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Case Report

Acute Profound Thrombocytopenia Following Tirofiban Administration after Primary PCI for Total Occlusion of the Right Coronary Artery - A Case Report

Dr. Sanjay R. Tarlekar

Consulting Interventional Cardiologist & Physician, Navi Mumbai, Maharashtra, India.

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Corresponding Author:

Dr. Sanjay R. Tarlekar
Consulting Interventional Cardiologist & Physician, Navi Mumbai, Maharashtra, India.

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ABSTRACT

Acute severe thrombocytopenia is a rare but important complication associated with glycoprotein IIb/IIIa inhibitors such as tirofiban. Rapid recognition and prompt withdrawal of the offending agent are essential to avoid potentially fatal bleeding complications. We present a case of acute profound thrombocytopenia developing within 8 hours of tirofiban administration in a patient undergoing primary PCI for myocardial infarction (STEMI) with total right coronary artery occlusion. Early recognition, immediate drug discontinuation, and supportive care led to prompt recovery. This case emphasizes the critical need for routine platelet monitoring following GPI administration.

Keywords: Tirofiban, Thrombocytopenia, Primary PCI, Glycoprotein IIb/IIIa Inhibitor, Right Coronary Artery.

INTRODUCTION

Strong platelet antiaggregants called GP (Glycoprotein) IIb/IIIa receptor antagonists are being utilised more frequently in PCI (Percutaneous Coronary Intervention) and ACS (Acute Coronary Syndrome). Tirofiban is a tiny non-peptide ligand-mimetic GPI that inhibits platelet aggregation by reversibly attaching to the arginine-glycine-aspartic acid (RGD) recognition site of GP IIb/IIIa.[1]

It is mainly used for patients having PCI and for the treatment of ACS. Tirofiban has been shown to dramatically lower the risk of MI (Myocardial Infarction) and composite cardiac outcomes (death, MI, and revascularisation) when used in conjunction with other GPRAs such as abciximab and eptifibatide.[2] Tirofiban has rarely been linked to thrombocytopenia, while showing a mortality benefit in patients with ACS and during PCI. Research has shown that between 0.4% and 5.6% of people experience tirofiban-induced thrombocytopenia, which is defined as a platelet count of less than 100,000/µL within 72 hours of tirofiban exposure.[3] Clinical trials also show that 0.1% to 0.5% of patients experience severe tirofiban-induced thrombocytopenia, which is defined as a platelet count below 50,000/µL.[4]

Tirofiban is linked to thrombocytopenia, with an incidence ranging from 0.4% to 5.6%, although being generally regarded as safe. Acute profound thrombocytopenia (<20,000/µL) is very uncommon, although severe thrombocytopenia (<50,000/µL) occurs in 0.1-0.5% of cases. The danger of bleeding and death are greatly increased by this immune-mediated illness.[5]

We describe a patient who had acute myocardial infarction (STEMI), underwent PCI, and experienced significant thrombocytopenia within 24 hours of receiving tirofiban.

CASE PRESENTATION

A 44-year-old gentleman presented with an acute onset of severe chest pain. His prior history was noteworthy since he had previously had percutaneous coronary intervention to the left anterior descending artery and coronary artery bypass graft surgery. ECG showed. He was diagnosed with acute myocardial infarction. Echocardiography revealed severe LV dysfunction with EF of 25-30% with global hypokinesia, more pronounced in the inferoseptum, anteroseptum and apex, along with RV dysfunction. The right coronary artery showed 100% stenosis with thrombotic blockage on coronary angiography. The patient underwent successful primary percutaneous coronary intervention to the RCA with 3 X 39 mm DES stent. Periprocedurally, he received dual antiplatelet therapy, tirofiban infusion, and unfractionated heparin. Approximately 8 hours after the procedure, the platelet count acutely dropped to below 20,000/µL (Baseline platelet count was 1.52 lakhs per cu.mm). Haemolyticanaemia and microangiopathic processes, such as TTP (Thrombotic Thrombocytopenic Purpura), were effectively ruled out by a peripheral smear that revealed decreased platelets and was negative for schistocytes. A platelet factor 4 (PF4)/heparin enzyme-linked immunosorbent assay was conducted because the patient had received a short course of heparin; the findings were negative, so ruling out HIT.

Tirofiban and heparin were immediately discontinued. The patient received one unit of single donor platelet transfusion. Serial monitoring demonstrated progressive recovery of platelet counts, which increased to greater than 100,000/µL before discharge, days after the procedure. No major bleeding complications occurred during hospitalization.

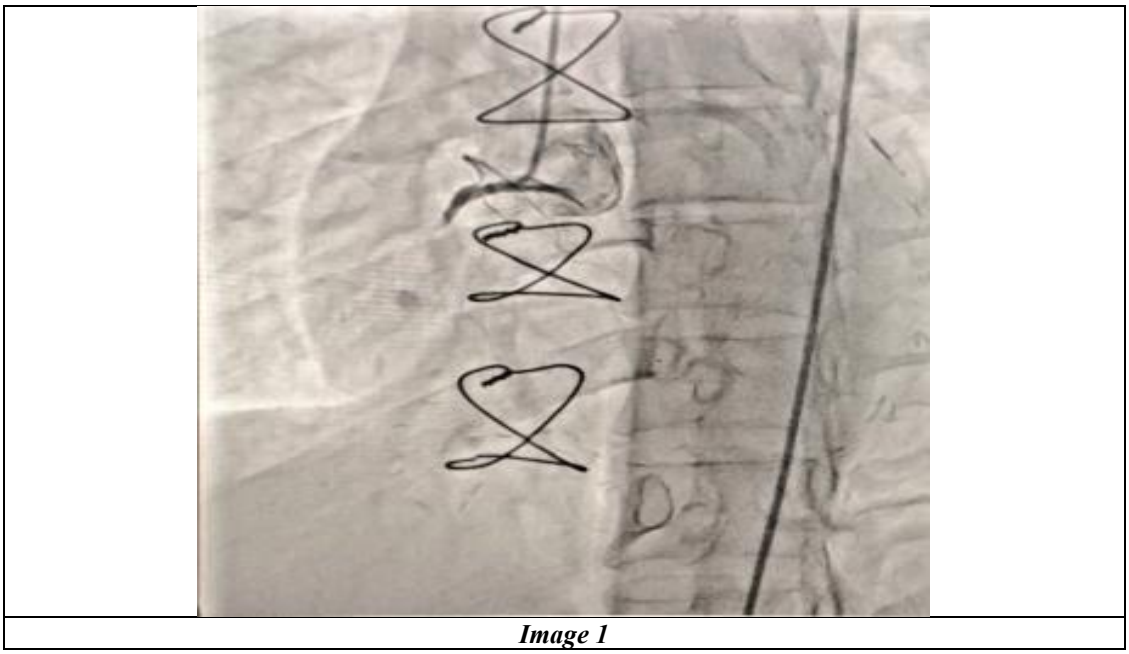
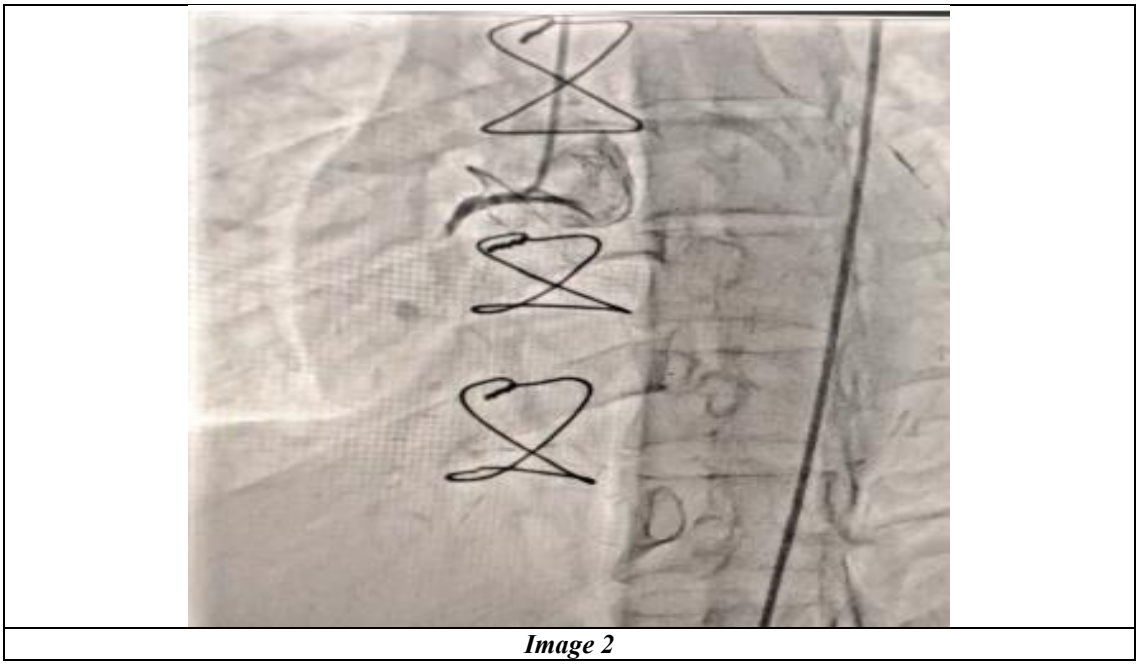


Image 1



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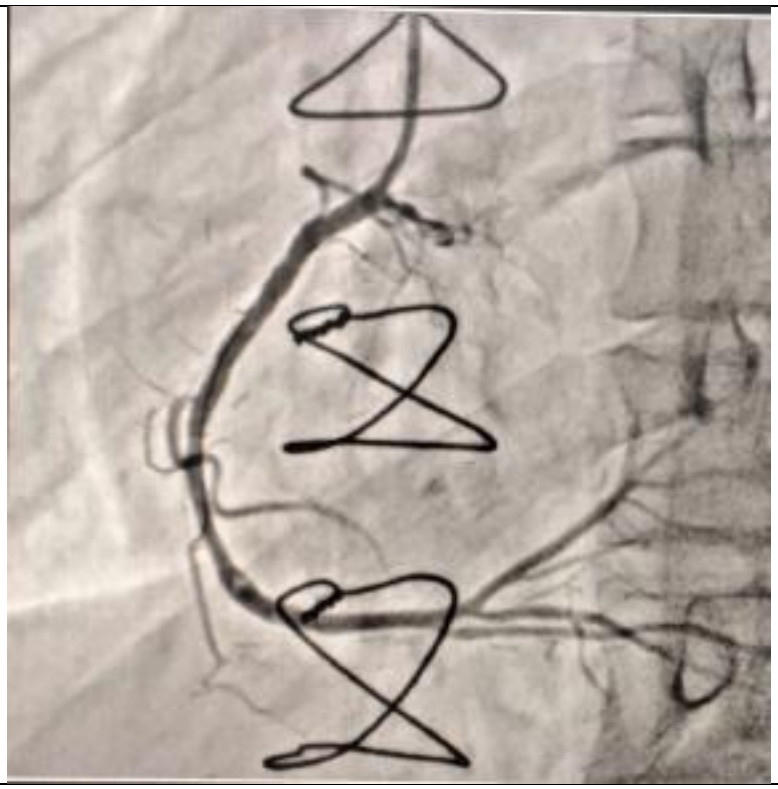

Image 3

DISCUSSION

Tirofiban binds to Gp IIb/IIIa to function as a reversible antagonist of fibrinogen. It prevents platelet aggregation and is only given intravenously. The PRISM trial, which showed a decrease in death, myocardial infarction, or refractory ischaemia in patients when paired with aspirin and heparin and aspirin, supports its use.[6] Tirofiban-induced severe thrombocytopenia is an uncommon disease that affects between 0.1% and 0.5% of patients.[7] Age over 65, diabetes mellitus, congestive heart failure, and chronic kidney disease are some patient-specific risk factors that may put people at risk for this negative consequence.[3] The risk of thrombocytopenia may also be increased by concomitant use of other anticoagulants, such as heparin.

DDAbs (Drug-Dependent Antibodies) are hypothesised to represent the immunological mechanism behind tirofiban-induced thrombocytopenia. Tirofiban induces structural changes in GP IIb/IIIa, exposing novel epitopes (LIBS). IgG antibodies bind to the GP IIb/IIIa complex only in presence of tirofiban. Antibody-coated platelets recognized by Fcγ receptors on macrophages in spleen/liver leading to rapid immune-mediated platelet destruction. Previous exposure to tirofiban may increase the likelihood of sensitization and accelerated thrombocytopenic response.[8]

Typical features of this clinical condition include the following:

- Onset: Within 24 hours (frequently 6-12 hours).
- Platelet nadir: Often <20,000/μL, can reach <1,000/μL.
- Bleeding symptoms: Petechia, hematuria, gingival bleeding, alveolar hemorrhage.

Differentiating tirofiban-induced thrombocytopenia from other comparable presentations, like HIT (Heparin-Induced Thrombocytopenia), is crucial. The timing and start of these two situations are key differentiators. The majority of HIT instances happen five to ten days after heparin exposure, but tirofiban-induced thrombocytopenia usually develops more quickly within hours to a few days. Furthermore, thrombosis rather than bleeding problems is the primary manifestation of HIT.[9]

Management primarily includes the following steps:

- Immediate discontinuation of the offending agent.
- Serial platelet monitoring.
- Avoidance of future exposure to tirofiban.
- Platelet transfusion in selected patients with severe thrombocytopenia or bleeding manifestations.

Early recognition remains essential to prevent catastrophic bleeding events. Corticosteroids (IV methylprednisolone) and IVIG may be considered for profound thrombocytopenia.[5]

After stopping tirofiban, platelet counts usually improve over time as long as there are no side effects, such as bleeding. The question of whether there are any substitute tirofiban agents emerges. The evidence for the use of bivalirudin, a direct thrombin inhibitor, in ACS derives mostly from its usage as a heparin substitute in patients with HIT.[10]
Some of the essential learning points from the case are as follows:

- Tirofiban can cause rapid and severe immune-mediated thrombocytopenia.
- Platelet count monitoring should be performed within the first 6–24 hours after administration.
- Immediate discontinuation of tirofiban usually results in platelet recovery.
- Differentiation from heparin-induced thrombocytopenia is essential.
- Prior exposure to glycoprotein IIb/IIIa inhibitors may predispose to rapid-onset thrombocytopenia.

CONCLUSION

This case highlights the possibility of acute profound thrombocytopenia following tirofiban exposure, particularly in previously exposed patients. Early platelet monitoring and immediate discontinuation of the suspected agent are crucial for favorable outcomes.

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