Skin Rash in a Stage 4 CKD Patient Treated for Hyperkalemia

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Case Description
A 63-year-old man whose history was significant for CKD stage 4 (baseline serum creatinine of 3.2 mg/dl) secondary to IgA nephropathy, proven by biopsy specimen at age 39, and chronic recurrent hyperkalemia presented with worsening, bilateral, lower-extremity, nonhealing, ulcerative lesions.

Further history revealed that he had been prescribed patiromer, to treat hyperkalemia, 4 weeks before the current presentation. Two weeks after the initiation of patiromer, the patient developed bilateral, lower-extremity, erythematous swelling, accompanied by a painful, petechial rash. Subsequently, the patient was treated with oral antibiotic for a presumed diagnosis of cellulitis.

Examination was significant for large, exudative ulcers and purpuric macules associated with weeping edema (Figure 1). Laboratory workup was remarkable for potassium (4.7 mmol/L), BUN (94 mg/dl), creatinine (4.1 mg/dl), and white blood cell count (11.3 cells/mm3). Deep venous thrombosis was ruled out via a lower-extremity venous Doppler ultrasound. A complete workup for GN was unremarkable. The wound cultures grew _Pseudomonas aeruginosa_, which was sensitive to ciprofloxacin.

A skin biopsy specimen showed small-vessel vasculitis, with intraepidermal and subepidermal neutrophilic aggregates, and positive direct immunofluorescence for fibrin along luminal blood vessels (Figure 2). No evidence for IgA deposition was noted on immunofluorescence exam.

The patient was discharged on high-dose, tapering prednisone and oral ciprofloxacin. The patient had complete resolution of the skin lesions 4 weeks later.

Discussion
Patiromer, approved in 2015 for outpatient treatment of hyperkalemia, is a nonabsorbed, sodium-free, potassium-binding polymer that exchanges calcium in the gastrointestinal tract, thereby increasing fecal potassium excretion (1–3). Several clinical trials have demonstrated the efficacy of patiromer in lowering potassium among patients with hyperkalemia who are on concomitant renin-angiotensin-aldosterone system inhibitors for heart failure, diabetic nephropathy, and CKD (3,4). Gastrointestinal symptoms are the most common adverse effects of patiromer (3,4). Notably, no previous cases of vasculitis were reported in association with patiromer.

Leukocytoclastic vasculitis is a small-vessel inflammatory disease characterized by deposition of immune complexes. The pathogenesis of the immune-complex formation is attributed to several factors, including infections (viral and bacterial), medications, and chemicals. Clinically, leukocytoclastic vasculitis manifests as palpable purpura of the lower extremities and is often accompanied by abdominal pain, arthralgia, and kidney involvement. A skin biopsy is often performed to confirm the clinical diagnosis (5).

Review of the literature revealed no cases of leukocytoclastic vasculitis reported in association with patiromer. The absence of IgA deposition on the skin

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biopsy specimen ruled out the role of IgA nephropathy with IgA vasculitis in the current presentation. Because it was the only new medication that was introduced to our patient, we hypothesized that the vasculitic skin lesions seen in our patient are probably due to patiromer. The exact mechanisms by which patiromer triggers cutaneous leukocytoclastic vasculitis remain to be elucidated.

Teaching Points

- Patiromer is increasingly used in managing hyperkalemia in patients with CKD.
- The development of purpuric rash in patients with CKD who are administered patiromer should raise the suspicion of vasculitis as a possible complication of patiromer.
- Cutaneous vasculitis is treated by discontinuing patiromer immediately and initiating high-dose steroid.

Disclosures

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Informed consent was obtained from the patient.

Author Contributions

A. Almehmi conceptualized the study, provided supervision, and reviewed and edited the manuscript; S. Almehmi was responsible for resources; and S. Almehmi and H. Boge wrote the original draft and were responsible for visualization.

References


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