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Case Report

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Vancomycin-Induced Toxic Epidermal Necrolysis in a Patient with End-Stage Renal Disease: A Case report

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Abstract

Toxic Epidermal Necrolysis (TEN) is a rare, life-threatening mucocutaneous hypersensitivity reaction, most often triggered by drugs. We report a case of a 60-year-old woman with End-Stage Renal Disease (ESRD) on intermittent hemodialysis who developed TEN following vancomycin exposure. The patient initially presented with buccal swelling and Upper Gastrointestinal Bleeding (UGIB), followed by rapidly progressive desquamative skin lesions involving over 80% of her body surface area. Management required a multidisciplinary approach including dermatological care, critical care support, parenteral nutrition and renal replacement therapy with minimal anticoagulation. Despite aggressive supportive care, the patient died from complications related to progressive TEN and sepsis. This case highlights the risk of Severe Cutaneous Adverse Reactions (SCARs) in ESRD patients, the importance of early recognition of atypical presentations and the complexities of managing TEN in the setting of renal impairment.

Keywords: Toxic epidermal necrolysis; Vancomycin; End-stage renal disease; Dialysis; Mucocutaneous reaction

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Introduction

Toxic Epidermal Necrolysis (TEN) is a rare but potentially fatal dermatological emergency characterized by extensive epidermal detachment and mucosal involvement. It is most commonly drug-induced, with medications such as sulfonamides, antiepileptics, NSAIDs and allopurinol being frequent triggers. Vancomycin, though less commonly associated, has been implicated in Severe Cutaneous Adverse Reactions (SCARs), particularly in individuals with impaired renal function [1,2]. The incidence of TEN is estimated at 0.4-1.9 per million per year, with mortality ranging from 25% to over 35% [3].

Case Presentation

A 60-year-old female with a history of ESRD on intermittent hemodialysis *via* left internal jugular catheter, type 2 diabetes mellitus (5 years) and hypertension (1 year) presented with signs of Catheter-Related Bloodstream Infection (CRBSI). She was empirically started on intravenous vancomycin. Blood cultures later confirmed Extended-Spectrum Beta-Lactamase (ESBL) producing Klebsiella pneumoniae.

During dialysis, she developed sudden swelling over the left buccal region. There was no history of orofacial trauma or bleeding. Examination revealed a 1 cm × 2 cm bluish lesion on the left buccal mucosa, which ruptured and bled persistently (~300 mL). She remained hemodynamically stable. Heparin-induced thrombocyte-openia was considered and dialysis continued without anticoagulation.

Laboratory investigations revealed prolonged aPTT (116.5 s), INR 1.19 and low-normal platelet counts. She was admitted to ICU for observation. Upper GI endoscopy revealed a bleeding linear mid-esophageal ulcer, controlled with endoscopic epinephrine injection.

By day 3, she developed erythematous, desquamative lesions involving >80% of her body surface area, affecting the face, trunk and limbs, with severe mucosal

involvement. A diagnosis of TEN was made. Vancomycin was immediately discontinued **(Figures 1 and 2)**.

Management included intravenous methylprednisolone (500 mg/day × 3 days) followed by oral prednisolone 30 mg daily, betamethasone and tacrolimus ointments, mupirocin prophylaxis, sterile dressings and thermoregulation. Later, was initiated.

Severe oral ulcerations necessitated total parenteral nutrition. Despite targeted meropenem for ongoing ESBL bacteremia, she remained septic. Hemodialysis was continued using Slow Low Efficiency Dialysis (SLEDD) due to hemodynamic instability. Platelets declined to 26 × 10^3/μL.

Although skin re-epithelialization improved by >50%, mucosal ulcers remained painful and bleeding. She deteriorated with worsening systemic sepsis and passed away on day 59 of admission due to multiorgan failure.

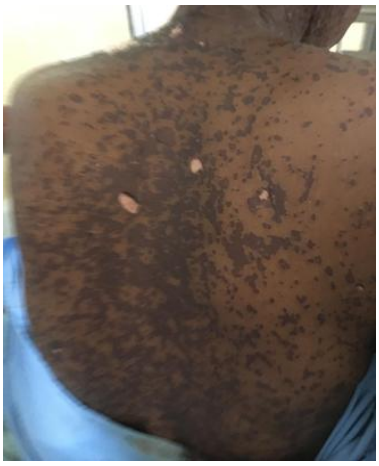

Figure 1: Extensive desquamative lesions affecting >80% of body surface area in the patient with toxic epidermal necrolysis.

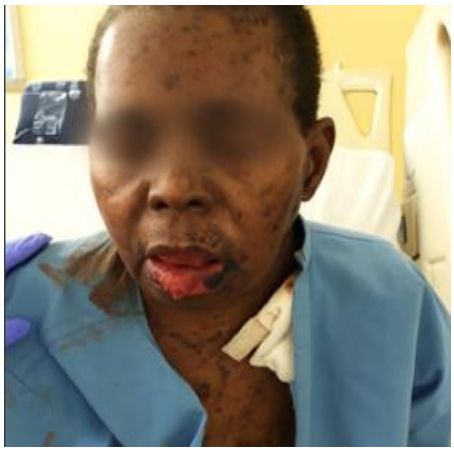

Figure 2: Severe mucosal ulcerations involving the oral cavity.

Discussion

This case demonstrates a rare and fatal occurrence of vancomycin-induced TEN in a patient with ESRD. TEN is typically precipitated by drugs, with over 80% of cases linked to recent medication exposure [3]. Vancomycin is increasingly recognized as a SCAR-inducing agent, especially in patients with renal impairment [1,2].

Our patient's initial presentation with mucosal ulceration and upper GI bleeding is highly unusual for TEN, where mucocutaneous involvement typically appears concurrently. This atypical progression complicated the early diagnosis. Delay in recognizing early signs may have contributed to worse outcomes.

Corticosteroids are controversial in TEN. While some studies show benefit in halting progression, others raise concerns about infection risk [5]. In our case, steroid escalation to intravenous pulse therapy coincided with partial skin improvement but did not prevent systemic deterioration, likely due to persistent sepsis and immunosuppression.

ESRD patients are more vulnerable to SCARs due to altered drug metabolism, impaired immunity and polypharmacy [6]. Early identification, withdrawal of the culprit drug and multidisciplinary supportive care are key. In suspected vancomycin-induced TEN, alternative antimicrobials with adjusted dosing and close dermatologic monitoring are essential.

This case adds to the limited reports of vancomycin-associated TEN in ESRD patients and underscores the need for heightened clinical vigilance, especially when managing high-risk drugs in immunocompromised populations.

Conclusion

Vancomycin-induced TEN is rare but potentially fatal, especially in patients with ESRD. Atypical presentations such as upper gastrointestinal bleeding may delay diagnosis. Prompt drug withdrawal, supportive care and a multidisciplinary approach are essential. This case reinforces the importance of cautious antibiotic use in renal failure and early recognition of SCARs.

Declarations

Ethics approval and consent to participate

Written informed consent for medical data usage and hospital case reporting was obtained from the patient's next of kin.

Consent for publication

The patient's next of kin provided written consent for publication of this case report and accompanying clinical images.

Availability of data and materials

All relevant data are included in the case report. Additional clinical details are available from the corresponding author upon reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' Contributions

Huda Akrabi: Conceptualization, clinical management, manuscript writing.

Datius Mutalemwa: Case documentation, literature review.

Kajiru Kilonzo: Discussion drafting.

Abel Mwanga: Final review and editing.

All authors reviewed and approved the final manuscript.

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