A case of Vancomycin-induced thrombocytopenia in the coronary care unit

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Learning Objectives

Vancomycin therapy can be a cause of drug-induced thrombocytopenia and should be on the differential diagnosis when a patient presents with acute onset thrombocytopenia with no other explanation.

Case

A 67 year-old male was admitted to the coronary care unit (CCU) for management of NSTEMI with concern for cardiogenic shock and possible septic component due to lower extremity wound infection.

On arrival to the ED, the patient had the following vital signs: HR 88bpm, RR 28bpm, BP 86/57mmHg, SpO_2 99% on 5L/min.

The patient was started on a heparin infusion for possible non-ST-elevation myocardial infarction and was covered with broad spectrum antibiotics (Vancomycin and Cefepime).

Repeated CBC 40 minutes, 12 hours and 15 hours after receiving the first dose of Vancomycin showed platelets (PLT) levels of $93x10^9$/L, $8x10^9$/L and $1x10^9$/L respectively. For which heparin infusion and antibiotic therapy were discontinued at that time.

Differential diagnosis at hour 15 of admission:
- Heparin-induced thrombocytopenia (HIT)
- Disseminated intravascular coagulation (DIC)
- Drug-induced thrombocytopenia
- Immune thrombocytopenia

4T´s score showed a low-probability (<5%) for HIT, in addition to negative HIT antibodies. Repeated laboratory studies showed a markedly elevated Fibrinogen (944mg/dL). The patient received platelet transfusion and a daily dose (130g) of intravenous immunoglobulin (IVIG), with improvement of platelet count to $60x10^9$/L.

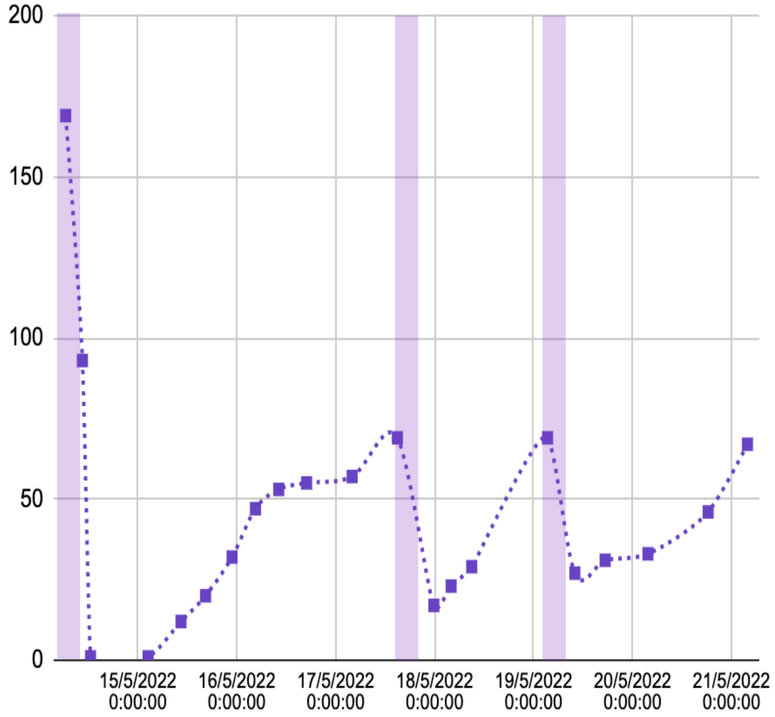
On day three of hospitalization there was a new concern for right lower extremity necrotizing fasciitis, for which he received Piperacillin/Tazobactam and a second dose of Vancomycin with a new drop in platelet count to $17x10^9$/L, which then improved after another dose of IVIG.

Case Continued

On day 5 of hospitalization, it was decided to administer another dose of Vancomycin due to worsening right lower extremity wound infection, which produced another drop in PLT count from $69x10^9$/L (3 hours prior to vancomycin administration) to $27x10^9$/L. with no other explainable cause for the intermittent drops in PLT count.

At that point, Vancomycin was identified as the most likely culprit of the thrombocytopenia due to the association between the time of administration and observed drop in PLT counts.
After no further Vancomycin doses, PLT counts kept improving until they returned to patient´s baseline levels.

Naranjo adverse drug reaction probability scale was used to determine these events as an adverse effect of the administration of Vancomycin. Based on the case, a score of 5 was obtained indicating a probable cause.



Vancomycin administration

Naranjo Scale Questions[1]	Score
1. Are there previous conclusive reports on this reaction?	+1
2. Did an adverse event appear after suspected drug was given?	+2
3. Did the adverse reaciton improve when the drug was discontinued?	+1
4. Did the adverse reaction appear when the drug was readministered?	+2
5. Are there alternative causes that could have caused the reaction?	-1
6. Did the reaction reappear when a placebo was given?	0
7. Was the drug detected in any body fluid in toxic concentrations	0
8. Was the reaction more severe when the dose was increased or less severe when it was decreased?	+1
9. Did the patient have a similar reaction on previous exposure?	0
10. Was the adverse event confirmed by any objective evidence?	0
Total score	**6**

Discussion

•Drug-induced thrombocytopenia is a common entity seen most frequently with other type of antibiotics (penicillins, linezolid, sulfamethoxazole and ceftriaxone). But using Naranjo scale we obtained a score of 6 (probable), which shows that in complex cases Vancomycin should always be considered as a cause.[1]

•Diagnosis of Vancomycin-Induced Thrombocytopenia is made by detecting Vancomycin-dependent antiplatelet antibodies in patient´s serum or by seeing improvements in PLT counts after discontinuation of the drug in the setting of no other apparent cause.[2]

•Onset of thrombocytopenia is usually described from hours to weeks after initial exposure[2,3], but this case showed that onset can be even after a few minutes/hours after initial exposure.

References

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