and a reporting bias due to increased hospitalizations for rotavirus-associated infections cannot be excluded. Recurrence rates in the paediatric population are around 25%, and mortality rates are low, with one death seen among 82 patients with CDI in one study.

We report a case of recurrent CDI in a 10-year-old child who was successfully treated with fidaxomicin, a newly approved macrocyclic antibiotic.

A 10-year-old patient presented with a history of five previous instances of CDI over 1 year, related to multiple use of antimicrobial therapy for recurrent pneumonia. He presented with a history of several days’ diarrhoea, lethargy and reduced intake. Physical exam findings were significant for dry skin with cold extremities and a soft, mildly distended abdomen upon palpation. On admission he had a fever of 101.3°F and a white blood cell count of $16 \times 10^9$/L with an elevated neutrophil count of 78%. His mother had initiated oral vancomycin therapy 2 days prior to admission with no significant improvement. He had a known history of chromosomal disorder, microcephaly, seizures and gastric tube (G tube) feeding. It is to be noted that G tube feeding can also contribute to diarrhoea. His previous episode of CDI was more than 10 weeks ago and he had finished his tapering 6 week course of oral vancomycin 2 weeks prior to his admission. His workup on admission showed positive faecal leukocytes, and a stool specimen was positive by PCR for C. difficile toxin. The stool specimen gave negative culture and test results for other bacterial pathogens, ova and parasites; blood cultures were also negative.

Owing to multiple recurrences of CDI following therapy with vancomycin he was started on 200 mg fidaxomicin twice daily, the tablet being crushed, mixed with water and given through a G tube. The patient’s diarrhoea improved within 24 h and he was discharged on the third day. He finished the 10 day course of fidaxomicin while at home and remained symptom free for his 1 month follow-up appointment. A few months later the patient developed pneumonia, which was treated with clarithromycin, and he subsequently developed another episode of CDI with a similar presentation to that seen in the prior episode. The patient was treated with fidaxomicin again and his symptoms resolved in 24 h.

Treatment of recurrent CDI in the paediatric population is often difficult owing to limited approved therapeutic options. Current options include metronidazole and vancomycin, which are associated with a similar recurrence rate (27.1% and 24.0%, respectively), while metronidazole has a higher rate of treatment failure (22.4% versus 14.2%). However, an alternative to vancomycin and metronidazole is now available in the form of fidaxomicin, a novel macrocyclic antibiotic approved in the USA in 2011 for use in adults. Fidaxomicin works by inhibiting DNA-dependent RNA polymerase and thus RNA synthesis. It has a $t_1/2$ of 11.7 h and an MIC range of 0.02–0.25 mg/L. Compared with vancomycin, fidaxomicin has a post-antibiotic effect of >24 h and has poor activity against normal Gram-negative and Gram-positive flora in the gut. Fidaxomicin also results in lower spore counts after treatment than vancomycin and is associated with less recurrence. The current disadvantage of fidaxomicin is its cost of use, although due to recent legislation, effective on 1 October 2012, Medicare set the maximum hospital add-on payment at S$868, which covers half of the average cost. The current recommended dose for adults is 200 mg twice daily and our patient was started on this regimen, despite his young age, in view of his past history of recurrence of CDI. The route of administration was via a G tube in view of negligible gastrointestinal absorption.

We believe this is the first reported case of the use of fidaxomicin for the treatment of CDI in a child in the literature.

We have obtained verbal consent for this case report from the patient’s guardian.

**Funding**

This study was carried out as part of our routine work.

**Transparency declarations**

None to declare.

**References**


**Elevated serum procalcitonin in anaphylaxis**

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Sir,

Procalcitonin (PCT) has been shown to be a promising diagnostic and prognostic marker in bacterial infection including sepsis, and PCT-guided antibiotic use has been proposed in some clinical settings. However, elevated PCT levels have also been reported in non-infectious conditions such as systemic trauma, burns, extensive surgery and severe pancreatitis. In addition, elevated serum levels of PCT have been reported in systemic autoimmune diseases.

Since patients with systemic autoimmune diseases are normally immunocompromised because of their treatment, for example with corticosteroids and immunosuppressant agents, it is essential to discriminate between infection and disease flare in febrile patients. Corticosteroids and some other immunosuppressive agents do not seem to suppress the PCT response, in contrast to C-reactive protein (CRP). Thus, PCT appears to have a greater diagnostic value than CRP for differentiating bacterial infection from a disease flare in febrile patients under these conditions. However, we report a case in which anaphylaxis was found to be the cause of an elevated serum PCT concentration during the treatment of a patient with a systemic autoimmune disease. The patient has provided permission to publish these features of his/her case, and the identity of the patient has been protected. We obtained informed consent from the patient on discharge.

The patient, who had been diagnosed with an active systemic autoimmune disease, was treated with 30 mg/day prednisolone. The patient was also given trimethoprim/sulfamethoxazole for prophylaxis against Pneumocystis pneumonia. Twelve days later, the patient had developed nausea, vomiting and high fever. The patient’s vital signs were as follows: body temperature, 38.9°C; blood pressure, 84/54 mm Hg; pulse rate, 116 beats/min; and peripheral oxygen saturation, 96% in room air. Breath sounds were normal and respiratory distress was not observed. The chest and abdominal radiographs demonstrated no significant findings.

Laboratory investigations revealed 8.42 mg/dL CRP, 10,730/mm³ white blood cells, 519×10⁴/mm³ red blood cells, 11.8×10⁴/mm³ platelets and 28.12 ng/mL PCT. Coagulation studies indicated a hypercoagulable state, such as elevated concentrations of fibrin/fibrinogen degradation products (260.8 μg/mL), D-dimer (124.0 μg/mL) and fibrinogen monomer complex (>150.0 μg/mL). Immunological studies suggested that the patient’s autoimmune disease was not in an acute flare-up phase.

Based on the presumptive diagnosis of sepsis, the patient was treated with the broad-spectrum antibiotic meropenem, although blood culture had not revealed any pathogen. The patient was maintained on 30 mg/day prednisolone intravenous-ly, but trimethoprim/sulfamethoxazole was discontinued. The symptoms resolved and laboratory investigations normalized in a week. Meropenem was stopped and trimethoprim/sulfamethoxazole recommenced, after which nausea, vomiting, hypotension and high fever immediately recurred, with 13.63 mg/dL CRP, 14,550/mm³ white blood cells and 14.28 ng/mL PCT. The diagnosis of anaphylaxis triggered by trimethoprim/sulfamethoxazole was made on the basis of clinical criteria.

In this case, the hyperthermia and hypotension along with elevated CRP and PCT levels on admission were suggestive of a diagnosis of septic shock. Furthermore, the presence of a hypercoagulable state along with thrombocytopenia suggested disseminated intravascular coagulopathy, which would be consistent with this diagnosis. However, anaphylaxis may also result in the consumption of coagulation factors, and this case was finally diagnosed as an anaphylactic reaction to trimethoprim/sulfamethoxazole after an unintended re-treatment. High serum levels of PCT have not been reported in anaphylaxis until now.

In anaphylaxis, skin and respiratory tract involvement are reported at a frequency of 80%–90% and 70%, respectively, neither of which was evident in this case. Instead, involvement of the gastrointestinal tract and cardiovascular system, which is observed in fewer than half of cases, were the main manifestations. We postulate that the daily use of 30 mg of prednisolone for the treatment of a systemic autoimmune disease may have attenuated the symptoms of anaphylaxis.

Although many mechanisms have been proposed to induce the release of PCT, proinflammatory cytokines, such as tumour necrosis factor-α, interleukin-6 and interleukin-1, appear to play important roles. Tumour necrosis factor-α and interleukin-6 are also elevated in anaphylaxis. Such a cytokine storm in anaphylaxis might have mediated PCT elevation in this case.

PCT is primarily used as a surrogate biomarker of bacterial infections. However, we report here a case of marked elevation of PCT levels that was induced by drug anaphylaxis. Based on our experience, anaphylaxis should be considered as a potential differential diagnosis for PCT elevation in the absence of severe bacterial infection.

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References