Case Report

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Ameboma mimicking cecal growth in a patient with autoimmune hepatitis/primary sclerosing cholangitis overlap syndrome

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Introduction

Ameboma is a relatively uncommon manifestation of amebiasis, more common in cecum and the ascending colon. It may mimic inflammatory bowel disease, colon cancer, tuberculosis or fungal infection on colonoscopy. We report a case of ulcero-proliferative cecal ameboma in a patient with autoimmune hepatitis / primary sclerosing cholangitis (AIH / PSC) overlap syndrome. We wish to highlight the importance of differentiating the cecal ameboma from malignancy (high risk in PSC) and its treatment before initiating corticosteroids (for treatment of AIH) from this case.

Case report

A 43-year-old teetotaler with no history of liver disease, blood transfusion, complementary and alternative medication was found to have abnormal liver function tests (LFTs) during routine health check up with raised aspartate aminotransferase (AST) 700 IU/L, alanine aminotransferase (ALT) 1,308 IU/L, alkaline phosphatase (ALP) 358 IU/L and total bilirubin (T.Bil) 3.7 mg/dL. Evaluation for etiology of liver disease revealed negative hepatitis viral serologies, normal serum copper, ceruloplasmin, antinuclear antibody (ANA) titers of 1:80, serum IgG of 2,144 mg/dL and positive antimitochondrial antibodies (AMA). Ultrasonography and Magnetic Resonance Cholangio-Pancreatography (MRCP) were unremarkable. Liver biopsy showed moderate mixed portal and mild lobular inflammation with moderate interface activity, periportal focal bridging fibrosis, occasional duct paucity in portal tracts with no significant ductulitis or granuloma based on which he was diagnosed to have AIH/PBC overlap syndrome.

His complete blood count (CBC) were normal with a platelet count of 2,10,000/mm^3. Surveillance colonoscopy to look for associated colitis revealed an ulcero-proliferative growth in the cecum which on biopsy revealed no evidence of dysplasia or malignancy, a preserved architecture, crypt abscesses, superficial ulcerations, moderate mixed inflammatory infiltrates in the lamina propria and Periodic Acid Schiff (PAS) positive amebic trophozoites in the slough. He was diagnosed to have Ameboma was treated with oral Metronidazole for 10 days followed by diloxamide furoate for 2 weeks. Follow-up colonoscopy revealed focal hyperemia and normal histology.

Discussion

Amoeboma is an uncommon manifestation of amoebiasis, and can mimic both carcinoma and inflammatory bowel disease.[1] Contrast-enhanced tomography and colonoscopy are sensitive tools for the work up of differential diagnosis of a cecal mass. Endoscopic evaluation yields a definitive diagnosis in about 66% of cases. Because ameboma is a rare condition, it is usually discovered at laparotomy.[2] Only a few cases have been reported where the diagnosis was made by biopsy through a colonoscopy and successfully treated with pharmacotherapy.[2] Patients with inflammatory bowel disease may also have amebiasis posing a considerable diagnostic challenge in endemic countries.

Corticosteroids are the first line treatment of AIH. However, they are also a known risk factor for developing fulminant amebic colitis in patients with symptomatic or asymptomatic intestinal amebiasis. Fulminant amebic colitis, though uncommon, is associated with high mortality and morbidity, with case fatality rates ranging from 40% to 89% by causing life-threatening complications such as bowel necrosis, toxic megacolon, perforation and peritonitis.[3,4]

In patients in endemic areas or those with recent travel history to these areas, amebic infection should be considered before using corticosteroids. An accurate diagnosis of ameboma is critical as in our case to avoid potentially fatal fulminant colitis especially in tropics (Figure 1) (Figure 2).

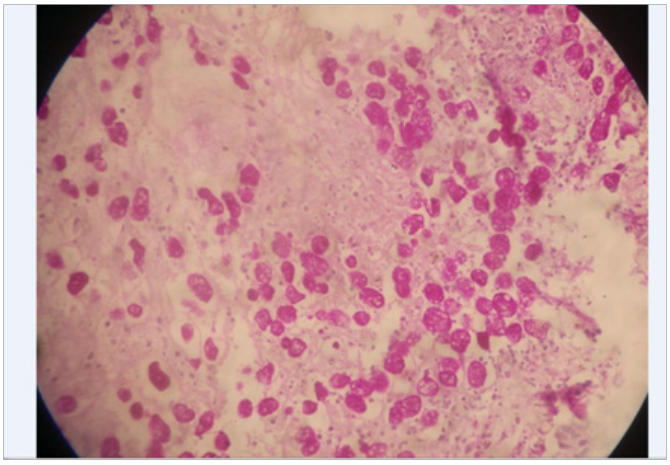

Figure 1 amoebic trophozoite forms highlighted by PAS stain(HE 40x).

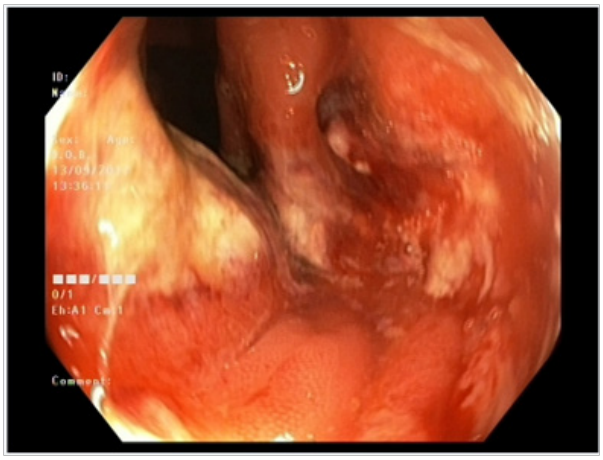

Figure 2 Colonoscopic view showing ulceroproliferative cecal growth.

In conclusion we report a rare case of ameboma presenting with an ulcero-proliferative cecal growth in an AIH / PSC overlap patient which was successfully treated with pharmacotherapy prior to treatment with corticosteroids. More awareness and lower threshold for suspicion among medical providers for amebiasis and its timely treatment before starting steroids for AIH in overlap syndromes will help avoid serious complications of steroids in amebiasis.

Acknowledgments

None.

Conflict of interest

The author declares there is no conflict of interest.

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