

Study of the incidence of Ruptured amoebic liver abscess with caecal perforation.

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I. Introduction

Amoebiasis is a parasitic infection caused by the protozoan, *Entamoeba histolytica*, that infects 10% of the world's population, resulting in 100,000 deaths/year.^[1] The colon and liver are the principal organs affected in amoebiasis. The parasite exists in two forms: a motile form called the trophozoite, and a cyst form, responsible for human transmission of the infection. The trophozoite inhabits the colon where it produces lesions of amoebic colitis. Invasion of the colonic mucosa leads to dissemination of the organism to extracolonic sites, predominantly the liver. Infection by *E. histolytica* causes a spectrum of intestinal illnesses as asymptomatic infection, symptomatic noninvasive infection, acute proctocolitis (dysentery), and fulminant colitis with perforation.

The most common complications of amoebic liver abscess (ALA) arise from rupture of the abscess into surrounding organs or anatomical spaces. Communication occurs in the peritoneum, viscera, and large vessels on one side of the diaphragm and the pleura, bronchi, lungs, and pericardium on the other side. Invasive intestinal amoebiasis presenting as cecal perforation is a rare entity. It is associated with high mortality and dismal outcome.^[2] Ruptured liver abscess with invasive intestinal amoebiasis is even rarer.

II. Patients and Methods

Patients presenting to Surgery emergency of nalanda medical college hospital, patna diagnosed to have liver abscess with clinical signs of generalized peritonitis were evaluated. Ultrasound of all patients showed liver abscess with breach in liver parenchyma with mild to moderate free fluid in the abdomen. A review of these data was performed to document the clinical presentation, etiology, diagnostic work-up, treatment, morbidity, and mortality. Four out of 24 such patients were found to have ruptured liver abscess with cecal perforation. Amoebic serology was positive in all 4 patients. Of 4 patients, 3 were male and 1 female. Mean age was 39.5 years. Procedure to be done was decided based on intra-operative findings. From oct 2017 sept 2018, we have operated 32 cases of ruptured liver abscess of which 4 patients had cecal perforation along with ruptured liver abscess.

III. Results

All patients presented with generalised abdominal pain, fever, and distension of abdomen. Clinically, all these patients had frank signs of peritonitis. Two out of four patients had free air under the diaphragm. All these patients were malnourished with body mass index of <18. In all these patients, serum albumin was <3 g%. Intraoperatively, all 4 patients had cecal perforation of which in 1 patient it was a sealed perforation and in 1 multiple perforations in cecum and ascending colon were present.

Three patients underwent limited resection, and right hemicolectomy was done in one patient who had multiple perforations in the cecum and ascending colon. Exteriorization of bowel was done in all patients. The drain was placed in liver abscess cavity in all patients. Resected specimen in two patients showed multiple flask-shaped ulcers with yellowish plaques. Microscopically, numerous amoebic trophozoites were present in the necrotic debris of the colon ulcers in all cases.

Postoperatively, two patients had wound infection which was managed conservatively with antibiotics. All patients were given intravenous Metronidazole along with a broad-spectrum antibiotic. One patient developed bilateral pneumonia and subsequently respiratory failure and expired.

IV. Discussion

ALA is a common presentation of infection by *E. histolytica*, however, carries high morbidity and mortality. It is the most common extra intestinal form of invasive amoebiasis. Indeed, an estimated 100,000 people succumb to ALA each year.^[31] Trophozoites that successfully penetrate the colonic mucosal barrier cause invasive disease enter the portal system and travel to the liver. Amoebic colitis and ALA rarely occur simultaneously, and the colonic lesions are usually silent; direct extension to the liver and lymphatic spread do not occur. The cecum is the most common site of amoebic colitis, and the right lobe of the liver is more commonly affected because of drainage of the right portal branch from the right side of the colon. The condition usually starts as diffuse amoebic hepatitis; liver cells undergo liquefactive necrosis, starting in the center and spreading peripherally to produce a cavity full of blood and liquefied liver tissue resembling anchovy sauce; it has no odor and is sterile. The fluid itself is free from any amoebae, which may be found at the expanding edge of the abscess cavity with little inflammation. Amoebae are known to lyse neutrophils, and the release of neutrophilic mediators may promote hepatocyte death and extension of the abscess. Secondary bacterial infection may occur spontaneously, altering the color, odor, and consistency of the pus. Lack of fibrotic response by the surrounding tissue with centrifugal extension results in extension of the abscess to the Glisson capsule, which is resistant to the amoebae. Generally, ALAs are solitary, large, and located in the right liver. Left lobe abscesses are less common, but because of the smaller volume of the left liver, abscesses in this location are more prone to rupture the capsule.

Amoebic serology is highly sensitive and specific in the differentiation between pyogenic and amoebic hepatic abscess. Enzyme immunoassay (EIA) is simple, rapid, inexpensive, and more sensitive, and it has now largely replaced indirect hemagglutination test and counter immunoelectrophoresis.^[41] EIA detects antibodies specific for *E. histolytica* in approximately 95% of patients with extra intestinal amoebiasis, 70% with active intestinal infection, and 10% who are asymptomatic cyst passers.

Primary surgical therapy is indicated in patients with intraperitoneal rupture, in which complete exploration and lavage of the abdomen are indicated.^[51] In some other patients, surgery may be indicated for the treatment of complications of the percutaneous drainage, such as bleeding or intraperitoneal leakage of pus. Patients with an underlying pathology that needs surgical resolution can undergo treatment for the liver abscess during the same operation, such as cases of acute cholecystitis presenting with an adjacent abscess. Large, multiloculated abscesses containing thick pus are more prone to not respond to percutaneous treatment. In these patients, complete evacuation and removal of necrotic tissue and debris may be more easily achieved surgically.^[61]

Peritonitis with amoebiasis is due to rupture of an ALA in 78% of patients and perforated or necrotizing amoebic colitis in the other 22%. Spontaneous rupture of ALA with colon may occur in 2.7%–17% of cases.^{[71],[81],[91]}

Fulminant colitis which is a known variant of amoebic colitis develops rapidly and presents with features of acute abdomen and loose stools.^[101] Various factors including male gender, increased age, signs of peritonitis and abdominal pain, leukocytosis, electrolyte disturbances, and hypoalbuminemia are associated with the development of fulminant amoebic colitis in patients who have invasive intestinal amoebiasis.^[111] Intraoperatively, the fulminating colitis presents as an inflamed, extremely friable colon, wrapped with omentum, and underlying full-thickness necrosis and perforation. The colon is so friable that it can disintegrate with any form of manipulation.^[121] Resection of the necrotic colon is the treatment of choice.^[131] There is a high risk of suture breakdown in tissue containing amoebae, and exteriorization of the bowel rather than repair should be the way to go.

V. Conclusion

Simultaneous occurrence of Fulminant colitis, cecal perforation and rupture of ALA is extremely rare, and such cases carry a very poor prognosis. Cecal perforation may spread to the appendix and lead to gangrenous appendicitis. Eggleston *et al.* documented a study of 26 patients with amoebic colonic perforation, wherein all 6 patients with concomitant liver disease died.^[141] Simultaneous rupture of ALA and colonic perforation is an indicator of extremely poor prognosis, and these patients should receive intensive medical and surgical management.

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