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Tirofiban-Associated Thrombocytopenia: A Case Report

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Abstract

Tirofiban, a competitive inhibitor of glycoprotein IIb/IIIa receptors on platelet membranes that assist in aggregation, is widely used in high-risk acute coronary syndrome (ACS). We present a case of a 53-year-old man with acute coronary syndrome and triple vessel disease who developed thrombocytopenia after first exposure to tirofiban. He presented to our hospital after his left-sided chest pain had subsided, and he was started on tirofiban 0.15 µg/kg/min; baseline platelets were 264 × 10^3/µL. Within hours, repeat testing showed a profound fall to 10 × 10^3/µL, prompting immediate discontinuation of tirofiban, holding aspirin and ticagrelor, hematology consultation, and transfusion of two apheresis platelet units. Platelet counts started to gradually rise back during the following days, and the patient remained free of bleeding complications. On day 3 of admission, dual antiplatelets were restarted as he was considered high risk, and percutaneous coronary intervention (PCI) was carried out successfully on day 4, addressing the right coronary artery (RCA) and left anterior descending artery (LAD). Staged PCI of the remaining vessels was performed two months after discharge without complications. Our patient's presentation is highly consistent with tirofiban-associated thrombocytopenia, as other alternative causes were deemed unlikely. Early detection, prompt discontinuation, and appropriate supportive care and monitoring are vital to ensure safe recovery and revascularization. Incorporating simple pre-procedure risk stratification, individualizing dosing, and early platelet monitoring may reduce complications while preserving ischemic benefit.

Categories: Hematology, Cardiology
Keywords: acute coronary syndrome, percutaneous coronary intervention, thrombocytopenia, tirofiban, tirofiban-associated thrombocytopenia

Introduction

Tirofiban, a competitive inhibitor of glycoprotein IIb/ IIIa receptors, which are present on platelet membranes and assist in platelet aggregation, has been widely used due to its notable clinical benefit in the treatment of acute coronary syndrome (ACS), specifically in high-risk patients who undergo emergency percutaneous coronary intervention (PCI) [1]. It is important to note that tirofiban is an antiplatelet agent, not a fibrinolytic drug, and its evidence-based role is selective in peri-procedural or bail-out use in patients with high thrombus burden or technically complex PCI anatomy, rather than routine therapy across all patients with ACS. However, with the expanding use of glycoprotein IIb/ IIIa receptor antagonists, an increasing number of cases have been reported to have tirofiban-induced thrombocytopenia (TIT), with an incidence ranging from 0.4% to 5.6% [2].

Drug-induced immune thrombocytopenia (DITP) is considered a life-threatening clinical syndrome that is challenging to diagnose due to various underlying mechanisms and the difficulty in detecting drug-dependent antibodies [3]. Although there is no definitive understanding of the underlying mechanism behind developing TIT, accumulating evidence suggests that it might be autoimmune due to the detection of antibodies that recognize glycoprotein IIb/ IIIa receptors in the presence of agents, such as tirofiban [4]. Drug-dependent antibody testing (such as flow cytometry-based assays detecting tirofiban-dependent anti-GPIIb/IIIa antibodies) represents the gold standard for confirming TIT [5]. However, such testing is not widely accessible in routine clinical practice.

Onset of TIT typically occurs within hours to days of drug exposure. Although severe cases carry a risk of life-threatening hemorrhage, the prognosis remains favorable with prompt discontinuation of the offending agent [2]. Despite its clinical significance, the precise mechanisms and optimal risk stratification strategies for TIT remain incompletely understood, underscoring the value of reporting well-characterized cases. In this case report, we present a patient who developed TIT within 12 hours of first drug exposure.

Case Presentation

A 53-year-old man, originally from the Philippines, with a known history of hypertension and prediabetes,

initially presented to the emergency department of another hospital two days earlier with left-sided chest pain, diaphoresis, and dizziness. He was noted to be hypotensive and bradycardic. After prompt admission to the intensive care unit (ICU), his laboratory results revealed raised troponin T and creatine kinase-MB (CK-MB). One day prior to presenting in our hospital, he underwent an angiogram that revealed triple vessel disease with 80%-90% stenosis in the left anterior descending artery (LAD), diffuse ostial stenosis of 90% in the diagonal branch of the left anterior descending artery, discrete eccentric stenosis of 80%-90% in the left circumflex artery (LCx), ostial stenosis of 80%-90% in the obtuse marginal artery 3, and 99% stenosis in the right coronary artery (RCA) with notable heavy luminal thrombus in the distal region. Subsequently, the patient was advised to undergo coronary artery bypass grafting (CABG), for which he attended our hospital for further review the following day (Table *1*).

Timeline	Event
2 days before	Presented to ED in another hospital, diagnosed with ACS
1 day before	Underwent angiogram revealing triple vessel disease, CABG recommended
Day 0 (11/07)	Presented to our ED, admitted to ICU and started on tirofiban, aspirin and ticagrelor continued; baseline platelets: 264 × 10^3/μL
Day 1 (12/07)	Patient was continued on tirofiban; laboratory results revealed platelets dropping to 10 × 10^3/μL; tirofiban stopped immediately, aspirin and ticagrelor put on hold, hematologist consulted, two units of platelets transfused; platelets dropped further to 3 × 10^3/μL
Day 2 (13/07)	Platelets: 73 × 10^3/μL
Day 3 (14/07)	Patient stable, platelets increased to 88 × 10^3/μL, aspirin and ticagrelor resumed
Day 4 (15/07)	Angioplasty done
Day 5 (16/07)	Discharge with follow-up plan in clinic in one week and recommended staged PCI after two months; discharge platelets: 149 × 10^3/μL

TABLE 1: Timeline of important events

ED: emergency department, ACS: acute coronary syndrome, CABG: coronary artery bypass grafting, ICU: intensive care unit, PCI: percutaneous coronary intervention

On presentation to our emergency department the following day, the pain had already subsided, physical examination did not reveal any significant findings, and electrocardiography showed ST elevations in the inferior leads (II, III, and AVF) (Figure *1*). He was subsequently admitted to our intensive care unit as a case of acute coronary syndrome (ACS), as the symptoms had already resolved and he was hemodynamically stable. The interventional cardiologist was informed of the case, and it was decided to start the patient on tirofiban 0.15 mcg/kg/min intravenously for 24 hours while continuing his current management with aspirin 100 mg oral once daily, ticagrelor 90 mg oral twice daily, spironolactone 25 mg oral once daily, bisoprolol fumarate 2.5 mg oral once daily, and atorvastatin 40 mg oral once daily. Baseline laboratory tests were ordered, including full blood count (FBC), creatinine, CK-MB, troponin T, lipid profile, and HbA1c. Results showed elevated CK-MB of 9.82 μg/L, and the remaining blood test results were within normal ranges. Platelets were noted to be 264 × 10^3/μL (Figure *2*).

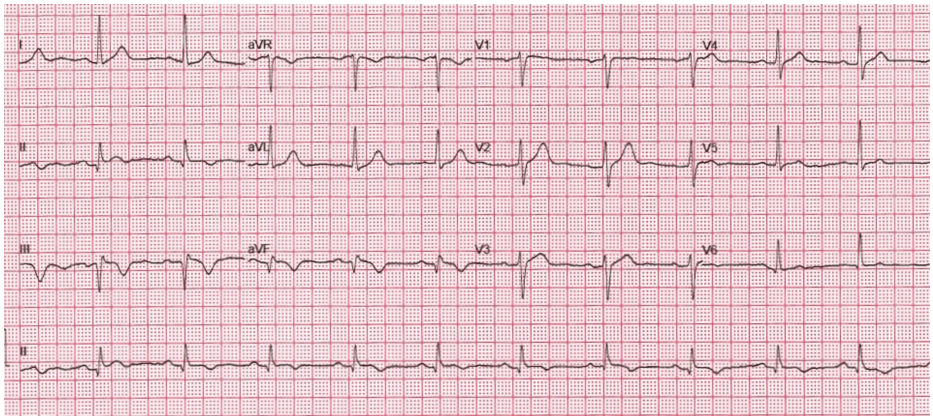


FIGURE 1: 12-lead ECG on admission

ST elevations in the inferior leads (II, III, and aVF) consistent with inferior myocardial infarction in a 53-year-old man presenting with resolving left-sided chest pain.

ECG: electrocardiogram

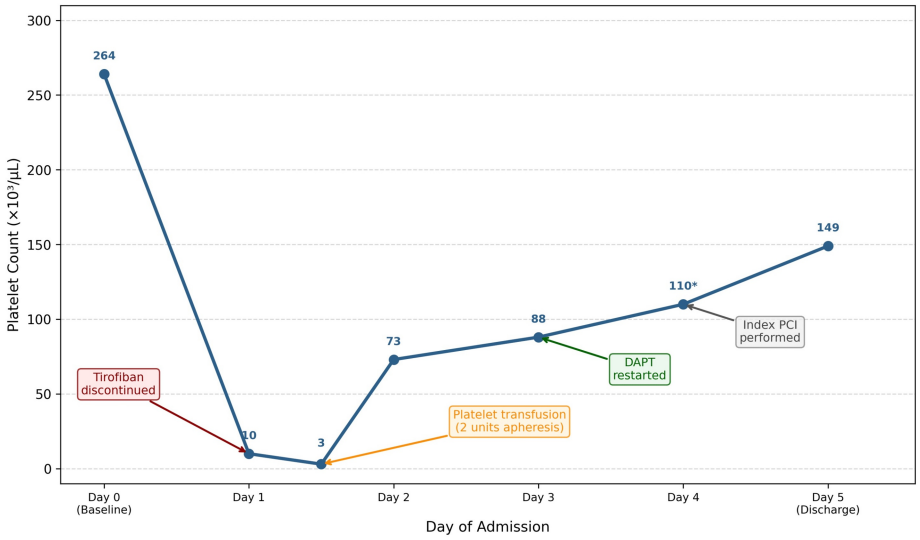


FIGURE 2: Platelet count trend during hospitalization

Serial platelet counts (× 10^3/μL) from admission to discharge demonstrating the profound nadir of 3 × 10^3/μL on day 1 following tirofiban initiation, with subsequent recovery following drug discontinuation and platelet transfusion. DAPT was resumed on day 3, and index PCI was performed on day 4.

DAPT: dual antiplatelet therapy, PCI: percutaneous coronary intervention

The following day (day 1), the responsible care provider was informed by the laboratory that, on repeat blood tests, platelet counts had decreased significantly to 10 × 10^3/μL. Tirofiban was immediately stopped, and it was decided to put aspirin and ticagrelor on hold due to the high risk of bleeding. The case was referred to the hematologist on call, who advised the transfusion of two units of apheresis platelets due to the profound thrombocytopenia and the associated high risk of bleeding. Full blood count was repeated after a few hours, which showed declining platelet counts reaching 3 × 10^3/μL. The post-transfusion nadir of 3 × 10^3/μL is attributed to continued immune-mediated platelet destruction by pre-existing drug-dependent antibodies that persist briefly after drug discontinuation. A peripheral blood smear performed at the time of the nadir confirmed true thrombocytopenia with no platelet clumping; repeat samples in both EDTA and citrate tubes yielded consistent results, definitively excluding pseudothrombocytopenia. Drug-dependent antibody testing was not feasible in our institution at the time of this case. There was no clinical or biochemical evidence of disseminated intravascular coagulation, sepsis, liver dysfunction, or bone marrow suppression. No prior exposure to tirofiban or any other GPIIb/IIIa inhibitor was documented. Clinical examination was not significant during that time, with no active bleeding, petechiae, or bruising noted.

The patient was monitored closely in the ICU for any possible complications, and laboratory results were repeated again the following day, which revealed increasing levels of platelets to 73 × 10^3/µL and eventually reaching 88 × 10^3/µL on day 3 of admission. After ensuring that the patient has stabilized, the discussion between the providing team was done, and a decision was made to restart the patient on dual antiplatelet therapy (aspirin and ticagrelor) as he has three vessel disease, hence categorizing him as high risk. The decision to resume antiplatelet therapy on day 3 was guided by three concurrent factors: platelet recovery to 88 × 10^3/µL (a level deemed sufficient to balance thrombotic and bleeding risk), the absence of active bleeding, and the urgent need to proceed with revascularization in a high-risk patient with triple vessel disease. Moreover, angioplasty was discussed within the surgical team and interventional cardiology, and they agreed that the left anterior descending artery is not suitable for grafting due to the significant diffuse stenosis; therefore, the decision to undergo angioplasty was taken instead. On day 4 of admission, the patient underwent angioplasty successfully, which showed stenosis in the RCA involving the proximal, mid, and distal regions reaching about 90%. The origin of the posterior interventricular artery (PDA) and posterior left ventricular arteries were noted to be involved. Good results were noted after stenting the RCA. Ballooning of the PDA and posterior left ventricular artery (PLV) was done as they were heavily calcified, which resulted in normal flow. There was severe stenosis from the proximal region to the end of the LAD; hence, ballooning was done, and a stent was placed in the mid-segment. The left circumflex artery was seen to have long proximal stenosis, and obtuse marginal artery 1 had ostial involvement. Pictures of the angiography are seen in Figure 3. There were no complications during the procedure.

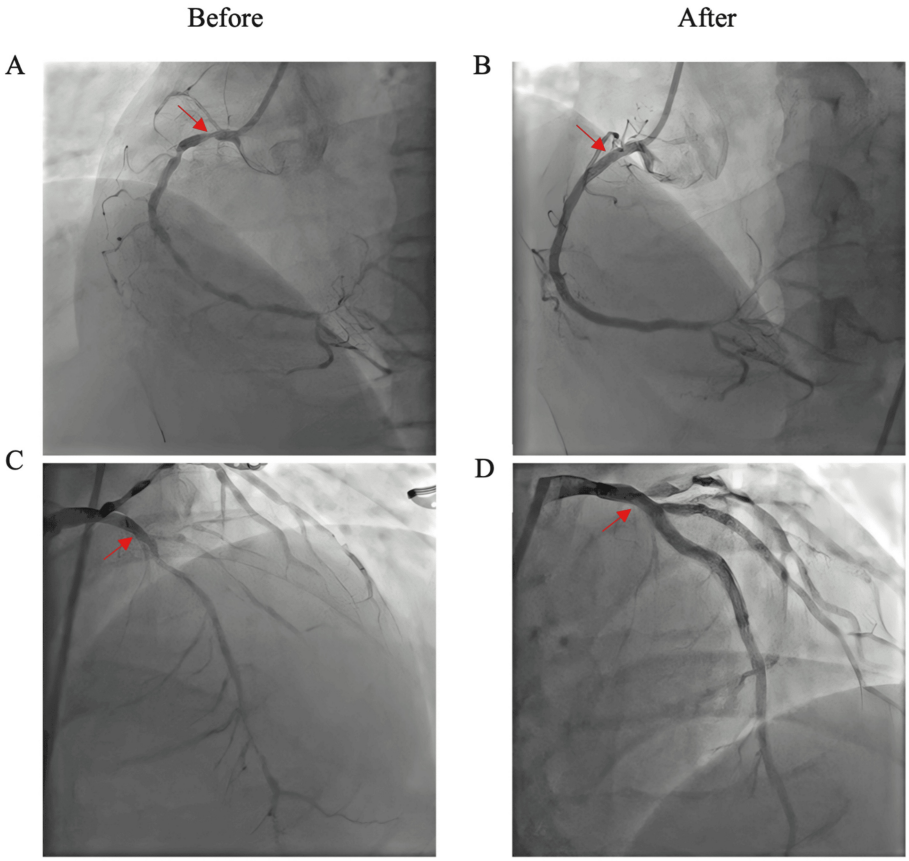


FIGURE 3: Coronary angiography before and after percutaneous intervention (day 4)

Panels A and B show the RCA before and after intervention, respectively. The RCA demonstrated significant stenosis involving the proximal, mid, and distal regions (approximately 90%), with involvement of the origin of the PDA and PLV artery. Following stenting of the RCA, ballooning of the PDA and PLV was performed due to heavy calcification, resulting in restoration of normal flow. Panels C and D show the LAD before and after intervention, respectively. Severe stenosis was present from the proximal to distal LAD; ballooning was performed throughout, with a stent placed in the mid-segment. The LCx demonstrated long proximal stenosis, and the OM1 had ostial involvement; these vessels were deferred for staged percutaneous coronary intervention.

RCA: right coronary artery, PDA: posterior interventricular artery, PLV: posterior left ventricular, LAD: left anterior descending artery, LCx: left circumflex artery, OM1: first obtuse marginal artery

The patient was discharged on day 5 with no complaints or notable bleeding. His last blood tests prior to discharge showed a platelet count of 149 × 10^3/µL. He was discharged on aspirin 100 mg oral once daily and clopidogrel 150 oral once daily. The switch from ticagrelor to clopidogrel at discharge was a deliberate decision by the treating team to de-escalate P2Y12 inhibition intensity in the context of the recent severe thrombocytopenic episode, balancing ischemic protection with hemorrhagic risk during the recovery and staged PCI planning period. A follow-up appointment was set for one week after discharge, and a recommendation was made for staged PCI of the remaining vessels (left circumflex artery and obtuse marginal artery 1) once the patient had fully recovered. The follow-up PCI was performed successfully after two months without complications.

Discussion

Patients presenting with acute coronary syndrome are commonly managed with multiple antithrombotic and antiplatelet drugs, increasing their risk of developing post-PCI thrombocytopenia, which is associated with higher rates of major adverse cardiac events [6]. This can, therefore, make it hard to determine which drug is the underlying precipitating agent for developing thrombocytopenia. A few cases in the literature have described aspirin-induced thrombocytopenia, possibly associating it with an allergic reaction to the drug [7]. Similarly, ticagrelor is rarely associated with drug-induced thrombocytopenia [8,9]. As both agents have been used in the management of our patient for a few days before starting tirofiban, it is unlikely that they are the culprits that have induced thrombocytopenia in this case. Moreover, since our patient was not started on heparin, there was no suspicion of heparin-induced thrombocytopenia. Pseudothrombocytopenia, a major differential observed in about one-third of patients undergoing PCI and managed with GPIIb/IIIa antagonists, was excluded by performing a peripheral blood smear and repeating the blood counts [10]. Based on the clinical course, the diagnosis is best classified as highly probable tirofiban-associated thrombocytopenia. Application of the Naranjo Adverse Drug Reaction Probability Scale yielded a score of 7, classifying the reaction as a “probable” causal relationship, given the temporal association between tirofiban initiation and the platelet nadir, recovery upon discontinuation, and systematic exclusion of alternative aetiologies [11]. Drug-dependent antibody testing, which would provide definitive confirmation, was not performed due to its limited availability at our institution [5].

Although the underlying mechanism of TIT has not been definitively identified in the literature, Bougie et al. have shown that the conformation of neoepitope or a ligand-induced binding site (LIBS) expressed by GPIIb/IIIa is altered following drug binding, causing antibodies to recognize them and hence platelet destruction [4]. The further decline in platelet count to 3 × 10^3/µL despite drug discontinuation and platelet transfusion is consistent with the persistence of pre-existing drug-dependent antibodies, which continue to opsonize both endogenous and transfused platelets until the drug is fully cleared from circulation, typically within one hour of tirofiban discontinuation [12]. In rare cases, thrombocytopenia occurring within hours of the first exposure is believed to be due to pre-existing, naturally occurring antibodies [4]. Furthermore, a case report showed that repeated administration of tirofiban may cause a faster onset of thrombocytopenia and slower recovery due to the possibility of antibodies being retained following the primary exposure to the drug [13].

Studies currently suggest categorizing the different presentations of TIT such as the following: (1) acute severe thrombocytopenia occurring within 12 hours of first exposure, (2) acute severe thrombocytopenia occurring within 12 hours of second exposure, (3) delayed thrombocytopenia occurring within 5-7 days from exposure, and (4) anaphylaxis after first or second exposure [14]. The degree of thrombocytopenia was defined in most studies as mild if the platelet count was <100,000/µL, severe if the platelet count was <50,000/µL, and profound if the platelet count was <20,000/µL [15]. This classification can guide the management of TIT, which is similar to that of DITP, while closely correlating it with the clinical findings. Some patients have been reported to present with symptoms such as fever, dyspnea, hypotension, and anaphylactic reaction following exposure to GPIIb/IIIa inhibitors, while life-threatening bleeding events are rare [16]. Close monitoring is recommended in patients whose platelet counts fall between 50,000/µL and 100,000/µL, and active treatment with GPIIb/IIIa inhibitors should only be stopped if the platelet counts continue to fall or bleeding complications occur. If the platelet counts fall below <50,000/µL, then the administration of GPIIb/IIIa inhibitors should be stopped immediately; this is due to the clearance of tirofiban from the circulation one hour after discontinuation [12,15]. If platelet counts fall under 10 × 10^9/L and the patients have severe bleeding, all anticoagulants and antiplatelet drugs should be stopped, and platelet transfusion is recommended [17,18]. In cases of refractory bleeding, plasmapheresis and hemodialysis can be utilized to accelerate drug elimination from circulation. Additional treatment options include corticosteroid therapy and immunoglobulin infusions [15].

Higher doses of tirofiban have been shown to reduce major adverse cardiac events. However, they are also associated with increased hemorrhagic risk through the drug’s expected antiplatelet mechanism. This bleeding risk may be further compounded by tirofiban-induced thrombocytopenia, which, as this case illustrates, can cause a profound, unpredictable drop in platelet count entirely independent of dose. Therefore, while dosing decisions should account for a patient’s individual bleeding risk and renal function, clinicians should recognize that thrombocytopenia represents an additional and largely unpredictable hemorrhagic hazard, rather than a dose-dependent side effect that can be mitigated by dose reduction alone

[15,19].

From a cardiological perspective, it is important to acknowledge that the initiation of a 24-hour tirofiban infusion in the present case, in a patient whose chest pain had resolved, who had Q-waves on ECG, and who was already on dual oral antiplatelet therapy, is difficult to reconcile with contemporary selective-use guidelines. Current evidence and guideline recommendations favor targeted, peri-procedural or bail-out GPIIb/IIIa inhibitor use in patients with ongoing ischemia or high angiographic thrombus burden at the time of PCI, as any ischemic benefit must be weighed against increased hemorrhagic risk. The primary justification in this case was the angiographically documented heavy luminal thrombus in the distal RCA. Even when tirofiban is initiated with perceived angiographic justification, the risk of profound thrombocytopenia warrants robust pre-treatment risk stratification and early platelet monitoring, particularly given the compounding bleeding risk from concomitant dual oral antiplatelet therapy.

Yi et al. suggested a pre-procedure risk model to predict tirofiban-induced thrombocytopenia in patients undergoing PCI [20]. Five independent factors were identified in the score model: age ⩾65 years (2 points), white blood cells ⩾12 × 10^9/L (1 point), history of diabetes mellitus (2 points), history of congestive heart failure (2 points), and history of chronic kidney disease (2 points). Accordingly, 6-8 points, 4-5 points, and ⩽3 points indicate high probability, intermediate probability, and low probability, respectively. This scoring system can assist physicians in preventing TIT and appropriately monitoring patients according to their risk score, possibly decreasing unnecessary regular monitoring in low-risk patients. Applying this score to our patient yielded a total score of 0 points, placing him in the low-probability category for TIT. Paradoxically, despite this low predicted risk, the patient developed profound thrombocytopenia (nadir 3 × 10^3/μL) within hours of first exposure. This underscores that even patients classified as low risk may develop severe TIT, likely mediated by pre-existing naturally occurring drug-dependent antibodies, and supports the argument for early platelet monitoring in all patients receiving tirofiban regardless of predicted risk category.

Conclusions

We present a case of acute profound thrombocytopenia, highly consistent with tirofiban-associated thrombocytopenia, that developed within hours of first exposure in a patient with ACS and multi-vessel coronary disease. The patient was managed by promptly discontinuing tirofiban, holding aspirin and ticagrelor, and transfusing two units of apheresis platelets under close ICU monitoring, leading to rapid platelet recovery without bleeding complications. On day 4 of admission, the patient underwent successful index angioplasty of the right coronary artery and left anterior descending artery. He was discharged on dual antiplatelet therapy and subsequently underwent staged PCI of the remaining vessels two months later without complications.

This case reinforces the need for early recognition and prompt management of GPIIb/IIIa inhibitor-related thrombocytopenia, which can occur even on first exposure and in patients classified as low risk by pre-procedure scoring systems. Importantly, it also highlights that the clinical indication for GPIIb/IIIa inhibitor initiation should be carefully justified; use in a post-infarction patient with resolved symptoms and without ongoing ischemia represents a debatable departure from current guideline recommendations and constitutes an important learning point. Simple pre-procedure risk assessment, individualized dosing, and early platelet monitoring may reduce complications while preserving ischemic benefit. Further research is needed to clarify the mechanisms of tirofiban-associated thrombocytopenia, identify patients at highest risk, and refine evidence-based monitoring and management protocols.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Ayatullah Hegazy, Mohamed Hassan, Aya Majwal, Mahmoud Marashi

Acquisition, analysis, or interpretation of data: Ayatullah Hegazy, Talib Majwal

Drafting of the manuscript: Ayatullah Hegazy, Mohamed Hassan, Aya Majwal

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Supervision: Mohamed Hassan, Talib Majwal, Mahmoud Marashi

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all

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