Case Report: Rectal Perforation Caused by Schistosoma haematobium

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Abstract. A 31-year-old woman from Cameroon was admitted to the University of Strasbourg Hospital in December 2007 with pelvic pain and fever that developed over three days. Her condition rapidly worsened and she underwent emergency exploratory celioscopy. Surgeons found peritoneal and retrouterine abscesses. The high rectum had a 4-cm perforation with infiltrated, friable, and irregular edges. A biopsy specimen of this pseudotumoral specimen showed many Schistosoma haematobium eggs with an inflammatory reaction surrounding the eggs. The patient was treated with praziquantel (40 mg/kg/day) for 5 days and a 4-week course of antibiotic therapy. Her progress was good and digestive continuity surgery was performed four months later. Schistosomiasis frequently involves rectal mucosa, but perforation is unusual. Our review of the literature found only two cases of colon perforation associated with S. mansoni infection. To our knowledge, this is the first case of rectal perforation caused by S. haematobium described in the literature.

INTRODUCTION

Schistosomiasis is a trematode infection endemic to tropical areas. Nevertheless, in countries not endemic for this infection, the clinician can encounter imported forms in migrant populations or travelers. The adult parasite can survive several years within the perivesical or mesenteric venous plexus and the first symptoms can appear belatedly (up to 30 years after the initial infestation). Several clinical manifestations have been observed, each stemming from particular species: genito-urinary tract infection caused by Schistosoma haematobium, digestive problems caused by S. mansoni or S. intercalatum, and hepatobiliary infestation by S. mansoni, S. japonicum, or S. mekongi. Digestive lesions are most frequently caused by rectal or large bowel involvement. Symptoms are not specific and endoscopy can show an inflammatory gut wall, pseudopolyposis, ulcerations, and superficial bleeding. In a few cases, colorectal stenosis or pseudotumoral lesions has been described. Microscopically, lesions are characterized by inflammation that can sometimes be granulomatous around the eggs laid by the adult worms located in the adjacent venous plexus. With this reaction, relatively substantial fibrosis can be seen, depending on how long the lesions have been present. We report a patient from Cameroon with a rectal perforation with peritonitis secondary to an infection with S. haematobium.

CASE REPORT

A 31-year-old woman from Cameroon living in France for five years was admitted to the University of Strasbourg Hospital in December 2007 because of pelvic pain associated with fever that developed over three days. She had a history of appendicular peritonitis in 1990 and a secondary episode of pelviperitonitis caused by salpingitis in 2006. Ultrasonography showed pelvic abscesses. The condition of the patient deteriorated rapidly and she underwent emergency exploratory celioscopy. Surgeons found an armored pelvis and a retrouterine purulent collection. Laparotomy showed an inflammatory high rectum and rectosigmoid junction with a pseudotumoral aspect.

DISCUSSION

This case motivated us to consider a rectal perforation secondary to parietal lesions generated by the parasitic infection. A review of the literature found two cases of colon perforation associated with invasive schistosomiasis: one case of colon perforation associated with S. japonicum and a case of sigmoid perforation associated with S. mansoni. To our knowledge, the case reported herein is the first case of rectal perforation caused by schistosomiasis. Nevertheless, the pathogenic role of the schistosomes must be discussed. Schistosomes can be involved in appendicular syndromes, even in true peritonitis. According to some investigators, this association would be purely fortuitous for people living in schistosomiasi-endemic areas. Other investigators believe that schistosomes are directly implicated. Two pathogenic pathways have been described to explain this occurrence with appendicitis. The first pathway considers the role of feces with its passage through inflammatory and fibrotic mucosa infected with the parasite. The second pathway is a direct inflammatory

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lesion of the appendix induced by an immunologic granulomatous reaction around eggs, leading to tissue destruction and appendicitis. A granulomatous reaction is not systematically observed, even when the mucosa or the submucosa show signs of active inflammation.

In a retrospective study based on 843 specimens from appendicectomies performed in Nigeria between 1991 and 2004, Badmos and others found schistosomes in 35 specimens, 23 of which were associated with active inflammation: acute suppurative inflammation in 17 cases and active granulomas with tissue eosinophilia in six cases. In our patient, several elements directed us to the diagnosis of a rectal perforation caused by parasitic infection. The first element was the presence of viable schistosome eggs, which indicated active infection. The second element was the presence of acute submucosa inflammation. The absence of a granulomatous reaction does not eliminate the diagnosis because it is not systematically associated with schistosomiasis. The pseudotumoral macroscopic aspect is also interesting in our patient. It is known to be an unusual type of schistosomiasis. The pseudotumoral macroscopic aspect is also interesting in our patient. It is known to be an unusual type of schistosomiasis. O’Leary and Mattia reported a case of secondary infertility and bladder mass related to S. haematobium infection. The bladder mass was large (3 cm) and irregular but not ulcerated.

In our patient, we believe that perforation could be favored in cases of anterior pelvic infection and perhaps friable mucosa. These elements led us to diagnose rectal perforation secondary to the parasitic infection. The presence of S. aureus and E. coli in pelvic abscesses is also interesting. It seems that parasitic diseases should have a central role in the development of bacterial infections by switching the immune system from a T helper 1 to a T helper 2 response, contributing to granuloma formation, eosinophilia, and susceptibility to bacterial and fungal infections. Other investigators have reported a relationship between S. mansoni infection and pyogenic liver abscesses caused by S. aureus in experimentally infected mice and in humans. These findings may also relate pelvic abscesses and shistosomiasis in our patient.

The question can also be raised regarding the pathogenicity of S. haematobium in the rectum, which is usually associated with urinary tract pathology. Nevertheless, its presence in rectal mucosa is not surprising and diagnosis of schistosomiasis can be made by rectal biopsy. In addition, vesical lesions usually observed do not differ from those observed in the gut wall: granulomatous infection, fibrosis, and ulcerations. Finally, the existence of subspecies with variable virulence could explain severe forms of schistosomiasis.

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REFERENCES