"AMOEBIC COLITIS MIMICKING COLONIC CARCINOMA: A CASE REPORT"

Case report by:

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SIGNIFICANCE:

The clinical presentation of *Entamoeba histolytica* can mimic more common diseases such as colonic malignancy and IBD. Moreover, endoscopic diagnosiscan be non specific and can be easily overlooked. We report a case of amoebic colitis in which the presentation, radiologic and endoscopic findings closely resembled colonic carcinoma. Additional knowledge of its atypical presentation would help in addressing clinical questions, sparing inappropriate evaluations and avoiding surgeries.

CLINICAL PRESENTATION:

A 60 year old, female, presented diarrhea and weight loss for 8 weeks. She also complained of easy fatigability and lightheadedness. Clinical examination revealed a pale patient with dry skin and buccal mucosa. Abdomen was soft withnon-tender palpable mass in the right iliac fossa.

MANAGEMENT:

Work ups:

CBC showed anemia and fecalysis was consistent with inflammatory diarrhea with no parasites seen. Abdominal CT revealed circumferential wall thickening in the ascending colon extending into the cecum. Colonoscopy revealed circumferential wall thickening with some areas of indurated ulcers from cecum to ascending colon with narrowing of the lumen. Histopathologic report revealed trophozoites with ingested RBC amidst the inflammation and hemorrhage. TB PCR and CEA were unremarkable.

Diagnosis:

Based on colonoscopy with biopsy result

Treatment:

Recommended treatment with Metronidazole and Diloxanide furoate. Response to treatment was monitored by repeat colonoscopy.

RECOMMENDATION:

Presumptive diagnosis based on a lesion's appearance is important for initial work up but only objective data enables definitive diagnosis. High index of suspicion, detailed history taking and early histologic confirmation are crucial for diagnosis, and are essential to avoid unnecessary surgery.

KEYWORDS: Case report, Entamoebahistolytica, Amoebic colitis, Colonic Carcinoma

INTRODUCTION

Amoebiasis is an infectious disease caused by *Entameba histolytica*, and is the second leading cause of death from parasitic diseases worldwide [1]. Most mortality and morbidity from amoebiasis occurs in developing countries because the infection is commonly acquired by ingestion of contaminated food or water and is therefore endemic in countries with poor sanitation. In the Philippines, the prevalence of amoebisias has not been well documented. In textbooks, the prevalence rate is indicated to be 30-33%, but biomedical surveys have contested this result and reported a prevalence of 2-8% [2]. Earlier this year, amoebiasis was considered as one of the top causes in student absences in the country [3].

Infection occurs following ingestion of cysts in fecally contaminated food or water [4]. Approximately 80 to 90% of infected persons remain asymptomatic. Only 10-20% of infected persons develop invasive disease characterized by invasion of colonic epithelium by trophozoites. It can also gain access to the bloodstream and gain spread hematogenously to establish infection to distant sites commonly in the liver. Malnutrition, infancy, old age, pregnancy and patients receiving glucocorticoid are said to be at an increased risk for fulminant disease [5].

More than 90% of patients with amoebic colitis presents with diarrhea. Other symptoms, such as tenesmus and fever, are highly variable. In addition, weight loss is common and usually associated with chronicity of the illness.

The gold standard for diagnosis of amebic colitis remains full colonoscopy with biopsy since the most commonly affected sites are the cecum and ascending colon. Colonoscopic finding is also variable from simply appearing indurated with no ulcerations to classical finding of multiple punctate ulcers 2 to 10 mm seen intervening a normal colonic mucosa. In severe cases, ulcers can coalesced and epithelium may appear necrotic. [5].

Although the disease is relatively common in the Philippines, it is of particular note in that its presentation can be varied and mimic more common conditions, such as colonic malignancy and inflammatory bowel disease. We report a case of colonic amoebiasis in which the presenting symptoms, radiologic and endoscopic findings closely resembled colonic carcinoma.

CASE REPORT

A 60 year old female presented in our institution with a 8 week history of diarrhea. Patient claimed to have 8 episodes of watery, non-bloody, non-mucoid, brownish stool a day amounting to 1 to ½ cup per bout. She also noted weight loss of 7 kg within 2 months. Patient also complained of easy fatigability, giddiness and lightheadedness for a month. No other associated symptoms such as abdominal pain, fever, nausea, vomiting nor tenesmus. Patient is a known hypertensive maintained with Amlodipine 10 mg tab daily. There was no reported intake of other medications, herbal drugs nor food supplements. Her father died of lymphoma. There was no other heredofamilial diseases disclosed and no history of recent domestic nor foreign travel. She claimed to have good eating practices and uses purified water for drinking purposes. She is not fond of

eating street food nor ready-to-eat foods being offered outside their house.

She was initially seen by a general practitioner in the province wherein she was subsequently admitted due to symptomatic anemia and was transfused with three units of packed red blood cells. She was also given Ciprofloxacin 500 mg tab twice daily for 1 week. Whole abdominal ultrasound was also requested revealing 11.4 x 4.1 cm colonic mass with echogenic center in the right iliac region and also finding of diffuse fatty liver. The physician, alarmed with the findings on the ultrasound, advised her to seek further consult to specialist.

Patient sought consult to our institution with the same complaint. Clinical examination revealed a pale patient with dry skin and buccal mucosa . Abdomen was soft with non tender palpable mass in the right iliac fossa. There was no organomegaly and no palpable peripheral lymph nodes. Digital rectal examination showed watery brown stools. Examination of other systems was unremarkable. Patient was initially admitted under the General Surgery service with consideration of colonic mass, probably malignant. The patient was worked up with a working impression of colonic carcinoma. Laboratory results revealed anemia (hgb 92 g/L) and leukocytosis (13.4 10*9/L). The fecalysis revealed pus cells of 4-10/hpf and red blood cells of 25/hpf. No ova nor parasites seen. Other blood chemistry results were unremarkable. CEA was also requested. CT scan of her abdomen was performed and revealed circumferential wall thickening in the ascending colon that extended into the caecum (Figure 1) approximately 13 cm in length which yielded an impression of colonic neoplasm. Patient was also referred to Gastroenterology service for colonoscopy and biopsy.



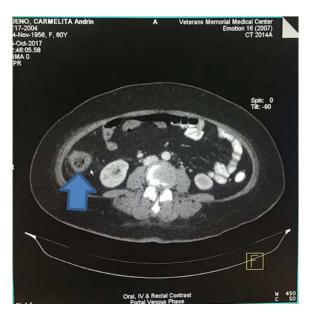


Fig. 1. Abdominal Ct scan (coronal and axial view) showing circumferential wall thickening at the cecum and ascending colon, approximately 13 cm in length

The colonoscopy was performed which revealed circumferential wall thickening with some areas of indurated ulcers from cecum to ascending colon with associated narrowing of the lumen (figure 2a and 2b). Endoscopic diagnosis was non specific colitis cecum to ascending colon s/p multiple random biopsy. The colonoscopic features closely resembled colonic carcinoma hence it was considered although out the diagnostic process. Multiple biopsies were obtained for histopathologic examinations and TB PCR.





Fig 2a. Fig 2b.

Fig. 2a and 2b. circumferential wall thickening with friable mucosa and with some areas of indurated ulcers from cecum to ascending colon

Histological examination of the biopsies taken during colonoscopy demonstrated trophozoites of *Entamoeba histolytica* with ingested RBC seen amidst the inflammation and hemorrhage (Figure 3).

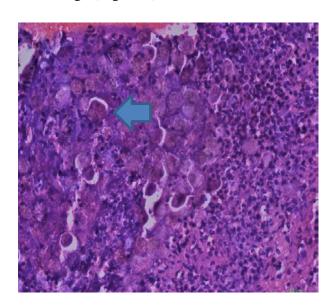


Fig 3. Amoebic trophozoites with ingested red blood cells seen amidst the inflammation and hemorrhage

Furthermore, the TB PCR was negative and CEA was also within normal levels. The patient was started with Metronidazole for 2 weeks followed by an antiluminal agent, Diloxanide Furoate for 10 days. A repeat colonoscopy was performedafter 6 weeks and revealed marked regression of the severely edematous and ulcerated mucosaleaving minimal areas of shallow ulcerations(figure 4a and 4b). The symptoms of the patient were markedly improved after treatment and was asymptomatic on subsequent follow ups. Furthermore, patient was advised for proper food handling and good hygiene at all times for prevention of infection.





Fig. 4a and 4b. marked regression of the previously noted thickened, friable, ulcerated mucosa from cecum to ascending colon leaving minimal areas of shallow ulcerations

DISCUSSION

Entameoba histolytica is one of the most important enteric pathogens affecting people worldwide, causing diarrheal disease. High rates of colonization with the parasite burden many developing countries [6]. The two main organ systems affected by the parasite are the GI tract and the liver. The presentation of intestinal amoebiasis ranges from an asymptomatic carrier state, colitis, through abscess formation to perforation. Almost 90% patients with symptomatic amebic colitis present with a history of bloody or watery diarrhea. Moreover, abdominal pain,tenesmus and fever are highly variable symptoms [5]. The insidious onset with fever and bloody stools absent in most cases complicates the diagnosis. The present case is atypical in that the patient had no preceding febrile episode, bloody diarrhea, and a definite travel history.

Four case reports have discussed amebic colitis mimicking carcinoma of the colon. Two of which presented with anobstructing right sided colonic mass hence emergency surgery was performed[7,8]. The other case report proceeded to surgical resection of the mass due to the possibility of colonic malignancy. Diagnosis of amoebic colitis was made by pathological examination of the resected specimen. There were also amebae parasites present within the

inflammatory cellular exudates in most of theulcers examined microscopically, but there was no granuloma formation, dysplasia or malignancy seen [10]. Another case report presented with weight loss and recurrent anemia which was initially worked up as colonic malignancy but histology revealed presence of free flying lumps of *Entamoeba histolytica*.

The presentation and endoscopic findings of amoebic colitis can also mimic inflammatory bowel diease. In a case report, a patient with repeated rectal blood loss without diarrhea underwent colonoscopy showing discontinuous ulcerative enterocolitis of the ileocecal region. Because of a high suspicion of Crohn's disease, patient was started withEntocort 9mg daily.Histological examination of the biopsies revealed the causative trophozoites of *Entamoeba hystolitica* hence the patient was started with Metronidazole. Fortunately, there was no serious complications occurred. This report emphasized the importance to exclude amoebic colitis prior to undertaking steroid therapy in patients suspected with Crohn's disease [9].

In developing countries the diagnosis is most commonly made by identification of cysts or motile trophozoites on a saline wet mount of stool specimen. The drawbacks of this method, however, include its low sensitivity and false positive results due to the identification of noninvasive Entamoeba species such as *Entamoeba dispar* infection. Therefore, this method should not be relied upon [5]. The diagnosis can also be based on the detection in stool of E. histolytica by specific antigen or DNA and by the presence of antiamebic antibodies in the serum. The serum indirect hemaglutination antibody (IHA) and the enzyme-linked immunosorbent assay (ELISA) are highly sensitive (90-100%) and turn positive within 7-10 days of infection. Serologic tests accurately distinguish *Entamoeba dispar* from *Entamoeba histolytica* infections since antiamebic antibodies do not develop in patients infected from *Entamoeba dispar*. From 70 to 85% of patients with acute amebic colitis have detectable antiamebic antibodies on presentation[5]. However, these tests are unable to distinguish between active or prior invasive infection since antibodies can remain detectable for as long as 20 years [9]

The gold standard for diagnosis of amebic colitis remains colonoscopy with biopsy [5]. The cecum and ascending colon are the most common affected sites complicating the diagnosis since GI TB and Crohn's disease are also common on these sites. Hence, these disease entities should be considered and ruled out as well. Histopathological findings associated with amebic colitis include diffuse, nonspecific mucosal thickening, focal ulceration with or without amoebae. The amoeba parasites are lying on top of the ulcera, sometimes extending in the submucosa. The ingested erythrocytes such as seen in figure 3 help to distinguish true parasites from swollen and degenerated epithelial cells.

The patient was treated with Metronidazole 500 mg three times a day for 2 weeks then started with an antiluminal agent, Diloxanide 500 mg for 10 days. Nitroimidazoles is the mainstay of the therapy for invasive amoebiasis. Either Metronidazole 500-750mg 3 times daily for 10 days or Tinidazole 2 g once daily for 3 to 5 days to eliminate invasive trophozoites [5] In the rare case of fulminant amoebic colitis it is practical to add broad-spectrum antibiotics to treat intestinal bacteria that may spill into the peritoneum. In addition, surgical intervention is required for complications such as acute abdomen, gastrointestinal bleeding, or toxic megacolon. In about 40-60% of the patients receiving Nitroimidazole, parasites are not eradicated. Therefore, Nitroimidazole treatment should be followed by Paromomycin or the second-line agent Diloxanide

furoate to eradicate luminal infection and prevent recurrent disease [10]. After combination therapy, the cure rates reaches approximately 86–90%[12]. Monotherapy with Metronidazole may be an adequate treatment as revealed in a casereport whereinMetronidazole 400mg twice daily was prescribed for six weeks in a patient with amebic colitis mimicking colon carcinoma. The patient remained asymptomatic during treatment. However, combination anti-amoebic therapy still displays less parasitological failure as compared to monotherapy albeit criticism ofthe lack of well designed studies. The role of monotheraphy versus combination therapy needs further evaluation with better designed studies [13].

There is no definite time when to perform repeat colonoscopy for monitoring of treatment response in patients with amebic colitis. In a case report of an amoebic colitis mimicking colonic carcinoma, colonoscopy surveillance performed after completion of therapy showed regression of the colonic lesions leaving a single area of raw ulcerated mucosa at transverse colon [13]. In countries where there is a high prevalence of *Entamoeba histolytica*, such as in Mexico and El Salvador, patients are empirically given with Metronidazole and Ciprofloxacinwhen infection from this parasites is highly suspected. After six weeks, a full colonoscopy is performed to evaluate treatment response [11]. In a case report of a patient from Taiwan with amoebomamimicking colon carcinoma, the follow-up colonoscopyshowed marked improvement after 4 months and complete remission after 8 months post treatment with Metronidazole and Iodoquinol [12]. For our case, a surveillance colonoscopy was done after 6 weeks which revealed marked regression of the thickened, ulcerated, friable mucosa leaving only several areas of shallow ulcerations on the previously involved segments.

Amoebiasis is relatively common in developing countries and it should be included as one of the differential diagnoses of a seemingly colonic mass. The clinical presentations as well as endoscopic and radiologic findings are highly variable such that it can cause a diagnostic dilemma which can further lead to additional unnecessary work up and even surgeries. In previous reports of unsuspected amoebic colitis presenting as different colonic pathology, misdiagnosis, delay in antibiotic treatment and institution of immunosuppressive therapy have resulted in serious morbidity and mortality [9]. Therefore, it is imperative to emphasize that presumptive diagnosis based on a lesion's appearance could be important only for theinitial therapy or work up but only objective data should enable definitive diagnosis and specific therapy. Furthermore, a high index of suspicion, detailed history taking and early histologic confirmation are crucial for diagnosis, and are essential to avoid unnecessary surgery.

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