CASE REPORT

Escherichia coli 0157 enterohaemorrhagic colitis associated with pyelonephritis: CT findings

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ABSTRACT. Escherichia coli 0157:H7 is increasingly being recognized as a cause of infectious colitis, which typically results in bloody diarrhoea in an afebrile patient. The absence of fever often means that an infectious process is not considered in the differential diagnosis, particularly as this organism will not be detected in routine stool cultures. Inappropriate antibiotic therapy may increase the risk of development of haemolytic uraemic syndrome, a potentially fatal complication of this form of colitis, hence the importance of accurate diagnosis. On CT, it is characterized by severe diffuse colonic wall thickening, with little or no pericolic inflammatory changes. The radiologist may be the first to suspect the correct diagnosis and so should be aware of its imaging appearances. We report the case of a 19-year-old man who presented with typical radiological findings of enterohaemorrhagic colitis and whose CT also showed evidence of acute pyelonephritis; we suggest that this combination of abnormalities should further heighten radiologists’ suspicions of infection due to E. coli 0157:H7, despite the absence of fever.

Enterohaemorrhagic colitis due to Escherichia coli 0157:H7 typically results in bloody diarrhoea in an afebrile patient; if unrecognized and inappropriately treated, it can be fatal. A specific culture medium is required for growth, and this organism is not detected on routine stool cultures. Although the CT appearances of this condition are non-specific, the radiologist may be the first to suggest the diagnosis based on typical imaging findings occurring in the correct clinical setting.

Case report

A previously healthy 19-year-old man presented to the emergency department with a 3-day history of severe central abdominal pain, nausea, vomiting and diarrhoea. On the day of presentation, the diarrhoea had become bloody and more profuse. The patient denied any recent weight loss, had not travelled abroad recently, and had no recent history of antibiotic use. He reported having eaten some frozen lasagne 3 days previously, and becoming nauseated shortly afterwards.

The patient was apyrexial at the time of presentation. His abdomen was mildly tender to palpation but clinical examination revealed no other abnormality. His white cell count was slightly elevated at 11.7 × 10⁹ l⁻¹; however, his complete blood count was otherwise normal. Liver and renal function tests were also within normal limits.

CT of the abdomen was performed and showed diffuse circumferential thickening of up to 17 mm in the colonic wall, extending from the caecum to the rectum, with avid enhancement of the colonic mucosa (Figure 1). There was minimal pericolic fat-stranding (confined to the left side of the abdomen), and no free intraperitoneal fluid was demonstrated. The terminal ileum appeared normal, and no lymphadenopathy was evident. An additional CT finding was the presence of multiple wedge-shaped low attenuation areas in both kidneys (Figure 2). Endoscopy showed a diffusely oedematous colon with hyperaemic mucosa (Figure 3).

The differential diagnosis at this point included inflammatory bowel disease and infectious colitis. The renal abnormalities raised the possibility of concomitant pyelonephritis. Empiric antibiotic therapy was commenced using co-trimoxazole. Corticosteroid therapy was also initiated.

Although the patient’s blood and urine cultures were negative, a faecal sample (obtained for culture prior to the administration of antibiotics) was positive for E. coli serotype 0157. In view of this, the antibiotic and corticosteroid therapy was discontinued. With supportive management, comprising intravenous fluids and bowel rest, the patient’s symptoms gradually resolved and he was discharged home 4 days following admission. A follow-up CT performed 6 weeks later showed complete resolution of the colonic and renal abnormalities.

Discussion

Enterohaemorrhagic colitis associated with the E. coli serotype 0157:H7 was first reported in 1983 following an
outbreak of over 700 cases of the illness linked to consumption of undercooked hamburgers from a chain of fast-food restaurants [1]. With increased recognition and the development of commercially available testing, it is now estimated that this serotype is responsible for over 20,000 infections and 250 deaths each year in the USA [2]. The majority of cases result from the consumption of undercooked contaminated beef, usually ground beef. E. coli 0157:H7 is present in the gastrointestinal tracts of approximately 1% of healthy cattle [2].

Infection has also been reported following the consumption of contaminated spinach, lettuce and bean sprouts, and direct person-to-person transmission can also occur [3]. The highest rate of infection is seen in children younger than 5 years of age [4]. Only a very small number of surviving bacteria are required to produce symptomatic infection [5]. The typical incubation period before the onset of symptoms is 3–4 days, but can occur within 1 day or as long as 8 days after ingestion of contaminated food [2].

E. coli 0157:H7 colitis is associated with a spectrum of clinical manifestations, including non-bloody diarrhoea, bloody diarrhoea, acute abdomen, haemolytic uraemic syndrome (HUS), thrombocytocytic thrombocytopenic purpura and death [6]. Unlike most bacterial enteric diseases, it characteristically results in an afebrile illness [5, 7]; consequently, non-infectious aetiologies are usually considered more likely by the treating clinician.

Figure 1. Coronal and transverse CT images obtained following intravenous and oral contrast administration show marked diffuse colonic wall thickening (arrows) with avid mucosal enhancement. There is minimal pericolic fat stranding confined to the descending colon (curved arrows).

Figure 2. Coronal and coned transverse images show multiple wedge-shaped striated areas of low density in both kidneys (arrowheads), appearances that are typical of pyelonephritis.
Typically, patients initially complain of abdominal cramps and watery diarrhoea that is succeeded, on average 48 h later, by the onset of bloody diarrhoea [4]. This persists for a median of 4 days, but ongoing haemorrhage for up to 22 days has been described [5]. Nausea and vomiting may be present in up to 50% of cases [4].

In addition to the absence of pyrexia, the condition may manifest in several other features that suggest a non-infectious aetiology. Abdominal pain and tenderness may be severe, suggesting a surgical emergency. The volume of bloody diarrhoea can be large, and there are often relatively few faecal leucocytes. E. coli 0157:H7 is not identified on routine stool cultures; sorbitol-MacConkey agar is required for colony growth, after which a commercially available antiserum will confirm the serotype. If this test is not specifically requested by the treating physician, the diagnosis may be delayed or missed. Clinically, the differential diagnosis often includes intussusception in children, inflammatory bowel disease in adults, and ischaemic colitis in the elderly. Other infectious colitides, caused by organisms such as Shigella and Campylobacter, may also be considered in the differential diagnosis, as may pseudomembranous colitis, diverticulitis and appendicitis, depending upon the site of abdominal pain [4].

The pathogenesis of colonic injury in this condition is secondary to a group of toxins elaborated by E. coli 0157:H7, known as verotoxins or Shiga-like toxins [8]. The bacteria adhere to the colonic mucosa, and the subsequent toxin release causes microvascular ischaemia. As a result, the imaging appearances are those of colonic ischaemia: plain radiographs may show thumbprinting, whereas CT demonstrates low-density thickening of the colonic wall owing to oedema [9]. Although these changes can involve the entire colon, as in the case presented here, several papers have reported a right-sided predominance, which may be caused by relative faecal stasis in the caecum and ascending colon or to higher concentrations of bile salts at this site [7, 10]. Colonic involvement is usually contiguous, without skip lesions [5]. Pericolic inflammatory changes have been described, as has mesenteric lymphadenopathy [4, 11]; however, these findings are more typically absent [9].

The degree of wall thickening is usually more severe than that seen with other infectious colitides such as Salmonella or Campylobacter [7].

With the increasingly widespread use of CT for the evaluation of patients with abdominal pain, despite its non-specific imaging appearances, radiologists should consider colitis due to E. coli 0157:H7 in their differential diagnoses for diseases causing severe bowel wall thickening, with absent or relatively mild pericolic changes, in patients with acute abdominal pain and bloody diarrhoea.

The majority of patients recover fully in 5–10 days without specific treatment [3], with standard care consisting of fluid resuscitation and bowel rest [4]. However, in a small proportion of cases (2–7%), HUS may develop [5]. HUS results in a microangiopathic haemolytic anaemia, thrombocytopenia and, in severe cases, renal failure and central nervous system injury, and usually occurs within 14 days of the onset of diarrhoea [2]. HUS develops more commonly in patients with bloody diarrhoea; it is postulated that the loss of intestinal integrity results in systemic exposure to verotoxins, bacterial lipopolysaccharides and inflammatory mediators, precipitating HUS [4]. This potentially fatal complication is more common in children and in the elderly [5], and appears to be more frequent in patients who have been inappropriately treated with antibiotics or antimotility agents [3, 10], hence the importance of early accurate diagnosis of E. coli 0157:H7 colitis. Steroid therapy, however, has no beneficial or detrimental effect on the clinical course or outcome [10].

Although unusual, extraintestinal isolation of the bacterium has been reported. E. coli 0157:H7 cystitis has been described [12]. Positive urine culture in a patient with diarrhoea but no urinary symptoms has also been reported, as has positive blood culture in an elderly patient who subsequently died [6]. In the case presented here, CT also showed ill-defined wedge-shaped areas of low attenuation — typical appearances of acute pyelonephritis [13]. Although our patient’s urine culture was negative, it appears likely that there was haematogenous seeding of the kidneys by E. coli, resulting in pyelonephritis. Although urinary tract infection by other enteropathogenic organisms such as Salmonella enteritidis has been reported, it is very rare, usually occurs in patients with diabetes mellitus, immunosuppression or anatomic urinary tract abnormalities, presents as cystitis rather than pyelonephritis, and is not typically associated with concomitant gastrointestinal symptoms [14, 15]. Consequently, we suggest that, in the correct clinical setting, the combined CT findings of severe colonic wall thickening and wedge-shaped hypoattenuating renal lesions should make the radiologist highly suspicious of E. coli 0157:H7 colitis.

References


