8] Serum (or plasma) ferritin, serum iron concentration, Transferrin saturation, hepcidin concentration and reticulocyte were not conducted in this case because of cost issue, thus the diagnostic approach used is based on degree of anemia, where severe anemia is often found in IDA, while ACD is more often mild or moderate. In adults, IDA found in the clinic is almost identical to chronic bleeding. The most important cause of IDA in men is gastrointestinal blood loss. Gastrointestinal bleeding can be occult, in which case of IDA could be the only evidence of luminal pathology.[8] In a review of five prospective study of upper endoscopy and colonoscopy in patients with occult GI bleeding, 29 to 56% had an upper GI tract source. Causes of occult bleeding in the upper GI tract include esophagitis, Cameron ulcers (a linear erosion in a hiatal hernia), gastric and duodenal ulcers, vascular ectasias, gastric cancer, and

gastric antral vascular ectasia. In patients older than 40 years, vascular ectasias and non-steroidal anti-inflammatory drug-induced ulcers are the most common causes.[7]

The most common vascular ectasia was gastric antral vascular ectasia (GAVE) and portal hypertensive gastropathy (PHG), about 4% of non-variceal UGIB.[10] In PHG, there seem to be abnormalities in the mucosal microcirculation because of portal hypertension. Two specific factors that have gained considerable attention include hypoxia and inflammation, which may or may not be interrelated. Hypoxia seems to be related to the dysregulation of the mucosal microcirculation. The abnormality in turn may lead to epithelial cell injury and set up a milieu in which there is overproduction of oxygen free radicals, nitric oxide, tumor necrosis factor-a, endothelin-1, prostaglandins, and/or other factors that cause cell injury. Tumor necrosis factor-a, an inflammatory cytokine, seems to be prominent. Further, because of these changes and injury to the epithelium, it has been proposed that the abnormal mucosa in PHG is unable to repair normally and thus may be predisposed to bleeding.[17] The diagnosis of PHG is made by endoscopy and typically shows a snake-skin mosaic pattern (mild subtype), which may have superimposed red signs (severe PHG) and is most commonly located in the proximal stomach (fundus and body). Esophagogastroduodenoscopy (EGD) not evaluate in this patient because of cost issue. As of a stepwise evaluation is need to prove this vascular ectasis exist in this patient by found underlying chronic illness such liver disease that cause portal hypertension.

Liver function tests and ultrasound examination should be performed among patients with harmful alcohol use and/or alcohol-use disorder (AUD) because of strong risk factor for develop of alcoholic liver disease (ALD) as a screening.[11-13] Liver biopsy is still considered the gold standard for establishing a definite diagnosis of ALD. However, liver biopsy is an invasive procedure, with significant morbidity, and is generally not recommended in routine clinical practice for all patients with suspected ALD. ALD is one of the main causes of chronic liver disease worldwide and accounts for up to 48% of cirrhosis associated deaths in the United States.[11,12] Increase echogenicity, splenomegaly and mild ascites in USG are suggestive of cirrhosis.[14] Ascites results from hypoalbuminemia and portal hypertension.[14] Thus portal hypertension can be very suggestive and high probability cause PHG in this study case.

A low platelet count might also be a marker of cirrhosis and is generally the result of platelet sequestration in the spleen as a result of portal hypertension.[14] But in our case presented with thrombocytosis, that very rare in patient be suggestive of chronic liver disease.[15] The possibility of reactive thrombocytosis in this patient could be due to the transient process such as acute inflammatory process, acute bleeding or sustained process such as IDA because of occult bleeding.[16] The pathophysiology of reactive thrombocytosis in IDA remains incompletely understood. The role erythropoietin (EPO) plays in iron-deficiency-related thrombocytosis has garnered much interest. It has been suggested that homology between the receptor for EPO (EPO-R) and TPO (MPL) may underlie this EPO-induced platelet rise, but in vitro studies have shown that EPO does not interact directly with MPL and more likely plays a synergistic role along with TPO in stimulating platelet production.[15,17]

CONCLUSION

A carefully evaluation of anemia can lead to underlying cause of disease. In this case we diagnose cirrhosis in patient with stepwise evaluation of anemia, even in limited of supportive examination and some other confounding presentation that is thrombocytosis. Cirrhosis and its related complications remain a prominent global health concern despite advances in understanding and treating the disorder. Early diagnosis and intervention strategies may reduce the impact of cirrhosis.

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