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One Case of Primary Thrombocythemia with Concealed Hypokalemia Complicated by Acute Myocardial Infarction

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Abstract

Medical history summary: Male, 47 years old, was admitted to the hospital due to "dizziness accompanied by chest tightness and pain for more than 8 days". One week ago, the patient experienced chest tightness, chest pain accompanied by profuse sweating for 3 hours and underwent emergency percutaneous coronary intervention (PCI) at a local hospital. The procedure revealed left main stem occlusion with subsequent left main stem to left anterior descending artery percutaneous transluminal coronary angioplasty (PTCA). After the procedure, the patient experienced hemodynamic instability, recurrent ventricular fibrillation, and critical condition, thus transferred to our hospital for further treatment. **Symptoms and signs:** The patient is in a comatose state, unresponsive to stimuli, with bilateral dilated pupils measuring 2.0 mm, exhibiting reduced sensitivity to light reflex, and recurrent fever. Coarse breath sounds can be heard in both lungs, with audible moist rales. Irregular breathing pattern is observed, and heart sounds vary in intensity. No pathological murmurs are auscultated in any valve auscultation area. **Diagnostic methods:** Coronary angiography results at the local hospital showed complete occlusion of the left main stem, and left main stem to left anterior descending artery percutaneous transluminal coronary angioplasty (PTCA) was performed. However, the distal guidewire did not pass through. After admission, blood tests showed a Troponin T level of 1.44 ng/ml and a Myoglobin level of 312 ng/ml. The platelet count was 1390 × 10^9/L. Von Willebrand factor (vWF) activity was measured at 201.9%. Bone marrow aspiration biopsy showed active bone marrow proliferation and platelet clustering. The peripheral blood smear also showed platelet clustering. JAK-2 gene testing was positive, confirming the diagnosis of primary thrombocytosis. **Treat-**

ment methods: The patient is assisted with mechanical ventilation and intra-aortic balloon counterpulsation to improve coronary blood flow. Electrolyte levels are closely monitored, especially maintaining plasma potassium levels between 4.0 and 4.5 mmol/l. Hydroxyurea 500 mg is administered for platelet reduction. Anticoagulants and antiplatelet agents are used rationally to prevent further infarction or bleeding. Antiarrhythmic, lipid-lowering, gastroprotective, hepatoprotective, and heart failure treatment are also provided. **Clinical outcome:** The family members chose to withdraw treatment and signed for discharge due to a combination of reasons, including economic constraints and uncertainty about the prognosis due to the long disease course. Acute myocardial infarction has gradually become one of the leading causes of death in our country. As a "green channel" disease, corresponding diagnostic and treatment protocols have been established in China, and significant progress has been made in emergency care. There are strict regulations for the time taken from the catheterization lab to the cardiac intensive care unit, and standardized treatments are provided to patients once they enter the intensive care unit. Research results show that the incidence of acute myocardial infarction in patients with primary thrombocythemia within 10 years is 9.4%. This type of disease is rare and difficult to cure, posing significant challenges to medical and nursing professionals. In order to benefit future patients, we have documented individual cases of treatment and nursing care for these patients. The research results show that these patients exhibit resistance to traditional oral anticoagulant drugs and require alternative anticoagulants. Additionally, there are significant differences in serum and plasma potassium levels among patients. Therefore, when making clinical diagnoses, it is necessary to carefully distinguish between the two. Particularly, nursing personnel should possess dialectical thinking when supplementing potassium levels in patients in order to reduce the incidence of malignant arrhythmias and mortality rates.

Keywords

Primary Thrombocythemia, Acute ST-Segment Elevation Myocardial Infarction, Concealed Hypokalemia, Thrombosis, Bleeding

1. Introduction

Primary thrombocythemia (essential thrombocythemia, ET) is a rare myeloproliferative disorder characterized by persistent elevation of platelet (PLT) count and impaired PLT function. The incidence is approximately 1 - 2 cases per 100,000 individuals [1]. One-third of patients with primary thrombocythemia develop venous and arterial thrombosis, which is more commonly observed in the limbs, internal carotid arteries, and other visceral arteries. Isolated thrombosis within the coronary arteries is extremely rare. Studies have shown that the incidence of acute myocardial infarction within 10 years among patients with

primary thrombocythemia is 9.4% [2] [3]. In this case, the thromboelastography performed on the 9th day after myocardial infarction revealed 0% inhibition with aspirin, leading to a switch to anticoagulation therapy with Xinweining. Additionally, this patient, who had primary thrombocythemia, developed recurrent ventricular fibrillation due to concealed hypokalemia, posing new challenges in managing arrhythmias and maintaining electrolyte balance. By reporting this case, the aim is to increase understanding of this condition and provide clinical reference.

2. Clinical Data

2.1. General Data

The patient, male, 47 years old, experienced sudden dizziness without apparent cause 7 days ago, accompanied by chest tightness, shortness of breath, profuse sweating, nausea, and vomiting in the precordial area. They sought medical attention at a local hospital where an electrocardiogram (ECG) was performed. The ECG showed ST segment elevation of approximately 0.1 - 0.3 mV in leads V1-V6 and leads I and aVL, poor R-wave progression, and a low amplitude of approximately 0.3 mV in leads II, III, and aVF. Troponin T level was measured at 0.723 ng/ml, indicating acute extensive anterior and high lateral wall myocardial infarction. Emergency percutaneous coronary intervention (PCI) was performed, revealing total occlusion of the left main trunk with no forward blood flow (TIMI 0 grade). Multiple attempts at aspiration thrombectomy from the proximal segment of the left anterior descending artery to the left main trunk achieved successful thrombus removal, with visualization of the circumflex artery and major first diagonal branch. The distal end of the left anterior descending artery did not show contrast filling despite repeated attempts to advance the guidewire. Intracoronary injection of tirofiban (10 ml) was administered, and subsequent angiography showed residual stenosis of 30% in the distal section of the left main trunk, with a small amount of thrombus present. There was also a small amount of thrombus observed at the opening of the circumflex artery. Blood flow improved to TIMI grade 3. After the surgery, the patient was transferred to the Coronary Care Unit (CCU) for monitoring and treatment. They were provided with assisted ventilation through an endotracheal tube connected to a ventilator. Treatment included anticoagulation, antiplatelet therapy, lipid-lowering medication, antiarrhythmics, and maintenance of fluid and electrolyte balance. During this period, the patient experienced recurrent ventricular fibrillation and required treatments such as electrical defibrillation and cardioversion to restore sinus rhythm. Previous cardiac echocardiography at an external hospital showed an ejection fraction (EF) of 25%, with enlargement of the left atrium and left ventricle, as well as decreased diastolic and systolic function of the left ventricle. The patient's vital signs were unstable, and their condition was critical with a poor prognosis. The patient's family requested transfer to our hospital for further treatment. The patient has no history of hypertension,

hyperlipidemia, diabetes, or a family history of coronary heart disease. Their BMI is within the normal range, and they do not smoke but consume alcohol in moderation. Eight years ago, they were hospitalized for chest pain and underwent coronary angiography, which showed no abnormalities.

Physical examination: T: 37.6˚C, R: 25 breaths per minute, BP: 144/80 mmHg, heart rate: 104 beats per minute. The patient is in a comatose state and unresponsive. Pupils are equally dilated, with a diameter of 2.0 mm, and show reduced sensitivity to light reflex. Coarse breath sounds are heard in both lungs, with audible moist rales. There is no pleural friction rub. No prominence is observed in the precordial region, and the apical impulse is normal. There is no tremor or pericardial friction sensation. The cardiac dullness is normal. The heart rate is regularly irregular, and the heart sounds are unequal in strength. No pathological murmurs are heard in the auscultation areas of the valves.

2.2. Inspection

During hospitalization, the initial diagnosis combination of bone marrow smears indicated a consistent picture with primary thrombocythemia. The combination for plasma cell disorders, B-cell lymphoproliferative disorders monitoring, B-ALL combination, T-ALL combination, and myeloid leukemia antigen detection did not show any specific abnormalities. JAK2 gene was positive (**Figures 1-3**).

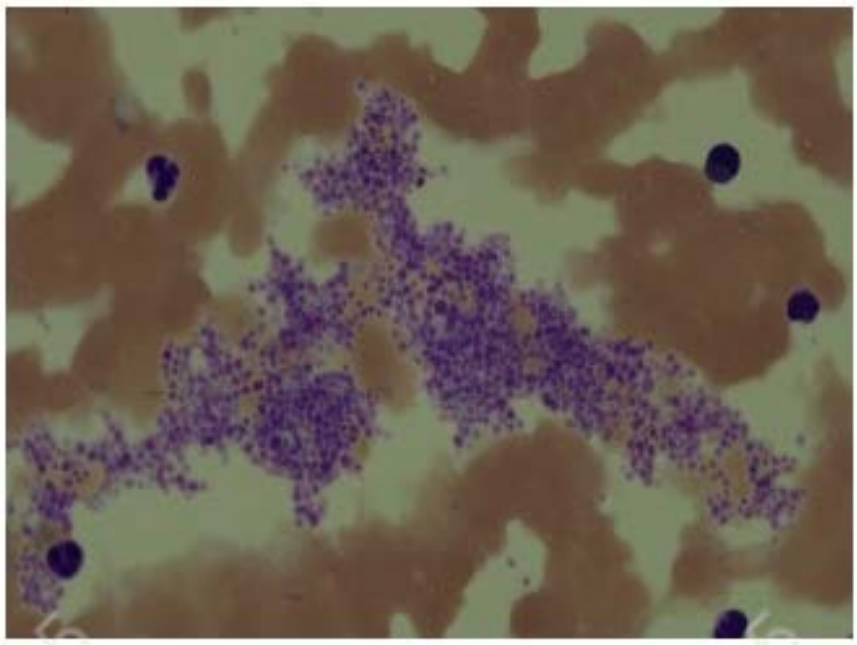

Figure 1. Initial diagnosis based on bone marrow smear shows active bone marrow proliferation, with 81% of the cells being from the granulocyte series, indicating an increased ratio. The majority of the granulocytes are segmented neutrophils. The erythroid series accounts for 6% of the cells, indicating a decreased ratio, but their morphology is generally normal. A total of 14 megakaryocytes are observed in the entire slide, with 13 of them being granular megakaryocytes. The platelets are clustered together.

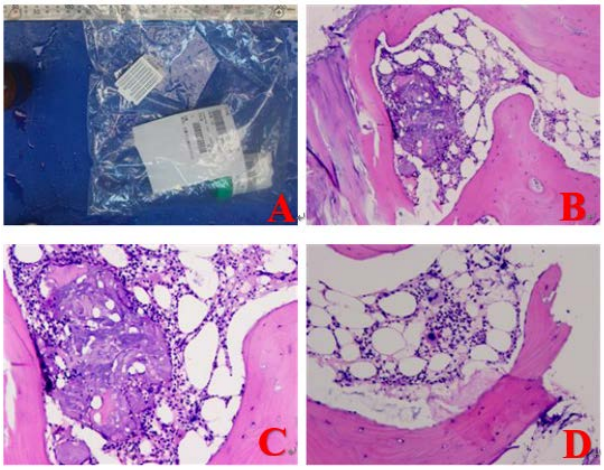

Figure 2. The tissue biopsy of the left posterior superior iliac spine mainly shows bone tissue, with focal hematopoietic tissue observed. The ratio of granulocytes to red blood cells is approximately normal, with predominantly immature stages present, including segmented nuclear macrophages. (A): CK positive immunohistochemical staining ×400; (B): CK positive immunohistochemical staining ×400; (C): CK positive immunohistochemical staining ×400; (D): CK positive immunohistochemical staining ×400.

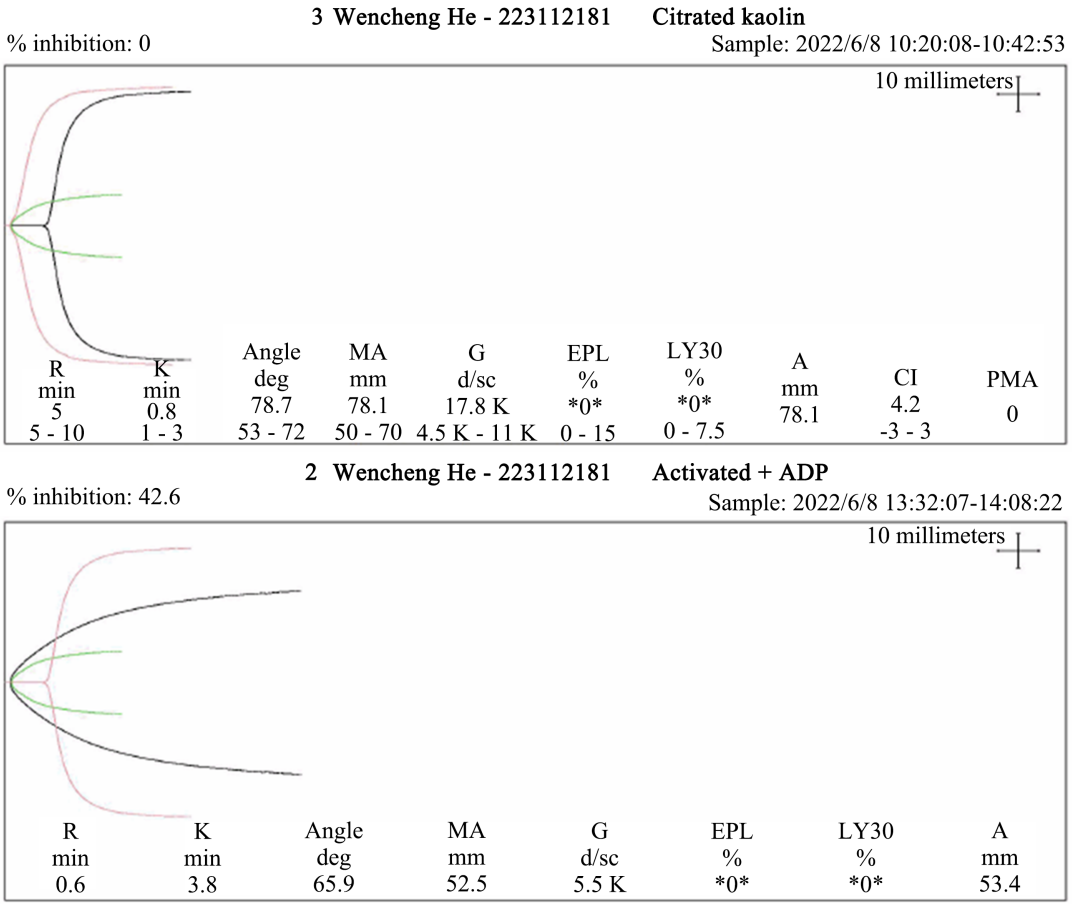

Figure 3. According to the thromboelastography (TEG) test, the fibrinogen reaction level is high, indicating increased platelet function. The platelet AA inhibition rate is 0.0%, and the ADP inhibition rate is 42.6%, suggesting insufficient antiplatelet effect of aspirin.

Upon admission, bedside echocardiography showed changes consistent with coronary heart disease and myocardial infarction. There is left atrial enlargement and significant left ventricular enlargement, with significantly reduced left ventricular systolic function. The right ventricular systolic function is normal. No specific abnormalities were found on bedside chest x-ray. Laboratory tests showed high-sensitivity troponin T at 1.44 ng/ml, NT-proBNP at 10,576 pg/ml, myoglobin at 312 ng/ml. The white blood cell count was 10.5 × 10^9/L, with a neutrophil count of 17.32 × 10^9/L and red blood cell count of 4.59 × 10^9/L. The platelet count was 1390 × 10^9/L, with a plateletcrit of 1.271 × 10^9/L. Venous potassium level was 5.57 mmol/L, and arterial blood gas analysis showed a potassium level of 3.8 mmol/L. D-dimer was 2.14 mg/L, fibrinogen was 6.83 g/L, total cholesterol was 2.8 mmol/L, high-density lipoprotein cholesterol was 0.78 mmol/L, and low-density lipoprotein cholesterol was 1.69 mmol/L. Cytomegalovirus antibodies were greater than 250 IU/ml. Sputum culture grew Moraxella catarrhalis. The von Willebrand factor (vWF) activity was 201.9%, and there were no abnormalities in the red/white blood cell morphology examination (Figures 4-6).

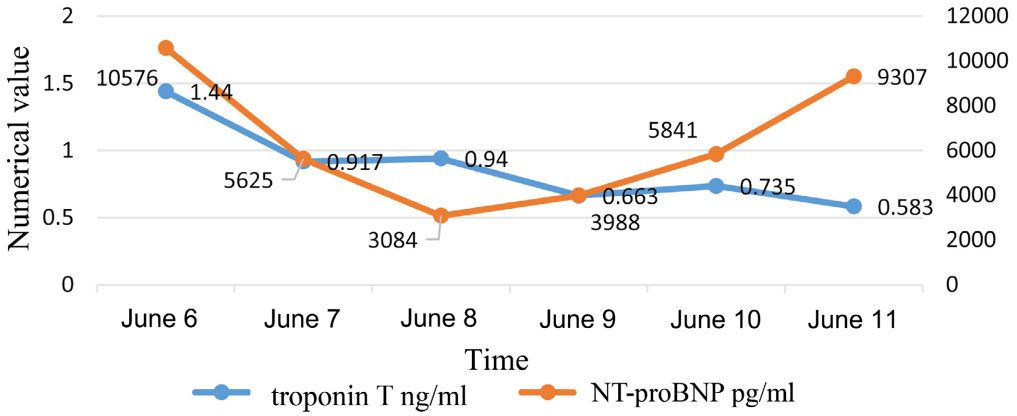

Figure 4. The dynamic changes of troponin T and NT-proBNP after admission.

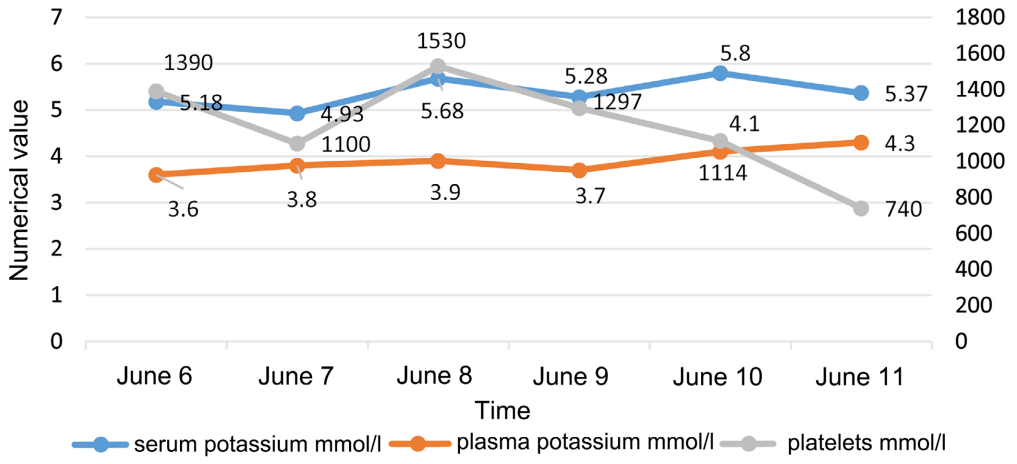

Figure 5. The dynamic changes of blood potassium and platelets after admission.

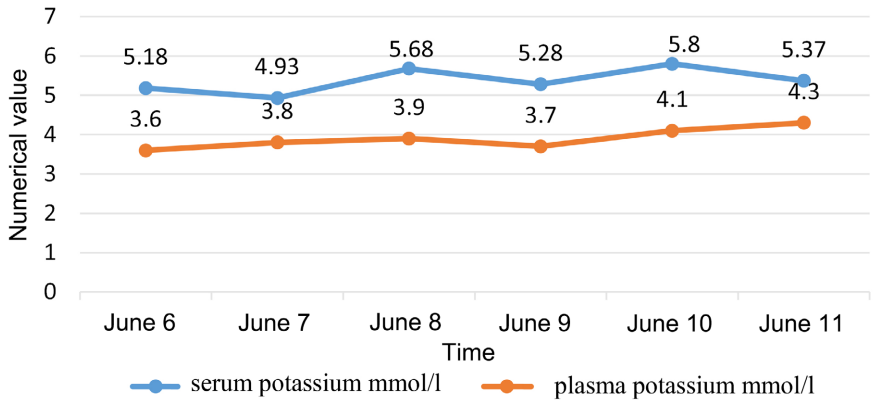

Figure 6. The dynamic changes of serum potassium and plasma potassium after admission.

3. Diagnosis and Differential Diagnosis

3.1. Aortic Dissection

Supporting point: dizziness accompanied by chest tightness, with elevated blood pressure during attacks. Non-supporting point: bilateral upper limb blood pressure is essentially symmetrical, no typical back tearing pain. Conclusion: Basically ruled out.

3.2. Pulmonary Embolism

Supporting point: dizziness accompanied by chest tightness, elevated D-dimer levels. Non-supporting point: no significant respiratory distress. Conclusion: Not currently supported.

Based on the patient's clinical presentation, electrocardiogram results, laboratory tests, bone marrow smear, and imaging results, the final diagnosis is primary thrombocythemia with acute ST-segment elevation myocardial infarction.

4. Treatment

After admission, the patient had unstable hemodynamics, heart failure, and developed pulmonary infection and liver damage. Tracheal intubation was performed and assisted ventilation was provided through a ventilator. Intra-aortic balloon counterpulsation was used to improve coronary blood flow. Antibiotics such as Mephenesin and Stabilokexin were administered to treat the infection. Aspirin and Berlinta were given for antiplatelet therapy. Ruige was used for pain relief, Lidocaine for controlling arrhythmias, Iloprost for heart rate control, Levosimendan and Sacubitril/Valsartan for heart failure, and Norepinephrine to maintain blood pressure. Politera was prescribed for lipid regulation. Consultations with the Digestive Department resulted in the use of Tianqinganmei to protect the liver, and consultations with the Nutritional Department helped with nutritional support treatment.

Since this patient has no high-risk factors for coronary artery disease and the possibility of an acute coronary event related to thrombocytosis is considered, a

consultation with the Hematology Department is requested to further investigate the cause of thrombocytosis. As the results of the bone marrow puncture are not yet available and the patient has unstable hemodynamics, platelet apheresis cannot be performed. Therefore, oral hydroxyurea is prescribed to lower the platelet count. The results of the bone marrow puncture indicate primary thrombocythemia. The Hematology Department suggests waiting for the BCR/ABL fusion gene results to rule out chronic myeloid leukemia and increasing the dosage of hydroxyurea. The patient's platelet count gradually decreases. Early malignant arrhythmias are common in patients with myocardial infarction, so it is particularly important to maintain electrolyte balance and anticoagulant/antiplatelet therapy. Thromboelastography results of this patient show high platelet function with zero inhibition rate of aspirin, indicating poor antiplatelet efficacy. Therefore, aspirin was discontinued and replaced with heparin for anticoagulant therapy. This patient had repeated ventricular tachycardia and ventricular fibrillation in another hospital, and also briefly experienced ventricular tachycardia after admission to our hospital. Controlling arrhythmias and maintaining electrolyte balance, especially blood potassium levels, are crucial. However, this patient had hypokalemia with a large gap between serum potassium and blood gas potassium levels. After correctly identifying the patient's hypokalemia, daily potassium supplementation was provided, and the patient no longer experienced life-threatening ventricular arrhythmias.

5. Treatment Results, Follow-Up and Outcomes

After treatment, the patient's platelet count decreased to 740 × 10^9/L, cardiac troponin T decreased to 0.583 ng/ml, NT-proBNP decreased to 9307 pg/ml, and plasma potassium was maintained around 4.5 mmol/L. During hospitalization, there were no thrombotic events, bleeding episodes, or fatal arrhythmias. The patient's family voluntarily requested discharge.

6. Discussion

Thrombosis formation is a crucial step in the development of acute myocardial infarction, and platelets play a vital role in this process. In most cases, platelets are involved in the intermediate stage, where after plaque rupture, the irregularly exposed subendothelial matrix triggers platelet adhesion and aggregation. In the case of primary thrombocythemia, the increased platelet count can be the initiating factor for arterial thrombotic events. An increased platelet count can elevate blood viscosity and promote thrombus formation. On the other hand, in patients with essential thrombocythemia (ET), changes in platelet glycoprotein receptor on the platelet surface, increased platelet P-selectin and platelet reaction protein expression, activation of platelet glycoprotein IIb/IIIa receptor, JAK2/V617F gene mutation, and other factors can reduce sensitivity to antiplatelet aggregation drugs or activate platelets, thus promoting thrombus formation.

Research has shown that patients with JAK2/V617F gene positivity have a

higher risk of thrombosis occurrence [1], and in this case, the patient is JAK2 gene positive. Previous studies have indicated that the left anterior descending artery is the most common site of occlusion, but occlusions can also occur in the right coronary artery. This may be related to the high blood flow pressure and shear stress in the left anterior descending artery, making it more susceptible to endothelial damage [4]. In this case, the patient had complete occlusion of the left main coronary artery, and after thrombus aspiration, the distal guidewire could not pass through, which is consistent with previous studies. In patients with essential thrombocythemia (ET), not only is there a risk of thrombosis due to abnormal platelet function, but there is also a risk of bleeding.

Studies have shown that patients with extreme thrombocytosis (>1500 × 10^9/L) have a significantly increased risk of bleeding, and the use of antiplatelet medications can also lead to a significant increase in bleeding risk. For AMI patients with concurrent essential thrombocythemia (ET), the use of antiplatelet aggregation and anticoagulant drugs should be individualized, with monitoring for any signs of bleeding tendency. Research has indicated that thromboelastography can better guide the antiplatelet therapy of myocardial infarction patients and reduce the risk of bleeding.

Based on the above situation, in order to ensure the safe and effective implementation of clinical treatment work, we conducted synchronous testing of potassium levels in serum and plasma of the patient during the same time period. The experimental samples were all patient blood specimens, which avoided factors such as hemolysis and ensured that the single variable was the blood component. The research results show a positive correlation between platelet levels and the difference in serum and plasma potassium levels. In other words, the higher the platelet count, the greater the difference between serum and plasma potassium, and the corresponding increase in the incidence of latent hypokalemia. This study further confirms previous theoretical findings.

On the 9th day after the onset of the illness, the thromboelastography results of this patient showed high platelet function, with an aspirin inhibition rate of 0, indicating aspirin resistance. Therefore, it was promptly switched to the anticoagulant medication Xinfu Ning to reduce the risk of thrombosis. Xinfu Ning is a non-peptide glycoprotein IIb/IIIa receptor antagonist, and this receptor is the main antibody on the platelet surface that is involved in platelet aggregation. Blocking this receptor can strongly inhibit platelet adhesion and aggregation, exerting a powerful anti-thrombotic effect [5] [6].

Research has shown that when platelets are >988 × 10^9/L, 17.2% of patients experience pseudohyperkalemia [7]. Pseudohyperkalemia was initially reported in 1955 by Hartmann and Mellinghoff. It refers to the phenomenon where serum potassium levels measured outside the body are higher than normal, while the potassium concentration in the intracellular plasma is within the normal range. If the difference between the two is greater than 0.4 - 0.5 mmol/L, it is called pseudohyperkalemia.

Most cases of pseudohyperkalemia are caused by improper blood collection and mishandling of blood specimens, leading to hemolysis and subsequent release of potassium ions from intracellular space. Since hemoglobin and lactate dehydrogenase can be released simultaneously from damaged cells, this phenomenon can be detected and reported through routine chemical tests. Less commonly, pseudohyperkalemia can also be caused by erythrocytosis and thrombocytosis. When the platelet count exceeds >500 × 10^9/L, excessive potassium can be released from the platelets during the process of venous blood clotting, resulting in a serum potassium level that is 0.5 mmol/L higher than plasma potassium levels (with a sensitivity of 71% and specificity of 89%) [8] [9] [10].

Research has shown that for every increase of 100 × 10^9/L in platelet count, the measured value of serum potassium increases by approximately 0.15 mmol/L [11]. In this case, the patient has a platelet count as high as 1300 × 10^9/L, with a serum potassium level of 5.18 mmol/L and a plasma potassium level of 3.6 mmol/L. Although hyperkalemia did not occur, significant differences were observed between the two measurements through repeated blood sampling and simultaneous measurement of serum and plasma potassium levels. Based on the comparison of the results and the patient's clinical presentation, it was determined that this patient may have early occult hypokalemia. The patient has hemodynamic instability and is at risk of potentially fatal ventricular arrhythmias. Correctly assessing the patient's potassium levels can prevent the occurrence of malignant ventricular arrhythmias and avoid the occurrence of serious complications.

7. Conclusion

Through this case, the following insights can be obtained: Primary thrombocythemia presenting solely as coronary artery thrombosis with acute ST-segment elevation myocardial infarction is relatively rare. Effective antiplatelet aggregation drugs and anticoagulants are crucial for the treatment of acute myocardial infarction. Early thromboelastography should be performed to guide the administration of antiplatelet drugs in myocardial infarction patients. In patients with primary thrombocythemia, simultaneous measurement of serum potassium and plasma potassium levels should be conducted. Treatment should be guided based on the potassium levels to avoid pseudo-hyperkalemia or covert hypokalemia, which can lead to fatal ventricular arrhythmias and other events. Considering that these patients are relatively rare and some of them have difficulties in accessing timely treatment, the treatment and nursing protocols for patients with essential thrombocythemia (ET) complicated with acute myocardial infarction (AMI) still need to be improved. In order to achieve better nursing outcomes, it is necessary to accumulate more individual cases, integrate nursing routines, and refine nursing bundles for reference by colleagues, contributing to the well-being of patients. We hereby declare that human materials or human data are used in accordance with the Helsinki Declaration.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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