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Case Report

Normal platelet count and leukocytosis findings in chronic liver disease patient with portal hypertension: a rare case

Felicity Pheeyen Marthadinata[1], Ketut Suryana[2]*

[1]Department of Internal Medicine, Wangaya Hospital, Denpasar, Bali, Indonesia
[2]Department of Internal Medicine, Merpati Clinic, HIV and Allergy-Clinical Immunology Services Unit, Wangaya Hospital, Denpasar, Bali, Indonesia

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***Correspondence:**
Dr. Ketut Suryana,
E-mail: ketutsuryana@gmail.com

ABSTRACT

Haematologic abnormalities are commonly encountered in chronic liver disease (CLD) due to hypersplenism occurred. Hypersplenism in CLD is a major cause of peripheral pancytopenia in patients with hepatic cirrhosis and portal hypertensive gastropathy and is characterized by splenomegaly. Peripheral pancytopenia is defined as a reduction in all three major constituents of the blood to below lower normal range, manifesting as anemia, leukopenia, and thrombocytopenia all occurring at the same time. We report an unusual case, a 44-year-old female patient, no splenomegaly, presented with severe anemia, leukocytosis and normal platelet which are rarely found in CLD.

Keywords: CLD, Portal hypertensive gastropathy, Hypersplenism, Leukocytosis, Normal platelet

INTRODUCTION

Thrombocytopenia, is the single most common haematological manifestation of CLD. As the incidence of thrombocytopenia in CLD is 70% in patients with cirrhosis versus 6% in patients without cirrhosis. In the United States, nearly 4 million adults (1.6% of the population) have CLD.[1]

Lu et al presented the clinical data from 183 patients found that 80.5% of peripheral cytopenia was caused by hypersplenism and it was also found clinical data of 322 hypersplenism patients with decreased peripheral blood cells, admitted with cirrhotic portal hypertension, was retrospectively studied over the last 17 years. In 64% (206/322) of patients, more than 2 kinds of blood cell were decreased, including 89 cases of pancytopenia (43.2%), 52 cases of WBC (white blood cell/leukocyte) + PLT (platelet / thrombocyte) decrease (25.2%), 29 cases of RBC (red blood cell / erythrocyte) + PLT decrease (14.1%), and 36 cases of WBC + RBC decrease (17.5%); in 36% (116/322) of patients, single type blood cell decrease occurred, including 31 cases of PLT decrease (26.7%), 29 cases of WBC decrease (25%) and 56 cases of RBC decrease (48.3%).[2]

CASE REPORT

A 44-year-old female patient, with obesity, complained of hematemesis-melena about 12 hours before came to the emergency department, followed by worsen weakness but still aware to order. Never had symptoms like these before. She no complained of headache change in vision, nose or ear problems, or sore throat cardiopulmonary, genitourinary or dermatologic signs and symptoms. No prior history of hepatitis viral or liver dysfunction, gastrointestinal disorder, diabetes, hypertension, alcohol and prolonged NSAID drugs consumption. No history of underwent any endoscopic ultrasound (EUS), colonoscopy or surgical history in the past. She used to be a housewife, with no heavy activity besides daily activities at her home. She admitted gain her weight after marriage, in normal way, but not sure about how many kilograms and never measuring her body.

Upon physical examination, the patient appeared alert and oriented, but pale. Her blood pressure 101/61 mmHg, with a heart rare 101 beats/min. Her respiration rate was 20 breaths/min, and her oxygen saturation was 96% on room air. She was afebrile, with a temperature 68.1ºF (36.1ºC). Her body mass index (BMI) was 30.04 (height 158 cm, weight 75 kilograms).

Sclera was found no icteric, conjunctivae were pale. Tongue and gums are normal. The chest examination revealed the lungs were clearly bilaterally, no wheezes, rales, or rhonchi. The heart rate and rhythm were regular, and no murmurs or gallops were detected. Abdominal examination revealed the abdomen non-distended, normal active bowel sounds, soft, with no obvious masses, hepatomegaly or splenomegaly. Gynaecologic examination was also unremarkable.

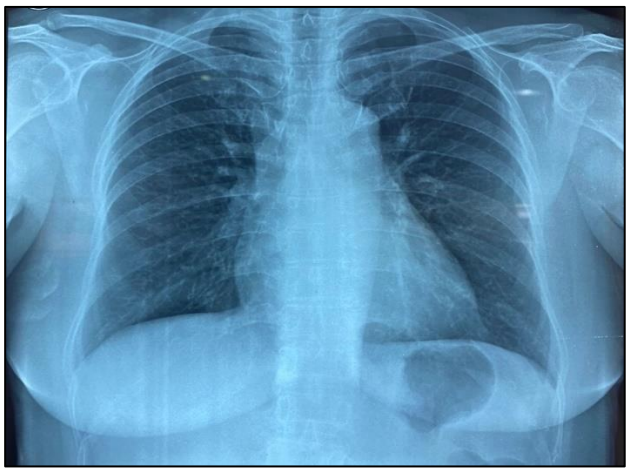

Figure 1: Normal chest X-ray.

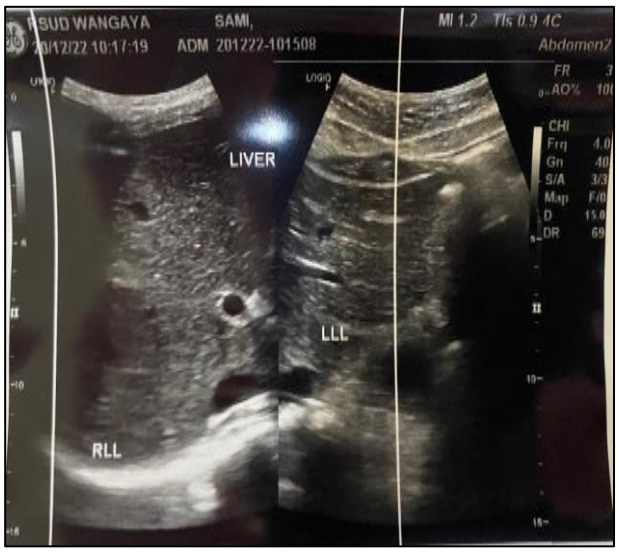

Figure 2: Ultrasound of the liver showed generally increase echogenicity.

Laboratory testing yielded the following results: severe anemia (hemoglobin 5 g/dL; hematocrit, 15.2), leukocytosis (WBC, 20.02×10^3/uL) and with normal MCV 85.9 fL, MCH 28.2 pg, MCHC 32.9 g/L and platelet (347×10^3/uL; total protein decreased with albumin decreased (2.7 g/dL), globulin slightly decreased (2.1 g/dL), fasting blood glucose increased 169 mg/dL, liver, renal function test and electrolyte appeared normal. The result of anti HCV rapid test and HBsAg rapid test were negative. Urine and stool were not examined because the patient has no complaints with the organs concerned. Normal chest X-ray. Abdominal ultrasound (AUS) was performed to asses underlying disease which cause massive gastrointestinal bleeding. This showed generally increased echogenicity as seen as CLD with minimal ascites no splenomegaly.

She was diagnosed with gastrointestinal bleeding in CLD (Child Pugh score 9-B) with PHG, severe anemia, hypoalbuminemia with mild ascites. The patient was treated with crystalloid fluid, a third-generation cephalosporin antibiotic (cefoperazone) twice daily IV for precaution to prevent infection, proton pump inhibitor (esomeprazole 40 mg) IV twice daily, tranexamic acid 500 mg IV three times daily, lactulose oral solution 15 mL three times daily, non-cardioselective beta blocker (propranolol) 10 mg twice daily, antagonist of aldosterone (spironolactone) 50 mg once daily.

A total of 5 units of packed RBCs were transfused resulting in a hemoglobin value of 8.1 g/dl. The condition of the patient gradually improved and she discharged from hospital. Gastroscopy and colonoscopy were scheduled but her spouse and family refused any further investigation.

DISCUSSION

Qamar, et al were analyzed a database of 213 subjects in with compensated cirrhosis without esophageal varices. Subjects were followed for approximately 9 years until the development of varices or variceal bleeding or completion of the study; 84 subjects developed varices. Abnormal haematological was defined as anemia at baseline (hemoglobin, ≤13.5 g/dL for men and 11.5 g/dL for women), leukopenia (WBC≤4000/mm^3), or thrombocytopenia (PLT ≤150,000/mm^3).[2]

Bashour et al did retrospective chart review of 235 patients who underwent a liver biopsy, of the cirrhotic patients, 64% were noted to have platelet counts consistently below 150×10^3/uL, they also had a significantly lower hematocrit and PLT count.[3]

Patients with cirrhosis, a long-term CLD, develop abnormal from multiple factors, including hypersplenism due to overactivity function of the spleen. Not only haematological abnormalities such as peripheral cytopenia but also hematologic indices (HI) are frequently abnormal in patients with cirrhosis. The haematological indices such as mean corpuscular volume (MCV), mean corpuscular Hb (MCH), mean corpuscular

Hb concentration (MCHC), red cell distribution width (RDW) are also been measured to make sure the abnormalities.[4] In this case report we found normal result for HI whereas in some studies examining the occurrence of abnormal HI have reported a prevalence of anemia, thrombocytopenia, and leukopenia (alone or in combination) in between 6% and 77% of patients with cirrhosis.[5] Thrombocytopenia is the most common and first abnormal HI to occur in patients with cirrhosis, followed by leukopenia and anemia. A combination of leukopenia and thrombocytopenia at baseline predicted increased morbidity and mortality.[6]

The pathophysiology of thrombocytopenia in CLD has long been associated with the hypothesis of hypersplenism, where portal hypertension causes pooling and sequestration of all corpuscular elements of the blood, predominantly thrombocytes, in the enlarged and congested spleen. Other mechanisms of importance include bone marrow suppression by toxic substances, such as alcohol or viral infection, and immunological removal of platelets from the circulation. The discovery of the cytokine thrombopoietin has led to the elucidation of a central mechanism. Thrombopoietin is predominantly produced by the liver and is reduced when liver cell mass is severely damaged. This leads to reduced thrombopoiesis in the bone marrow and consequently to thrombocytopenia in the peripheral blood of patients with advanced-stage liver disease.[7,8]

Thrombocytopenia can occur in both the compensated and decompensated stages, but without interventional procedures, there is little or no risk of spontaneous bleeding because of the low platelet count, although the presence of thrombocytopenia can predict the presence of varices that may be at risk of rupturing.[8,9]

In the other hand, it has been well established that cells of the immune system play an important role in the pathogenesis of obesity and metabolic syndrome related chronic disease. Leukocytes are the major source of cytokines, which orchestrate the effectiveness of innate immunity by inducing local inflammation and systemic acute responses, which produce pro-inflammatory cytokines such as TNF-α and IL-6. Innate and adaptive immune become dysfunction, which lead the major component of cirrhosis, also referred to as cirrhosis-associated immune dysfunction syndrome.[10-12]

Liver parenchymal texture is a characteristic that is somewhat subjective and has low sensitivity for the detection of cirrhosis. However, in a series of 103 patients with CLD it has been shown that liver parenchymal texture (graded as fine echotexture, mildly coarse, coarse and highly coarse) has a statistically significant correlation with the degree of fibrosis. When combined with two more features (liver surface nodularity and liver edge), correlation with the degree of fibrosis increased. When compared to echotexture, liver surface nodularity has better accuracy for the presence of cirrhosis reaching both a sensitivity and specificity. In order to provide a fluid-tissue interface, ascites needs to be present for optimal evaluation. Once ascites is present, cirrhosis is generally more advanced and less of a diagnostic challenge.[13]

CONCLUSION

This case of normal platelet and leukocytosis could occur in CLD patient with PHG without any symptoms before. That is why clinician should be aware of this condition, not all CLD patient will have pancytopenia laboratory findings. Careful surveillance is mandatory with regular clinical examination and laboratory blood tests, especially for those have long-term consumption of who considered at high risk, for example patients who alcohol, NSAID drugs or another drug related hepatotoxicity, and herbal medicine. Therefore, we hope that our case report will be a reminder for how to anticipate an unusual finding in CLD patient.

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