

Case Report

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Acute appendicitis related to *Enterobius vermicularis* infestation in a geriatric patient: a case report and literature review

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ABSTRACT

Acute appendicitis (AA) is a common surgical emergency with a multifactorial aetiology, rarely associated with intestinal parasites. This report highlights the case of a 93-year-old female who developed AA in association with *Enterobius vermicularis* (EV), a rare finding in this age group. The patient underwent successful laparoscopic appendectomy followed by antiparasitic therapy, with a favourable outcome. Parasitosis may mimic typical AA or represent a coincidental finding. This case emphasizes the importance of considering parasitosis in the differential diagnosis of AA, especially in atypical presentations or when contemplating non-surgical management. Laparoscopic appendectomy with antiparasitic treatment remains the standard of care.

Keywords: *Enterobius vermicularis*, Acute appendicitis, Appendectomy, Pinworms

INTRODUCTION

Acute appendicitis (AA) is a common cause of emergency abdominal surgery, mainly affecting young adults.¹⁻⁵ It has a multifactorial aetiology and intestinal parasites like *Enterobius vermicularis* (EV) have rarely been implicated.^{1,2,4} The exact role of EV in AA is unclear, as it may mimic appendicitis symptoms without causing true inflammation. This clinical case highlights an unusual presentation of AA in a geriatric patient with an underlying parasitic infestation.

CASE REPORT

A 93-year-old female patient, independent for activities of daily living, presented to the emergency department with a one-day history of abdominal pain and vomiting. The patient denied other symptoms, such as fever or urinary symptoms. Past medical history included dyslipidemia and arterial hypertension. She was medicated with trifusal 300 mg, sertraline 50 mg, atorvastatin 10 mg and perindopril 5 mg. She had no known drug allergies.

At physical examination, the patient was alert, oriented, hydrated, had normal vital signs and presented a slight cutaneous pallor. Abdomen was tender with abdominal pain and guarding in the right iliac fossa. No masses were noted. Complementary studies were performed.

Laboratory tests documented a slight elevation of CPR (1.02 mg/dl) with no other alterations. Computed tomography (CT) scan showed elongated ileocecal appendix with thickened walls, suggesting AA (Figure 1).

The patient underwent emergency surgery under general anaesthesia. Laparoscopy was performed, revealing an inflamed cecal appendix without any purulent fluid or contamination. A cluster of eggs was found after excision of the appendix. Surgery was uneventful and post-operative clinical evolution was favourable.

Pathology reported “acute suppurative appendicitis with periappendicitis and associated peritonitis triggered by parasitic colonization with *Enterobius vermicularis*”, live

parasites were found in the lumen of the appendix (Figures 2 and 3).

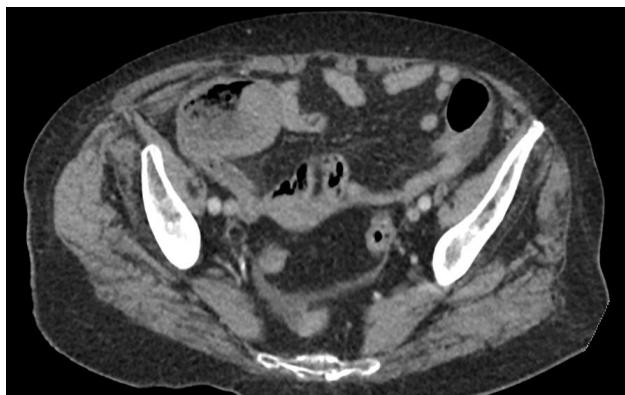


Figure 1: CT scan showing elongated ileocecal appendix with thickened walls.

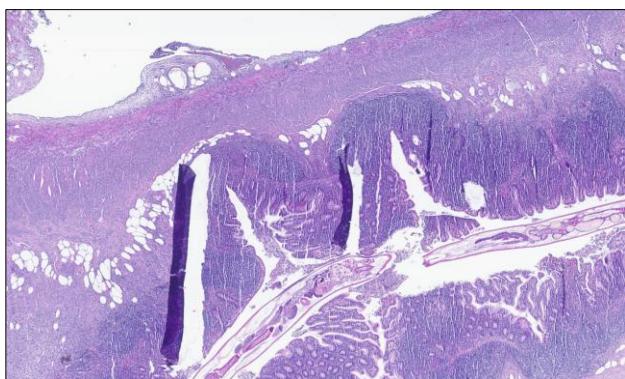


Figure 2: Parasites in the lumen of the appendix.



Figure 3: Detail of the parasite's lateral spur.

She completed treatment with albendazol. No complications or symptoms were reported in six weeks of follow-up.

DISCUSSION

AA is one of the most common causes for emergency abdominal surgery worldwide with an incidence around

8%, mostly in the second and third decades of life.¹⁻⁵ Its diagnosis is clinical. While the majority of cases are treated with laparoscopic appendectomy, currently it is acceptable to use only antibiotics to treat uncomplicated AA.^{1,4,6}

Regarding the pathophysiology of the disease, the main triggering factor is the obstruction of the appendiceal lumen, resulting in increased intraluminal pressure, bacterial overgrowth, distension and inflammation. The aetiology of AA is wide. The most common causes are fecal stasis, fecalites and lymphoid hyperplasia and among the rarest are tumours and intestinal parasites.^{1,2,4}

Regarding intestinal parasites, those most commonly linked to AA include EV, *Schistosoma* species, *Trichuris trichura*, *Taenia* species and *Ascaris lumbricoides*.^{1,2,6}

EV, also known as pinworm or *Oxyuris vermicularis*, is one of the most common nematode infections worldwide, both in low and high-income countries, particularly among children and institutionalized individuals.^{1,3,4,6} Infection in children can range from 4% to 38% in certain regions.^{1,7} Risk factors include age, children playing habits, previous infections, family size, overcrowding, lack of piped water, and incomplete parental literacy.⁵

This parasite is identified in the gastrointestinal system, most frequently in the ileum and cecum.^{2,4,5} It is only transferable by humans via the fecal-oral route, with infection acquired through the ingestion or, less commonly, inhalation of eggs from contaminated food, water, dust, or surfaces (viable for two to three weeks).^{1,5,7}

It's a self-limiting disease due to the short life cycle of the parasite, which takes about 2 to 6 weeks. Female worms deposit eggs in the perianal area, mostly at night, leading to the characteristic symptom of nocturnal perianal pruritus.^{1,3,5,8} Scratching due to pruritus can contaminate the fingers or under the fingernails causing autoinfection and consequently increase the length of the disease.^{1,3,8}

EV infection is often asymptomatic (about 40% of individuals). Symptomatic disease (enterobiosis or oxyuriasis) is generally mild, but a heavy worm burden can cause abdominal discomfort, nausea/vomiting and less commonly extra-intestinal organ involvement such as enterocutaneous fistula, urinary tract infection, mesenteric abscesses/granulomas, vulvovaginitis and salpingitis.^{1,3,7,9} Scratching can lead to skin lesions and secondary bacterial infections.^{1,8}

Diagnosis of enterobiosis is typically based on a history of intermittent perianal pruritus and can be confirmed by identifying eggs using the tape test (adhesive cellophane swab) or by direct visualization of worms in the stool or on the perianal skin.¹

Due to its possible location in the lumen of the appendix (first reported by Fabrius in 1634), EV has been linked to

AA. Its role in the pathogenesis of AA remains controversial and is the subject of ongoing debate in the medical literature.^{1,2,4,5}

EV was detected in 0.5% to 4.2% of appendectomy specimens, with some studies finding rates as high as 8%.^{2,4,6}

A literature review conducted by Laor identified four main parasitic infections associated with AA. They differentiated cases where EV infection mimics an AA (patient who clinically was diagnosed with AA but had a negative appendectomy) and cases where the author suggested that the EV infection was the cause of the AA. There were some indeterminate cases where the patient with AA is also positive for parasitic infection but the author does not conclude on a causal relationship. EV was mostly identified as a mimicker of AA.⁶ They conclude that an association between EV and AA in fact exists however, the causal relationship between EV and true AA is not definitively established.^{1,6} EV may be the causative agent of AA or an incidental finding.

The mechanisms proposed for EV-associated symptoms

Mechanical obstruction of the appendiceal lumen by adult worms or eggs, leading to increased intraluminal pressure, mucus accumulation, and secondary bacterial overgrowth promoting a true AA.^{1,3,5,7,10,11}

Appendiceal colic, where the presence of the parasite causes contraction and pain without true inflammation (mimicking symptoms of AA).^{3,5,7}

Direct mucosal irritation or hypersensitivity reactions, sometimes resulting in lymphoid hyperplasia or mild, chronic inflammatory infiltrates (mimicking symptoms of AA).³

Most cases described in literature are in the paediatric population and the majority of appendices containing EV do not show acute inflammation on histopathology, only lymphoid hyperplasia.^{1,2,4,7,10}

The importance of considering the possibility of a parasitic infection in patients presenting with symptoms of AA is especially relevant in cases where conservative treatment with antibiotics might be considered. In these patients, there is a risk of disease progression if antiparasitic agents are not administered, as it would not be a true case of AA.^{6,11} Laparoscopic appendectomy remains the treatment of choice for AA.¹ If pinworms are identified during surgery, thermal ablation or endoscopic suctioning should be used, as well as a specimen bag for extraction to avoid contamination.^{5,11} If EV are found in the pathological exam, antiparasitic treatment should be administered post-appendectomy.^{2,5} Household contacts of the patient should also be treated and the living environments cleaned to prevent reinfection.^{1,2}

EV's role as a direct etiological agent remains unproven and warrants further investigation. Careful clinical and pathological assessment is required to distinguish true AA from parasitic mimics and to guide appropriate management, however the diagnosis is usually confirmed postoperatively by pathological identification of the parasite.

CONCLUSION

AA has a multifactorial aetiology that occasionally includes intestinal parasites such as EV. Although EV infection can mimic the clinical presentation of AA through mechanisms like mechanical obstruction, mucosal irritation, or lymphoid hyperplasia, its direct causal role in true inflammatory appendicitis is still debated. Most cases involving EV show little to no histopathological evidence of acute inflammation, suggesting that parasitic infection often represents an incidental finding or a mimicker rather than a definitive cause. Given the diagnostic challenges, especially when considering conservative antibiotic management, it is essential to maintain a high index of suspicion for parasitic involvement in appropriate clinical contexts. Surgical treatment via laparoscopic appendectomy remains the gold standard, complemented by targeted antiparasitic therapy upon identification of EV, alongside measures to prevent reinfection in household contacts. Further research is needed to clarify the pathogenic relationship between EV and AA and to optimize management strategies for affected patients.

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