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Case Report

Dabigatran Triggers Polycythaemia Vera (PV) in a Patient with Masked PV

Fabio Abenavoli[1#], Shreya Sharma[2#], Viviana Mohilitchi[3], Andrea Piccin[3-6*]

[1]Plastic Surgeon, Avoca Clinic, Wicklow, Ireland

[2]Trinity College Dublin, Dublin, Ireland

[3]Department of Haematology, Mater Private Hospital, Dublin, Ireland

[4]Royal College of Surgeons in Ireland (RCSI), Dublin, Ireland

[5]Department of Engineering, University of Trento, Italy

[6]University of Medicine Innsbruck, Innsbruck, Austria

[#]These authors contributed equally and should be both be considered co-first author

***Corresponding Author:** Andrea Piccin, Department of Haematology, Mater Private Hospital, Dublin, Ireland

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Abstract

Polycythaemia Vera (PV) is a chronic myeloproliferative neoplasm characterised by erythrocytosis, thrombocytosis, and JAK2-driven clonal proliferation. Patients with masked PV (mPV) display borderline haemoglobin or haematocrit values with elevated platelet counts. We describe a patient with mPV, who developed overt PV following six months of dabigatran therapy. The chronology of events, exclusion of alternative causes, and biological plausibility suggest a potential association that warrants further investigation.

Keywords: Polycythaemia Vera; PV; Myeloproliferative Neoplasm; Masked PV; Mpv; Dabigatran.

Introduction

Myeloproliferative neoplasms (MPNs) comprise polycythaemia vera (PV), essential thrombocythaemia (ET), and primary myelofibrosis (PMF). These conditions are driven by recurrent somatic mutations in JAK2, CALR, or MPL. Polycythaemia vera (PV) is classically characterized by absolute erythrocytosis, frequently accompanied by thrombocytosis and an increased risk of arterial and venous thrombosis. However, a subset of patients presents with masked polycythaemia vera (mPV), in which haemoglobin and haematocrit values remain at or near diagnostic thresholds-often due to iron deficiency or plasma volume expansion—despite the presence of the JAK2 V617F mutation [REF]. These patients may subsequently progress to overt PV and appear to share a comparable vascular risk profile.

Dabigatran, a direct thrombin inhibitor, is widely used for the treatment and secondary prevention of venous thromboembolism. Its well-recognised adverse effects include bleeding and gastrointestinal intolerance [REF]; however, an association with erythrocytosis has not previously been reported. We describe the first case in which overt PV emerged during prolonged exposure to dabigatran (Pradaxa®), raising the hypothesis of a potential drug-disease interaction that may have contributed to the unmasking or acceleration of the underlying myeloproliferative process.

Case Presentation

A 69-year-old man with no previously documented history of MPN presented with acute limb-threatening ischaemia involving

the left lower extremity. Imaging studies demonstrated acute occlusion of the proximal hypogastric artery, deep femoral artery, and anterior tibial artery. The patient underwent urgent surgical intervention consisting of Fogarty catheter thrombectomy, followed by prosthetic bypass grafting due to persistent critical ischaemia. Although partial reperfusion was achieved, distal tissue necrosis progressed, ultimately necessitating surgical amputation of the third metatarsal. Following clinical stabilization, the patient was discharged on therapeutic low-molecular-weight heparin (LMWH) and subsequently transitioned to antiplatelet therapy with aspirin. Despite this, four months later he was readmitted with extensive deep venous thrombosis of the left lower limb, confirmed by duplex ultrasonography. Laboratory evaluation at the time of admission revealed mild leukocytosis (white blood cell count 11.49 × 10^9/L), erythrocytosis at the upper limit of normal (red blood cell count 5.92 × 10^6/μL; haemoglobin 16.2 g/dL; haematocrit 47.6%), and marked thrombocytosis (platelet count 475 × 10^9/L). The patient was initially treated with parenteral anticoagulation and subsequently commenced on vitamin K antagonist therapy (warfarin). However, due to persistence of thrombotic burden on follow-up imaging, despite therapeutic international normalised ratio (INR) values, anticoagulation was switched to dabigatran 150 mg PO BD. After six months of continuous dabigatran therapy, routine laboratory monitoring revealed overt erythrocytosis (red blood cell count 7.31 × 10^6/μL; haemoglobin 18.5 g/dL; haematocrit 58.4%), accompanied by progressive leukocytosis, persistent thrombocytosis, elevated lactate dehydrogenase levels, and the development of aquagenic pruritus. Given the evolving haematological abnormalities and clinical features suggestive of an underlying myeloproliferative disorder, molecular testing was performed and demonstrated the presence of the JAK2 V617F mutation, confirming the diagnosis of overt polycythaemia vera. Dabigatran was immediately discontinued and therapeutic phlebotomy was initiated. Two weeks later, the patient developed acute mesenteric ischaemia requiring emergent surgical intervention. Despite operative management, his clinical condition rapidly deteriorated. He deceased of multi-organ failure three days postoperatively.

Discussion

This case highlights a strong temporal association between the initiation of dabigatran (Pradaxa®) therapy and progression from masked polycythaemia vera (mPV) to overt polycythaemia vera (PV). At presentation, laboratory findings demonstrated a borderline haematocrit in the presence of thrombocytosis and recurrent thrombotic events, a constellation of features consistent with mPV, as previously described [1,2]. Six months after the introduction of dabigatran, the patient fulfilled the diagnostic criteria for overt PV, including marked erythrocytosis, JAK2 V617F positivity, elevated lactate dehydrogenase levels, and the development of aquagenic pruritus. Several pharmacological agents have been reported to induce secondary erythrocytosis, including androgens, sodium–glucose cotransporter-2 (SGLT2) inhibitors, erythropoiesis-stimulating agents, and anti-angiogenic therapies [3]. In contrast, anticoagulants-including direct oral anticoagulants-have not previously been associated with erythrocytosis or acceleration of myeloproliferative neoplasms. Experimental data suggest that thrombin plays a role in megakaryopoiesis and may interact with erythroid differentiation pathways, providing a potential biological framework for a drug–disease interaction. The temporal relationship between dabigatran exposure and haematological progression, the stability of concomitant medications, and the exclusion of alternative causes of erythrocytosis support the hypothesis that dabigatran may have contributed to the unmasking or acceleration of an underlying myeloproliferative process. Although a causal relationship cannot be definitively established, the observed sequence of events raises clinically relevant concerns.

Conclusion

Masked polycythaemia vera carries a thrombotic risk comparable to that of overt PV and requires careful clinical and laboratory surveillance. This case suggests that dabigatran may unmask or accelerate latent PV in susceptible individuals. While causality cannot be confirmed, heightened awareness and close monitoring of full blood counts should be considered in patients with JAK2 positivity or suspected myeloproliferative neoplasms who are treated with direct oral anticoagulants.

Learning Points

- Masked polycythaemia vera (mPV) may present with borderline erythrocytosis and thrombocytosis despite carrying full thrombotic risk.
- Dabigatran has not previously been associated with erythrocytosis or progression of mPV to overt PV.
- A temporal association between dabigatran exposure and overt PV manifestation should prompt further investigation.
- Clinicians should consider periodic monitoring of blood counts in JAK2-positive patients receiving DOACs.

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