Vancomycin Associated Acute Interstitial Nephritis in a Patient with Infective Endocarditis

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Introduction

- Vancomycin, a bacterial cell wall inhibitor, can cause acute kidney injury, defined by a creatinine rise over 0.5 mg/dL after several days of therapy.
- Risk factors include high doses, prolonged use, other nephrotoxic drugs, and reduced GFR.
- While it usually leads to acute tubular necrosis (ATN), it can also rarely cause acute interstitial nephritis (AIN).
- This case highlights vancomycin-induced AIN in a patien with supratherapeutic levels.

Patient Presentation

- A 70-year-old male with chronic kidney disease, hypertension, type 2 diabetes, and chronic heart failure with a biventricular ICD presented with fatigue. Physical exam was unremarkable except for hypotension.
- Labs showed leukocytosis with neutrophilic predominance, lactic acidosis, and elevated C-reactive protein.
- Blood cultures were drawn and he was started on vancomycin and cefepime, for suspected sepsis
- Blood cultures showed a growth of gram positive cocci. Considering the patient had ICD, he underwent transesophageal echocardiogram (TEE). TEE was suggestive of a vegetation attached to the right ventricular lead of ICD and his ICD was explanted.
- He was maintained on IV vancomycin.
- Final blood cultures showed growth of methicillin sensitive staphylococcus aureus, sensitive to vancomycin and cefazolin.
- His kidney function remained stable until the 9th day of vancomycin therapy where it worsened.
- Vancomycin level was checked which was elevated to 40mcg/mL (Normal range 10-20 mcg/mL).
- Vancomycin was immediately stopped, and switched to IV cefazolin.
- Over the period of next 10 days, his creatinine continued to worsen and increased to 5.5mg/dL. On the 10th day after stopping vancomycin therapy, his creatinine plateaued around 5mg/dL and vancomycin level also came down to 19mcg/mL at the same time.

Figure 1. Kidney Biopsy

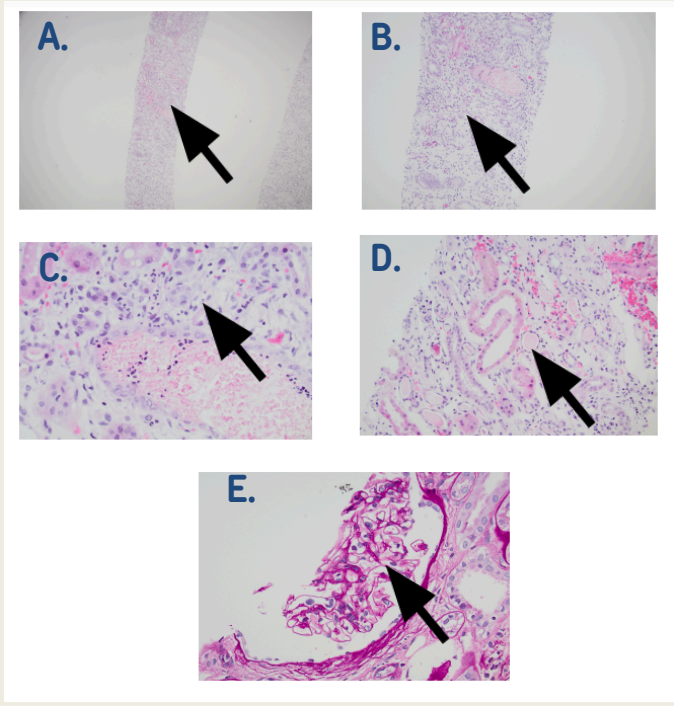

Figure 2. Timeline

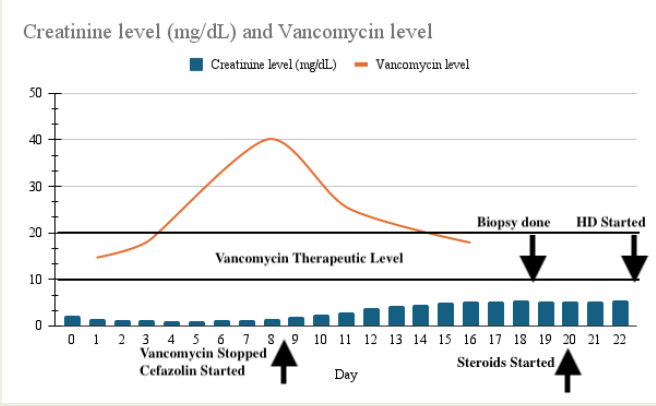

Figure 2. Timeline showing the temporal chain of events

Figure 1. Kidney Biopsy

- **A.** Pathology slides showing mild to moderate interstitial inflammation (40x, H&E)
- **B.** Mixed inflammation including plasma cells and occasional eosinophils (100x, H&E).
- **C.** Collections of neutrophils are present, both in the interstitium and within the tubules (400x, H&E).
- **D.** There is also a component of acute tubular injury (200x, H&E).
- **E.** The glomeruli present do not show glomerulonephritis (400x, PAS).

Clinical Course

- The patient underwent a renal biopsy, which was consistent with AIN (Figure 1).
- High-dose corticosteroids were initiated, but without improvement in renal function, hemodialysis was started.
- This slowly improved the patient's renal function, and he was discharged with outpatient follow-up with nephrology for evaluation of renal recovery.

Discussion

- Vancomycin is typically linked to acute tubular necrosis (ATN), but it can also cause acute interstitial nephritis (AIN) in rare cases.
- Monitoring vancomycin levels (usually before the 4th dose after a change) is crucial, as nephrotoxicity may not appear in routine labs until days or weeks after starting the drug.
- In this case, the patient developed acute renal failure after 9 days of therapy, with supratherapeutic levels found.
- A renal biopsy confirmed AIN, given the temporal events vancomycin being the most likely culprit.
- Treatment involves discontinuing vancomycin, trying high-dose corticosteroids, and, if necessary, starting hemodialysis.

References

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