A Rare Case of Kayexalate and CMV Colitis in a Patient of Sarcoidosis and Chronic Kidney Disease

Sir,

Colitis is a common manifestation of cytomegalovirus (CMV) reactivation / *de novo* infection in immunocompromised hosts but is rarely seen with sodium polystyrene sulfonate (SPS) treatment. Co-existence of both causing colitis is still rarer. SPS can cause colonic necrosis. Although its effectiveness and safety has been disputed, SPS remains widely used for hyperkalemia. CMV infection and reactivation are known complications after solid organ transplantation but is not well reported in patients on long-term steroids.

A 60-year-old male patient was a known case of type 2 diabetes mellitus, hypertension, hypothyroidism, chronic kidney disease stage III, coronary artery disease (CAD) post percutaneous transluminal angioplasty (PTCA), and sarcoidosis. He presented to the emergency department with a 30-days history of lower abdominal pain and diarrhea. Patient was taking oral anti-diabetic, anti-hypertensive medications (on tab amlodipine 10 mg once a day) and was on oral steroids for sarcoidosis. Patient had persistent hyperkalemia with type IV renal tubular acidosis secondary to diabetic nephropathy for which he was taking oral SPS.

On presentation, the patient was afebrile and blood pressure and heart rate were 104/78 mmHg and 125 bpm respectively. Patient was dehydrated and his physical examination was significant for lower abdominal tenderness. Routine blood tests revealed Hb: 12.1 g/dL, total leucocyte count (TLC): 7005/dl, lymphocytopenia: 2800/dl, platelet count: 169 thousand/μL, random blood sugar: 165 mg/dL, HbA1C: 7.4%, hyperkalemia: 5.84 mmol/L, serum sodium: 125 mmol/L, and an elevated serum creatinine: 3.78 mg/dL and blood urea nitrogen (BUN): 98 mg/dL, Angiotensin converting enzyme (ACE) level was 16 U/L. Arterial blood gas analysis was significant for compensated metabolic acidosis (pH: 7.38; Pco2: 29.8; bicarbonate: 17.3 mmol/L; anion gap: 6.9). Urine anion gap was positive (26.1). Stool routine and microscopy was negative for ova, cyst, and trophozoites and stool culture was also negative. Stool for Clostridium deficile toxin was negative. USG whole abdomen showed diffuse concentric thickening of colon. Non-contrast abdominal CT scan showed mild diffuse concentric thickening in ascending, descending, and sigmoid colon. Maximum thickness of colon was 8 mm. The patient was given IV fluids and IV antibiotics. For persistent hyperkalemia, SPS was continued and insulin with dextrose was administered. The dose of SPS was 15 G thrice a day for four days during the present hospitalization. Prior to the present illness, he was taking oral SPS intermittently for the past six months. Patient gradually improved and creatinine decreased. Colonoscopy was planned in view of abdominal symptoms, which showed multiple scattered ulcers of varying sizes from 2 mm × 2 mm to 6 mm × 6 mm in transverse, descending, and sigmoid colon. Multiple biopsies were taken from the colon. Histological work-up of colonic biopsies revealed active colitis. Abundant purplish SPS crystals with fish scale appearance were noted within the neutrophilic exudates of the ulcers slough [Figure 1a-c]. There was enlargement of endothelial cells. In addition, the small vessels in lamina propria showed presence of intranuclear inclusions within the endothelial lining. Immunostaining for CMV was positive within these inclusions [Figure 1d]. Following biopsy findings SPS treatment was immediately stopped, and CMV blood PCR was sent, which was found positive. CMV infection was treated with oral valgancyclovir until plasma CMV DNA load became undetectable (for three weeks). It was followed by oral valgancyclovir prophylaxis for four weeks. Patient was later discharged in a stable condition. However, he died 4 months later due to severe metabolic acidosis following dialysis.

Impairment of renal function in cases of acute kidney injury and/or chronic kidney disease and many drugs like ACE inhibitors/angiotensin II receptor blockers (ARBs)/potassium sparing diuretics that interfere with K^+ excretion can result in

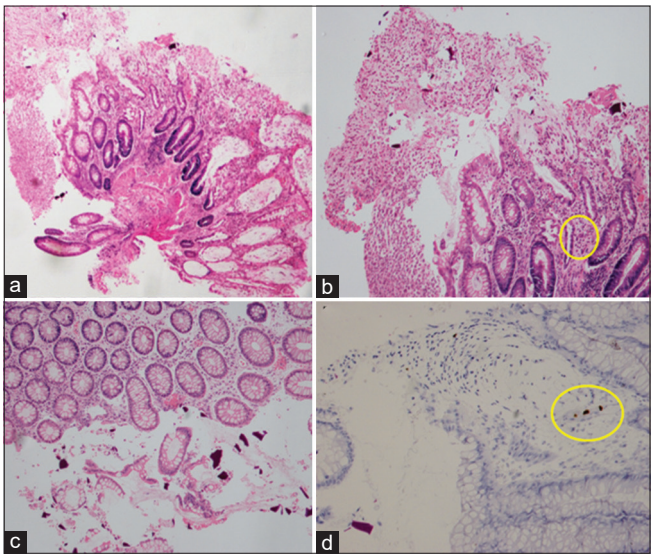

Figure 1: (a-c) Microscopy showing ulcerated colonic mucosa with purplish crystals in ulcer slough (H and E) (d) Showing CMV positive inclusions within endothelial cells (CMV IHC ×20 in yellow circle)

hyperkalemia.[1] The management of hyperkalemia includes dietary modifications, diuretics, modifying drugs, and sodium polystyrene sulfonate (SPS).

SPS causes potassium removal via the colon, thereby bypassing the impaired renal excretory mechanisms. If the patient develops constipation, this agent is ineffective that is why this agent is usually given with lactulose. SPS administration is rarely associated with the development of colitis. It causes intestinal necrosis especially when used with sorbitol. Jacob *et al*. have described six patients developing Kayexalate colitis.[2]

CMV colitis is usually seen in immunocompromised patients and rarely in immunocompetent host. Diarrhea, fever, and abdominal pain are common presenting symptoms. The diagnosis is confirmed by the presence of classical intra-nuclear owl's eye inclusions with CMV immunohistochemistry (IHC) positivity. Management includes antiviral treatment with ganciclovir or valganciclovir and reduction of immunosuppressive treatment if possible.[3] Gurtler *et al*. have described a case of hemorrhagic duodenitis secondary to SPS and CMV duodenitis.[4]

To conclude, this case highlights need to identify kayexalate-induced colitis and to increase its awareness among pathologists.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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