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Case Report

CHRONOLOGICAL DEVELOPMENT OF CLINICAL FEATURES IN A CASE OF FULMINANT AMEBIC COLITIS: A CASE REPORT

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Abstract

Introduction: Amebiasis is a parasitic infection usually limited to endemic areas, caused by the protozoa *Entamoeba histolytica*. Fulminant amebic colitis is its rare, rapidly progressing, life threatening variant reported from many countries all over the world. **Case Report:** Present case started as malaise and fever not responding to antibiotics. Fever was spiking by third and fourth days with postprandial rigors, relative bradycardia and hypotension. Abdominal pain was rapidly increasing with flatulence and distension on the fourth day evening. Abdominal distention and pain were relieved and temperature was normal after the first intravenous infusion of metronidazole. **Conclusion:** Fulminant amebic colitis may be seen in any country all over the world due to increasing international travel, import of food materials, HIV infection and immune deficiency. None of the clinical features is specific or diagnostic for fulminant amebic colitis. Fulminant amebic colitis should be considered in all cases of acute abdomen, and fever not responding to antibiotics with postprandial rigors and/or pain abdomen. Suspicion and immediate institution of specific treatment (Intravenous metronidazole infusion) is life saving. All cases should be evaluated for colonic perforations and surgical intervention.

Key Words: Amebiasis; Entamoeba histolytica; Fulminant amebic colitis; postprandial rigors.

Introduction

Amebiasis is a parasitic infection caused by the protozoon, Entamoeba histolytica. Transmission of amebic infection is by ingestion of cysts from feces of infected case. Intestinal amebic infection includes five clinical syndromes: asymptomatic colonization (cyst passage), acute amebic colitis, fulminant amebic colitis, chronic amebic colitis and ameboma. Amebic liver abscess is the commonest extra-intestinal infection by the parasite and other sites of involvement include pleura, peritoneum, pericardium, and brain.

Asymptomatic cyst passage usually resolves without treatment and many such cases actually may have E dispar. Patients with acute amebic colitis usually present with lower abdominal pain and diarrhea with or without fever. Many patients of amebic colitis in endemic areas present with colonic tenderness and constipation with or without flatulence. Some of these chronic amoebic colitis patients may present with backache

probably due to spasm of the underlying spinal muscles. Ameboma is a rare manifestation that may present as a tender mass with or without symptomatic dysentery.

Fulminant amebic colitis is rare, rapidly progressing, and life threatening. Though amoebiasis is an infection of endemic areas, cases of fulminant amebic colitis were reported from many countries all over the world probably due to increasing international travel, import of food materials from the endemic areas, HIV infection and immune deficiency.[1-10] More than 50% mortality was reported for fulminant amebic colitis.[11-14] Early suspicion of the disease and institution of specific treatment avoiding delay for investigations were recommended for better prognosis.[11-13] Majority cases of fulminant amebic colitis in literature were hospital admissions after colonic perforations and the signs and symptoms during early stages were not clear.[1-14] We are presenting the chronological development of clinical features in a case of fulminant amebic colitis. Though all cases of fulminant amebic colitis do not present in the same way, the present case shall enrich our knowledge to facilitate early suspicion of the disease and institution of specific treatment.

Case Report

The patient in the present case is an Indian, 66 year old male, vegetarian, not hypertensive, not diabetic, and otherwise healthy. He ate a small piece of food purchased from the market on that day, at about 11:30 am. He started feeling sick and little unease by 7 pm. Next morning he woke up with mild fever, myalgia, and altered taste as in toxaemia of bacterial infections. Temperature was 37.2 °C (99° F). Blood counts were within normal limits. Cefexim 200 mg was given to the patient twice daily by oral route.

On the third day temperature was 39.4 °C (103° F), and pulse rate was 80 per minute (relative bradycardia). He had chills and rigors in the afternoon and at night. Chills and rigors started about 3 hours after food and lasted for 20 to 30 minutes. Blood film was negative for parasites. Urinalysis did not reveal any abnormality. Food intake was reduced.

On the fourth day temperature was 40° C to 40°.5 C (104° to 105° F). There was mild, vague abdominal pain in the right paraspinal area. Metronidazole 400 mg was given to the patient in the morning and afternoon by oral route. Chills and rigors occurred in the afternoon. Blood pressure was 80 / 50 mm Hg at 7 pm. Intravenous 5% dextrose saline infusion was started. Pain abdomen was increasing with flatulence and distension of the whole transverse colon. The patient felt as if the colon was thinning out to rupture. Abdomen was soft, distended without marked tenderness. Metronidazole 500 mg in 100 ml was started as intravenous infusion. Few minutes before the infusion was about to finish, the patient felt as if many tiny bubbles were bursting in the wall of whole length of transverse colon in a span of few seconds. Flatus was pushed down and the abdominal pain was relieved in a few minutes. Temperature was normal after one to two hours. Oral intake was stopped and patient was maintained on intravenous fluids for three days. Cefexim was stopped. Gentamycin 80 mg in 2 ml and metronidazole 500 mg in 100 ml were given by intravenous route, 8 hourly for 5 days. And, patient was advised oral metronidazole 400 mg. 8 hourly for another ten days.

On the fifth day, small quantity of bloody stool was passed in the morning. Stool was negative for parasites. Temperature and pulse rate were normal. Blood pressure was 120/80 mm Hg. Epigastric tenderness was mild. Ultrasound scan of the abdomen was showing gallbladder sludge and mild dehydration changes in the kidneys. There was no mass lesion, no colonic distension and no evidence of perforation. Fasting blood sugar was 98 mg/dl, serum creatinine was 1.4 mg/dl, erythrocyte sedimentation

rate was 64 mm at the end of first hour and blood counts were within normal limits. On the sixth day, few shreds of mucus membrane were passed in stool. The patient was allowed oral intake on the eighth day.

Discussion

Clinical features of fulminant amebic colitis include rapid onset and progression, severe bloody diarrhea, high spiking fever, unstable vital signs, abdominal distension, severe abdominal pain, colonic perforations and amebic liver abscesses.[1-14] The infection may start as fulminant amebic colitis from inception or acute amebic colitis or chronic amebic colitis may progress to fulminant amebic colitis.[1,2,4,6,8,10] Accordingly, the clinical history and presentation may vary considerably from patient to patient.

Diarrhea was not observed in the present case. Bloody stool passed in the morning of fifth day, probably, suggests that bloody diarrhea, colonic perforations, septicemia and liver abscesses might have followed if the intravenous metronidazole was delayed. Though diarrhea is a usual feature of amebiasis, some cases of fulminant amebic colitis in literature were reported without diarrhea. [1, 2] Diarrhea may be absent in chronic amoebic colitis, ameboma and in asymptomatic cyst passers.

The disease, in the present case, started as malaise and fever not responding to antibiotics. Fever was spiking by third and fourth days with postprandial rigors. Relative bradycardia was recorded on third day and low blood pressure on the fourth day (unstable vital signs).

Abdominal pain and distension were rapidly increasing by 7 pm on the fourth day, qualifying the fulminant nature of the disease. Abdominal pain, starting early or late, is a constant feature in all cases of fulminant amebic colitis.[1-14] Abdominal pain, probably, is due to colonic serosal inflammation or stretch by increasing flatulence and distension. Initial right paraspinal pain might be originating from the right part of the transverse colon. Acute amebic colitis may present with lower abdominal pain. Colonic tenderness is common in chronic amebic colitis. Ameboma may be with or without marked tenderness. Abdominal pain is diffuse with rebound tenderness in cases of peritonitis after colonic perforations. [1,4,11,12] High spiking fever, unstable vital signs, abdominal distension and abdominal pain in the present case were unassociated with colonic perforations and were probably due to fulminant amebic colitis.

Dehydration changes in the kidneys on ultrasound scan and marginal elevation of serum creatinine were, probably, due to low blood pressure and renal hypo perfusion. Tissue necrosis due to diffuse parasitic infiltration of the transverse colon might have caused ESR elevation and blood stained stool after metronidazole infusion.

There were no signs and symptoms indicating colonic disease till the abdominal pain on the fourth day. Postprandial timing of rigors on third and fourth days was probably indicative of intestinal pathology. Rigors and their postprandial timing were not recorded in fulminant amebic colitis cases in literature.

None of the clinical features is specific or diagnostic for fulminant amebic colitis. Amebic trophozoits or cysts may be demonstrated in stool in all clinical syndromes of intestinal amebiasis. Parasites in fulminant amebic colitis penetrate deep into the colonic wall and only 20% cases were reported in literature as stool positive.[2,11] The diagnosis of fulminant amebic colitis has to be established in a suspected clinical syndrome by demonstrating diffuse infiltration of parasites in to the colonic muscle or by clinical response to the specific treatment (metronidazole infusion).

Suspecting fulminant amebic colitis in the present case, when abdominal distention and pain were rapidly increasing, intravenous infusion of metronidazole was started without delaying for investigations in view

of impending colonic perforations. [11-13] Stool could be examined only after intravenous metronidazole, and was found negative for parasites. Oral metronidazole on the fourth day, probably, failed to develop required blood concentrations rapidly and was ineffective. Colon biopsy was not considered for demonstrating the parasites as the patient recovered completely with the first intravenous infusion of metronidazole. The patient's description 'as if many tiny bubbles were bursting ……. in a span of few seconds', probably corresponds to the killing of parasites diffusely infiltrating the wall of the transverse colon.

Conclusion

Fulminant amebic colitis may be seen in any country all over the world due to increasing international travel, import of food materials, HIV infection and immune deficiency. None of the clinical features is specific or diagnostic for fulminant amebic colitis. Fulminant amebic colitis should be suspected in all cases of acute abdomen, fever not responding to antibiotics with postprandial rigors and / or pain abdomen. Immediate institution of specific antiamebic treatment (intravenous metronidazole infusion) without delaying for investigations is life saving. All cases should be investigated for colonic perforations and evaluated for surgical intervention.

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