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Case Report

Colonic necrosis and perforation due to calcium polystyrene sulfonate in a uraemic patient: a case report

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Abstract
Sodium or calcium polystyrene sulfonate (Kayexalate or analog) is an ion-exchange resin commonly used to treat hyperkalaemia in patients with chronic kidney disease. It is known to cause digestive complications, such as nausea, vomiting and constipation. Although rare, colonic necrosis and perforation are very severe complications associated with the medication. In this case report, we present a case of calcium polystyrene sulfonate-induced colonic necrosis and perforation to remind clinicians of this rare, but dangerous, toxicity associated with this commonly used medication.

Keywords: calcium polystyrene sulfonate; chronic kidney disease; colon perforation; hyperkalaemia

Background

Colonic necrosis has been described as a rare complication after the administration of Kayexalate [1, 2]. In this case study, we present a case of calcium polystyrene sulfonate-induced colonic necrosis and perforation to remind clinicians of this rare, but dangerous, toxicity associated with this commonly used medication.

Case report

A 78-year-old woman with Stage 4 chronic kidney disease (CKD) due to chronic pyelonephritis, and a right hypoplastic kidney was presented to our emergency department with a 2-day history of abdominal pain. Her medical history included epilepsy that was treated with carbamazepine, hypertension and hyperlipidaemia, for which she received carvedilol and atorvastatin. Because of the persistent hyperkalaemia, she was treated with calcium polystyrene sulfonate at 30 g/day (anti-potassium granule; Assos Drug, Istanbul, Turkey). As a result of the CKD, she was prescribed calcitriol for a mineral and bone disorder, darbepoetin alpha for anaemia and sodium hydrogen carbonate for acidosis. Upon initial physical examination, her abdomen was non-distended with normoactive bowel sounds, but there was diffuse tenderness in her abdomen. Significant laboratory values at the time of admission included sodium 138 mEq/L, potassium 4.6 mEq/L, pH 7.20, bicarbonate 13 mEq/L, Blood Urea Nitrogen 71 mg/dL, creatinine 2.6 mg/dL, leukocytes 15 500/mm^3, eosinophils 110/mm^3, haematocrit 32.7%, platelets 221 000/mm^3 and C-reactive protein 10.9 mg/dL. On the second day of hospitalization, the abdominal pain worsened and free air under the diaphragm was found on abdominal radiography. She was taken immediately to the operating room for a laparotomy. Her sigmoid colon was found to be necrotic and perforated. A biopsy was performed and the perforated segment was repaired by primary closure. The full-cut biopsy contained the perforated area and layers of colonic wall. The surface of the deep ulcer contained necroinflammatory debris and various sized fragments of basophilic crystalloid material with angulated margins on microscopic examination (Figure 1a and b). Also, there were no features of chronic colitis, including inflammatory bowel disease or chronic ischaemic colitis. This characteristic finding is consistent with Kayexalate crystals.

Discussion

Sodium polystyrene sulfonate can also bind intraluminal calcium, leading to constipation, fecal impaction and subsequent bowel obstruction or perforation [1]. The real incidence of colonic necrosis after Kayexalate remains unknown. Gerstman *et al.* [2] reported a 0.27% overall incidence, with a higher incidence (1.8%) during the post-operative period. Lillemoe *et al.* originally reported colonic necrosis due to Kayexalate–sorbitol enemas, along with experimental evidence suggesting that the necrosis was due to sorbitol rather than the Kayexalate in presence of uraemia. Extensive transmural necrosis was noted in rats receiving enemas of sorbitol or Kayexalate in sorbitol in both the uraemic and non-uraemic groups [3]. As in this case report, renal failure may be an important facilitating factor in the pathogenesis of the necrosis. In contrast to the experimental data, our case was treated with calcium polystyrene sulfonate orally, not rectally. This case report shows that colonic necrosis is caused not only by Kayexalate–sorbitol, but also by

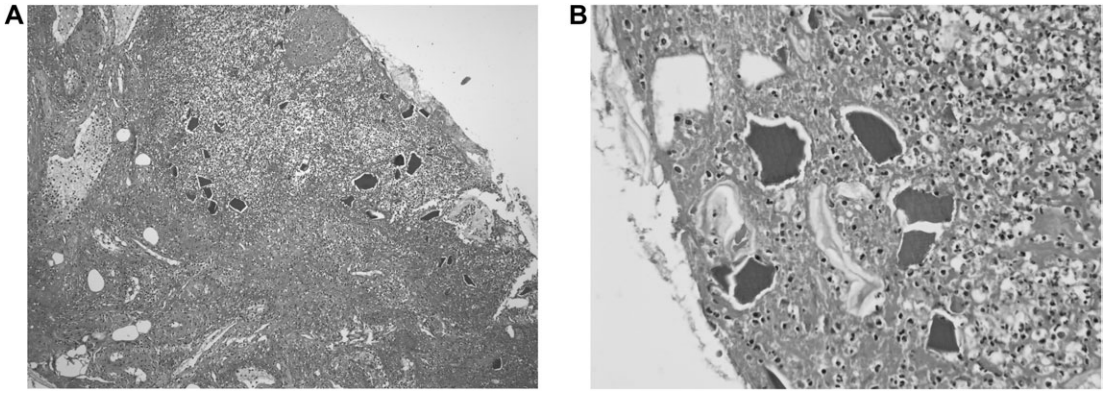

Fig. 1. (**A**) The basophilic crystalloid material in the necroinflammatory background of the ulcer surface (haematoxylin and eosin, original magnification, ×100). (**B**) The basophilic crystalloid material in the necroinflammatory background of the ulcer surface (haematoxylin and eosin, original magnification, ×400).

a calcium analog. Rashid *et al*. noted that Kayexalate in sorbitol given as an enema or orally to treat hyperkalaemia has been reported to induce intestinal necrosis in uraemic patients. They studied clinical and pathologic features of 15 patients and observed Kayexalate crystals in tissue specimens from surgical resections and endoscopic biopsies [4]. The mechanism of the necrosis and perforation is unknown. One possibility is elevated renin levels, commonly seen in renal insufficiency, that predispose the patient to non-occlusive mesenteric ischaemia via angiotensin-mediated vasoconstriction [5]. One gram of Kayexalate possesses a theoretical *in vitro* exchange capacity of 2–3.1 mEq of potassium and *in vivo* capacity of ~1 mEq [6]. Emmett *et al.* [7] reported that *in vivo* potassium-binding capacity may be lower than previously estimated, more on the order of 0.4–0.8 mEq/g of Kayexalate resin. In contrast to other minor digestive complications associated with Kayexalate treatment, colonic perforation results in significant morbidity and mortality.

As a result, potassium exchange resins may, although rarely, induce a colonic perforation, and this diagnosis should be considered in a patient treated as such in case of acute abdomen. The clinicians must be aware of the possible rare and serious complications of potassium exchange resins.

Conflict of interest statement. None declared.

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