



Apixaban Induced Thrombocytopenia: A Rare Event

Dhruv Kotecha*

Department of Clinical Case Report, India

*Corresponding Author: Dhruv Kotecha, Department of Clinical Case Report, India.

Received: January 03, 2025

Published: March 18, 2025

© All rights are reserved by **Dhruv Kotecha**.

Abstract

Thrombocytopenia is a common clinical condition caused by drugs specifically heparin induced. However to overcome this novel oral anticoagulants have been introduced to overcome this side effect. However still in 1% of cases novel oral anticoagulants too can cause thrombocytopenia. Here we present one such case where thrombocytopenia was caused by APIXABAN and reverted back by Fondaparinaux.

Keywords: Thrombocytopenia; Platelets; Blood

Introduction

Thrombocytopenia is a condition characterized by a decrease in the number of platelets in the blood. A platelet count of less than $150 \times 10^9/L$ is considered thrombocytopenia. Causes of Thrombocytopenia includes variety of factors, including like Bone marrow failure or suppression, Infections, Malignancies, and Medications etc. Mild thrombocytopenia often does not cause symptoms, but more severe cases can lead to Easy bruising, Easy bleeding, Petechiae, Purpura and Haemorrhages. Heparin Induced Thrombocytopenia is well known complication heard of in patients who are on Heparin or heparin analogues [1-3]. So Novel oral anticoagulants have been introduced in cases where there is Heparin induced thrombocytopenia [4]. However very rare about 1% is only reported cases of thrombocytopenia caused by novel oral anticoagulants [5].

Case Report

47 Years old, Male, K/C/O CA Lung (Mesothelioma) Post Op Pneumonectomy with metastasis to anterior pericardium and inter-costal muscles. Patient had right pleural effusion for which ICD insertion was done. Post ICD removal after 7 days patient developed right upper limb edema with occasional shortness of breath. Post complains 7 days later patient underwent Doppler of right upper limb which showed Right subclavian and axillary, proximal brachial veins non compressible and did not revealed evident colour flow on Doppler study suggestive of deep vein thrombosis. Further CT thorax with contrast study done which showed thrombosis of brachial, axillary and subclavian vein up to level of confluence with left subclavian vein. Left renal vein thrombosis. Pulmonary emboli

involving right pulmonary artery just above postoperative clips and metastatic involvement of pericardium, intercostal muscles on right side and superior surface of liver. On admission laboratory findings suggestive of neutropenic sepsis with Hb of 7.3 mg/dl, Total leucocyte 920 and platelets of 1, 23,000. Patient's CRP level was 96. Patient was started on broad spectrum antibiotic, low molecular weight heparin and GCSF injections. Patient was then shifted to NOACS on 2nd day and laboratory investigation suggested Hb of 9 mg/dl, Total leucocyte 5360 and platelets of 76000. Platelets were decreasing and TLC normalized so PCT done suggestive of normal. Apixaban had been started and on 4th day lab findings suggestive of 36000. Apixaban stopped for 24 hrs. And repeat lab findings suggestive of improved platelets to 59000 on 5th day evening and on 6th day morning platelets were about 97000. Patient restarted on Injection Fondaparinux this time and on follow up after 3 days Patients lab investigation showed Hb of 9.7 gm, Total Leucocyte 8840 and platelets of 440000. Repeat USG Doppler of Right Upper limb showed recanalization of involved veins and started on tab rivoroxaban.

Discussion and Conclusion

Apixaban, A Newer oral anticoagulant, working by directly inhibiting factor Xa, formed by both intrinsic and extrinsic pathways, thus preventing thrombin formation from prothrombin, fibrin formation from fibrinogen and ultimately the clot. Its use has been widely accepted for prevention of stroke and other thrombo-embolic events. Since it has shown superiority over warfarin in prevention of systemic embolism and reduced risk of bleeding, and use being more frequent, side effects such as Thrombocytopenia

should be considered while starting on Apixaban. Very few cases has been reported but uncommon side effects need to be monitored and reported. In our case, Patient started developing Thrombocytopenia after Apixaban has been started. All possible causes of Thrombocytopenia (other drug induced, Sepsis, TTP, DIC) had been ruled out. Platelet count started to increase after stopping the drug and hence it has to be attributed as Apixaban induced Thrombocytopenia.

Possible mechanisms of Apixaban-induced thrombocytopenia include immune-mediated reaction via drug-dependent antibodies, or bone marrow suppression. The rapidity of improvement in platelet number favours an immune-mediated hypothesis, since bone marrow recovery typically needs more than 7 days [6]. Since it is a relatively new and its prescription is rapidly increasing, even uncommon side effects need to be monitored closely and reported. This is especially important now with increased use of NOAC for treatment of HIT.

Bibliography

1. Philip Young-Ill Choi, *et al.* "Results of an international survey of opinions on the definitions and treatments for heparin-induced thrombocytopenia: communication from the ISTH SSC Subcommittee on Platelet Immunology". *Journal of Thrombosis and Haemostasis* 22.6 (2024): 1772-1778.
2. Daniel C Dees. "Heparin Induced Thrombocytopenia Testing". *Clinics in Laboratory Medicine* 44.3 (2024): 541-550.
3. Hogan M and Berger JS. "Heparin-induced thrombocytopenia (HIT): Review of incidence, diagnosis, and management". *Vascular Medicine* 25.2 (2020): 160-173.
4. Skelly JW, *et al.* "Novel oral anticoagulants for heparin-induced thrombocytopenia". *Journal of Thrombosis and Haemostasis* 42.2 (2016): 172-178.
5. Snellgrove O. "Case report: apixaban-induced thrombocytopenia". *Clinical Case Report* 5.3 (2017): 268-269.
6. A Greinacher, *et al.* "Autoimmune heparin-induced thrombocytopenia". *Journal of Thrombosis and Haemostasis* 15.11 (2017): 2099-2114.