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# Enterobius vermicularis in colon mimicking tubercular ulcers

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#### **KEYWORDS**

# Enterobius vermicularis, Colon mimicking, tubercular ulcers

## ABSTRACT

Enterobius vermicular is a parasite can lead to inflammation and symptoms in rare cases however it can manifest as ulceration in colon which may mimic tubercular ulcer. A 40-year-oldman presented with pain abdomen. The pathologic findings confirmed the diagnosis of *E. vermicularis* in the colonic biopsy. It is important to be aware of this resemblance to avoid unnecessary surgical intervention and simple treatment measures which can cure the patient.

# Introduction

Enterobius vermicularis is the common helmintic parasite known, affecting all members of society regardless of age, gender, and social status [1]. They typically reside in the caecum, appendix and distal ileum, where they adhere to the mucosa [2]. infections Although many asymptomatic, perianal itching, especially at night, is the most common symptom [1]. However there are a lot of atypical presentations described in the literature, for example, infections of the kidneys [3] and infections of the female genital tract [4] as well as many other presentations.

Typically the diagnosis rests upon applying cellophane or scotch tape to the

perianal skin in the morning, removing it, and detecting eggs using the microscope [1, 5]. The worms can however be seen during endoscopy [6], and both the worm and its eggs can be found in histological specimens [5, 7]. Once diagnosed the infection is eradicated with two doses of Mebendzole two weeks apart as well as hygienic measures [8]. We here report a case of a man who presented with diarrhea. Inflammatory bowel disease was suspected and a colonoscopy showed ulcerated lesions suggestive of tubercular ulcersbut colonic Enterobius biopsy revealed eggs of vermicularis. Enterobius vermicularis has reported various to cause manifestations but presenting as tubercular ulcer is rare.

#### **Case Presentation**

A 40-year-old man presented with pain abdomen and an exacerbation of diarrhea during the past two months. During the past two months, he had been having an increased amount of watery-thin diarrhea 5-6 days a week. He now had symptoms also during the night and with no relation to food intake. In addition abdominal cramps were sustained during the days of diarrhea. His blood counts and biochemistry were within normal limits.

The colonoscopy showed erosions which increased distally up until the caecum, where ulcerations upon a erythematic base was seen. In the distal ileum pattern was seen together with multiple erosions. The morphology suggested tubercular ulcers and multiple biopsies were taken. Biopsy showed no crypt abscesses and no granulomas. In ileum lymphoid hyperplasia with germinal centers was found as well as focal neutrophilic infiltrates. In caecum and ascending colon a few spots with cryptitis were found. The most remarkable finding a female larvae of Enterobius vermicularis in the intestinal lumen (Figure 1). The patient was therefore given a single dose of Mebendazole with a second dose two weeks later. The MRI was normal. A follow-up colonoscopy five months later revealed a macroscopically normal colon and distal ileum; biopsies were taken and they revealed lymphoid hyperplasia in the distal ileum and a normal caecum and colon. Fecal calprotectin was normalized during the following months and he remains symptom free.

### **Discussion**

To our knowledge no case of *Enterobius* vermicularis mimicking tuberculosis have been reported in the literature. It is believed

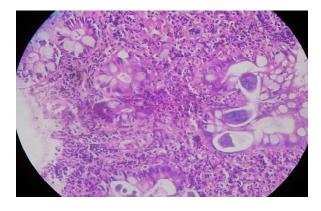
that Enterobius vermicularis cannot penetrate the intestinal mucosa unless there is some insult to the mucosal barrier. They are however known to be associated with colonic ulcerations but the question of causation remains unanswered. In two of the previous cases presented by Beattie et al. [9] and McDonald and Hourihane [2] a reasonable mechanism of mucosal damage was present. In the case presented by McDonald and Hourihane [2] a perforated appendix was deemed reason behind the symptomatic Enterobius vermicularis.

Fernandez-Flores and Dajil [10] have described that the worms attach themselves to the mucosa using their heads [2]. This may cause the ulceration necessary for the pinworms to become invasive.

The colon alone can be affected by Tuberculosis though this is much less common (5 of 159 cases). The duodenum and/or jejunum can be affected by TB but this is rare as well (2 of 159 cases).[1]. There are two primary types of lesions associated with intestinal tuberculosis, ulcerative and ulcero-hypertrophic.

For ulcerative lesions, areas of disease are moderately indurated and are marked by an increase in mesenteric fat and the circumference is studded with nodules of variable size. Mesenteric lymph nodes are often enlarged. Characteristic caseation may be found after examining numerous lymph nodes. The ulcers may be single or multiple, in the latter case variable lengths of uninvolved mucosa being present between. Characteristically, the established lesion consists of an annular ulcer involving the entire circumference affecting a segment generally less than 3 cm in length. The lumen in this region is narrowed, sometimes measuring less than 1 cm in diameter, resulting in a napkin-ring type of stricture.

**Fig.1** Colonoscopic biopsy showing the parasite *Enterobius vermicularis* 



Ulceration is relatively superficial and does ordinarily penetrate muscularispropria. The ulcers present a variable appearance. The ulcer bed is covered with a necrotic slough. It may be coarsely granular, often showing small pseudopolyps, or the mucosal folds may be replaced by mamillated a Sometimes, the mucosal folds are evened out and scattered with irregularly disposed mucosal erosions. Where the ulcers are well defined, the margins may be undermined or sloping, or flush with the surface. In an occasional case, the ulcer presents a stellate appearance with a deep excavation bordered overhanging sharply hypertrophic which are intensely mucosal shelves hyperemic. The ulcer in such cases tends to be disposed along the longitudinal axis and the neighboring mucosal folds tend to converge upon the ulcer. The thickness of the wall underlying the ulcer bed is variable; it may be thinned or may appear hypertrophic and scarred, streaked with vellowish areas of necrosis. Ulcerohypertrophic lesions usually affect the ileocecal region, the patient presenting with a large lump in the right iliac fossa. The ileocecal region, mesenteric fat, and their constituent lymph nod-s are seen to constitute a large mass with extensive adhesions. The ileocecal angle is distorted and often obtuse. On opening, the wall is

seen to be markedly thickened, occasionally in a tubular form, measuring up to 3 cm in thickness.

The mucosal changes are quite variable. There may be a prominent 'cobblestoning' or pseudopolyposis or the mucosal folds may be flattened and the surface shows disposed irregularly furrows mostly converging upon the constriction. It may be mentioned here that there are no sharp differences between ulcerative and ulcerohypertrophic lesions and the two can occur at the same time. A deformed, patulous ileocecal valve with heaped up mucosal folds is suggestive of intestinal tuberculous. microscopy of tubercular ulcer shows these features which were absent in this biopsy. Exuberant granulomatous tissue extending onto the serosa. Mesenteric fat, enlarged lymph nodes, fibrosis, and hypertrophy of the muscularis.Granulomas in the mucosa or the Peyer's patches. The structure of the caseating granulomas, which is considered a diagnostic finding, is well known. The peripheral part of the granuloma contains a zone of infiltration by an admixture of lymphocytes, plasma cells, and giant cells of the Langhans variety. As in the case presented by Beattie et al. we could demonstrate a normal colon and distal ileum during a follow-up colonoscopy. In our case we could also show a normalized fecal calprotectin indicating that the mucosal inflammation had ceased.

The question whether the worm we found in the lumen could have caused this inflammatory reaction does not have a definitive answer. In the case reported by McDonald and Hourihane worms were indeed present in the intestinal wall. However in the cases presented by Fernandez-Flores and Dajil the worm was found solely in the lumen covered by eosinophils. The worm was described as

being found at the base of an ulcer in the case presented by Beattie et al. but the worm was deemed invasive by the authors. Lastly in a case where *Enterobius vermicularis* caused eosinophilicileocolitis no worms were found in biopsy specimens obtained during colonoscopy instead the worm was found during stool examination.

## **Conclusion**

The histopathological finding of a pinworm and the lasting remission after Mebendazole treatment lean towards the notion that Enterobius vermicularis infection is the cause of his symptoms. Since the patient responded well to Mebendazole and the inflammatory infiltrates found in the biopsy specimen were not typical for tubercular ulcer, we believe that our patients symptoms were caused by Enterobius vermicularis. It is today unknown if Enterobius vermicularis is an invasive pathogen or if structural damage is required for invasive infection. Enterobius vermicularis might be an important differential diagnosis, since this easily treated parasite infection can greatly reduce the patients quality of li

### References

- 1.Symmers WSC. Pathology of oxyuriasis; with special reference to granulomas due to the presence of Oxyurisvermicularis (*Enterobius vermicularis*) and its ova in the tissues.AMA Arch Pathol. 1950 Oct;50(4):475–516.
- Ma Y, Chandrasoma P. Pathology of the small intestine. In: Chandrasoma P, ed. Gastrointestinal Pathology. Stamford, CT: Appleton & Lange; 1999:189-223.
- 3. Gonzalez Martin MA, Diaz Oller J, Gonzalez Perez F, et al. Colorectal tuberculosis. Rev EspEnfermApar Dig. 1989;75:712-716.

- 4. Singh V, Kumar P, Kamal J. Clinicocolonoscopic profile of colonic tuberculosis. Am J Gastroenterol. 1996;91:565-568.
- 5. Nagi B, Kochhar R, Bhasin DK, et al. Colorectal tuberculosis. EurRadiol. 2003:13:1907-1912.
- 6. Deodhar SD, Patel VC, Bharucha MA, et al. Primary tuberculosis of the large bowel. J Postgrad Med. 1986;32:161-162.
- 7. Lax JD, Haroutiounian G, Attia A. Tuberculosis of the rectum in a patient with acquired immunodeficiency syndrome. Report of a case.Dis Colon Rectum. 1988;31:347-349.
- 8. Marshall JB. Tuberculosis of the gastrointestinal tract and peritoneum. Am J Gastroenterol. 1993;88:989-999.
- 9. Stiefelhagen P. Suspected Crohn disease diagnosis: endoscopy alone is not reliable. MMW Fortschr Med. 2005;147:13-14.
- 10. Heinrich S, Hetzer FH, Bauerfeind P, et al. Retroperitoneal perforation of the colon caused by colonic tuberculosis: report of a case. Dis Colon Rectum. 2004;47:2211-2214.