Amoebic Anal Fistula: New Insight Into an Old Disease

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ABSTRACT

A 67-year-old gentleman underwent fistulectomy for low trans-sphincteric anal fistula along with curettage for an associated abscess extending proximally for half a centimeter into the intersphincteric plane. The roof of the cavity became clearly visible after satisfactory culmination of the surgical procedure. Histopathological examination of the fistulous tract and the curetted granulation tissue revealed presence of multiple trophozoites of Entamoeba histolytica exhibiting erythrophagocytosis in the background of mixed inflammatory infiltrate. This case report provides the outlook that yields the novel insight into the possible role of Entamoeba histolytica in the pathogenesis and persistence of the fistulous tract.

Key words: anal fistula, Entamoeba histolytica, fistula.

INTRODUCTION

Amoebiasis is an infection caused by an enteric protozoan Entamoeba histolytica. This parasite has a worldwide distribution and the prevalence is especially higher in tropical developing countries where poor socioeconomic and low sanitary conditions prevail.¹ Amoebiasis is a major health problem in China, Mexico, the eastern part of South America, south-east and west Africa, and the whole of south-east Asia including the Indian subcontinent.² Entamoeba histolytica infection remains clinically silent in 90% of patients, where it can be present as a persistent carrier, and the remaining 10% of patients develop a variety of intestinal and extraintestinal manifestations. Intestinal
amoebiasis predominantly involves cecum and rectosigmoid part of colon. Intestinal invasive amoebiasis may be associated with a number of anatomical/physiological/pathological alterations including acute colitis, toxic megacolon, amoeboma and appendicitis.\textsuperscript{3,4} Anal fistula as a complication of invasive amoebiasis is a relatively lesser known entity.

CASE ILLUSTRATION

A 67-year-old non-diabetic gentleman presented with repeated episodes of pus discharge in and around anus for one year duration. He did not complain of altered bowel habits, mucus or blood discharge per rectum. He reported to have undergone incision and drainage for a perianal abscess 12 years back and after which he had remained well and asymptomatic before presenting with his present complaints. Abdominal examination was unremarkable. On perineal examination, there was a \textit{2×2 cm\textsuperscript{2}} area of tender induration located at 3 O’clock with central sloughing of its overlying skin causing persistent discharge of pus from within. The patient was advised to undergo debridement for the slough and evacuation of the pus from the burst perianal abscess under anesthesia, but he refused. A course of broad spectrum antibiotic (ciprofloxacin and metronidazole) for 5 days along with other supportive measures was started. The patient reported back to the outpatient department after a month and complained of persistent discharge of pus from the perianal wound. A repeat anorectal examination confirmed a low perianal fistula with an external opening at 3 O’clock, 2 cm away from anal verge and an internal opening at 6 O’clock within the distal 1 cm of the anal canal. There was no other abnormality palpable in upper anal canal or lower rectum. Rectosigmoidoscopy was normal. The patient was advised to undergo fistulectomy. Intraoperative study done by injecting methylene blue from the external opening confirmed the presence of the internal opening at the 6 O’clock position at the dentate line. Fistulectomy for low trans-sphincteric anal fistula along with curettage for an associated abscess extending proximally for half a centimeter into the intersphincteric plane was done. The patient was prescribed intravenous ciprofloxacin and metronidazole for first 24 hours and then continued these drugs orally for next seven days. Histopathological examination of the tract showed presence of multiple trophozoites of Entamoeba histolytica exhibiting erythrophagocytosis along with mixed inflammatory infiltrate in the background. (Figure 1) PAS staining clearly demonstrated trophozoites. (Figure 2) Post fistulectomy, the wound healed completely in 4 weeks. The patient is well after 9 months of follow-up.

Figure 1. Shows anal canal squamous epithelium with multiple trophozoites (black arrows) showing erythrophagocytosis, and mixed inflammatory infiltrate (H&E 100X).

Figure 2. Shows PAS positive Trophozoites (black arrows) (H&E 100X).
DISCUSSION

Anal fistula may result from a number of infectious and non-infectious causes. Most of the anal fistula have infectious etiologies consequent to cryptoglandular sepsis\(^5\) and arise posteriorly as it is here that the densest concentration of anal glands is located. This cryptoglandular sepsis accounts for more than 90% of cases of fistulae.\(^6\) Other infectious causes include pelvic sepsis, tuberculosis, actinomycosis and sexually transmitted diseases. The non-infectious causes include inflammatory bowel disease and various anorectal operative procedures. Anal fistula may be the first presenting feature of an underlying anorectal tumour such as squamous cloacogenic adenocarcinoma and anal glandular malignancy.\(^7\) Fistula may occasionally follow sclerotherapy, internal sphincterotomy or closed haemorrhoidectomy.\(^8\)

Histopathological report of our patient brought us an inexplicable diagnosis. Even extensive literature failed to reveal any previous report of association of amoebiasis with anal fistula. Presence of amoebic trophozoites in the fistulous tract raises the critical issue that whether they were merely bystanders after having migrated into the fistulous tract from the original place of residence in the large gut or have actually proved pathogenic for the formation and persistence of fistula. The concentration of trophozoites in the fistulous tract was fairly large in number and many were exhibiting erythrophagocytosis. Moreover, the additional presence of inflammatory infiltrate in the fistulous tract further points towards the invasiveness of the trophozoites. This inflammatory infiltrate was not as dense as in usually evident in bacterial infection, though the type of the chronic cell infiltrate suggests long drawn inflammatory reaction consequent to the persistence of amoebic infection in the epithelized and fibrosing fistulous tract. The increasing fibrosis may lead to decreased vascularity of the area and consequent decreased or nearly sub pharmacological drug dose delivery in the affected fistula area causing the amoeba to survive. These factors indicate pathogenic nature of the amoeba which can suggest the etiopathogenesis by its trophozoites in the development of the anal fistula in the present case.

From where the trophozoites invaded the anal canal wall is another issue to be understood. We believe anal glands and crypts might have provided the route for migration to trophozoites during repeated acts of defecation and further accentuating their inflammation with passage of time that’s preceding the formation of the clinically overt abscess and fistula. Though our patient received full course of metronidazole for the treatment of ruptured perianal abscess, yet he developed amoebic anal fistula. This may be related to the inability of antiamoebic drugs to penetrate the fistulous tract for effective medical cure as stated above.

CONCLUSION

This case report highlights the possible role of amoebiasis in the pathogenesis of anal fistula. Considering high prevalence of intestinal amoebiasis in tropical countries, the findings of this case report underscores the need for further research to establish causative association of amoebiasis and anal fistula.

REFERENCES