Section: Pediatrics

Case Report

CASE REPORT ON INVASIVE AMOEBIASIS

Khaja Amer Khan[1], Ahmed Khan[2], Mohammed Atif Ahmed[2], Arbaaz Shahid[2]

[1]*Assistant professor, Department of Pediatrics, Shadan Institute of Medical Sciences, Hyderabad, Telangana, India.*
[2]*Post Graduate, Department of Pediatrics, Shadan Institute of Medical Sciences, Hyderabad, Telangana, India.*

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Corresponding Author:
Dr. Khaja Amer Khan,
Assistant Professor, Department of Pediatrics, Shadan Institute of Medical Sciences, Hyderabad, Telangana, India.
Email: aamer.k42@gmail.com

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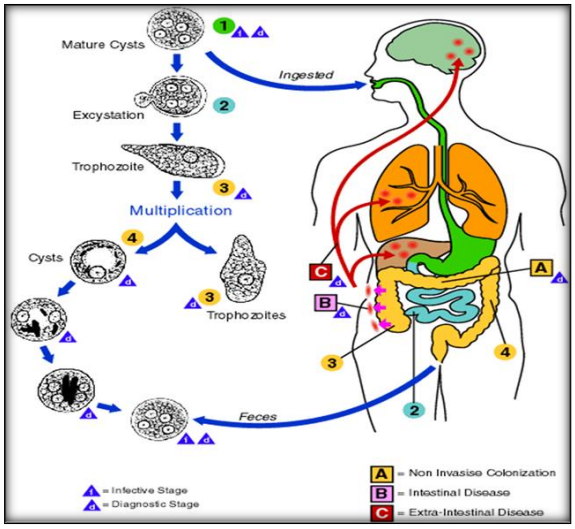
ABSTRACT

Amoebiasis is present all over the world though most cases occur in the developing world. About 480 million people are currently infected with about 40 million new cases per year with significant symptoms. This results in the death of between 40,000-1,00,000 people a year. Our case reports a 12 year old male child in Hyderabad hailing from a below average socio economic region and consumption of infested food and water. Presented in septic shock with colon perforation who required an emergency laparotomy and complications of pleural effusion and soft tissue involvement (due to metastasis). Reporting this case with a view for health care workers as how a simple disease can lead to so many complications when presented with non typical symptoms and how with protocolised treatment, morbidity and mortality can be reduced.

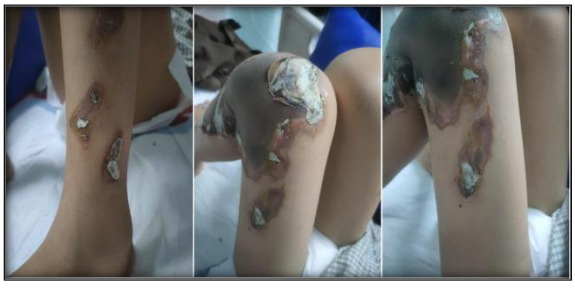
Keywords: Invasive amoebiasis; Entamoeba histolytica; necrotizing fasciitis; pleural effusion; pediatric colonic perforation; metronidazole; paromomycin.

INTRODUCTION

Amoebiasis is caused by organism Entamoeba histolytica, usually caused by ingestion of contaminated water, unwashed fruits or vegetables. The organism has at least seven different species that can inhabit the human intestine and one that can be found in human oral cavity.



Following excystation, the emerging trophozoites migrate to the large intestine, can become virulent and invasive, where they start to destroy the mucous epithelial barrier, thus inducing the overproduction of mucus, killing host cells, and provoking inflammation, subsequently causing amoebic colitis. Metastatic amoebiasis occurs secondary to hematogenous or lymphatic spread and involvement of distant organs causing abscesses in and around liver, lungs, kidney, spleen, brain or adrenals. Cutaneous amoebiasis is due to the direct spread around anus and is characterised by extensive gangrenous destruction of the skin. These lesions mimic condyloma, epithelioma or carcinoma.

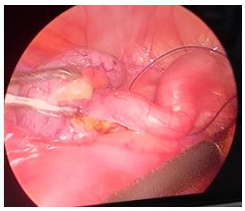


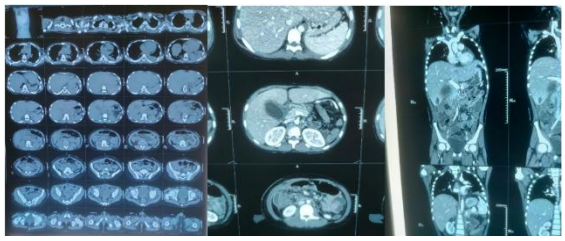
CASE REPORT

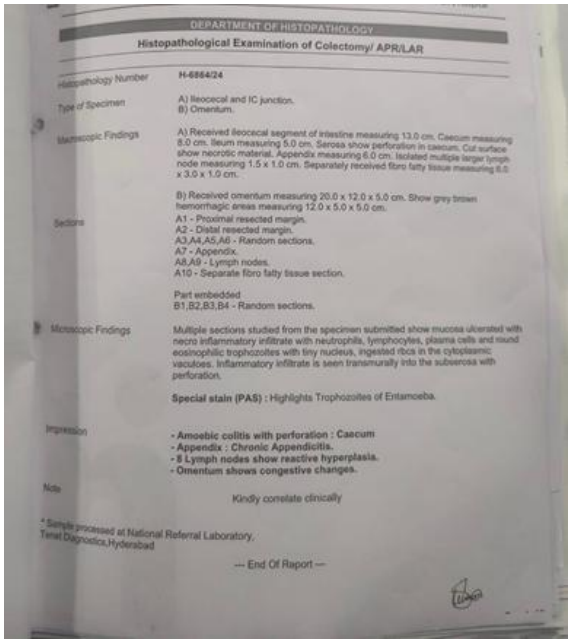
A 12 year old adolescent presented to the emergency department with severe pain abdomen and yellowish discolouration of eyes, been taking treatment since a week at other outside hospital in

the form of iv fluids, anti emetics, anti spasmodics and antibiotics including metronidazole.

Physical examination revealed the patients general condition to be compromised with diffuse pain located throughout the abdomen. Diffuse tenderness was present with child having a dull look on his face. The point of maximum tenderness was located in the right iliac region.







DEPARTMENT OF HISTOPATHOLOGY

Histopathological Examination of Colectomy/ APR/LAR

Histopathology Number: H-6864/24

Type of Specimen: A) Ileocecal and IC junction. B) Omentum.

Macroscopic Findings: A) Received ileocecal segment of intestine measuring 13.0 cm. Caecum measuring 8.0 cm. Ileum measuring 5.0 cm. Serosa show perforation in caecum. Cut surface show necrotic material. Appendix measuring 6.0 cm. Isolated multiple larger lymph node measuring 1.5 x 1.0 cm. Separately received fibro fatty tissue measuring 8.0 x 3.0 x 1.0 cm.

B) Received omentum measuring 20.0 x 12.0 x 5.0 cm. Show grey brown hemorrhagic areas measuring 12.0 x 5.0 x 5.0 cm.

Sections: A1 - Proximal resected margin. A2 - Distal resected margin. A3,A4,A5,A6 - Random sections. A7 - Appendix. A8,A9 - Lymph nodes. A10 - Separate fibro fatty tissue section.

Part embedded B1,B2,B3,B4 - Random sections.

Microscopic Findings: Multiple sections studied from the specimen submitted show mucosa ulcerated with necro inflammatory infiltrate with neutrophils, lymphocytes, plasma cells and round eosinophilic trophozoites with tiny nucleus, ingested rbcs in the cytoplasmic vacuoles. Inflammatory infiltrate is seen transmurally into the subserosa with perforation.

Special stain (PAS) : Highlights Trophozoites of Entamoeba.

Impression:
- Amoebic colitis with perforation : Caecum
- Appendix : Chronic Appendicitis.
- 8 Lymph nodes show reactive hyperplasia.
- Omentum shows congestive changes.

Note: Kindly correlate clinically

* Sample processed at National Referral Laboratory, Tenet Diagnostics,Hyderabad

--- End Of Raport ---

Laboratory investigations showed significant leucocytosis (wbc 25000cell/cumm). Coagulation profile was deranged (PT25 APTT55). LFT was abnormal with TSB 7mg/dl with raised liver enzymes (SGPT 267U/L SGOT 113U/L), C reactive protein positive (52.3mg/l). Other routine pathological tests done were negative. CECT abdomen was done which revealed dilated mid distal ileal loops upto IC junction with air fluid levels.IC junction and cecum grossly dilated with diffuse wall thickening, features suggestive of infective colitis with impending perforation and retrograde small bowel obstruction. Explorative laparotomy was done and resection of distal ileum, cecum and ascending colon was performed. The resected samples were then sent for histopathological evaluation.

Post operative course: Child was then kept on mechanical ventilator. He then developed massive left sided pleural effusion for which ICD was put and managed. He had persistent fever spikes. The child also developed necrotising facitis of both lower limb which extended progressively. Inspite of broad spectrum antibiotic coverage, the child had persistent fever spikes. HPE report then showed amoebic colitis with perforation. Metronidazole double dose (invasive amoebiasis dose) was started followed by paromomycin resulting in significant improvement in signs and symptoms over next 10 days.

DISCUSSION

Invasive amoebiasis, caused by Entamoeba histolytica, remains a significant public health concern in developing countries where sanitation and access to clean water are compromised. The case presented here emphasizes the devastating complications that can arise from delayed diagnosis and treatment of an otherwise treatable condition.

Globally, E. histolytica infects nearly 480 million individuals annually, with 40 million manifesting clinically significant disease and up to 100,000 fatalities reported each year.[1] Although most infections are asymptomatic or limited to mild colitis, a small percentage can develop invasive disease characterized by extra-intestinal dissemination. In this child, the disease manifested in its most severe form—fulminant amoebic colitis leading to intestinal perforation and systemic complications including pleural effusion and necrotizing fasciitis.

Delayed Presentation and Diagnostic Challenges: The patient had received symptomatic treatment at a peripheral center before being referred in septic shock, highlighting a common issue in low-resource settings: inadequate suspicion and diagnostic capacity for amoebiasis. At the time of presentation, clinical features and laboratory investigations, including markedly elevated WBC count and deranged liver function tests, pointed toward a severe systemic infection. Radiological evaluation via CECT abdomen confirmed features of impending colonic perforation, necessitating emergency surgical intervention.

In the diagnosis of amoebiasis, the gold standard remains microscopic detection of cysts and trophozoites in stool, which is both inexpensive and direct. However, this method lacks sensitivity and specificity, and fails to distinguish between pathogenic E. histolytica and non-pathogenic E. dispar or E. moshkovskii.[2] In this case, stool examination was impractical due to the patient's postoperative condition and critical illness, a limitation often encountered in emergency settings. Thus, the final diagnosis rested on histopathological

evaluation of the resected bowel, which confirmed invasive amoebic colitis.

Unusual and Severe Complications: Amoebic colitis can rarely lead to transmural necrosis and perforation, with a mortality rate ranging from 40% to 80% once perforation occurs.[3] The present case further complicated with necrotizing fasciitis and pleural effusion. Necrotizing fasciitis, although extremely rare in amoebiasis, has been reported in immunocompromised or critically ill patients as a result of hematogenous dissemination of trophozoites or secondary bacterial infection.[4] Pleural involvement in amoebiasis typically occurs secondary to rupture of a hepatic abscess into the pleural cavity; however, in this patient, it may have arisen via hematogenous spread or as a systemic inflammatory response.

Management and Therapeutic Response: Initial treatment with standard-dose metronidazole failed to achieve clinical improvement. It was only after administration of a high-dose regimen, recommended for invasive amoebiasis, followed by luminal amoebicide paromomycin, that the patient's clinical condition improved significantly. This highlights the importance of adhering to protocolized therapy for invasive amoebiasis: a tissue amoebicide (e.g., metronidazole or tinidazole) to eradicate trophozoites followed by a luminal agent (e.g., paromomycin, diloxanide furoate) to clear cysts from the intestine and prevent recurrence.[5]

The occurrence of such a complex and life-threatening manifestation of a preventable and treatable parasitic disease in a child from a socioeconomically disadvantaged background also highlights the broader public health challenge. Improved sanitation, health education, and access to timely medical care remain essential components in reducing the morbidity and mortality associated with amoebiasis.

CONCLUSION

Invasive amoebiasis can present with fulminant colitis and life-threatening extra-intestinal complications, even in pediatric patients from resource-limited settings. Early recognition—supported by radiological and histopathological confirmation—is essential to guide prompt surgical intervention and appropriate antimicrobial therapy. Adherence to a two-step treatment regimen, combining a tissue amoebicide (high-dose metronidazole) with a luminal agent (paromomycin), was pivotal in achieving clinical recovery. This case underscores the need for heightened clinical suspicion and protocolized management to reduce morbidity and mortality associated with severe amoebic infections.

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