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Vancomycin Induced Thrombocytopenia Complicating Permanent Pacemaker Placement: A Case Report

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Abstract Introduction: Vancomycin is widely used in patients with methicillin resistant staphylococcus aureus (MRSA) infections or with other infections unresponsive to other antibiotics. Vancomycin-related adverse effects, including nephrotoxicity, ototoxicity, red-man syndrome, and neutropenia have been reported. Vancomycin induced thrombocytopenia is a rare complication of vancomycin that many physicians are unaware of. When an acute decline in platelet count or bleeding occur in patients on vancomycin, vancomycin- induced thrombocytopenia should be considered. Case: We present a case of acute severe thrombocytopenia that occurred within 12 hours after vancomycin infusion in a patient that received permanent pacemaker placement. Vancomycin was discontinued and patient's platelets returned to baseline five days after vancomycin was discontinued. Conclusion: This case report highlights the importance of physicians being aware of vancomycin induced thrombocytopenia, a rare but reversible complication of vancomycin, which if diagnosed early will lead to early discontinuation of vancomycin, early resolution of thrombocytopenia and reduced hospital stay.

Keywords: *vancomycin, thrombocytopenia, case report*

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1. Background

Vancomycin has activity against gram positive organisms including methicillin resistant staphylococcus aureus (MRSA) and is widely prescribed for hospital-acquired infections, device-related infections, and treatment of resistant organisms. [1,2]

Vancomycin has been well recognized as a cause of reversible neutropenia, red man syndrome, ototoxicity and nephrotoxicity, but the incidence of vancomycin induced immune thrombocytopenia is rare. [1,2] We present a case of acute severe thrombocytopenia that occurred within 12 hours after vancomycin infusion in a patient that received permanent pacemaker placement.

This case report highlights the importance of physicians being aware of this rare but reversible complication of vancomycin, which, if diagnosed early, could potentially avoid worsening patient status and prolonged hospital stay.

2. Case Report

70-year-old female with history of atrial fibrillation and tachy-brady syndrome is hospitalized with complaints of palpitations and difficulty breathing. On admission, blood pressure was 141/60, heart rate 128, respiratory rate 20, and temperature 98F. Apart from fast irregular heartbeats, patient's physical examination was unremarkable. EKG showed atrial fibrillation with rapid ventricular rate. Electrolytes were normal apart from a chronic hyponatremia of 130 meq/L. White blood cell (WBC) was 12.9, hemoglobin 12.1g/dl and platelets 256,000. Urinalysis, renal and hepatic function tests were normal. Upon consultation with cardiology, the patient had previously been recommended for permanent pacemaker (PPM) for treatment of tachy-brady syndrome. She was then admitted and scheduled for pacemaker placement the following day. Patient was not started on heparin prophylaxis for venous thromboembolism on admission because she was scheduled for surgery the following day.

On day two of admission, prior to PPM placement, the patient received a preoperative dose of vancomycin. Vancomycin was chosen due to her documented allergy to penicillin and sulfa medications. Shortly after the procedure, the patient suddenly became hypotensive. The performing physician noted only 50 cc of blood loss at the time which would not account for her sudden loss of blood pressure. Physical exam revealed no postoperative hematoma. Patient received normal saline boluses and was started on norepinephrine to maintain her blood pressure.

Post-procedure chest x-ray ruled out pneumothorax. Bedside echo showed thickened pericardial fat and small pericardial effusion, which was not different from baseline. CT chest with contrast, CT abdomen/pelvis, and urinalysis were unremarkable. An immediate postoperative blood count showed leukocytosis congruent with her initial labs. However, the labs showed an acute severe normocytic anemia (hemoglobin 6.6) and acute severe thrombocytopenia (platelets 5,000) (Table 1 and Figure 1). Heparin PF4 antibody was negative. Lactate dehydrogenase (LDH) was low while bilirubin, serum cortisol, reticulocytes, fibrinogen, prothrombin time, partial thromboplastin time, and haptoglobin were normal. Her blood microscopy did not reveal any schistocytes. Therefore, hemolysis was ruled out and concluded that the patient may have suffered blood loss from profound thrombocytopenia. Due to her elevated leukocyte count, an infection was suspected so vancomycin was continued, and she was empirically started on metronidazole and levofloxacin. Patient received one unit of platelets, one unit of fresh frozen plasma (FFP) and three units of packed red blood cells (PRBC). Post transfusion, hemoglobin was 10g/dl and platelet count was 50,000.

On day three of hospital admission, the patient received a second dose of vancomycin. Repeat labs resulted in a platelet drop from 50,000 to 15,000. Hemoglobin similarly dropped from 10g/dl to 8.6g/dl, while the white blood cells dropped from 19,100/ml to 8,400/ml. The patient received one more unit of platelets and another unit of packed RBCs. At that time, the patient noted that on three prior occasions while undergoing surgery in other facilities, she developed low platelet count. Without any other likely cause to be found, the vancomycin was immediately discontinued. The patient's platelets returned to baseline five days after vancomycin was discontinued. Once the patient's platelets returned to baseline, her hemoglobin remained stable. [Table 1 and Figure 1].

Table 1. Showing change in platelet count with vancomycin administration

Days	Platelet count	Remark
Day 1	219,000	
Day 2: 6am	203,000	First dose of vancomycin given
Day 2: 6pm	5,000	1 unit of platelets, 1 unit of fresh frozen plasma transfused
Day 3: 6am	50,000	Second dose of vancomycin given
Day 3: 12pm	15,000	1 unit of platelet given
Day 3: 9pm	27,000	Vancomycin discontinued
Day 4: 6am	31,000	
Day 4: 6pm	54,000	
Day 5	83,000	
Day 6	147,000	
Day 7	208,000	
Day 8	246,000	

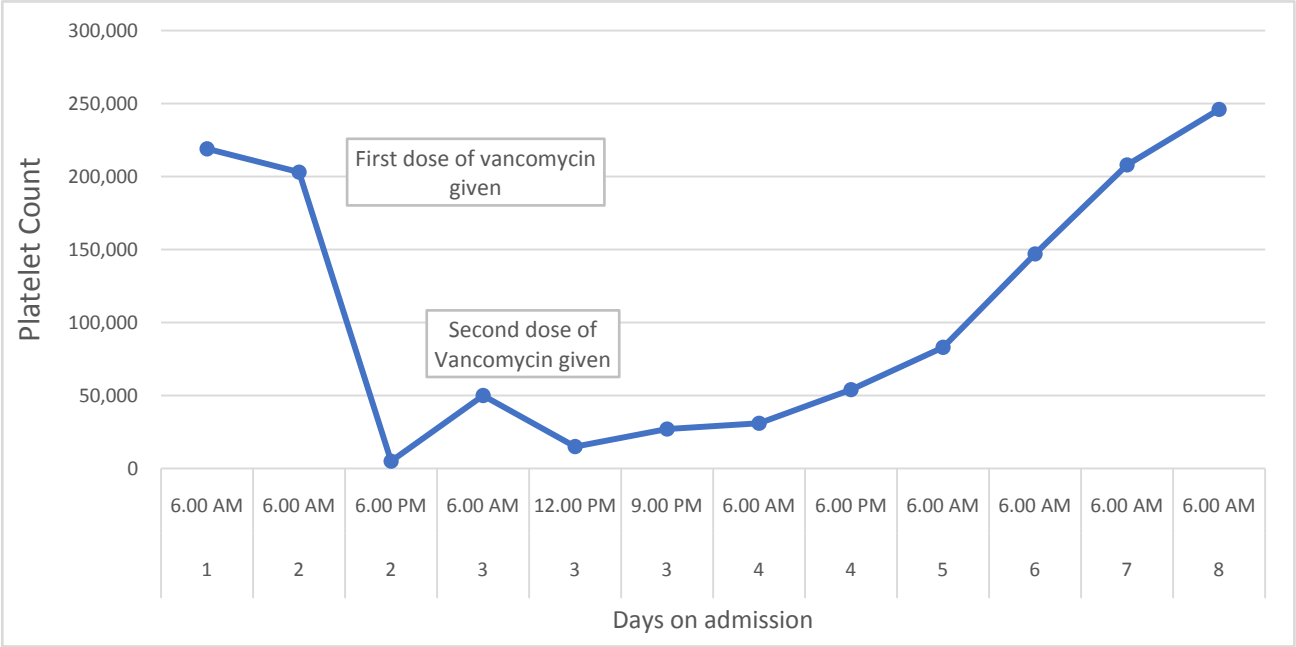

Figure 1. Showing change in platelet count with vancomycin administration

3. Discussion

Vancomycin-dependent, platelet-reactive antibodies of the IgG class, IgM class, or both have been associated with thrombocytopenia in patients that received vancomycin. [2-12] In a review of 29 patients with vancomycin induced thrombocytopenia, the platelet count dropped to 13,600/mm^3 on average (range: 1,000 to 60,000), which represented a 93% drop from pretreatment values (range: 76% to 99%). [1] The nadir for the platelet count was reached about 8 days after vancomycin was initiated (range: 1 to 27 days) and platelet levels returned to baseline in about 7 days after vancomycin was discontinued (range: 4 to 17 days). It was also noted that thrombocytopenia may persist longer in patients with renal failure, most likely due to delayed clearance of vancomycin. [1,12] These findings were similar to a review of 30 case reports that showed a mean time to platelet nadir count of 8 days and return of platelet counts to normal within 5 to 6 days of discontinuation of vancomycin in reported cases. [13] For our patient, the platelet count returned to baseline within 5 days after the discontinuation of vancomycin, which was within the range documented in the above studies.

Acute severe thrombocytopenic episodes that developed within 24 hours after infusion of vancomycin have been reported in patients who have been previously exposed to vancomycin. [1,8,13] In these patients, the acute severe thrombocytopenia is likely due to antibodies persisting from the previous exposure to Vancomycin. [1] Our patient developed acute severe thrombocytopenia within 12 hours of vancomycin infusion and did not have a known history of prior exposure to vancomycin. However, she has had multiple surgeries at other hospitals and presumably could have received vancomycin during those procedures. The very rare possibility that naturally occurring antibodies causing the acute thrombocytopenia in our patient could not be excluded. The possibility of heparin induced thrombocytopenia (HIT) was ruled out in our patient because the patient did not receive heparin during her hospital admission and heparin PF4 antibody was negative. In addition, acute severe drop of platelet count to 5,000 is not consistent with the usual picture in HIT. [14]

In conclusion, this case report highlights the importance of physicians being aware of vancomycin induced thrombocytopenia, a rare but reversible complication of vancomycin, which if diagnosed early will lead to early discontinuation of vancomycin, early resolution of thrombocytopenia and reduced hospital stay.

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