ORIGINAL RESEARCH PAPER
Volume - 14 | Issue - 05 | May - 2025 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

TOXIC MEGACOLON IN A PATIENT OF AMOEBIC LIVER ABSCESS WITHOUT AMOEBIC COLITIS

Microbiology

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ABSTRACT

Amoebiasis is asymptomatic in up to 90% of infected persons, in the others it presents as diarrhea (amoebic dysentery) or extraintestinal disease (mainly hepatic abscesses). Rarely (< 3% of cases), the disease takes a fulminant course characterized by necrotizing colitis that may lead to toxic megacolon and requiring emergent total colectomy due to the occurrence of colon perforation and peritonitis, with a mortality rate of more than 40% . We report a case of toxic megacolon in a middle-aged man, due to infection with Entamoeba histolytica acquired in a endemic zone of amoebiasis, presented as liver abscess without any clinical and endoscopic evidence of amoebic colitis.

KEYWORDS

Toxic Megacolon, Amoebic Colitis, Amoebic Liver Abscess

INTRODUCTION

Amoebiasis is a parasitic infection caused by the protozoon, Entamoeba histolytica, that infects 10% of the world's population. Infection by E. histolytica causes a spectrum of intestinal illnesses as aymptomatic infection, symptomatic noninvasive infection, acute protocolitis (dysentery), and fulminant colitis with various complications.

Fulminant amoebic colitis, however, is a rare complication of amoebic dysentery, It presents with rapid onset of severe bloody diarrhea, severe abdominal pain, and high fever. The amoebic invasion of the colonic wall leads to a fierce superacute course of massive necrosis of wide segments of the entire colon. The extensive necrosis usually affects all layers of the bowel wall. Toxic megacolon and intestinal perforation are common in this setting ,the mechanism in development of toxic megacolon are not clearly delineated but chemical mediators such as nitric oxide and interleukins may play a pivotal role in pathogenesis.

Very few case reports are available in this regard, that's too available in the setting of fulminant amoebic colitis with a grave prognosis, we herein report a case of " toxic megacolon in a patient of amoebic liver abscess without amoebic colitis " that has responded to conservative medical management and has a good prognosis.

Case Report

A 38 yr. old man admitted in our hospital with complaints of right upper abdomen pain and fever for 7 days.

This pain was gradual in onset, moderate in severity, continuous dull aching in nature. He had intermittent fever with chills.

For the last 3 days patient had sudden onset pain abdomen of increased severity, which was generalised, continuous, associated with abdominal distension, constipation, obstipation and few episodes of vomitings. Patient was a chronic alcoholic for last 8 yrs.

At addmission, patient was febrile, on examination pulse rate was 108/min, B.P was 110/74 mm of Hg, Abdomen was distended with generalised tenderness and guarding with sluggish bowel sounds.

Laboratory investigations revealed leukocytosis (TLC-15560/ mL), ESR(100mm 1st hr), AST (78.78 U/L) all other biochemical investigations were within normal limits.

X ray abdomen erect posture showed a distended transverse colon loop with loss of haustrations and of more than 6 cm of diameter. (fig- 1)

USG abdomen revealed a 74x40 mm size, ill defined, heterogenous lesion, non-liquified with internal echogenic foci noted in lt. lobe of liver. GB wall was thickened with peri GB cuffing and there as minimal collections in B/L pleural cavities, Bowel loops were dilated. A possibility of lt. lobe evolving liver abscess was kept by the radiologist.

Given the acute nature of the pathology , pt. started on conservative medical management with withdrawl of all drugs or agents that precipitates toxic megacolon. CECT abdomen with oral and I.V contrast was postponed. Pt. was kept NBM, started on continuous Ryle's tube aspiration , higher antibiotic with ciprofloxacin and metronidazole, regular maintenance of serum electrolytes, blood sugar and monitoring of daily TLC and vitals were done. On the 2nd day itself , fever came down, abdominal distension and tenderness has decreased and pt. had started to pass flatus, though tachycardia was still there. USG guided aspiration of liver abscess was tried but was non-aspirable, amoebic serology was sent for lab investigation and was found to be positive.

Stool samples of patient was collected in a wide mouthed sterile container .

Stool wet mount examination was done for entamoeba histolytica which was found negative.

E. histolytica rapid antigen test was done.The prototype E. histolytica rapid antigen test was supplied by TechLab, Inc. it was specifically designed to identify E. histolytica-specific antigen in stool samples. For this study, 200 microl of stool sample was added to 500 microl of the diluent supplied with the test kit in a 1-ml tube. The samples were mixed by lightly vortexing them. They were then centrifuged at 1,500g for 2 min, and 500 l of supernatant was resuspended in a new tube. Two drops of conjugate (60 l), consisting of 7F4 antibody coupled to horseradish peroxidase (HRP) in a buffered solution, was added to the sample and the tube was again vortexed. The sample-conjugate mixture was incubated at room temperature for 15 min. A total of 400 microl of the sample was then added to a membrane through a sample well and was allowed to migrate over two antibody-striped lines: a control line and a test line. The control line bound to the conjugate regardless of the sample antigen content and indicated whether the test ran properly. The test line contained antibodies specific for E. histolytica lectin and trapped antigen conjugate complexes if they were present in the sample. The sample was allowed to incubate at room temperature for 10 min. The reaction well was then washed with 500 l of wash solution. Two drops of the substrate solution was added to the reaction well containing the HRP enzyme conjugate. The test was allowed to develop for 10 min before the sample was classified as positive or negative.The total assay time was approximately 35 min.

E. histolytica rapid antibody test was done.The prototype E. histolytica rapid antibody test was supplied by TechLab, Inc. The 25 microl of serum sample was diluted with 225 microl of the buffer supplied with the test kit, and 250 microl of diluted sample was applied to the reaction well and allowed to completely absorb for 1 min. The reaction well was washed with 2 drops of membrane wash buffer, followed by a

second wash after complete absorption. The membrane that it flowed through had two dots. The control dot was designed to bind to any antihuman immunoglobulin G (IgG), while the test dot consisted of an E. histolytica-specific recombinant protein, termed LecA, which captured E. histolytica-specific IgG. Anti-human IgG conjugated to HRP was added to the reaction well and was allowed to react for 1 min. The well was again washed twice with 2 drops of membrane wash solution. To determine whether bound human IgG was present, 2 drops of substrate solution was added, and the HRP enzyme converted the substrate to a blue color if it was present on the control dot or the test dot. The test was allowed to develop for 5 min, and 2 drops of a stop solution was added before the test was read.

At the end of the 3rd day of medical management, pt. became afebrile, passed stool, abdominal distension got relieved and on examination guarding and tenderness were no more , bowel sounds were present. Pt. had a normal X ray abdomen erect posture on the day 4th morning.

On day 5th colonoscopy was done with normal mucosa and normal vascular pattern seen till 10 cm of terminal ileum, no evidence of ulcerations were present.

Patient started on clear liquids which was gradually increased , it was well tolerated with begining of passage of stool. All lab investigations were within normal limits on day 6th of treatment.

Patient continued on i.v management for 7 days and discharged thereafter on oral medications with ciprofloxacin and metronidazole for the next 7 days and on follow up pt. was symptom free , taking orally with a regular bowel habit. Repeat USG Abdomen revealed few air foci in the lt. lobe due to previous needle insertion with evidence of healed abscess. (fig-2)

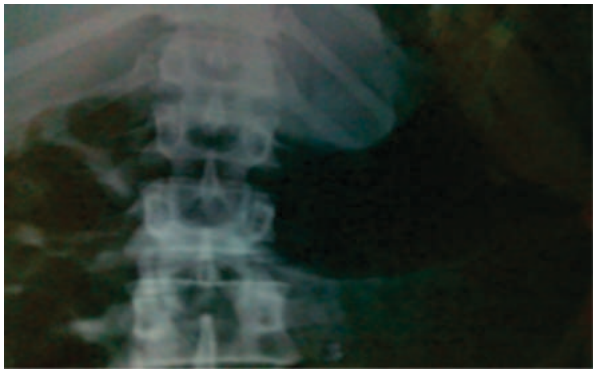

Fig – 1; Dilated Transverse Colon With Loss Of Haustra

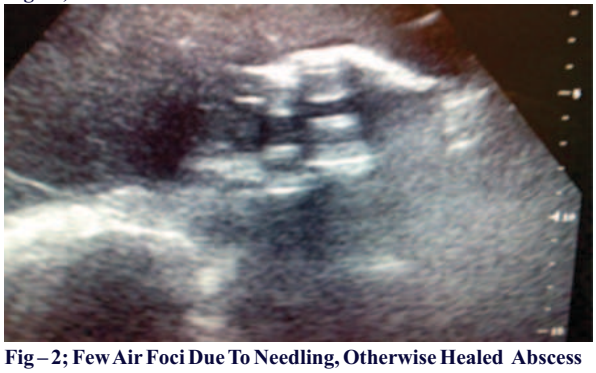

Fig – 2; Few Air Foci Due To Needling, Otherwise Healed Abscess

DISCUSSION

Amoebiasis is asymptomatic in majority of cases (> 90%) , rarely (< 3% of cases), the disease takes a fulminant course characterized by necrotizing colitis that may lead to toxic megacolon with a mortality rate of more than 40%[1.]

Uncomplicated amebic colitis is readily treated and has a mortality rate of less than 0.5% Complications necessitating surgical intervention develop in only 6% to 11% of patients with symptomatic disease. Risk factors for invasive disease include the virulence of the strain of E. histolytica, as well as host factors including genetic susceptibility, young age, pregnancy and impaired immunity (for example. due to treatment with corticosteroids, malignancy, malnutrition, alcoholism, and HIV infection). Chuah et al found that significant factors associated with the development of fulminant intestinal amebiasis in univariate analyses were male gender, being age over 60 years, and having an associated liver abscess, progressive abdominal pain, and signs of peritonitis, leukocytosis, hyponatremia, hypokalemia, and hypoalbuminemia.[2].

Management involves close medical attention, supportive care, and treatment of the underlying colitis. Possible exacerbating factors such as narcotic, loperamide and anti cholinergic medications must be withdrawn, and colonic decompression via tube drainage or positional techniques must be considered. Signs of progression or complications of the disease must be treated aggressively with surgical intervention, as delay is associated with even greater risk of mortality. [3][4]

Acute amebic colitis has a acute onset presenting with a 1- to 2 week history of,. rapid onset of severe bloody diarrhea, severe abdominal pain, tenesmus and high fever. Toxic megacolon and intestinal perforation are common. The perforations may be microscopic and usually sealed off by the adjacent omentum or bowels.[5].In this condition emergent laparotomy with procto- colectomy may be required.

In our case clinical sign and symptoms were not of amoebic colitis also in the background history of liver abscess pt. had developed acute onset paralytic ileus of large bowel resulting in toxic megacolon. As presentation was not that fulminant and also there was no evidence of bowel perforation, we decided to go with a conservative medical management with an option for surgical consultation open if there were no response to treatment in the first 48 to 72 hrs.

Here is case, probably the first one of, "toxic megacolon " which is reported in the absence of fulminant amoebic colitis with presence of " amoebic liver abscess" and thus has a good response to conservative medical management.

As there was no amoebic invasion of the colonic wall leading to a fierce superacute course of massive necrosis, and also endoscopically, transmural ischemic necrosis with multiple ulcers and yellowish plaque-like lesions were absent.

The pathogenesis of toxic megacolon in this case is entirely due to systemic and local inflammatory effects of amoebic liver abscess in the left lobe .

This is mainly due to two factors,

1) Inflammation leads sequentially to the release of inflammatory mediators in the systemic circulation , increased inducible nitric oxide synthase, generation of excessive nitric oxide, and inteleukins and colonic dilatation.
2) Transverse colon is in close proximity of a superficial liver abscess in the lt. lobe, these inflammatory mediators also have a local effects playing in colonic dilatation.

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