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Case Report

Persistent abdominal pain of a middle aged old female– Think of Amoebic Colitis

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Abstract:

Amebic colitis, also known as amebiasis, is a gastrointestinal disorder caused by invasion of the intestine by the protozoan parasite, Entamoeba histolytica. Although primarily a disease found in underdeveloped countries, this condition may exist in patients who have recently travelled these area. Obtaining an accurate history and testing are crucial for correctly diagnosing and treating amebic colitis. Here we are reporting a case of Amoebic Colitis who has presented with severe abdominal pain and diarrhoea.

Key Words: *Amoebic colitis, Amoebiasis, Amebomas, Entamoeba Histolytica, Metronidazole.*

Introduction:

Entamoeba histolytica is one of the commonest parasitic infections worldwide, infecting about 50 million people.[1] The World Health Organization suggests that this pathogen is responsible for 70,000 deaths annually, second only to malaria as the leading cause of death from a parasitic infection worldwide.[2] Amebiasis due to invasive infection with *Entamoeba histolytica* is said to affect up to 50 million individuals worldwide. Undiagnosed amebiasis can lead to fulminant intestinal infection and liver abscesses that are associated with a high mortality rate and cause about 100,000 deaths every year. Amebiasis is most prevalent in developing countries, but a high incidence is also encountered in Western countries, among Human Immunodeficiency Virus-infected individuals and travelers returning from endemic areas. Amebiasis classically presents with chronic dysentery and is one of the important differential diagnoses of inflammatory bowel disorders in areas where it is highly prevalent. The rising incidence of inflammatory bowel disease (IBD) in many of these countries has increased the importance of being able to differentiate them from amebiasis. This is particularly important because amebiasis can be cured by antibiotics, whereas IBD requires long-term anti-inflammatory therapy. The use of immunosuppressants for IBD could precipitate fulminant disease or dissemination of infection in individuals with amebiasis. Amebiasis is classically associated with discrete, flask-shaped mucosal ulcers, commonly located in the cecum and the rectum, but irregular ulcers surrounded by edema and erythema, resembling those seen in IBD, have also been described[3]. The prolonged nature of the infection and the intense mucosal damage that are often associated with amebiasis are factors that could contribute to distortion of the mucosal architecture and induce significant chronic inflammation, both of which are classical features of IBD.

Case Report:

71 years old Bangladeshi lady presented to us with the complaint of lower abdominal pain for 2 to 3 months which is cramping in nature, moderate to severe in intensity without any radiation & passage of loose stool mixed with fresh blood for last 15 days. There was no aggravating or reliving factor of her pain ..She also had some sorts of continuously present abdominal discomfort for last 2 to 3 months associated with flatulence, abdominal bloating sensation & nausea. She developed fever (highest recorded temp was 102 at home) for last 2 days which was continued in nature. Regarding past medical history-she is known case of HTN, Sick sinus syndrome (SSS) & Parkinson‘s Disease. She is on regular medication for above mentioned conditions. Her Physical examinations revealed that she was consious ,oriented ,anaemic, non-icteric ,no lymphadenopathy. BP- 140/80 mmHg.Pulse-56 b/m, abdomen- Soft, moderately tender left hypochondriac & lumber region associated with a swollen lumpy mass in left iliac fossa. Lungs- B/S vesicular, no added sound, 1st & 2nd H.S are normal. Involuntary movement presentin hands & face -clinical feature of parkinson's .Laboratory Investigations revealed CBC: Tc-WBC : 19,500/mm3, N-90.4%, lymphocyte- 6.3 %, Hb : 10.4%, S.electrolytes:Na-133 mmol/l, K- 3.15 mmol/l. S.creatinine: 1.17 mg/dl, Blood C/S & urine C/S showed no growth. We did a colonoscopy, findings of which is suggestive of Amoebic Colitis but Koch's colitis needed to be ruled out (fig. 1 & 2). Biopsy of colonic tissue confirmed the diagnosis. Report showed necrotic material admixed with mucin, proteinaceous exudate, and blood clot lining ulcers, trophozoite and cyst are present.

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Patient was treated with I/V fluid ,adequate nutritional Support & Inj. Metronidazole 500mg I/V- 8 hourly initially & then orally- for total 10 days., Other previous ongoing treatment of SSS & Parkinson´s disease is continued.

On follow up visit after 10 days, all symptoms of the Patient have been resolved with improved general well being, except loss of appetite. She gained weight & abdominal pain & discomfort were subsided.Clinical examination showed soft & non-tender abdomen,lumpy feeling of thickened gut wall disappear on deep palpation.

On 2nd follow up after 15 days she recovered from side effect of oral Metronidazole, with improved appetite. There was no subsequent recurrence of abdominal pain.

Discussion

Amoebiasis is the second most common cause for mortality among parasitic infections caused by Entamoeba histolytica. The word "histolytica" stands for eating up the tissue which was vividly demonstrated in the index case. 90% of the individuals are asymptomatic and only 0.5% of them have a risk of developing invasive disease and fulminant colitis. It is usually common in immunocopmpromised individuals, pregnant women and malnourished children.[4] Diffuse ulceration of the ileum and colon with perforation and serositis can occur due to inflammatory bowel disease, ischaemic colitis, vasculitis and infective causes. In this context, gross and microscopic examination of the vessels is an extremely important adjunct to a proper diagnosis. Moreover, a diffuse ulcer with shaggy necrotic base suggests an infective/ inflammatory origin rather than a vascular insult. Ulcerative colitis is a disease that mostly involves the rectosigmoid although ileo-caecal region can be involved, however complete sparing of the descending colon and rectosigmoid should arouse the suspicion of an alternative diagnosis. Moreover, a transmural involvement is rare in the setting of ulcerative colitis, though it can happen in toxic megacolon. The index case however did not show any luminal dilatation or thinning of the wall precluding a gross diagnosis of toxic megacolon. Cröhn's disease involves the small and large intestine in patches with typical skip areas and transmural involvement is also common.[4,5] In the index case, no skip area, creeping fat or fistula was noted. Microscopically, there were no features of activity or chronicity to suggest a possibility of inflammatory bowel disease. Amoebiasis, as mentioned can be associated with rarer complications such as amoebic brain abscess. Surgical management is warranted in cases of fulminant colitis, peritonitis and abscess formation. Male gender, peritonitis, hypo-albuminaemia and electrolyte imbalance are some poor prognostic factors in fulminant colitis. Early surgical intervention is such cases have been found to decrease the mortality which is 55- 87.5% on conservative management. It is important to rule out an infectious colitis in the form of co-infection or super-infection prior to the beginning of steroid therapy in a suspected case of inflammatory bowel disease to prevent its devastating effects. The definitive diagnosis is hence based on the histo-pathological examination of the specimen. This again highlights the importance of having a suspicion of amoebic colitis even in such rare presentations as an early surgical intervention and appropriate amoebicidal agent can be life saving. Fulminant colitis, toxic megacolon complicate less than 0.5% of cases, whereas multiple colonic perforations have been reported in upto 75% of cases described by Chen et al.[6] Computed tomography scan is the established gold standard in assessing the presence and complications of amoebic and solid organ involvement. Trophozoites are also able to cause host cell death by causing the cell to enter apoptosis, or programmed cell death. This process adds to the ability of the ameba to invade the mucosa via channels created by macrophages called to destroy the dying epithelial cell . Once in the host's mucosa, the trophozoite is able to continue its tissue destruction in a lateral direction. This destruction results in an ulcer that is characteristically "flask-shaped," with a wide base and a thin neck that extends to the intestinal lumen. [7] This ulcer produces bloody stools that are a hallmark of amebic colitis .[8] The host is at risk of developing paralytic ileus, sloughing of colonic mucosa, and perforation of the GI tract, causing fulminant amebic colitis, which has a mortality rate of greater than 40%. Toxic megacolon may also develop, particularly if the patient has been treated with corticosteroids, which inhibit the inflammatory process and allow E. histolytica to continue attacking the now further compromised host. Trophozoites in the mucosa may be able to escape the intestinal mucosa and invade the liver and brain.[9] Entering the liver through the venous portal system, E. histolytica attacks hepatocytes and neutrophils, producing liver abscesses and causing further liver damage due to the release of cytokines from the damaged neutrophils. Abscesses consist of soft, necrotic, acellular yellow-brown debris, described as "anchovy paste.[10] Brain abscesses due to amebic invasion are rare, with death occurring in about half of patients developing this complication. Trophozoites are able to encase themselves in cysts, which are then excreted in the feces. An individual coming into contact with the infected feces is then at risk for developing amebiasis.Ulcers with undermining edges and hemorrhagic material in their bases separated by normal mucosa are classically described as the mucosal change associated with amebiasis. UC and CD, unlike amebiasis, show ulceration in association with other mucosal changes such as edema, erythema, loss of vascularity, friability, and nodularity. Amebiasis is also known to produce mass lesions called amebomas. Amebiasis can rarely present with perforation or toxic dilatation of the colon, Amebic trophozoites invade the mucosa and digest the tissue.This was evident in the mucosal biopsies as necrotic material admixed with mucin, proteinaceous exudate, and blood clot lining ulcers and was seen in all the cases of amebiasis reviewed. Trophozoite forms of ameba were seen in the necrotic material lining sites of ulceration, in separate fragments of necrotic material as well as over intact mucosa. Chronic inflammation and architectural alteration are the histological hallmarks of IBD. In infectious colitis, chronic inflammation is confined to the superficial part of the mucosa, unlike IBD that shows basal plasmacytosis.

Treatment-Colonization with *E. histolytica* should be treated

with a luminal agent alone; *E. dispar* infection does not require treatment. Oral drugs that are effective against luminal infection include diloxanide furoate, paromomycin and iodoquinol. The recommended duration of treatment with paromomycin is 7 days, with diloxanide furoate is 10 days, and with iodoquinol is 20 days. In a case in which luminal agents cannot be used, it seems a reasonable (if unproven) approach to treat luminal infection with metronidazole and test for cure with the stool antigen detection test.

Conclusion:

Amebic colitis can present a true medical emergency. Accurate history taking, including questions of recent travel to underdeveloped countries, is essential . Microscopy of stool samples for evidence of E. histolytica is no longer considered diagnostic. Amebic trophozoites are most frequently located within the necrotic material, mucin, proteinaceous material, and blood clot lining ulcers. Treatment includes maintenance of the airway, breathing, and circulation, as well as antibiotics such as metronidazole and paromomycin .[10] Broad-spectrum antibiotics should be included for fulminant amebic colitis . The use of loperamide to control diarrhea is discouraged. Persons who are planning to travel to underdeveloped countries should be educated about food and water preparation. Early diagnosis and specific treatment can prevent the devastating outcome of Amebic Colitis as describe above.

References:

1. van Hal SJ, Stark DJ, Fotedar R. Amoebiasis: current status in Australia. Med J Aust. 2007; 186(8): 412-6
2. WHO. Life in the 21st Century: a vision for all. The World Health Report 1998. Geneva, Switzerland: World Health Organization; 1998
3. World Health Organization (WHO). Amoebiasis. WHO Wkly Epidemiol Rec 1997;72:97-100.
4. Gonzales ML, Dans LF, Martinez EG . Antiamoebic drugs for treating amoebic colitis. Cochrane Database Syst Rev (2009).CD006085
5. Petri WA Jr, Singh U. Diagnosis and management of amebiasis. Clin Infect Dis 1999;29:1117-25.
6. Chen WJ, Chen KM, Lin M . Colon perforation in amebiasis. Arch Surg. 1971;103(6):676-680
7. Pai SA. Amebic colitis can mimic tuberculosis and inflammatory bowel disease on endoscopy and biopsy. Int J Surg Pathol 2009;17(2):116-21.
8. Gill GV, Beeching NJ. Tropical Medicine. 5th ed. Oxford, UK: Blackwell Publishing; 2004:153-159
9. Ravdin JI. Pathogenesis of disease caused by Entamoeba histolytica Studies of adherence, secreted toxins, and contact-dependent cytolysis. Rev Infect Dis 1986;8(2):247-60.
10. Ravdin JI, Stauffer WM. Entamoeba histolytica (amebiasis). In:Mandell GL, Bennett JE, Dolin R, eds. Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases. Vol 2. 6th ed. Philadelphia, PA: Churchill Livingstone; 2005:3097-3111.
11. Gupta SS, Singh O, Shukla S, Raj MK . Acute fulminant necrotizing amoebic colitis: a rare and fatal complication of amoebiasis: a case report. Cases J 2009;2:6557.

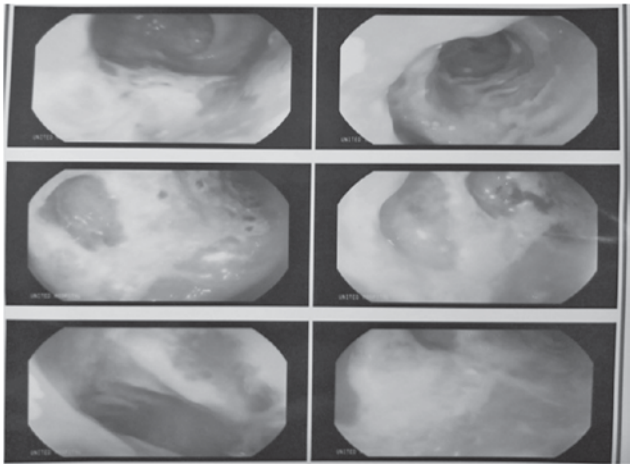

Figure : 1

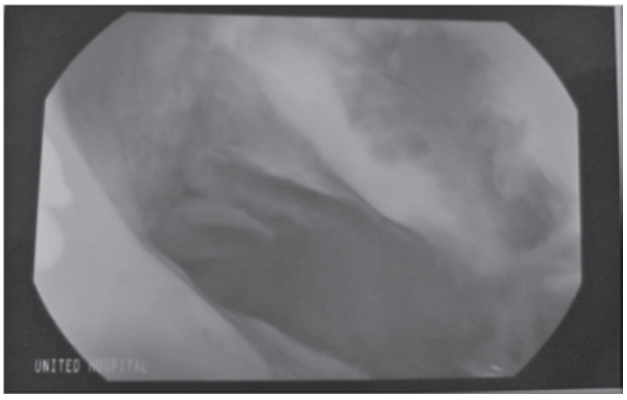

Figure: 2