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LETTER TO THE EDITOR
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Vancomycin-Induced Immune Thrombocytopenia in an Older Patient in the Intensive Care Unit: A Case Report

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To the Editor,

Thrombocytopenia is a frequent finding in patients treated in intensive care units, and determining its exact cause is essential for proper management. Among many possible etiologies, drug-related immune thrombocytopenia (DITP) is uncommon but clinically relevant. The disorder occurs when drug-dependent antibodies react with platelet surface glycoproteins, mainly GPIb/IX/V or GPIIb/IIIa, leading to immune-mediated destruction of platelets. Early recognition and discontinuation of the suspected medication are crucial to avoid potentially severe bleeding events (1-4).

Vancomycin is a glycopeptide antibiotic that inhibits bacterial cell wall synthesis and is used to treat gram-positive infections, particularly methicillin-resistant Staphylococcus aureus (5). In rare cases, vancomycin may trigger immune-mediated thrombocytopenia through a quinine-like mechanism. The drug can promote the formation of drug-dependent antibodies that bind to platelet glycoproteins such as GPIIb/IIIa, leading to immune destruction and platelet clearance (2, 6). In this letter, we describe a patient with septic shock who developed vancomycin-associated immune thrombocytopenia during intensive care treatment.

A 65-year-old woman with a history of ischemic cerebrovascular disease was admitted to the intensive care unit (ICU) due to fever and deterioration in her general condition. She was immobilized and had chronic comorbidities including diabetes mellitus, hypertension, and recent ischemic stroke. On admission, she fulfilled the criteria for septic shock secondary to a urinary tract infection, with an increase of more than two points in the Sequential Organ Failure Assessment (SOFA) score, hypotension requiring vasopressor support, and lactate levels exceeding 2 mmol/L. She was started on intravenous hydration, empiric broad-spectrum antibiotics (meropenem and vancomycin), vasopressors, and corticosteroids.

Initial laboratory tests revealed leukocytosis, elevated inflammatory markers (CRP and procalcitonin), increased urea and creatinine levels consistent with acute kidney injury, and a platelet count of $129\times10^3/\mu L$. Coagulation tests (INR and aPTT) and liver enzymes were within normal limits, while lactate was elevated. The peripheral smear confirmed the platelet count (approximately $120\times10^3/\mu L$) and ruled out platelet clumping. During follow-up, infection markers regressed, renal function normalized, and vasopressor requirements decreased. However, the platelet count continued to decline despite overall clinical improvement.

Over the next several days, the patient's platelet values fell progressively, reaching below $20\times10^3/\mu L$. A pooled platelet transfusion was administered, but the post-transfusion count did not show a meaningful increase. The patient's clinical course and laboratory data suggested isolated thrombocytopenia without signs of disseminated intravascular coagulation (DIC), as fibrinogen, D-dimer, and coagulation parameters remained within normal limits. Serum lactate dehydrogenase (LDH) was 214 U/L (135–214 U/L), total and indirect bilirubin levels were 0.4 and 0.1 mg/dL respectively (reference: 0.1–1.2 mg/dL and 0–0.5 mg/dL), and no schistocytes were observed in the peripheral smear, excluding hemolysis. The absence of renal deterioration or microangiopathic findings ruled out

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thrombotic microangiopathies such as hemolytic uremic syndrome (HUS), atypical HUS, and thrombotic thrombocytopenic purpura.

Since meropenem was considered more likely to cause thrombocytopenia, it was discontinued after Staphylococcus hominis was isolated, while vancomycin therapy was continued. Despite infection resolution, the platelet count failed to recover, excluding sepsis-related thrombocytopenia. The temporal relationship between vancomycin exposure and platelet decline, along with the absence of other etiologies, strongly suggested vancomycin-induced immune thrombocytopenia. The patient had not received heparin or low-molecular-weight heparin, excluding heparin-induced thrombocytopenia.

Given the possibility of immune-mediated thrombocytopenia, corticosteroid therapy was intensified to prednisolone 1 mg/kg/day. Despite three days of treatment, there was no platelet recovery, and intravenous immunoglobulin (IVIG) was initiated at 1 g/kg/day for two consecutive days. Vancomycin therapy was discontinued concurrently. Following drug withdrawal and IVIG administration, the platelet count increased markedly, reaching normal levels within two weeks. As the platelet count normalized and infection markers stabilized, corticosteroid therapy was gradually tapered and discontinued. The patient was discharged from the ICU.

Vancomycin-associated thrombocytopenia is an uncommon but clinically important adverse reaction that has been reported only in a limited number of cases (7). Literature data emphasize the importance of recognizing the condition early, promptly discontinuing the causative drug, and initiating appropriate therapeutic interventions, including corticosteroids, IVIG, and platelet transfusions when active bleeding is suspected (1). In our case, corticosteroid treatment had already been started for septic shock, but platelet recovery occurred only after IVIG administration. This observation suggests that, in critically ill patients receiving corticosteroids for septic shock, IVIG can be prioritized as a first-line option for drug-induced immune thrombocytopenia, avoiding unnecessary escalation of steroid therapy and potential adverse effects.

Sincerely.

DESCRIPTIONS

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