



## Short communication

## Colonic necrosis and perforation following oral sodium polystyrene sulfonate (Resonium A<sup>®</sup>/Kayexalate<sup>®</sup>) in a burn patient

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### 1. Introduction

Colonic necrosis and perforation is rare in burn patients, however, it can lead to serious morbidity and mortality. Here we present a case of unexplained colonic necrosis and perforation in a 53-year-old woman with 25% total body surface area burns. No identifiable cause of bowel necrosis was found except for the presence of sodium polystyrene sulfonate (SPS) crystals in the inflammatory debris on histological examination. Colonic necrosis has been described in the literature following the administration of SPS in sorbitol for hyperkalaemia in uraemic patients. However, this condition has never been documented in any burn patients previously.

### 2. Case Report

A 53-year-old, 160 kg woman with 25% total body surface area burns was admitted to the Intensive Care Unit following self-immolation with methylated spirits. She sustained deep partial thickness burns to her face, chest and arms and full thickness burns to her neck. She was electively intubated at the time of initial presentation because of facial involvement. She underwent debridement and grafting surgery twice during her admission. Two separated doses of 15 g oral Resonium A<sup>®</sup> were prescribed on days 1 and 9 for the treatment of hyperkalaemia. Fifteen days following the last dose of Resonium A<sup>®</sup>, her clinical condition deteriorated suddenly. She became febrile, hypotensive, tachycardic, tachypnoeic, lactic acidotic with renal impairment. She vomited faeculent material and produced 1000 ml of non-bloody diarrhoea. Her abdomen was grossly distended and tender. Urgent abdominal X-ray revealed pneumoperi-

toneum and colonic distension. She underwent an urgent laparotomy. At laparotomy, 3 l of faeculent material was found within the peritoneal cavity. The splenic flexure was inflamed with a cobblestone appearance. The serosal surface was covered with fibrinous exudate. Focal areas of necrosis were identified. There was no evidence of vascular compromise to the affected colonic segments. The rest of the large and small bowel appeared normal. Re-section of the splenic flexure was performed with formation of a colostomy and mucous fistula. The peritoneum was washed out with a copious amount of normal saline. "Re-look" laparotomy on the second day revealed no sign of further colonic necrosis. Her recovery followed a stormy course and was complicated by gram-negative septicaemia. She eventually died with multi-organ failure.

Histological examination of the specimen showed multiple discrete areas of deep ulceration with intramural necrosis, abscess formation and focal transmural penetration. There was active chronic inflammation and granulation tissue associated with the ulcer bases. SPS crystals were present in the inflammatory debris. No changes consistent with ischaemia were identified. There was no evidence of vasculitis. In the context of colonic necrosis with no identifiable cause and the presence of SPS crystals, a diagnosis of SPS induced colonic necrosis was made.

### 3. Discussion

Colonic ulceration and necrosis in burn patients is rare and usually occurs in the caecum related to non-specific ulceration or acute colonic pseudo-obstruction which is more commonly known as Ogilvie syndrome [1]. Still et al. reported a case of caecal perforation secondary to non-specific ulceration in a patient with 26% TBSA burns [2]. Some colonic lesions in burn patients were found to be ischaemic

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71 in nature in a retrospective study by Desai et al. [3]. Our  
72 patient represents a case of colonic necrosis and subsequent  
73 perforation as a result of the administration of SPS. Such a  
74 case has not been described in burn patients previously in  
75 the literature.

76 One of the treatments of hyperkalaemia is the oral or rec-  
77 tal administration of a cation exchange resin, such as SPS  
78 (also known as Resonium A<sup>®</sup> or Kayexalate<sup>®</sup>). SPS binds  
79 with intraluminal calcium and can cause constipation, faecal  
80 impaction and subsequent bowel obstruction or perforation  
81 [4]. Sorbitol is frequently administered with SPS in order  
82 to reduce faecal impaction and subsequent bowel obstruc-  
83 tion [4]. The mixture of SPS in sorbitol has been reported  
84 as an aetiological agent of colonic necrosis [4–9]. Thus  
85 far the literature has only reported cases of SPS–sorbitol  
86 mixture and colonic necrosis in association with uraemic  
87 patients [4,5]. Lillemoe et al. experimented with the ef-  
88 fects of SPS–sorbitol enemas on uraemic and non-uraemic  
89 rats. Administration of SPS alone was not found to cause  
90 any significant pathological changes. It was concluded that  
91 sorbitol was the responsible agent and SPS only a con-  
92 founder [7]. Despite our patient being given SPS without  
93 sorbitol, pathological examination revealed similar pathol-  
94 ogy as described by Rashid and Hamilton. They examined  
95 colonic specimens from nine patients with SPS–sorbitol in-  
96 duced colonic necrosis. The involved segments were found  
97 to have mucosal ulceration, dusky mucosal coloration, mural  
98 oedema, serosal fibrinous exudate, mucosal or transmural  
99 necrosis and perforation with luminal SPS crystals. Till  
100 date there have been no studies performed with human sub-  
101 jects to verify Lillemoe's conclusions. Therefore, the po-  
102 tential of SPS only induced colonic necrosis should not be  
103 discarded.

104 The precise mechanism of colonic necrosis from the ad-  
105 ministration of SPS–sorbitol remains unknown. Contribut-  
106 ing factors including uraemia, hypovolemia, hypotension  
107 and immunosuppression have been described in renal trans-  
108 plant patients [9]. These factors are shared by the majority  
109 of severely burned patients. Furthermore, patients with  
110 massive burns have associated increased angiotensin II and  
111 vasopressin activity in the splanchnic circulation causing  
112 vasoconstriction and thus, ischaemia [3]. In this instance, the

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gastrointestinal mucosa is placed at increasing risk for injury 114  
from pharmacological agents such as SPS–sorbitol [8]. 115

#### 4. Conclusions 116

This case report illustrates that SPS could induce colonic 117  
necrosis and perforation in addition to the other gastroen- 118  
terological complications associated with burn patients. 119  
We believe that this condition is under-recognised in burn 120  
patients. Although the exact mechanism of SPS induced 121  
colonic necrosis remains unknown, the prescribing clinician 122  
should be aware of the possible serious adverse effects as- 123  
sociated with its administration. Clinical suspicion should 124  
remain vigilant. Early recognition and prompt management 125  
is imperative for a good clinical outcome. 126

#### References 127

- [1] Ghoneim IE, Bang RL. Caecal perforation in a burn patient. *Burns* 128  
1995;21:619–21. 129
- [2] Still Jr JM, Scheirer RC, Law EJ. Caecal perforation due to colonic 130  
ulcer in a burn patient. *Burns* 1994;20:85–6. 131
- [3] Desai MH, Herndon DN, Rutan RL, Abston S, Linares HA. Ischemic 132  
intestinal complications in patients with burns. *Surg Gynecol Obstet* 133  
1991;172(4):257–61. 134
- [4] Dardik A, Moesinger RC, Efron G, Barbul A, Harrison MG. Acute 135  
abdomen with colonic necrosis induced by Kayexalate<sup>®</sup>–sorbitol. 136  
*South Med J* 2000;93(5):511–3. 137
- [5] Wootton FT, Rhodes DF, Lee WM, Fitts CT. Colonic necrosis with 138  
Kayexalate<sup>®</sup>–sorbitol enemas after renal transplantation. *Ann Int Med* 139  
1989;111(11):947–9. 140
- [6] Scott TR, Graham SM, Schweitzer EJ, Bartlett ST. Colonic necrosis 141  
following sodium polystyrene sulfonate (Kayexalate<sup>®</sup>)–sorbitol enema 142  
in a renal transplant patient. *Dis Colon Rectum* 1993;36:607–9. 143
- [7] Lillemoe KD, Romolo JL, Hamilton SR, Pennington LR, Burdick 144  
JF, Williams GM. Intestinal necrosis due to sodium polystyrene 145  
(Kayexalate<sup>®</sup>) in sorbitol enemas: clinical and experimental support 146  
for the hypothesis. *Surgery* 1987;101:267–72. 147
- [8] Abraham SC, Bhagavan BS, Lee LA, Rashid A, Wu T. Upper 148  
gastrointestinal tract injury in patients receiving Kayexalate<sup>®</sup> (sodium 149  
polystyrene sulfonate) in sorbitol. *Am J Surg Pathol* 2001;25:637–44. 150
- [9] Rashid A, Hamilton SR. Necrosis of the gastrointestinal tract in uremic 151  
patients as a result of sodium polystyrene sulfonate (Kayexalate<sup>®</sup>) in 152  
sorbitol. *Am J Surg Pathol* 1997;21:60–9. 153