Concurrent thrombocytopenia and thrombosis associated with Eptifibatide – A rare complication

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Introduction

Eptifibatide is a glycoprotein (GP) IIb/IIIa inhibitor most often used in patients with acute coronary syndrome (ACS) undergoing percutaneous coronary intervention (PCI). Acute thrombocytopenia is a rare complication of eptifibatide. Eptifibatide-induced thrombosis is exceptionally rare, and only a small number of cases have been reported in the literature. Here we present the case of a patient who developed acute severe thrombocytopenia within 24 hours of administration of eptifibatide during PCI with subsequent development of acute in-stent thrombosis.

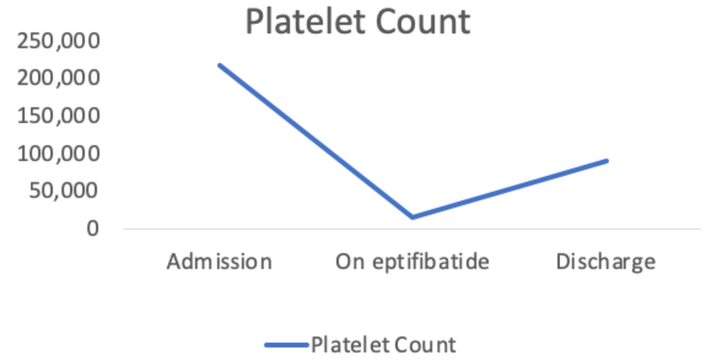
Case Presentation

A sixty-three-year-old gentleman with a past medical history significant for hypertension, hyperlipidemia, hypothyroidism, morbid obesity and type 2 diabetes presented to an outside hospital with chest pain and was found to have an inferior wall ST-elevation myocardial infarction. He underwent percutaneous coronary intervention during which he was given a double bolus of eptifibatide and maintained on an infusion for 18 hours. Notably, his platelet count on admission prior to catheterization was 217 x 103/μL and the following day, while still on the eptifibatide infusion, it had dropped to 15 x 103/μL.

The day after his catheterization, the patient started to complain of recurrent chest pain and was transferred to Geisinger Medical Center for hematologic evaluation in the setting of worsening ST elevations. Hematology evaluated the patient and thought that because he had had no prior exposure to heparin in the last 24 hours, his thrombocytopenia was less likely due to heparin-induced thrombocytopenia (HIT) and was most likely an adverse effect of eptifibatide. The patient underwent emergent repeat catheterization and was found to have a total occlusion of his RCA. Multiple attempts at percutaneous revascularization failed due to organized thrombus burden. A HIT panel was sent, which was negative, indicating that this complication was likely a result of eptifibatide. After discontinuation of eptifibatide his platelet count rapidly improved to 90 x 103/μL.

Discussion

Early clinical trials of the use of eptifibatide during PCI did not show any significant association between eptifibatide and thrombocytopenia. However, recently it is becoming more widely recognized as a rare but significant complication of eptifibatide use. While eptifibatide-induced thrombocytopenia is rare, eptifibatide-induced thrombosis is exceptionally rare. Because of its rarity, eptifibatide-induced thrombosis has the potential to be misinterpreted as heparin-induced thrombocytopenia (HIT). However, unlike HIT, prior exposure to eptifibatide is not a requirement for developing thrombocytopenia. Because our patient did not have recent exposure to heparin prior to catheterization, Type I HIT, which occurs within 24 hours of repeat exposure to heparin, was ruled out. Type II HIT, which develops within 5-10 days after heparin administration, was less likely as our patient's hematologic complications occurred within 24 hours of heparin administration.



Conclusion

Thrombocytopenia with subsequent thrombosis is an unusual complication of eptifibatide and as such might be misdiagnosed as HIT. It is imperative that clinicians recognize this phenomenon and understand the importance of early and frequent platelet monitoring in patients receiving eptifibatide during PCI.

References

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