

# CHRONIC CAECAL AMEBIASIS MIMICKING FEATURES SUGGESTIVE OF ABDOMINAL TUBERCULOSIS AND CROHN'S DISEASE: A CASE REPORT

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## ABSTRACT

A 35 year old man presented with fever, abdominal pain, diarrhea and recurrent oral ulceration for 2 years. Based on examination and investigations, initial diagnosis of abdominal tuberculosis (TB) was made, but there was no response to anti-tuberculosis treatment (ATT) which leads to search for other close differentials. Biopsy of right hemicolectomy specimen raised strong suspicion of chronic caecal amebiasis. Patient was started on amoebicidal medication for 3 weeks. He stayed symptom free on follow up after 3 weeks with healing of the perianal abscess and marked improvement in oral ulcers.

**KEY WORDS:** Abdominal TB, Anti-Tuberculosis Treatment, Caecal Amebiasis.

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## INTRODUCTION

Amebiasis, is a gastrointestinal disorder caused by invasion of the intestine by the protozoan parasite, *Entamoeba histolytica*. It usually involves the cecum and ascending colon. Clinical features of amebiasis, crohn's disease and abdominal tuberculosis (TB) overlap and at times it can be difficult to differentiate them from one another.<sup>1</sup> All these conditions can present with abdominal pain, fever, chronic diarrhea and a palpable mass in the right iliac fossa. Perianal abscesses and fistulas are common to both crohn's disease and complicated amebiasis.<sup>2</sup> Raised ESR, low serum albumin and calcium levels can occur in all these conditions.<sup>3</sup> In such circumstances, tissue diagnosis can be resorted to, which can differentiate caseous necrosis of abdominal TB from non-caseous granulomatous inflammation of Crohn's disease. It can

also highlight the ulcers and granulation tissue of caecal amebiasis alongwith trophozoites of *E.histolytica*. We present a case of chronic caecal amebiasis presented with chronic gastrointestinal symptoms.

## CASE REPORT

A 35 year old patient presented to our medical unit with complaints of abdominal pain, watery diarrhea, fever and recurrent oral ulceration for 2 years. History of weight loss was also there although it was not documented. He was a non-smoker and denied history of arthralgias, visual problems or any skin eruptions. He had taken 6 months conventional interferon and ribavirin therapy for hepatitis C in 2005, following which he had achieved sustained virologic response (SVR). He had a previous history of admission to the same ward in 2009 for

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the same complaints, and suspected to be having Non-Hodgkin lymphoma (with a neutropenic and monocytotic blood picture) but trephine biopsy of the bone marrow denied the suspicion, and no lymph node was there for biopsy. History of acute renal failure and hyperuricemia (uric acid= 16mg/dl) was also there, for which he had undergone 3 sessions of hemodialysis and had subsequently recovered.

On examination he had a blood pressure of 100/60 mmHg, pulse was 110/min and temperature was 100F. He was markedly emaciated, pale, and had multiple aphthous ulcers in the oral cavity. There was no lymphadenopathy, clubbing, conjunctivitis or arthritis. However there was tenderness on abdominal examination, with a palpable liver and a discharging perianal abscess was noted.

Investigations revealed an Hb of 5.7 gm/dl, TLC of 3,200/cmm with differential showing 92% lymphocytes and platelet count of 520,000/cmm. ESR was 105 mm/1st hour, Serum calcium was 7.4 mg/dl. Serum electrolytes, liver and renal function tests were normal. Serum albumin was 3gm/dl. HIV and hepatitis B profile were non-reactive and PCR for HCV was negative. Blood culture was negative for microbes. Mantoux test was negative. An ultrasound of the abdomen showed hepatomegaly with liver size of 17.8 cm but rest of the study was unremarkable. Barium enema was unrevealing, but barium follow through showed a thickened, contracted and deformed ileo-cecal area with narrowings and irregularities noted in the ileum. Patient was started on ATT and steroids on a provisional diagnosis of abdominal TB, with follow up advised after a month.

The patient returned after 2 months with deterioration of his clinical features. Baseline investigations were repeated and ESR was found to be 120 mm/1st hour. The patient was referred to surgical unit for a full thickness terminal ileal biopsy to get a tissue diagnosis to rule out Crohn's disease. On laparotomy, a polypoid, rounded hard mass was seen in the ileo-cecal region extending to the whole cecum along with generalized mesenteric adenopathy. Subsequently the patient underwent a right hemicolectomy with ileostomy, and specimen was sent for histopathology. Histopathology report showed ileo-cecal ulceration, granulation tissue formation and dense lymphoplasmacytic infiltrate. The features were non-specific and there was no evidence of granulomatous process or malignancy. No fungal or parasitic organisms were identified. Crohn's disease was considered in the differential diagnosis but the adjacent bowel showed normal architecture. Considering the localized ulceration, possibility of caecal amebiasis was raised. Following biopsy report, patient's stool specimen was sent for ova, cysts or trophozoites of *E. histolytica*, but the report was negative except for few WBCs. Based on clinical and microscopic (biopsy) suspicion, ATT was withheld, and patient was started on amoebicidal medication for 3 weeks. He stayed symptom free on follow up after 3 weeks with healing of the perianal abscess and marked improvement in oral ulcers.

## DISCUSSION

Our patient had amebiasis with its complications but due to similar nature of clinical features, was mis-diagnosed as abdominal TB and Crohn's disease. Because amebiasis patients erroneously treated for inflammatory bowel disease with glucocorticoids can develop fulminant colitis, accurate initial diagnosis is critical.<sup>4</sup> The major diagnostic challenge for the clinicians is to distinguish the illness from other causes of bloody diarrhea. The

differential diagnosis includes the causes of bacterial dysentery, such as *Shigella*, *Salmonella* and *Campylobacter* species and enteroinvasive or enterohemorrhagic *Escherichia coli*, non-infectious diseases, including inflammatory bowel disease and ischemic colitis.<sup>5,6</sup> Nondysenteric amebiasis may be misdiagnosed as irritable bowel syndrome, regional enteritis or diverticulitis. A right-sided colonic mass may also be mistaken for cancer, TB, actinomycosis or lymphoma. In amebic dysentery, stools are usually less frequent and watery than those in bacillary dysentery. They characteristically contain tenacious mucus and flecks of blood. Unlike stools in shigellosis, salmonellosis and ulcerative colitis, amebic stools do not contain large numbers of WBCs because trophozoites lyse them.

Unusual complications include the formation of enterocutaneous, rectovaginal, and enterovesicular fistulas and ameboma.<sup>7</sup> The polypoid cecal mass in our patient could most likely be an ameboma. Amebomas occur rarely resulting from formation of annular granulation tissue, usually in the cecum and in the ascending colon. Microscopic sections of an ameboma usually reveal dirty necrosis, neutrophils, lymphocytes, eosinophils and granulation tissue. In the dirty necrosis, scattered trophozoites of *E. histolytica* may be seen. Dense inflammatory infiltrate is seen in the submucosa, muscularis propria and serosa. Periodic acid-Schiff (PAS) staining demonstrates bright pink trophozoites. Because ameboma is a rare condition, it is usually discovered only at laparotomy.<sup>8</sup> Only a few cases of colonic amebomas have been reported where the diagnosis was made on colonoscopic biopsy and successfully treated with pharmacotherapy. Metronidazole remains the mainstay of treatment for amebiasis.<sup>9</sup> Surgery is rarely required and is indicated only in cases of diagnostic uncertainty or if any complication occurs.<sup>10</sup>

Microscopic stool examination for trophozoites from a single stool sample

in amebic colitis is only 33-50% sensitive. Examination of 3 stool samples over no more than 10 days can improve the detection rate to 85-95%. Finding the trophozoites in the biopsy specimens may be difficult due to extensive cytolysis, especially in clinically unsuspected cases.<sup>11</sup> Trophozoites are better recognized after PAS staining.<sup>12</sup> This may be one reason why trophozoites could not be visualized in stool and biopsy specimens.

Our patient also had recurrent oral ulceration most likely due to deficiency of vitamin B12, folic acid and iron, associated with the chronic diarrhea.<sup>13</sup> Chronic diarrhea in association with any disease can also lead to hyperuricemia.<sup>14</sup> The persistent diarrhea causes metabolic acidosis and subsequent renal compensation by increasing bicarbonate reabsorption and excretion of hydrogen rich urine, leading to acidic urine and favouring hyperuricemia and uric acid stones.<sup>14</sup> Hypovolemia and rhabdomyolysis can be other possible mechanisms involved.<sup>15</sup>

Caecal amebiasis may be mistaken for abdominal TB or Crohn's disease both of which have a protracted treatment. Owing to its short treatment course and complete cure, clinicians should keep a low threshold for investigating patients for amebic colitis, whenever they present with overlapping symptoms of chronic diarrhea, abdominal pain and fever.

## REFERENCES

- Petri WA Jr, Singh U. Diagnosis and management of amebiasis. *Clin Infect Dis* 1999; 29: 1117-25.
- De Villiers JP, Durra G. Case report: amoebic abscess of the brain. *Clin Radiol*. 1998; 53: 307-9.
- Thomas PD, Forbes A, Green J, Howdle P, Long R, Playford R, et al. Guidelines for the investigation of chronic diarrhoea, 2nd edition. *Gut* 2003; 52 (Suppl 5): v1-15.
- Boulware DR, Stauffer WM, Hendel-Patterson BR. Maltreatment of *Strongyloides* infection: Case series and worldwide physicians-in training-survey. *Am J Med* 2007; 120: 545-8.
- Kelly P, Menzies I, Crane R. Responses of small intestinal architecture and function

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- over time to environmental factors in a tropical population. *Am J Trop Med Hyg* 2004; 70: 412-19.
6. Owens SR, Greenon JK. The pathology of malabsorption: Current concepts. *Histopathology* 2007; 50: 64-82.
  7. Majeed SK, Ghazanfar A, Ashraf J. Caecal amoeboma simulating malignant neoplasia, ileocaecal tuberculosis and Crohn's disease. *J Coll Physicians Surg Pak* 2003; 13: 116-17.
  8. Mendell GL, Benett JE, Douglas RE. Principles and practice of infectious diseases: 6 th ed. New York: Churchill Livingstone; 2005.
  9. Sharma D, Patel LK, Vaidya VV. Amoeboma of ascending colon with multiple liver abscesses. *J Assoc Physicians India* 2001; 49: 579-80.
  10. Ng DC, Kwok SY, Cheng Y, Chung CC, Li MK. Colonic amoebic abscess mimicking carcinoma of the colon. *Hong Kong Med J* 2006; 12: 71-3.
  11. Ray G, Iqbal N. Right colonic mass with hepatic lesion-remember amoeboma? *Indian J Gastroenterol* 2006; 25: 272.
  12. Radovanovic ZL, Katic VV, Nagorni AV, Zivkovic VV, Stankovic TD, Trenkic MS. Clinical diagnostic problems associated with caecal amoeboma: Case report and review of the literature. *Pathol Res Pract* 2007; 203: 823-5.
  13. Thompson D, Milford-Ward A, Whicher JT. The value of acute phase protein measurements in clinical practice. *Annals Clin Biochem* 1992; 29(2): 123-31.
  14. Knight CJ. The investigation of chronic diarrhoea. *Current Paediatr* 2003; 13(2), 89-94.
  15. Ronco C, Inguggiato P, Bordoni V. Rasburicase therapy in acute hyperuricemia and renal dysfunction. *Contrib Nephrol* 2005; 147: 115-23.

**CONFLICT OF INTEREST**

Author declares no conflict of interest

**GRANT SUPPORT AND FINANCIAL DISCLOSURE**

NIL

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