Case Report

Case report: enteroaggregative and enteroinvasive *Escherichia coli* as a cause of acute abdomen

Abstract

Abdominal pain stands as one of the foremost reasons for consultation among pediatric patients, presenting a diagnostic challenge owing to its diverse underlying causes. The manifestation of abdominal pain varies according to age, associated symptoms, and pain localization. While frequently self-limited, certain conditions exist that endanger life and require urgent intervention. Acute abdomen denotes severe, non-traumatic abdominal pain resulting from inflammatory, ischemic, obstructive, infectious, gynecological, or metabolic etiologies, warranting immediate therapeutic intervention. Infectious processes that mimic acute abdominal conditions are relatively uncommon. Consequently, the identification of infectious gastroenteritis as a probable etiology of acute abdomen can prevent unnecessary surgical interventions in patients. This report details two cases: a 14-year-old pediatric patient presenting with acute abdominal pain, in whom appendiceal involvement was ruled out through ultrasonographic and computed tomography examinations, confirming the presence of enteroaggregative *Escherichia coli*; and a 10-year-old pediatric patient presenting with a sudden onset of abdominal pain. Computed tomography findings revealed an appendiceal fecalith without concurrent inflammation but accompanied by mesenteric adenitis. Despite conservative management without pain improvement, it was subsequently established that the patient was a carrier of enteroinvasive *Escherichia coli.* In both cases, antimicrobial treatment with rifaximin 200 mg every 8 hours is administered, leading to the resolution of the conditions without the need for hospital readmission or additional therapy.

Conclusion: Infectious conditions stemming from enteroaggregative and enteroinvasive *E. coli* can mimic acute abdomen and should be regarded as potential infectious etiologies when other more common causes have been ruled out.

Keywords: abdominal pain, acute abdomen, enteroaggregative *Escherichia coli*, enteroinvasive *Escherichia coli*

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Introduction

It has been described that strains of *Escherichia coli* causing diarrhea in humans are classified into five pathotypes: enteropathogenic *E. coli* (EPEC), enterotoxigenic *E. coli* (ETEC), enteroinvasive *E. coli* (EIEC), enteroaggregative *E. coli* (EAEC), and Shiga toxin/verotoxin-producing or enterohemorrhagic *E. coli* (STEC/VTEC/EHEC).[1,2] EAEC has been identified as a cause of diarrhea in children living in developing countries, particularly in cases of persistent diarrhea lasting more than 14 days. It stands as one of the primary contributors to morbidity and mortality in this context. Furthermore, it has been associated as a foodborne pathogen responsible for sporadic diarrhea in healthy adults and children in developed countries.[3] Its pathogenesis involves a proposed model in which the bacteria produce enterotoxins and mucosa-adhering cytotoxins, leading to a self-limiting watery diarrhea lacking mucus or blood, while concurrently inflicting damage to the intestinal mucosa.[1,3–5]

On the other hand, EIEC has been identified as an invasive strain causing dysentery-like diarrhea. It has been reported endemically in developing countries, accounting for a frequency of 1 to 5% of diarrhea episodes in the population. Its mechanism of pathogenicity involves the production of enterotoxins and mucosa-adhering cytotoxins, followed by colonization and invasion of colonic cells, subsequently leading to their destruction, and triggering an inflammatory process. This cascade results in self-limiting watery diarrhea, accompanied by the presence of mucus and blood.[6] It is uncommon for infectious processes to manifest as acute abdomen symptoms. Infections caused by enteroaggregative *Escherichia coli* (EAEC) and enteroinvasive *Escherichia coli* (EIEC) typically manifest with watery diarrhea rather than severe abdominal pain.[3,4,7] Abdominal pain is reported as an infrequent symptom, though when present, it can become the prominent symptom, mimicking an acute abdomen presentation.[4,8]

Cases of acute abdomen involving infectious processes are exceedingly rare; specific data in the literature are lacking due to their infrequent occurrence. Therefore, particular attention should be given to these cases in order to prevent unnecessary surgical procedures.[4] Gastroenteritis stands as the most common non-surgical cause of abdominal pain, whereas appendicitis ranks as the most frequent surgical condition.[8] In this article, we present two cases of challenging acute abdomen scenarios stemming from infections caused by enteroaggregative *E. coli* and enteroinvasive *E. coli.*

Case presentation

Case 1

14-year-old, female patient, with no significant medical history. Presented to the emergency department with a two-day history of abdominal pain, nausea, and reduced appetite. The patient was afebrile and had no history of vomiting or chills. Upon questioning, she described acute abdominal pain initially localized in the mesogastrium that migrated to the right iliac fossa, with an intensity of 8/10. The pain was constant, radiating to the back, and had a sudden onset. Abdominal distension and decreased consistency of fecal matter were also observed, with no mucus or blood. The patient and her family denied recent travel or consumption of food outside their home.

Upon admission, the patient exhibited stable vital signs. On physical examination, she manifested abdominal pain upon palpation, primarily in the right iliac fossa, with a positive rebound sign, positive McBurney´s sign, positive psoas sign, and positive percussion tenderness. There were no visceromegaly findings, and percussion resulted in generalized tympanic resonance with decreased peristalsis. The rest of the physical examination yielded no relevant data. An initial abdominal X-ray showed nonspecific findings, prompting a decision to perform an abdominal ultrasound (Figure 1), which reported mesenteric adenitis with the presence of free abdominal fluid and an indistinctly visualized appendix. An abdominal CT scan was also conducted (Figure 2), revealing mesenteric adenitis without significant changes. Management was initiated with intravenous hydration, fasting, and analgesia. The following laboratory tests were conducted: complete blood count revealing a hemoglobin level of 16.1 g/dL, hematocrit of 46.6%, platelet count of 257,000, white blood cells count of 8,000, neutrophils at 46% (3,680), lymphocytes at 47% (3,760), and monocytes at 4% (320). A general urine exam showed a pH of 6.5, specific gravity of 1.005, and no other abnormalities detected.

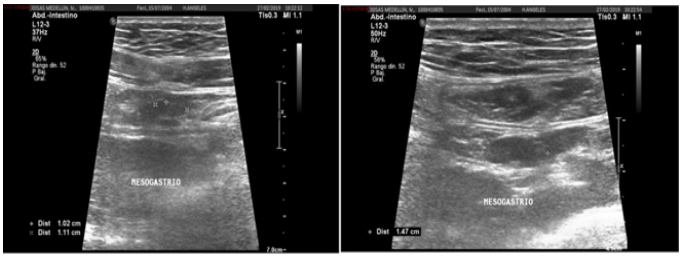

Figure 1 Abdominal ultrasound.

(a) Several lymph nodes with an inflamed appearance are observed.

(b) An inflamed lymph node measuring 14 mm, suggestive of mesenteric adenitis, and a small amount of free fluid in the right iliac fossa are noted.

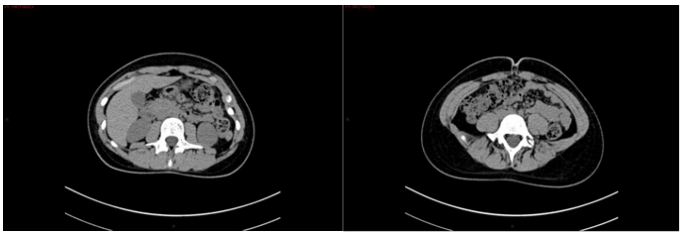

Figure 2 Axial abdominal CT scan.

(a) Free fluid is observed in the posterior cul-de-sac. Small umbilical hernia.

(b) Findings consistent with mesenteric adenitis.

After 24 hours of the initial evaluation, oral intake was initiated, but it was poorly accepted and tolerated. This led to nausea and an episode of emesis, accompanied by increased pain in the right iliac fossa, reaching an intensity of 10/10 with similar characteristics as before. The patient also experienced decreased fecal consistency. As a result, a fecal sample was collected for gastrointestinal pathogens panel using PCR, which yielded a positive result for enteroaggregative *E. coli.* Antimicrobial treatment was initiated with rifaximin at a dose of 200 mg every 8 hours. Following this, the patient showed improvement, characterized by a reduction in abdominal pain until it disappeared completely, while also exhibiting proper tolerance and acceptance of oral intake. A follow-up hematological analysis was contacted, revealing a hemoglobin concentration of 14.7 g/dL, a hematocrit value of 44%, a platelet count of 245,000, a total leukocyte count of 6,700, neutrophils accounting for 53% (3,550), lymphocytes at 41% (2,750) and monocytes at 4% (270). Due to the clinical improvement, the patient was subsequently discharged home and showed signs of favorable recovery without requiring readmission or further treatment.

Case 2

A 10-year-old male patient presents with an episode of intense and sudden abdominal pain, prompting him to seek outpatient medical attention. At the time of presentation, the patient was afebrile and displayed no history of vomiting. Directed questioning revealed that the patient had experienced an acute respiratory episode characterized by nasal congestion. Additionally, the patient described acute pain localized predominantly in the right iliac fossa, with increasing intensity. Notably, there were no associated symptoms reported. Both the patient and his family denied recent travel or consumption of food outside the home.

Upon admission, vital signs were within normal ranges. On upper airway examination, a hyperemic pharynx without exudate was noted. Clinically, there were no signs of peritoneal irritation, with the patient solely experiencing abdominal pain upon superficial and deep palpation in the right iliac fossa. The remainder of the physical examination did not yield any pertinent findings. Due to the escalation in pain intensity, a decision was made to perform an abdominal CT scan (Figures 3 & 4), revealing a retrocecal appendix the presence of an appendicolith within, but lacking signs of inflammation. Multiple peritoneal lymph nodes were detected, leading to the diagnosis of mesenteric adenitis, and ruling out acute appendicitis. Surgical intervention was deemed unnecessary. Based on the findings from the tomographic study, the need for surgical management due to appendiceal inflammation is ruled out, confirming the diagnosis of mesenteric adenitis. Expectant management is initiated with analgesia for pain control. However, due to inadequate response and the persistence of moderate-intensity pain, a fecal sample is collected for the performance of a gastrointestinal pathogens panel using PCR, which yielded a positive result for enteroinvasive *E. coli.*

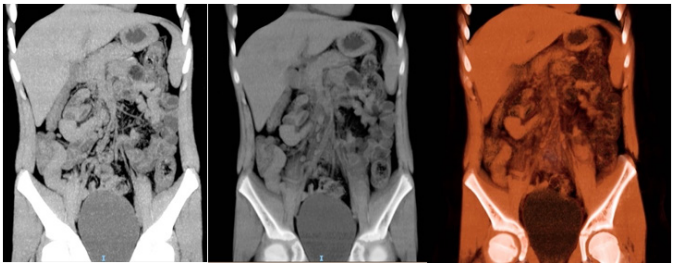

Figure 3 Coronally-sectioned abdominal CT scan.

(a, b) Findings consistent with mesenteric adenitis. Multiple oval-shaped images correspond to inflamed mesenteric lymph nodes. More than five mesenteric lymph nodes are observed with short axes exceeding 10 mm.

(c) Urinary bladder with a volume of 326 cc.

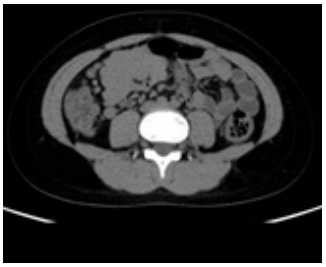

Figure 4 Axial abdominal CT scan.

(a) Retrocecal appendix of increased density, with a maximum transverse diameter up to 4.4 mm at the base, without peripheral inflammatory changes, and with the presence of a small appendicolith at the tip measuring more than 2 mm in diameter.

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Antimicrobial therapy was initiated with rifaximin at a dosage of 200 mg every 8 hours. Following this intervention, the patient exhibited improvement, characterized by a reduction in abdominal pain leading to its eventual resolution. The patient displayed commendable tolerance and adherence to oral food administration. Owing to the marked clinical improvement, the patient was discharged. The observed trajectory of recovery was favorable, resulting in no need for hospital admission or further treatment.

Discussion

The differential diagnosis of acute abdomen includes a range of medical and surgical conditions. Physicians are required to conduct a thorough clinical history and physical examination to minimize unnecessary surgical procedures. In both our cases, despite the presentation mimicking a surgical abdomen, immediate surgical pathology was initially ruled out through imaging studies, biochemical assessments, and clinical evaluation.[4] Enteroaggregative *Escherichia coli* (EAEC) is an emerging type of *E. coli* that is increasingly recognized as a causative agent of traveler´s diarrhea, diarrhea in children in developing countries, and persistent diarrhea in patients with human immunodeficiency virus (HIV).[4]

Irritable bowel syndrome (IBS) can mimic acute abdomen. In a study conducted in Poland in 2006, it was demonstrated that enteroaggregative *E. coli* (EAEC) was more prevalent in patients with IBS, with EAEC being present in 81.8% of the patients.[9] A study conducted in Germany in 1997, involving a pediatric population under 16 years of age with a sample size of 798 patients, demonstrated the presence of Enteroaggregative *Escherichia coli* in fecal samples using PCR in 16 children out of which five presented acute abdomen symptoms as their main clinical manifestation.[10]

In 1944, strains of *E. coli* capable of invading the colonic mucosa in a manner similar to Shigella were identified and termed Enteroinvasive *E. coli* (EIEC), exhibiting significant genetic similarities. Despite the new insights gained into the physiology and pathogenic mechanisms, as well as advancements in antibiotic management, infectious gastroenteritis caused by these bacteria persist as one of the top four caused of diarrheal diseases in children under five years of age in developing countries.[11] Given that both patients presented with intense abdominal pain and lacked other risk factors for *E. coli* infection, suspicion of such an infection was initially quite low. The presence of reduced stool consistency played a pivotal role in raising suspicion of an infectious pathology. It is imperative for both clinicians and surgeons to consider the possibility of an infectious etiology in patients presenting with abdominal pain and diminished fecal consistency. This approach is crucial in order to avoid unnecessary surgeries in patients who could benefit from antibiotic treatment.

Considering that many infections caused by EAEC and EIEC can be asymptomatic or self-limiting, the foundational approach to treatment is intravenous hydration. Nevertheless, for symptomatic patients, ciprofloxacin 500 mg twice a day for seven days or rifaximin 200-400 mg twice a day for three days con be administered. This therapeutic regimen has been shown to curtail the duration of diarrheal symptoms.[4,12] While these strains of *E. coli* are well-recognized causes of diarrhea, it´s essential to note that they can also lead to mesenteric adenitis and abdominal pain, thus mimicking a presentation of acute abdomen. Given the significance of establishing the etiology in our patients, it is advisable to consider requesting an infectious panel using PCR on stool samples, which can identify the pathogens responsible for the clinical presentation. Following the initiation of targeted treatment, rapid improvement was observed.

Conclusion

The etiological consideration of infectious origins should invariably be entertained when evaluating patients who present with abdominal pain suggestive of acute abdomen. Notably, pathogens such as Enteroaggregative *E. coli* and Enteroinvasive *E. coli* have demonstrated proclivity to induce mesenteric adenitis, thereby warranting thorough consideration as potential etiologic agents responsible for abdominal pain, which may simulate an acute abdomen presentation. This approach helps prevent unnecessary surgical interventions. Moreover, the administration of pharmacotherapeutic regimens involving specific antibiotics has proven capable to result in a reduction in the temporal duration of hospitalization, concomitantly with a discernible improvement in the symptomatic profile of afflicted patients.

Acknowledgments

None.

Conflicts of interest

The authors declare that there are no conflicts of interest.

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