

Platelet Abnormalities after Splenectomy for Hypersplenism in Decompensated Cirrhosis: A Case Report

Yucheng Lin¹, Hongcheng Wei^{1, *}

¹ Department of Gastroenterology, The First Affiliated Hospital of Jinan University, Guangzhou, 510000, China

* Corresponding author: Hongcheng Wei (Email: twchc@jnu.edu.cn)

Abstract: Patients in the decompensated stage of cirrhosis are usually associated with hepatic decompensation and portal hypertension, splenomegaly, hypersplenism, and abnormal or significantly reduced blood counts in at least one of the three blood cell lines, mainly platelets. At present, surgical removal of the spleen is still an essential treatment for hypersplenism. The majority of patients who undergo splenectomy can effectively improve the decrease in peripheral blood platelet count. In order to provide some reference value for the diagnosis and treatment of similar clinical cases in the future, we report a case of platelet reduction in hypersplenism in cirrhosis after splenectomy, in which platelets first rose to normal and then decreased abnormally.

Keywords: Cirrhosis, Cirrhotic decompensation, Hypersplenism, Splenectomy, Platelets.

1. Introduction

Cirrhosis is a pathological stage in which various chronic liver diseases progress to a stage characterized by diffuse fibrosis of the liver, pseudo lobule formation, and proliferation of blood vessels inside and outside the liver. The compensated stage of cirrhosis has no obvious clinical symptoms, while the decompensated stage is characterized by portal hypertension and severe impairment of liver function. Patients with cirrhosis often die from multiple organ failure due to complications such as ascites, gastrointestinal bleeding, sepsis, hepatic encephalopathy, hepatorenal syndrome and carcinoma [1]. Splenectomy reduces portal vein pressure and has a significant short-term effect in eliminating hypersplenism [2]. In clinical work, most patients with decompensated cirrhosis who undergo splenectomy can effectively improve the decrease in peripheral blood platelet count [3].

Presentation of case: A 63-year-old man was admitted to the hospital on May 13, 2021 with a complaint of abdominal distension for more than 4 months, aggravated for 1 month. The patient developed abdominal distension with depressed edema of both lower extremities with gingival bleeding with no obvious cause 4 months ago, no fever, no chills, no dizziness, no headache, no nausea, no vomiting, no acid reflux, no heartburn, no abdominal pain, no bloating, no urinary frequency, no urinary urgency, no urinary pain. He then went to the outpatient clinic of Yingde People's Hospital and underwent the following tests: Blood routine examination: WBC $3.3 \times 10^9/L$; PLT $26 \times 10^9/L$; RBC $3.97 \times 10^{12}/L$; CT pelvic and abdominal cavity: Cirrhotic decompensation (portal hypertension, splenomegaly, esophagogastric fundus and splenic vein varices), peritoneal effusion, hepatic S4 segment cyst, small left kidney stone, enlarged prostate, partial intestinal wall thickening and edema; Electronic Gastroscopy: Chronic gastritis, esophageal varices (mild). He was treated with anemia improvement therapy, leukocyte boosting therapy, liver protection therapy, and anti-hepatitis B virus therapy, but his symptoms did not improve significantly. On January 12, 2021, he came to our gastroenterology

department and completed tests suggestive of hypersplenism and trilineage hemocytopenia. We gave a combination of acid suppression and gastric protection, diuretic, albumin supplementation, anti-hepatitis B virus, and blood transfusion, and the patient was discharged after his symptoms improved. Meanwhile, we advised him to receive further treatment in surgery after discharge. The patient was then hospitalized in our gastrointestinal surgery department on March 8, 2021, and underwent "Laparoscopic splenectomy and Loosening of intestinal adhesions" under general anesthesia on March 16, 2021. Pathology of the spleen after surgery suggests hypersplenism. The postoperative blood count, which was repeated on March 17, 2021, showed a PLT of $142 \times 10^9/L$. After the patient was discharged from the hospital, he was regularly rechecked at the local health center. In the past 1 month, he had progressive decrease in blood cells of three lines with abdominal distension, no nausea or vomiting, no black stool, abdominal pain or diarrhea, no fever, chills or cough, no chest tightness or shortness of breath, and no edema of both lower limbs. The patient came to our hospital for further treatment and was admitted to our department for "Cirrhosis of The Liver with Esophageal Varices". Since the onset of this disease, the patient has had normal mental and sleep quality, normal bowel movements, and a weight gain of about 2.5 Kg in the last 2 months. The patient had a previous history of viral hepatitis B for more than 6 months, a history of smoking for more than 40 years, and a history of alcohol consumption for more than 40 years, drinking about 50 g of alcohol per day, and had quit drinking for 3 years.

Results of inspection: Body temperature was $36.7^\circ C$, pulse was 82 beats/min, respiration was 16 breaths/min, and blood pressure was 103/68 mmHg. He had a clear consciousness, good spirit, anemic face, no yellow staining of skin mucosa, no rash or bleeding point, no liver palm and spider nevus, no obvious abnormalities in heart and lung examination; the abdomen was flat, no abdominal wall varices were seen, liver and spleen were not touched under the ribs and no pressure pain, positive mobile turbid sounds in the abdomen, no edema in both lower limbs. The results of the examination after admission are as follows: Blood routine examination: WBC

3.82X10⁹/L, Hb 67g/L, PLT 36X10⁹/L; Liver Function: ALB 29.4g/L; ALT 22U/L; AST 34U/L; TBil/DBil 9.6/1.9umol/L; Blood Ammonia: 92ug/dl; Hepatitis B Series: HBsAg 225ng/ml, HBeAb 4PEIU/ml, HBcAb 10PEIU/ml; HBVDNA undetected. Abdominal ultrasound: consistent with cirrhosis sonogram; liver cyst sonogram; left lobe of liver atrophy is poorly displayed. Small amount of perihepatic effusion sonogram. No abnormality was seen in the biliopancreatic double kidney and bladder.

Diagnosis and treatment: After admission, he was treated with anemia correction, platelet raising, blood ammonia lowering, acid suppression, anti-hepatitis B virus, liver protection, hypoproteinemia correction, etc. At the same time, we had a consultation with the hematology department, and they recommended morphological examination of the bone marrow, flow cytology testing and bone marrow biopsy, and continued to treat with iron supplementation and platelet raising with Recombinant Human Thrombopoietin. A bone marrow aspiration biopsy was performed on May 15, 2021, and bone marrow cytology tests and histochemical staining revealed: blood film characteristics: normal peripheral blood leukocyte count. Red blood cells were variable in size and platelets were rare and scattered. Opinion: The bone marrow is actively proliferating, with granulocyte lineage proliferation predominant and erythroid lineage proliferation reduced, platelets are rare, according to the iron stain suggesting iron deficiency, please consider in the context of clinical considerations. Pathological diagnosis of bone marrow tissue: Conclusion: 1. The hematopoietic tissue was uneven in this case, with active proliferation of granulocyte lineage in the hyperplastic area and a high proportion of naive erythrocytes, and hyperplastic anemia was not excluded. 2. No significant decrease in megakaryocytes was seen, and it is recommended to evaluate the maturation status of megakaryocytes in combination with bone marrow smear. On May 19, 2021, we reviewed the blood test and showed that the PLT decreased to 28.70X10⁹/L, thus we changed to "Avatrombopag 20mg bid" to increase platelets. At the same time, the patient's gastroscopy report showed esophageal varices (degree III), and we performed an Endoscopic variceal ligation of esophageal veins on May 21, 2021. On May 25, 2021, the Blood routine examination showed that the PLT rose to 118X10⁹/L. The Avatrombopag was discontinued and the patient was later discharged with improved symptoms.

2. Discussion

Hypersplenism is a manifestation of decompensated cirrhosis, commonly with leukopenia, proliferative anemia, and thrombocytopenia in the peripheral blood, with thrombocytopenia being the most common [3]. The main mechanisms of thrombocytopenia in hypersplenism in cirrhosis are: inhibition of platelet production (bone marrow suppression, reduction of thrombopoietin (TPO)), platelet splenic retention (significant increase in the splenic blood pool, with 50% to 90% of platelets retained in the spleen, resulting in circulating thrombocytopenia due to increased platelet retention in the spleen from the blood circulation) and platelet destruction due to platelet autoantibodies, among other factors [4]. Currently, hypersplenism is considered to be the main cause of thrombocytopenia in patients with cirrhosis. Removing the spleen of patients with cirrhosis and relieving the destruction of peripheral blood cells by the spleen is one of the essential methods to improve peripheral blood

cytopenias in clinical practice [5]. Most patients with hypersplenism undergo splenectomy, which relieves the retention of platelets by the spleen, and platelets in the peripheral blood can rise to normal values, even higher than normal values [6]. When thrombocytopenia reappeared, it suggests that there may be other factors contributing to thrombocytopenia, including a decrease in TPO. Avatrombopag is a small molecule TPO receptor agonist that mimics the TPO effect and stimulates megakaryocyte proliferation and differentiation from bone marrow progenitor cells, thereby increasing platelet production [7]. In this case, platelets were low in a patient with hypersplenism in cirrhosis and could be raised to normal after splenectomy. However, thrombocytopenia recurred in this admission. After excluding hematological-related diseases, platelets could return to normal after symptomatic treatment with TPO receptor agonists to elevate platelets, suggesting that the patient may have TPO deficiency. Serum TPO levels in patients with chronic liver disease are closely associated with thrombocytopenia [8]. The decrease in platelets is not only considered to be due to hypersplenism [9]. Splenectomy alone may not completely eliminate the factor of thrombocytopenia [10]. In clinical work, it is still necessary to reexamine platelets regularly after encountering patients with hypersplenism in cirrhosis who have recovered their platelets to normal levels with surgical treatment to avoid delaying the patient's treatment.

3. Conclusion

The platelet count frequently decreases in patients with cirrhosis in combination with hypersplenism, and most of them can recover to normal or even higher than normal values after splenectomy. This case reported that the patient's platelets rose to normal and then appeared thrombocytopenia again, reminding clinicians to regularly review the patient's platelet level when encountering similar cases, and give corresponding treatment in time when abnormalities occur.

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