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Case Report

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Abciximab-induced thrombocytopenia: Correct management

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Abstract

Thrombocytopenia is a possible side effect of routinely administered medical agents. Abciximab is one of the three potent intravenous glycoprotein IIb/IIIa receptor inhibitors (along with eptifibatide and tirofiban) that have shown significant positive outcomes when used in patients with intracoronary thrombus. The use of abciximab was associated with the risk of thrombocytopenia. This case reported the development of thrombocytopenia and treatment management after using abciximab.

Keywords: Abciximab, thrombocytopenia, glycoprotein IIb/IIIa receptor inhibitors, side effect

1. Introduction

Glycoprotein (GP) IIb/IIIa receptor inhibitors are antiplatelet agents used in interventional cardiology in acute coronary syndrome. Thrombocytopenia may develop as a side effect due to these agents, and its incidence has been reported as 8-10%.

Thrombocytopenia can be considered pathophysiologically, as decreased production, increased destruction, and a disorder in platelet distribution. Many etiological factors can cause thrombocytopenia with different mechanisms, and drug-induced thrombocytopenia can also be observed. Drug-induced antibodies cause immune destruction of platelets. One of these drugs is abciximab, a glycoprotein (GP) IIb/IIIa receptor inhibitor that prevents platelet aggregation. This case aimed to review thrombocytopenia that developed after abciximab treatment.

2. Case Report

The patient, who applied at the emergency department with an epileptic seizure complaint, was hospitalized as we detected inferior myocardial infarction (MI) in their electrocardiography (ECG). We performed the right and left selective coronary arterial intervention procedure through the right femoral route on October 11, 2021, and administered 7500 UFH, 300 mg acetylsalicylic acid, 180 mg ticagrelor and one vial of abciximab. The patient was admitted to the Coronary Intensive Care Unit on October 11, 2021, discharged without any bleeding, hematoma or other complications at the intervention site. We hospitalized the patient after the outpatient visit on November 2, 2021, as we noticed thrombocytopenia, besides intraoral bleeding and hematuria. In the examinations of the patient, the values were WBC: 10000 mm^3 HGB: 10.1 g/dl PLT: 1000 mm^3. We performed a peripheral smear evaluation to differentiate pseudothrombocytopenia and evaluated the platelet level, which was in accordance with the peripheral smear. We started prednol 40 mg on November 3, 2021, and gave IVIg treatment as 1 mg/kg on November 3-4, 2021. We observed an increase in the course of thrombocytes in the follow-ups. On November 8, 2021, we discharged the patient with 107.000 mm^3 platelets in the examinations. We found the platelet as 247000 mm^3 in the examinations during the November 15, 2021 outpatient visit.

3. Discussion

Thrombocytopenia is defined as when circulating platelets are below the typical number. Pseudothrombocytopenia should be differentiated from true thrombocytopenia by repeating the platelet count using citrate anticoagulated blood. Acute thrombocytopenia is usually caused by pseudothrombocytopenia, drug-induced, idiopathic thrombocytopenic purpura gestational and less often chronic liver disease, myelodysplastic syndrome, congenital syndromes, or viral infections (1).

Today, drug-induced thrombocytopenia (DIT) is an increasingly common cause of isolated thrombocytopenia. Antibody-mediated platelet destruction should be suspected in patients who experience an acute decrease in platelet levels, usually within one or two weeks of starting a new drug (2). More than 300 drugs have been implicated in DIT. In the systematic review of individual patient data, the most commonly reported drugs with a definite or probable causal relationship to thrombocytopenia are quinine, quinidine, trimethoprim/sulfamethoxazole, vancomycin, penicillin, rifampin, carbamazepine, ceftriaxone, ibuprofen, myralaminplatin, and oxalimintazapine. GPIIb/IIIa inhibitors such as abciximab, tirofiban, and eptifibatide, have also been demonstrated. However, the most common drug involved in

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DIT is heparin (3). Abciximab is a potent antiplatelet agent that blocks platelet aggregation. It effectively prevents and treats acute ischemic complications of percutaneous coronary angiography and improves outcomes of high-risk procedures by reducing the incidence of major adverse cardiac events (4). The incidence of profound thrombocytopenia caused by abciximab is 8.5% and occurs rapidly (within 2-4 hours) after drug infusion; platelet values return to normal within 2-5 days after discontinuing abciximab but may take up to 10 days (5). Abciximab-induced acute severe thrombocytopenia (platelet count <20,000/μL) represents a rare, unpredictable, adverse severe complication that can cause fatal haemorrhagic events, including intracranial haemorrhage (6). Most cases are usually mild, but deaths have also been reported in some cases of severe thrombocytopenia (7). The reported incidence of thrombocytopenia is greater than 1% at the first exposure and more than 10% at the second exposure. Thrombocytopenia onset can take up to 8-10 days after the first dose of the drug, as in our patient (7, 8). The diagnosis of abciximab-induced thrombocytopenia should generally be considered after heparin-induced thrombocytopenia (HIT), pseudothrombocytopaenia, and disseminated intravascular coagulation (DIC) have been excluded (9). Since the immune response is drug-dependent, thrombocytopenia usually resolves after discontinuing the drug, removing the drug from the circulation and forming new platelets by the bone marrow (10). Treatment includes immediate discontinuation of the drug if the platelet count falls below 50 000/μL. Platelet transfusion should be considered if there is bleeding or if the platelet count falls below 20 000/μL. Although data on the efficacy of these supportive measures in acute situations is still lacking in severe thrombocytopenia cases, it may be reasonable to consider intravenous immunoglobulin (IVIg) and high-dose corticosteroids, as in our case (11).

Drug-induced thrombocytopenia is a severe condition that should be considered in the differential diagnosis of a patient with thrombocytopenia receiving drug therapy. It can be difficult to diagnose drug-induced thrombocytopenia, especially in its immune-mediated forms definitively. It was primarily diagnosed by excluding other causes of thrombocytopenia. The timing of thrombocytopenia is correlated with the administration of the high-risk drug (12). There are many case reports of thrombocytopenia in the literature after abciximab treatment (13). In this case, we aimed to present the development of severe acute thrombocytopenia caused by abciximab, its successful management, and treatment with discontinuation of abciximab. Therefore, patients who are given GP IIb/IIIa receptor blockers should be hospitalized and followed hematologically as these blockers may cause life-threatening adverse outcomes.

Informed consent

We obtained informed consent from all individual participants included in the study.

Conflict of interest

None to declare.

Acknowledgments

None to declare.

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