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Vancomycin induced thrombocytopenia: A case report

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Abstract
Vancomycin is a first-line drug for treating methicillin-resistant Staphylococcus aureus. Thrombocytopenia is a rare adverse reaction to vancomycin treatment. We describe the case of a 8 years old boy, known case of CKD Stage 5, who was admitted for continuous ambulatory peritoneal dialysis and had hospital acquired infection during stay. After 10 days of treatment with vancomycin, his platelet count reduced as low to 18 × 109/L. Vancomycin was discontinued following which platelet count returned to normal after 4 days. Patients, especially young children, receiving vancomycin for a long clinical course should undergo careful monitoring of laboratory indicators and blood tests.

Keywords: vancomycin. Adverse –reaction. Thrombocytopenia

Introduction
Vancomycin is a glycopeptide antibiotic commonly used in management of serious gram-positive bacterial infections that are resistant to other antibiotics, especially methicillin-resistant Staphylococcus aureus and coagulase-negative Staphylococcus. Use of vancomycin is most commonly attributed to side effects such as ototoxicity, nephrotoxicity, and erythrocyte syndrome. Vancomycin-induced thrombocytopenia (VIT) is a rare adverse reaction. Although isolated cases of VIT have been reported in adults and few cases of VIT in children, to our knowledge, there have been no reports of VIT in infants, and only 2 cases 9 [1, 2] documented in neonates and 1 case [3] in a young child (2 years). Here, we report a case of severe thrombocytopenia after the administration of intravenous vancomycin in 8 years old boy of chronic kidney disease.

Case Report
8 years old boy, known case of CKD Stage 5, was admitted for continuous ambulatory peritoneal dialysis initiation. Child underwent hemodialysis 6-7 times during that period. At two weeks of hospital stay, child developed high grade fever spikes. Possibility considered was health care associated infection- IV catheter related. Work up for the same were sent and child was started on IV vancomycin / meropenem. Child continued to have fever spikes despite IV antibiotics, thus IV amphotericin B was added on day 3 of fever and fungal work up was sent. Blood culture showed growth of Staphylococcus aureus sensitive to vancomycin/cloxacillin. His urine and peritoneal fluid cultures were sterile and serum galactomannan came to be positive. Child was continued on IV vancomycin/meropenem/amphotericin B. During the hospital stay, child became afebrile. Meropenem was stopped after 7 days and child was continued on vancomycin / amphotericin B and stopped after 14 days of completion. Over the hospital stay, he started to have thrombocytopenia from day 22 of hospital stay. Cause initially considered was sepsis – fungal (i/v/o child having fever with positive galactomannan (1.65ng/ml)), for which he was continued on IV amphotericin B. Despite being on amphotericin B and fall in CRP and no fresh fever spikes, child had persistent thrombocytopenia as low as 16000/cumm. He was given platelet transfusion twice i/v/o low platelet count (<20,000/cumm, though clinically he had no bleeds). Other causes considered were drug induced (vancomycin related) /immune mediated / myelofibrosis due to high PTH/ uremic myelosuppression /nutritional. Serial platelet workup showed rising levels of platelets following stoppage of IV vancomycin (after completion of 14 days of course). (Figure1.) Other workup sent for nutritional/immune causes came out to be negative. Hence, the cause attributed to thrombocytopenia considered was vancomycin induced since the platelet counts improved gradually after stoppage of vancomycin. At time of discharge, child was afebrile, hemodynamically stable, B.P-88/56 (50th centile) undergoing daily manual CAPD, on medical supplementation for CKD comorbidities.

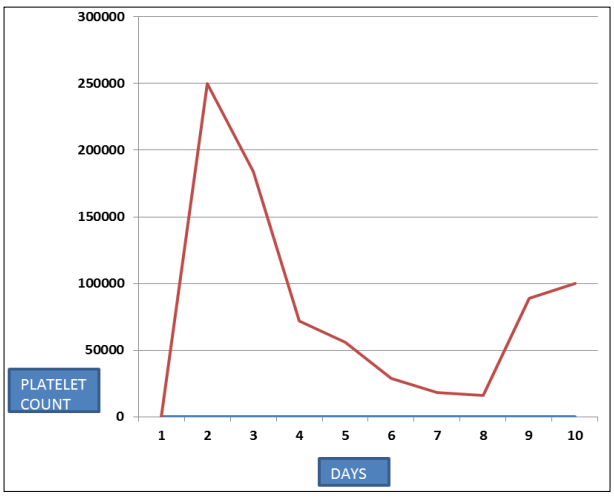

Fig 1: Demonstration of platelet drop on continuous use of vancomycin followed by improvement with discontinuation of drug.

Discussion

Vancomycin is associated with rare adverse event of thrombocytopenia, most probable mechanism understood is immune mediated mechanism. In immune thrombocytopenia purpura (ITP), platelet destruction is caused as antibodies bind to platelets leading to their clearance by the reticulo-endothelial system (RES), as well as by some degree of decreased production. ITP can be idiopathic, or related to viral infections, autoimmune disorders, lymphoproliferative disorders, or drugs [4, 5]. Classical causes of drug-induced thrombocytopenia are the quinine and quinine-like drugs [5]. The thrombocytopenia is typically sudden, severe, and may be accompanied by bleeding. The thrombocytopenia induced by these drugs is caused by antibody that is nonreactive in the absence of drug, but binds to epitopes on platelet membrane, glycoproteins IIb/IIIa or Ib/IX, when the sensitizing drug is present. Vancomycin can also be associated with marked thrombocytopenia.

Demonstrated first by Drygalski *et al*. who detected vancomycin-dependent antibodies platelet reactive antibodies (IgG and IgM) in patients clinically suspected of VIT [6]. Although the ITP generally develops rapidly, it usually resolves upon cessation of treatment and is drug-specific**.** Kenney *et al.* [7] contested that there are two immune mechanisms associated with VIT. One is the persistent presence of antibodies against vancomycin following vancomycin treatment, resulting in the rapid development of thrombocytopenia. Donnell *et al.* [8] reported a case of thrombocytopenia following vancomycin treatment within 4 hours. The other mechanism is the presence of an innate vancomycin-dependent antibody, resulting in a rare probability of thrombocytopenia upon the first administration of vancomycin. Although the detection of vancomycin-dependent antibodies is beneficial for diagnosing VIT, not all hospitals have appropriate testing conditions. As reported by Drygalski *et al*., the patient's platelet counts reached a nadir after approximately 8 days of treatment with vancomycin, with a mean value of 13.6 × 109/L. Hemorrhagic patients had a platelet count of 8.4 × 109/L, and asymptomatic patients had a platelet count of 35 × 109/L. The reported nadir of patients with other bleeding disorders is 2 × 109/L –10 × 109/L, and the average restoration time of platelet counts after discontinuing vancomycin was approximately 6 days[6]. Thus, it is necessary to discontinue vancomycin immediately in patients who are clinically suspected of VIT [9] and replacement with other drugs according to the results of drug sensitivity and the risk of drugs in young children should be considered. Blood transfusions, corticosteroids, and intravenous immunoglobulin are options, and plasma exchange can also be considered if thrombocytopenia is prolonged or accompanied by hemorrhagic signs and symptoms. Closely monitoring of platelet counts (once every 3 days) is recommended. Previous reports and this case suggest that VIT can occur in patients of all ages. Therefore, physicians prescribing vancomycin as the first choice for treating infections should be mindful of special populations, especially young children, although VIT is a rare adverse reaction. Patients with long courses should be monitored for laboratory indicators and hemorrhagic manifestations.

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Ethical Approval-Due permission was taken from institutional ethical committee.

Patient Consent

Written and informed consent was taken from patient for the publication of this case report.

References

1. Dilli D, Oguz SS, Dilmen U. A newborn with vancomycin-induced thrombocytopenia. Pharmacology. 2008; 82(4):285-6.
2. Kalra K, Mittal HG, Maria A. Vancomycin-induced thrombocytopenia in a newborn. Drug Metab Pers Ther. 2016; 31(4):235-7.
3. Bay A, Oner AF, Dogan M, Caksen H. A child with vancomycininduced thrombocytopenia. J Emerg Med. 2006; 30(1):99-100.
4. Aster RH, Bougie DW. "Drug-induced immune thrombocytopenia," The New England Journal of Medicine. 2007; 357(6):580-587.
5. Visentin GP, Liu CY. "Drug-induced thrombocytopenia," Hematology/Oncology Clinics of North America. 2007; 21(4):685-696.
6. Von Drygalski A, Curtis BR, Bougie DW, *et al*., "Vancomycin-induced immune thrombocytopenia," The New England Journal of Medicine. 2007; 356(9):904-910.
7. Kenney B, Tormey CA. Acute vancomycin-dependent immune thrombocytopenia as an anamnestic response. Platelets. 2008; 19(5):379-83.
8. O'Donnell E, Shepherd C, Neff A. Immune thrombocytopenia from vancomycin in orthopedic cement. Am J Hematol. 2007; 82(12):1122.
9. Gupta S, Sharma S, Menon N, Ahuja S, Dahdouh M. Case report of vancomycin-induced pancytopenia. Rev Soc Bras Med Trop. 2016; 49(2):258-9.