

# Amoebic colitis mimicking a colonic carcinoma

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## ABSTRACT

Amoebic colitis is reportedly common with prevalence reported to be in the range of 9 to 50% population depending on geographic and socioeconomic location. Presentation varies from normal to fulminant colitis and therefore endoscopic and subsequently histopathological studies are essential for diagnosis. We present an interesting case of a gentleman undergoing investigations for recurrent anaemia.

**Keywords:** Amoebic colitis, protozoa, *Entamoeba histolytica*, colorectal cancer, colitis

## INTRODUCTION

Amoebic colitis is commonly reported in tropical countries. <sup>1-4</sup> *Entamoeba histolytica* (*E. histolytica*), a protozoan that typically affects the liver and colon, usually enters via the fecal-oral route. Although majority of patients are asymptomatic, amoebic colitis may also present as fulminant colitis necessitating emergency treatment. <sup>1</sup> Symptoms may mimic inflammatory bowel disease and colonic malignancy. <sup>2, 5</sup> Investigations may reveal mass like lesions resembling colonic tumour. We report a case of an elderly gentleman with symptomatic anaemia undergoing investigations for suspected colorectal cancer.

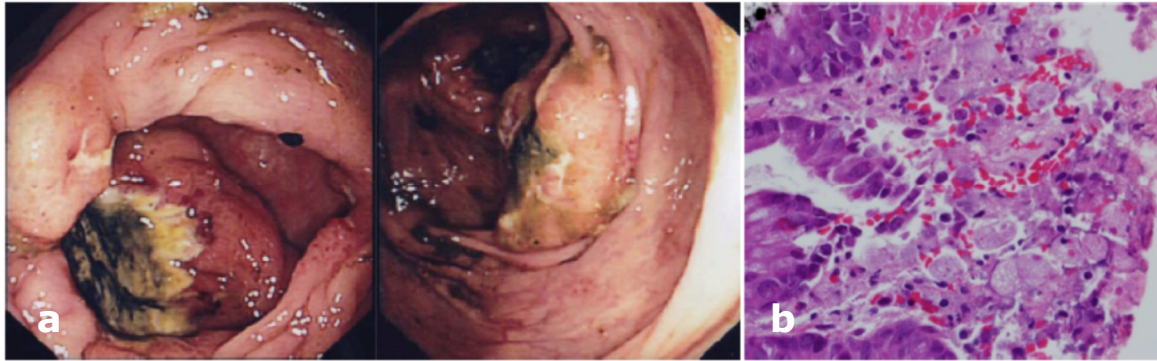
## CASE REPORT

A 71-year-old retired Malay gentleman with well controlled diabetes mellitus for 20 years presented with giddiness and lethargy for a month. He also reported loss of weight of 10 kg within three months. There was no fever,

no abdominal pain and no history of altered bowel habits. He was previously admitted to another hospital with symptomatic anaemia and was transfused with three pints of packed cell blood. He lives in an urban area and no recent travel was reported. Clinical examination revealed a cachectic and pale gentleman. No ecchymosis was seen. Abdomen was soft with no tenderness. There was no organomegaly and no palpable peripheral lymph nodes. Digital rectal examination showed brown stools. Examination of other systems was unremarkable.

Blood investigations showed hypochromic microcytic anaemia of 8.6 gm/dL. White cell count was normal with no evidence of eosinophilia. Iron level was low at 5.0umol/L, transferrin saturation of 15% and ferritin level 3,218 umol/L (patient on oral iron replacement). Full blood picture showed pictures suggestive of iron deficiency anaemia. Liver function test was normal with albumin level of 38 mmol/L. Stool for ova and parasite were negative. Upper gastrointestinal endoscopy was normal. Colonoscopy showed three

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**Figs. 1:** a) Colonoscopy showing an ulcerated lesion at the right colon affecting the caecum, ascending colon and also proximal transverse colon, b) Histology showing clumps of amoeba (PAS, x60).

mucosal masses with ulcerated surface seen at the caecum, proximal ascending colon and proximal transverse colon (Figure 1a). Histopathology studies showed lamina propria infiltrated by neutrophils, eosinophils, lymphocytes and plasma cells. No granuloma, no dysplasia or evidence of malignancy seen. Free lying clumps of *E. histolytica* trophozoites admixed with inflammatory exudates were seen (Figure 1b). A diagnosis of amoebic colitis was made and metronidazole 400mg twice daily was prescribed for six weeks.

The patient remained asymptomatic during treatment. Colonoscopy surveillance performed after completion of therapy showed regression of the colonic lesions leaving a single area of raw ulcerated mucosa at transverse colon.

## DISCUSSION

*E. histolytica* is the major cause of diarrhoea in the tropical countries and is the causative agent of amoebiasis and amoebic colitis. The reported prevalence rate in Malaysia vary between 1.0% and 40.7%.<sup>2</sup> In Asia, the reported prevalence rate is higher among developing countries especially in India followed by Indonesia.<sup>6</sup> It is now less common and even rare in well-developed nations due to improved standard of living. Higher prevalence has been noted in the immunocompromised

patients.

The infestation of the protozoan *E. histolytica* cyst is via the faecal-oral route. The cysts are digested into the intestinal lumen releasing trophozoites. The trophozoites reproduced by clonal expansion and subsequently forming cysts and a new cycle of infection is started. Liver and gastrointestinal tract are the main organs commonly affected by this type of protozoan.

Amoebic colitis can occurred at any part of colon with predilection at caecum and ascending colon.<sup>6</sup> The spectrums of disease presentation of amoebic colitis ranges from asymptomatic patients, symptomatic non-invasive infection (fever, abdominal pain, weight loss and anorexia), dysentery and fulminant colitis.<sup>5</sup> Amoebic colitis are also common among inflammatory bowel disease patient in particular ulcerative colitis.<sup>7</sup>

The diagnosis of amoebic colitis depends on demonstration of *E. histolytica*. The conventional method of diagnosis by stool microscopy is a test with poor sensitivity as compared to serology antigen test for *E. histolytica* which has both high sensitivity and specificity.<sup>5, 8, 9</sup> Colonoscopy with colonic biopsy although invasive, is now the gold standard for diagnosis as it reveals the

trophozoites confirmed via PAS or Geimsa stain. Typical histological finding from colonic biopsy is flask-shaped ulcer with exudate and necrotic tissue.<sup>8</sup> Amoebic colitis may masquerade as inflammatory bowel disease or colitis as evidence inflamed or haemorrhagic mucosal wall with inflammatory cells. However presence of amoebic trophozoites within inflammatory exudate differentiates the diagnosis. Therefore it is essential to ensure proper diagnosis to avoid unnecessary treatment with glucocorticoids and avoid mismanagement.

Treatment goal is to treat the invasive disease and eradicate the infective organism. Metronidazole (500mg-750mg three times daily) is considered to be drug of choice to treat amoebic colitis with duration five to 10 days follow by paromomycin (depend on availability and accessibility) which acts to eradicate colonisers intraluminally.<sup>5, 10</sup>

Other agents include Tinidazole (2g daily for three days) and iodoquinol (650mg three times daily for 20 days) which are effective against luminal forms of amoebic infestation. Chloroquine (1g daily for two days followed by 500mg daily for 14-21 days) is effective for extraluminal infestation in particular hepatic amoebiasis.

Monotherapy with metronidazole, which is widely available and accessible, may be adequate treatment as illustrated in this case. However, combination anti-amoebic therapy shows less parasitological failure compared to monotherapy albeit criticism of the lack of well designed studies.<sup>10</sup>

Surgical intervention is reserved for fulminant colitis with perforation.

In conclusion, amoebic colitis is not frequently encountered although reportedly common in tropical countries. The patient

may present with varying complaints including mimicking colorectal malignancy as demonstrated in this case. As such amoebic colitis should be considered as a differential diagnosis besides the more common diagnosis of colorectal cancer diagnosis or inflammatory bowel diseases particularly in endemic tropical region. Early histological confirmation of the disease allows eradication therapy, improvement of quality of life and avoids unnecessary surgical intervention. The role of monotherapy vs combination therapy needs to be evaluated further in better-designed studies.

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