

Differentiating Amoebic Ulcero-haemorrhagic Recto-colitis from Idiopathic Inflammatory Bowel Disease

Still a Diagnostic Dilemma

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ABSTRACT

The colon responds monomorphically to a variety of insults thus making it difficult to differentiate invasive amoebic colitis and inflammatory bowel disease (IBD). The authors present a case with chronic dysentery, haematochezia, anaemia and hypoproteinaemia. The endoscopic findings were suggestive of IBD. The stool examination was negative for trophozoites or cysts of parasites. The recto-colonic biopsy specimens showed mucosal inflammation with exudates containing amoebic trophozoites. The patient was successfully treated with metronidazole and iodoquinol. He recovered within two weeks and repeat colonoscopy four weeks after the treatment showed a normal rectum and colon. Clinicians should have a high level of suspicion for amoebic colitis in cases of colitis especially in regions where amoebiasis is still present. Efforts should be made to find the amoebic trophozoites in multiple stool and colonic biopsy specimens.

Diferenciar la Rectocolitis Úlcero-hemorrágica Amebiana de la Enfermedad Inflamatoria Idiopática del Intestino:

un Dilema Diagnóstico sin Resolver

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RESUMEN

El colon responde de manera monomórfica a una variedad de insultos, lo cual hace difícil distinguir entre la colitis amebiana invasiva y la enfermedad intestinal inflamatoria (EII). Los autores presentan un caso con disentería crónica, hematoquexia, anemia e hipoproteinemia. Los resultados endoscópicos apuntaban a una EII. El análisis de las heces fecales arrojó resultados negativos en cuanto a presencia de trofozoitos o quistes de parásitos. Esto condujo a un diagnóstico erróneo y el paciente fue tratado por una EII. Sin embargo, los especímenes de la biopsia rectocolónica mostraron una inflamación mucosal con exudados en los que se hallaban presentes trofozoitos amebianos. El paciente tuvo un tratamiento exitoso con metronidazol y iodoquinol. Se recuperó en dos semanas, y se le repitió la colonoscopia cuatro semanas después de que el tratamiento mostró un recto y colon normales. Los clínicos debían mostrar un alto nivel de sospecha ante la colitis amebiana, especialmente en aquellas regiones donde la amebiasis todavía está presente. Deben hacerse esfuerzos por encontrar trofozoitos amebianos en múltiples especímenes de heces fecales y biopsia colónica.

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INTRODUCTION

Amoebiasis is the second most common cause of parasitic death worldwide (1), and although its incidence has declined considerably in the Caribbean countries, clinical cases may still be found (2–5).

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Despite the availability of sophisticated investigative procedures, differentiating invasive colonic amoebiasis from idiopathic inflammatory bowel disease (IBD), may be difficult (6–8). This case is presented to remind clinicians of the similarities in the clinical endoscopic features of these two conditions and to highlight the difficulty in differentiating them.

CASE REPORT

A 38-year-old man presented to hospital with a four-month history of diarrhoea and intermittent haematochezia. He had

generalized body weakness, easy fatigability, mild abdominal pain, low grade pyrexia and 6.8 kg weight loss over a four-month period. He did not smoke, drink alcohol and was heterosexual. He denied the use of antibiotic prior to or during the illness.

On examination, his vital signs were normal except for tachycardia. His mucosal membranes were very pale, but the systemic examination was normal except for bloody stool found on rectal examination. Haemoglobin (Hb) was 4.3 gm/dl. There was a microcytic hypochromic picture on blood film and serum iron 10 ng/ml (normal range 37–181 ng/ml). The blood chemistry was normal except for low potassium of 2.5 mmol/l and the erythrocyte sedimentation rate was 15 mm/hr (Westergren). His HIV screening was negative and the stool was positive for occult blood but no cysts or trophozoites of parasites were found. Proctosigmoidoscopy (panel A) showed severe inflammation with cobblestone formation, bleeding and friability of the mucosa from the rectum to beyond the sigmoid colon and six biopsy specimens were taken from the rectum and sigmoid colon. In the absence of classical endoscopic findings of amoebic colitis and negative stool examination for parasites, the patient was treated as inflammatory bowel disease using prednisolone and mesalazine while waiting for the histology report. He was transfused four units of packed red blood cells and had potassium chloride supplement before being discharged. The patient was however re-admitted after two weeks with haematochezia and swollen feet and hands. His Hb was 7.78 gm/dl with a microcytic and hypochromic picture and low serum albumin.

Definitive diagnosis was made from the histologic report which showed active colitis with surface exudates containing *Entamoeba histolytica* trophozoites and the absence of granuloma. The previous treatment was stopped and he had a 10-day course of metronidazole at 750 mg thrice daily. He also had iodoquinol for another 20 days to eliminate the cysts. He made a dramatic recovery and returned to work within two weeks. The repeat proctocolonoscopy (panel B) four weeks after treatment showed normal sigmoid and descending colon, and rectum.

DISCUSSION

The dilemma in differentiating amoebic ulcero-haemorrhagic (AUH) colitis from IBD is more likely if amoebiasis is present in the community or when the patient has visited an endemic area.

This problem is compounded by the similarity in the symptomatology of the two diseases and the non-specific endoscopic findings, coupled with the absence of amoebic trophozoites and/or cysts in the stool in some cases of AUH colitis. Both AUH colitis and IBD may present with bloody mucoid diarrhoea, abdominal pain, frank haematochezia, anaemia and hypoproteinaemia. Both can present with constitutional and extra intestinal syndromes (1, 6–11).

The endoscopic findings in both AUH colitis and IBD may be non-specific, making definitive diagnosis difficult (9–11). Typical discrete flask-shaped ulcers of amoebic colitis may also be seen in Crohn's disease. Alternatively, continuous mucosal inflammation typical of ulcerative colitis can be seen in amoebic colitis. Although the anatomic extent of the lesions in these three causes of colitis varies, it is of most importance in differentiating Crohn's disease from ulcerative colitis than amoebic colitis from the IBD. Lesions from Crohn's disease apart from involving the small intestine, colon and anal region, usually spare the rectum whereas lesions of ulcerative colitis usually spare the small intestine but involves the rectum. The lesions of amoebic colitis although concentrated at the caecum can involve the entire colon, rectum and even the anal region (7, 9–12). In the index case, cobblestone lesions and the involvement of the rectum led one to a diagnosis of IBD. These similarities in the clinical and endoscopic features of AUH colitis and IBD may be due to the colon responding to varieties of insults in a monomorphic way (6). Adding to the difficulty in differentiating amoebic colitis from IBD is the possibility of not seeing the amoebic trophozoites and /or cyst in the stool in acute invasive colitis with diarrhoea, and the fact that serologic test are not readily available in all centres (6, 13). The sensitivity of stool microscopic examination in the diagnosis of amoebiasis can reach 100% in localized non-dysentery intestinal disease, but is only about 14–40% in invasive disease (6, 14). In the study by Vinayak *et al* (14), trophozoites of *Entamoeba histolytica* were seen in less than 40% of stool microscopy in patients with invasive intestinal disease. The sensitivity of antibody to *Entamoeba histolytica* detected by counter immunoelectrophoresis, indirect haemagglutination and immunofluorescent tests is about 64%, 80%, and 74% respectively in invasive colitis, but could be as low as 25% in non-invasive intestinal infection (14–16). Because of the similarities in their clinical and endoscopic features, the most accurate way of differentiating AUH colitis from IBD is to take multiple biopsy specimens and look for amoebic trophozoites on histology (13).

It is pertinent to mention that although these two diseases mimic each other, they can also coexist, further complicating the dilemma of differentiating between them (17, 18). Differentiating amoebic colitis from IBD and *vice versa* is very important because a delay in diagnosis and mistreatment in either case can be fatal, especially if steroids should be mistakenly used in AUH colitis, leading to disseminated amoebiasis (6, 8). Since amoebiasis still exist in some Caribbean countries, even if not in endemic proportion, it should be considered in cases of colitis in this region. The investigations that increase the sensitivity of identifying amoebiasis such as examining multiple stool and biopsy specimens for trophozoites and serologic tests should be done on cases of colitis especially in the islands where the infection exists.

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