CASE REPORT

Pseudomembranous colitis secondary to methicillin-resistant *Staphylococcus aureus* (MRSA)

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SUMMARY

A 37-year-old woman with a history of type II diabetes and Crohn's disease, status postcholecystectomy, presented with a >2-week history of cramping abdominal pain, nausea, non-bloody/non-bilious emesis and, later, diarrhoea. A flexible sigmoidoscopy was performed, revealing that 'a segmental pseudomembrane was found from rectum to sigmoid colon'. *Clostridium difficile* PCR on the stool was repeated twice and resulted negative both times. A food history prior to onset of symptoms was consistent with *Staphylococcal* food poisoning and a stool culture was positive for heavy growth of methicillin-resistant *Staphylococcus aureus* and the absence of enteric flora. The patient was successfully treated with oral vancomycin.

BACKGROUND

Prior to the implication of a cytotoxin produced by *Clostridium difficile* in 1978,¹ *Staphylococcus aureus* was felt to be a major cause of antibiotic-associated enterocolitis, as well as enterocolitis in patients with predisposing factors who had not been exposed to antibiotics. Owing to the predominance of *C. difficile* as the cause of pseudomembranous colitis, the appreciation of *S. aureus* as a potential aetiology has declined and been disputed.

Other rare causes of pseudomembranous colitis include cytomegalovirus, *Shigella dysenteriae*, *Escherichia coli* O157:H7, *Klebsiella oxytoca* and ischaemic colitis.² Our patient, with evidence of pseudomembranous colitis on flexible sigmoidoscopy, stool culture demonstrating an absence of enteric flora with a heavy growth of methicillin-resistant *S. aureus* (MRSA) and a negative stool *C. difficile* PCR, confirms this underappreciated diagnosis.

CASE PRESENTATION

A 37-year-old woman with a history of type II diabetes mellitus and Crohn's disease, status posthysterectomy, status postcholecystectomy, presented to the emergency department, with cramping abdominal pain, nausea, non-bloody/non-bilious emesis and diarrhoea, which had become progressively worse over the preceding 7–10 days. The patient stated that her Crohn's disease had been stable without medication for over 6 months, and the cramping pain was not typical of a flare of Crohn's disease. She was evaluated, chemistry and haematology testing was performed, and a CT of the abdomen was obtained. Other than a blood sugar level of 198 mg/dL, other laboratory tests were within normal range. CT of the abdomen was

interpreted as 'pericolonic stranding and colonic wall thickening involving predominantly the transverse colon, suggesting colitis'. A stool for *C. difficile* PCR was negative. The patient was diagnosed with colitis and discharged from the emergency department with prescriptions for ciprofloxacin and metronidazole. As a result of her extreme nausea, she was unable to tolerate the antibiotics. Therefore, after five more days with no resolution of symptoms and only two doses of the prescribed antibiotics retained, she returned to the hospital and was admitted. A food history before the onset of symptoms disclosed that the patient's aunt had developed severe nausea, vomiting and diarrhoea concurrently with the onset of the patient's symptoms, but the relative's illness had resolved within 72 h. The patient reported having eaten two devilled eggs from a take-out box of food from the restaurant at which the aunt had eaten prior to the onset of her gastrointestinal (GI) illness. This was suggestive of *Staphylococcal* food poisoning. Flexible sigmoidoscopy revealing pseudomembranous colitis—and a stool culture with 3+ growth of MRSA—confirmed the diagnosis. The patient was successfully treated with 7 days of oral vancomycin, 125 mg every 6 h.

INVESTIGATIONS

- ▶ A flexible sigmoidoscopy was performed, revealing that 'a segmental pseudomembrane was found from rectum to sigmoid colon. An area of mildly congested mucosa was found from rectum to sigmoid colon' (figure 1).
- ▶ *C. difficile* PCR was repeated twice and found to be negative both times, a test with a very high negative predictive value of 99%.³
- ▶ Stool culture demonstrated an absence of enteric flora, and was positive for 3+ growth of MRSA.
- ▶ A stool test for ova and parasites was negative.

DIFFERENTIAL DIAGNOSIS

The differential in this case included common causes of acute infectious diarrhoea: *E. coli*, *Shigella*, *Salmonella* and *Campylobacter*. Given the presence of pseudomembranes on flexible sigmoidoscopy, *C. difficile* was considered, but a negative PCR test ruled it out. Other rare causes of pseudomembranous colitis include cytomegalovirus, *S. dysenteriae*, *E. coli* O157:H7, *K. oxytoca*, *S. aureus* and ischaemic colitis. Given the history of Crohn's disease, an acute exacerbation of Crohn's disease was also included in the differential diagnosis.

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Figure 1 Flexible sigmoidoscopy demonstrating pseudomembranous colitis.

Stool culture demonstrated an absence of enteric flora, and was positive for a heavy 3+ growth of MRSA. This was highly suggestive of pseudomembranous colitis secondary to MRSA.

TREATMENT

Since the *S. aureus* isolate was methicillin resistant, the patient was treated with 7 days of oral vancomycin, 125 mg every 6 h.

OUTCOME AND FOLLOW-UP

In a follow-up telephone call 2 weeks later, the patient reported resolution of her symptoms.

DISCUSSION

Prior to the implication of a cytotoxin produced by *C. difficile* in 1978,¹ *S. aureus* was felt to be a major cause of antibiotic-associated enterocolitis, as well as enterocolitis in patients with predisposing factors who had not been exposed to antibiotics. Owing to the predominance of *C. difficile* as the cause of pseudomembranous colitis, the appreciation of *S. aureus* as a potential aetiology has declined. Other rare causes of pseudomembranous colitis include cytomegalovirus, *S. dysenteriae*, *E. coli* O157:H7, *K. oxytoca*, *S. enterica*, *Plesiomonas* and ischaemic colitis.^{2–4} Medications and chemicals such as cisplatin, cyclosporine, 5-fluorouracil and gold have been implicated too.⁴

Sweetser and colleagues described a case of pseudomembranous colitis secondary to *K. oxytoca* in a patient presenting with explosive diarrhoea, and colitis with non-response to empiric treatment with metronidazole. A selective stool culture was positive for *K. oxytoca*. The patient responded after metronidazole was discontinued, reflective of ongoing disruption in the colonic microflora.²

Gastroenteritis associated with *Staphylococci* can occur from ingestion of the preformed toxin in food, or from ingestion of the organism, which then produces the toxin after ingestion.⁵

Studies of prevalence for *C. difficile* and *S. aureus* in stool samples have produced inconsistent results with rates being similar in some studies, but *C. difficile* being up to 60 times more prevalent in another investigation.⁶ *S. aureus* strains produce toxins responsible for toxic shock syndrome, enterotoxins that function as superantigens, *Staphylococcal* enterotoxin A and the bicomponent leucotoxin LukE-LukD. Each of these has been implicated in cases of *Staphylococcal* enterocolitis.^{5–7}

Staphylococcal enterocolitis has most frequently been described in patients with underlying disease of the colon, infants, the elderly, persons with immune compromising conditions affecting the T cells, in individuals colonised with MRSA and in patients with decreased gastric acid production, either medication-induced or due to a medical condition.^{7–8}

If endoscopy is performed, areas of patchy erythema, ulcerations and necrosis can be seen. *S. aureus* enterocolitis can present with pseudomembranes that can be loosely adherent, and located in the upper and lower GI tract.⁹ This differs from pseudomembranes seen in *C. difficile* infection, which are tightly adherent, well demarcated and typically located only in the colon and ileocecal valve.¹ Treatment with vancomycin is usually successful, but in severe cases with acute abdomen, bowel gangrene, toxic megacolon or perforation, surgery is required.^{8–10}

The patient described in this case study had previously been diagnosed with Crohn's disease. Her initial presentation was suggestive of preformed *Staphylococcal* toxin-related gastroenteritis. However, her persistence of symptoms and development of colitis suggest ingestion of the organism. Treatment with ciprofloxacin and metronidazole likely resulted in disruption of the colonic flora leading to selection and overgrowth of MRSA, which possibly led to pseudomembranous colitis.

Learning points

- ▶ The commonest cause of pseudomembranous colitis is *Clostridium difficile*.
- ▶ In cases of pseudomembranous colitis that test negative for *C. difficile*, diagnostics for other causes should be pursued along with a food and medication history prior to the onset of symptoms.
- ▶ Continued empiric treatment with antimicrobials in such cases may further disrupt the normal colonic microflora, resulting in prolongation of symptoms.

Contributors KBP completed the history-taking and examination, and initial manuscript write-up. KJS attended on the case and contributed to decision-making, management of the patient, literature review as well as editing of the manuscript. EH assisted with literature review.

Competing interests None declared.

Patient consent Obtained.

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