Calcium polystyrene sulfonate-induced gastrointestinal tract necrosis and perforation

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To the Editor,

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Hyperkalaemia is defined as a high serum potassium level requiring urgent and specific treatments to prevent life-threatening complications. Cation-exchange resin, such as calcium polystyrene sulfonate (CPS), promotes potassium excretion in the intestinal/colonic lumen and is a treatment of choice when less aggressive treatment is allowed (1). We herein present three cases of CPS-induced gastrointestinal tract necrosis or perforation.

The patients were a 61-year-old man (Patient 1) and two women aged 74 and 89 years, respectively (Patients 2 and 3). All had multiple comorbidities such as chronic kidney disease or heart disease. Patients 1 and 2 were in a postoperative state. The CPS dosages were 60, 75, and 90 g/day orally for 2 to 3 days, with a total CPS dose of 210, 150, and 180 g without sorbitol, respectively. No patients had evidence of infection, vascular thrombosis, or other significant pre-existing causes of bowel necrosis/ perforation. No nonsteroidal anti-inflammatory and/or anticoagulant drugs had been prescribed within 3 weeks prior to the incidents. The surgical specimen from Patient 1 included hard, stony intestinal contents. The intestinal mucosa of all patients exhibited ischaemic changes, mucosal/transmural necrosis, and basophilic angulated crystalloid material with a fibrino-inflammatory process (Figure 1). Table 1 shows the details of each case.

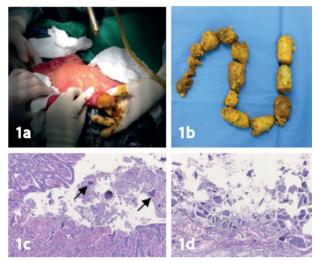


Figure 1. — Operative and pathological findings of the patients. (Pongsakorn Buraphat). **1a.** In Patient 1, an incision was made above the ileocecal valve. **1b.** In Patient 1, hard, stony contents were removed from the small bowel following resection of gangrenous intestine. **1c.** In Patient 2, the small intestinal mucosa showed multiple erosions with ischaemic changes and basophilic angulated crystals (black arrow) on the surface (haematoxylin-eosin stain; $10\times$). **1d.** In Patient 3, numerous basophilic angulated crystals with a fish scale appearance were observed adhering to the surface of the mucosa (haematoxylineosin stain; $10\times$).

Table 1. — Other details in this series

Parameters	Patient 1	Patient 2	Patient 3
First clinical presentation of the complication	Constipation for 2 days, abdominal pain, septic shock, respiratory failure	Sepsis, abdominal pain respiratory failure	Abdominal pain, constipation, septic shock, respiratory failure
Sorbitol co-prescription	No	No	No
Laxative prescription	Sennoside oral	No	Sennoside oral
Evidence of gut obstruction	Yes	No	Yes
Sites of perforation/ulcer	No perforation	Ileum, cecum	Sigmoid colon
Patient outcome	Died	Died	Discharged

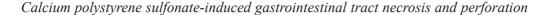
This report describes an unusual but fatal adverse event of CPS, which is commonly used in clinical practice. Although the definite mechanism of intestinal/colonic necrosis is unknown, several theories have been proposed, including an increasing inflammatory process due to comorbidities, resulting in compromised

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gastrointestinal mucosal integrity (2). The high water affinity of CPS to resins facilitates bulk formation with shear-thickening flow behaviour that leads to clumping and clogging of resin (3), especially in patients with poor gastrointestinal motility. This provokes resin impaction followed by gut obstruction, ischaemic necrosis, and perforation similar to findings in stercoral colonic perforation (4).

Although hyperkalaemia is life-threatening, correcting this condition by CPS might cause a fatal outcome. CPS is generally safe, but caution should be exercised when using it in high-risk patients, such as older patients and those with gastrointestinal hypomotility. Routine monitoring for early detection of complications is recommended.

Conflict of interest

None

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