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Case Report

Perforation of the Small Intestine with Acute Peritonitis Caused by *Enterobius Vermicularis*

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Abstract

Perianal and perineal pruritus is often associated with *Enterobius vermicularis* in children. Although this roundworm is common in pediatric practice, most doctors are unaware that it can cause appendicular colic with/or appendicitis, severe urogenital complications, bowel perforation, and peritonitis. We report a case of a young male who presented with signs and symptoms of acute peritonitis. During the operation, perforation of the jejunum with a purulent exudate under the transverse colon, a left lateral canal, a Douglas cavity were found as well as single enlarged mesenteric lymph nodes. Histological studies detected *Enterobius vermicularis* in the lumen of the appendix and jejunum, as well as in the purulent exudate in the intestinal wall and serosa. A mesenteric lymph node, histologically presented with chronic nonspecific lymphadenitis. In conclusion, infection with *Enterobius vermicularis* should be considered in peritonitis, appendicitis, and enlarged mesenteric lymph nodes, especially in young patients.

Keywords

ectopic enterobiasis, peritonitis, tuberculosis

INTRODUCTION

Enterobius vermicularis (EV) is the most common helminthic infection in humans. It is more commonly seen in young children in whom it presents with pruritis in the perianal area.[1] During the cycle of the parasite, the fertilized female descends into the rectum from its habitual location, the caecum and the colon, and lays its eggs in the anal folds and the surrounding areas. The deposited eggs mature in a few hours and contain fully developed larvae that are infective. The movements of the females and the laying of the eggs cause itching, predisposing the passage of the infection to the same patient or other patients through hands which have been contaminated. Parasitosis is easily diagnosed by means of specific parasitological methods (perianal swabs or the Scotch tape test). Self-infection is common and can cause long-term infections. It occurs either by the mechanism described or by retroinfection.[2] The worms typically reside in the caecum, appendix, and distal ileum, where they adhere to the mucosa. The female EV sometimes can be found in different places: female genitals, Fallopian tubes, ovaries, perineum. Other times, both in female and male patients, the parasite can be found in rare locations; the prostate, the urinary bladder, the ureter, the spleen, the peritoneum, the mucosa and/or the appendicular lumen, the intestinal wall, the liver, the lungs, the epididymis, and the conjunctival sac.[2] Clinicians often treat the complications of a parasitic infection and in most cases the diagnosis is histologically established as a surprise to the pathologist and shocks the treating team of clinicians. In this report, I present a case of coinfection between parasitosis and tuberculosis. The cause of perforation of the small intestine with peritonitis is parasitosis.

CASE REPORT

A 33-year-old man was admitted to the emergency department of our hospital with a history of constant pain mainly in the lower half of the abdomen, fever, lack of bowel movement, and flatulence. He had persistent abdominal pain, no nausea, and vomiting for a few days. The patient had been treated for active pulmonary tuberculosis three months earlier. At physical examination, he had pronounced muscular defense mainly in the lower abdomen, missing peristalsis. Bloomberg's and Mendel's signs were positive. Roentgenography of the abdomen: hydroaeric shadows. Roentgenography of the lung: left-sided pulmonary tuberculosis in remission. Laboratory tests: increased leukocytes and accelerated ESR. Biochemistry studies were within normal limits. Clinical and laboratory studies were compatible with acute appendicitis and peritonitis, possibly tuberculous. A surgical approach was performed with a Lenander incision, in which 150 ml of thick pus was evacuated around the appendix and cavum Douglas. During the revision of the abdominal cavity, a stenotic fragment at the lower part of the jejunum with a compacted wall and a perforation 7-8 mm was detected. There was scarce pus in the adjacent sections. The perforated bowel, appendix and mesenteric lymph node were resected and sent for histological assessment. No postsurgical complications were registered. The macroscopic and histopathological examination was performed at the Department of Pathology of Uni Hospital, Panagyurishte. Appendix 7 cm with slightly hyperemic serosa and lumen filled with fecal material. A resect small intestine 27/4 cm with a visible whitish plaque on the serosa ***(Fig. 1)****, small perforation defect and hyperemic mucosa. Lumen filled with intestinal contents. A lymph node measuring 1.1×0.6 cm with a homogeneous shear surface. Histopathological examination of the appendix showed a transverse and longitudinal section of female and male worms in a lumen with clearly visible Alae* ***(Fig. 2a)****. The appendicular mucosa was preserved. Submucosa showed reactive lymphoid follicles with germination centers. Mild chronic inflammation in the appendicular wall, consisting of lymphocytes and scarce eosinophils. A histological examination of the small intestine showed parasites and eggs in the lumen, disturbed mucosal sites and marked hyperemia with acute inflammation in the lamina propria and presence of eosinophils* ***(Fig. 2b)****. In the area of perforation, a visible course filled with parasites, eggs, and food debris with purulent inflammation was noticed* ***(Fig. 2c)****. In another part of the intestinal wall, there were parasitic eggs with degenerative changes* ***(Fig. 2d)****. A histological examination from the area of the whitish plaque on the small intestinal serosa presented parasites and parasitic eggs in the pus* ***(Fig. 2e)****. A histological examination of the lymph node showed chronic nonspecific lymphadenitis. Diagnosis: Perforation of the small intestine with acute peritonitis caused by Enterobius vermicularis.*

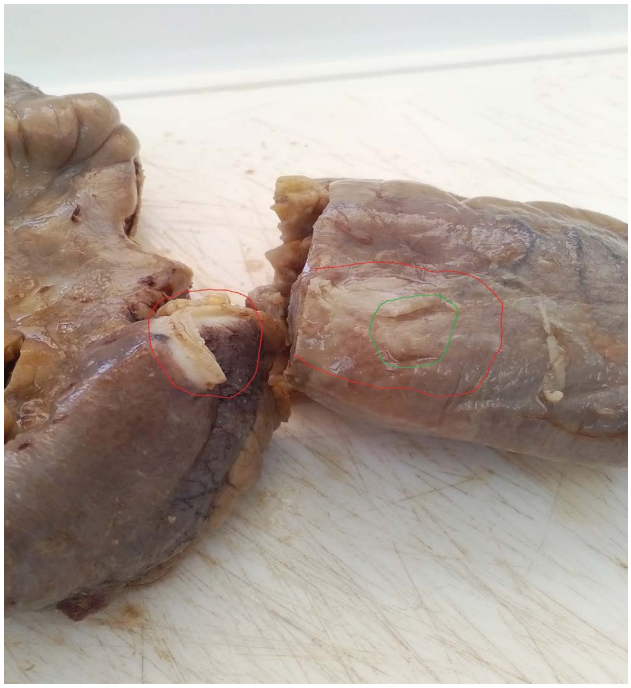
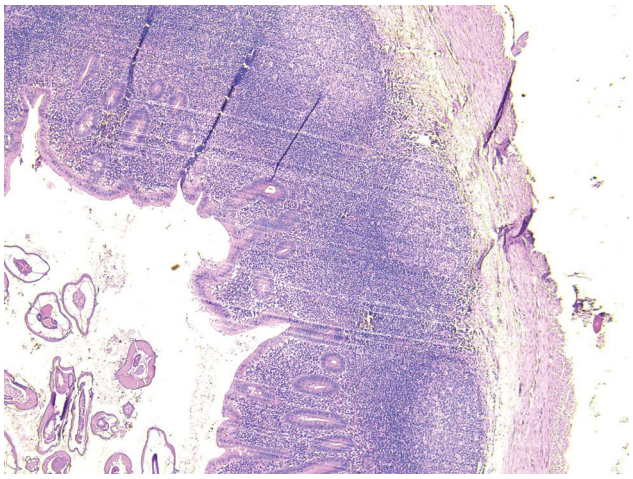


Figure 1. Macroscopic picture: small intestine with fibrinous-purulent exudate on the serosa outlined in red and micro-perforation outlined in green.

DISCUSSION

Enterobius vermicularis is the most common infection-causing helminth. It colonizes predominantly the intestinal tract and its discovery elsewhere is casuistry. It is more common in temperate climates and in school-age girls.[1] EV can cause the appendix to obstruct with appendix colic, but the direct connection with appendicitis remains controversial.[3] EV cannot migrate through tissues, peritoneal cavity entry occurs either via fallopian tubes or during appendicectomy.[4] Peritoneal cavity contamination can cause pelvic inflammatory disease, peritonitis or *Enterobius* granulomas.[2] Despite the presentation of Ariyarathenam AV, et al., our case shows that EV causes acute inflammation in the intestinal wall with the formation of abscesses, and subsequently, its passage into the peritoneal cavity. The combination of pulmonary tuberculosis and EV are bilateral agents that suppress the patient's immunity and create conditions for persistence in both diseases. *Enterobius vermicularis* can cause severe morbidity and can even be fatal when outside the gut.[5] In the reported case, perforation of the small intestine and acute peritonitis were successfully managed with subsequent antihelminthic therapy with Vermox (mebendazole). One month after treatment in the study control sample *Enterobius vermicularis* was not detected. The presence of tuberculosis and subsequent infection with *Enterobius vermicularis* in a patient with compromised immunity has created a prerequisite for penetration of the EV through the intestinal wall with subsequent perforation and peritonitis. From the reference in the Medline database via PubMed using the key words: "*Enterobius vermicularis*", "ectopic enterobiasis", "peritonitis",



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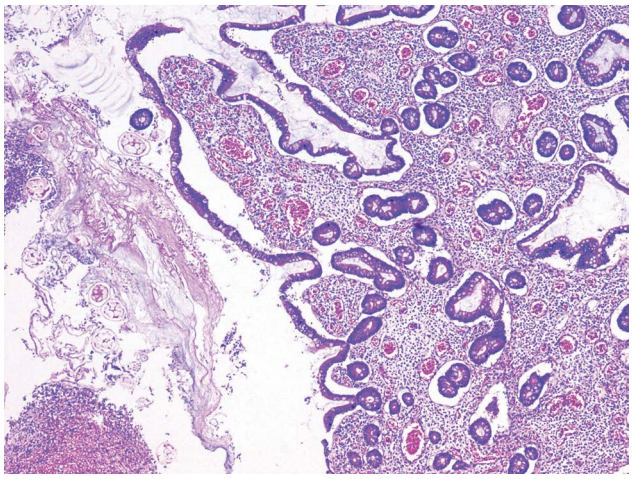

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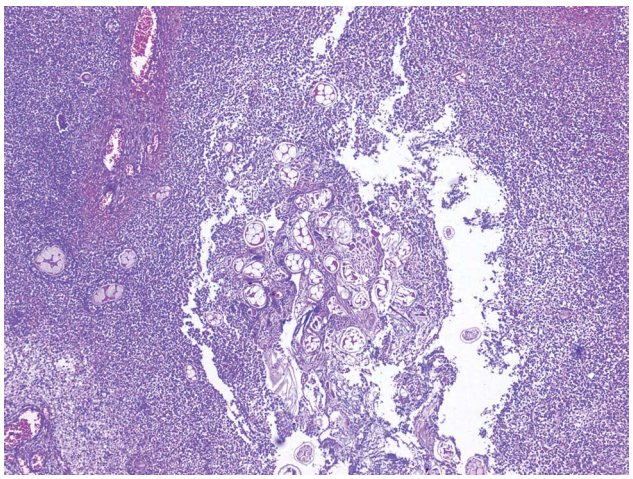

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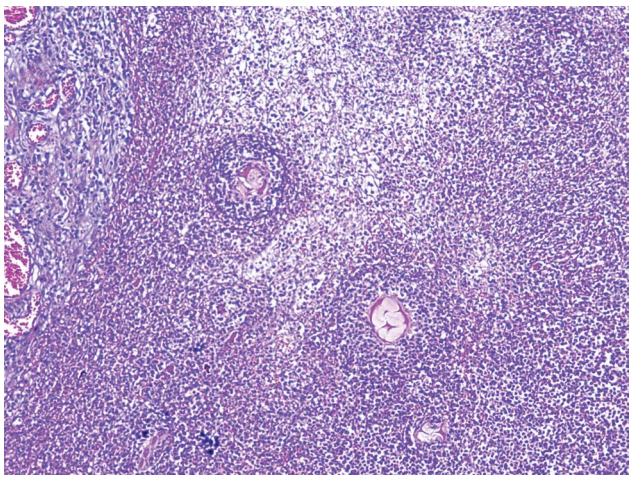

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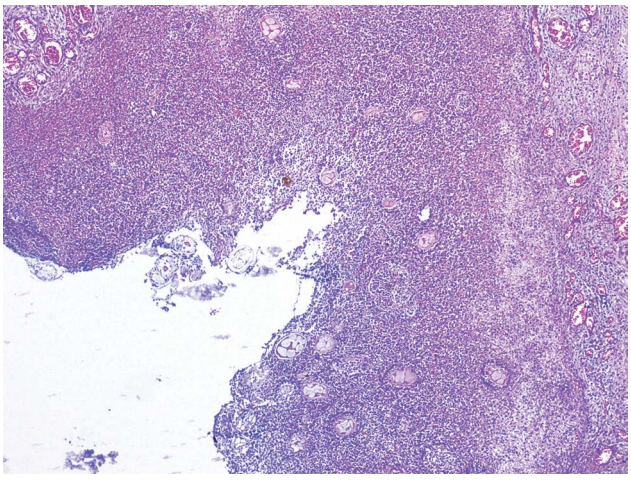

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Figure 2. Histology (H&E): **a.** *Enterobius vermicularis* in a lumen of an appendix with expressed follicular hyperplasia (magnification ×50); **b.** Small intestinal mucosa with pronounced hyperemia and inflammation in the lamina propria. In the lumen EV - eggs and parasites. (magnification ×50); **c.** Intestinal wall in the area of perforation with visible progress with purulent inflammation in the periphery and parasites and food debris in the center (magnification ×50); **d.** Small intestinal wall with purulent inflammation, including the presence of parasitic eggs with degenerative changes (enlargement ×100); **e.** Small intestinal serosa with purulent inflammation and parasitic forms (magnification ×50).

"tuberculosis" this was found to be the first reported case of coinfection between EV and tuberculosis associated with intestinal perforation, peritonitis and a favourable outcome for the patient. There are two possible options for affecting the peritoneum: migration of the female worm from the anus through the vagina with access to the female genital tract and peritoneum[6], and secondary involvement of the omentum and peritoneum by intestinal perforation caused by the worm.[7] Our case is in support of the fact that EV can cause inflammation in the intestinal mucosa and the wall, in this case causing diffuse purulent inflammation with subsequent intestinal perforation, peritonitis, and the discharge of the parasite with its eggs into the abdominal cavity. There are two theories explaining the ectopic existence of EV. According to the first theory, EV enters the ectopic site directly by penetrating the tissue, as is the case reported. The second theory assumes that ectopic involvement occurs by hematogenous and/or lymphatic spread of worms.[8] It explains the destruction of organs that lack direct contact with the intestinal wall.[2] Despite the possibility of a lymphatic/hematogenous spread of EV in our case, in the serial histological examination of an enlarged mesenteric lymph node, no specific process was detected. We assume that the increased reactive altered mesenteric lymph nodes, in this case, are due to co-infection: EV and pulmonary tuberculosis. Despite the reporting of mesenteric involvement by EV[9] lymph nodes in our case, this has not been identified. In the reported case, we present of EV in the appendix, where adult forms are found in its lumen, but there is no inflammation. Histological examination of the small intestine reveals parasites and eggs in the bowel, diffuse and focal purulent inflammation in the intestinal wall with parasite eggs and sites of granulomatous inflammation. Separate eggs and single parasites in the pus are detected in the serosa. Our case is a clear example that when EV is in the intestinal lumen, as in the appendix, it does not cause inflammation of the intestinal wall, but when it penetrates the intestinal wall, it provokes an acute and chronic inflammatory response with the development of purulent, purulent/necrotic and granulomatous reactions around eggs and dead worms.[9] The presence of eggs in inflammation indicates that they are more resistant to the effects of the immune system of the macroorganism.[6] Our case, as well as the other cases of extra-intestinal EV, as well and the lack of cases for "ectopic enterobiasis" in children, is consistent with the belief that EV occurs predominantly in immunocompromised patients, regardless of the type of spread. This case highlights the usefulness of histological examination of all specimens in search of EV, especially in endemic areas, during appendicectomy, to minimize the risk of contamination and to initiate early treatment for both the patient and the people who have been in contact with him.

CONCLUSIONS

Intestinal parasitosis, especially in young patients, should always be considered as a possible cause of acute abdomen in a patient affected by active tuberculosis or during recovery after therapy.

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Перфорация тонкой кишки при остром перитоните, вызванном *Enterobius Vermicularis*

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Резюме

Перианальный и промежностный зуд часто ассоциируется с *Enterobius vermicularis* у детей. Хотя эта аскарида часто встречается в педиатрической практике, большинство врачей не знают, что она может вызывать аппендикулярную колику с аппендицитом или без него, тяжелые урогенитальные осложнения, перфорацию кишечника и перитонит. Это случай молодого человека, у которого появились симптомы и признаки острого перитонита. Во время операции обнаружена перфорация тощей кишки с гнойным экссудатом под поперечной ободочной кишкой, левым боковым каналом, полостью Дугласа и единичным увеличенным мезентериальным лимфатическим узлом. Гистологическое исследование выявило *Enterobius vermicularis* в просвете отростка и тощей кишки, а также гнойный экссудат в стенке кишечника и серозной оболочке. Брыжеечный лимфатический сосуд гистологически представлен хроническим неспецифическим лимфаденитом. В заключение, инфекцию *Enterobius vermicularis* следует рассматривать при перитоните, аппендиците и увеличенных мезентериальных лимфатических узлах, особенно у молодых пациентов.

Ключевые слова

эктопический энтеробиоз, перитонит, туберкулёз