

A Study of Caecal Perforation in Cases of Ruptured Liver Abscess

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Abstract

Amoebic colitis is a common infection in all population groups across all geographical barriers and is an easily treatable disease in its routine manifestations. However, it has its set of complications that carry poor prognosis. Amoebic liver abscess is a common complication but its association with caecal perforation is rare, and carries worst prognosis and high mortality rate.

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I. Introduction

Amoebiasis with its subset disease spectrum is a common parasitic infection distributed amongst all socioeconomic groups of patients and regions, producing diarrhea, colitis, and amoebic liver abscess, predominantly in developing tropical countries. Around 80% of the infected patients stay asymptomatic, while the other 20% develops disease. Extra-amoebic colitis, likely amoebic liver abscess is the commonest cause contributing to mortality. The organism travels via portal vein to the liver and manifests as liver abscess. This resultant abscess may rupture into the peritoneal, pleural and pericardial cavities. Intra-Peritoneal rupture of amoebic liver abscess is reported in around 6 – 9%. While bowel perforation from amoebiasis is very rare and it is extremely rare to have both these complications occurring simultaneously.

Case 1

A 45 yr old non-alcoholic, non-smoker male patient admitted in emergency surgery ward with complaint of abdominal pain since 4 days and having past history of right open nephrectomy 2 years ago.

Patient did not have history of fever, nausea, vomiting, diarrhoea, burning or difficulty in micturition or any comorbidity.

On examination- patient is conscious, cooperative and well oriented to time, place and person.

Pulse-100/min, BP- 130/90, RR-28/min.

On chest examination- Right sided air entry decrease.

Per abdomen- Distended and Tenderness in RHC present.

CXR finding-Mild right sided pleural effusion noted.

USG abdomen- 130 cc right lobe of liver abscess with 20 cc sub diaphragmatic collection and thin strip of sub capsular fluid noted S/O - Ruptured liver abscess.

- Mildly inflamed terminal ileum and base of caecum s/o- typhlitis

- Moderate free fluid in RIF and pelvic cavity.

Patient first immediately resuscitated and blood investigation sent with plan of emergency Laparotomy.

Blood investigation: HB- 12.0 WBC- 12.5 PLT-129000.

SGOT- 528 SGPT-547 ALP-113

BILLIRUBIN T-0.8, D-0.4, I-0.4.

PROTEIN T-4.8, ALB-2.8, GLOB-2.0

UREA-114, CREAT- 2.0

Na⁺/K⁺/Cl⁻ -140/4.3/117.

Intraoperative finding- 1-1.5 cm² antero lateral caecal perforation found.

Around 100cc purulent pus aspirated from ruptured liver abscess.

OT-emergency exploratory Laparotomy +liver abscess drainage+ double barrel ileo-ascending stoma formation.

On POD 3- purulent discharge present from stitch line with wound gap.

On POD 7-
OT- secondary tension suturing taken over laparotomy wound.
On regular dressing purulent discharge still present from stich line of laparotomy wound.



FIG 1-Burst abdomen despite of tension suturing

On POD-11
OT- Reexploratory laparotomy with peritoneal lavage with tension suturing.
Patient kept intubated after OT for 2 days and then extubated and kept on o2 support.
Then regular dressing was done but gradually discharge present from stitch line

On POD- 27- patient presented with burst abdomen.
OT- Bogotá bag DCS over burst abdomen.
On HPE examination-Only partially autolytic changes are seen.
No evidence of granuloma or malignancy found.
At present POD-28, patient is in sicu with Bogotá bag in situ, stoma in situ, right ADK in situ and on supportive o2 support.



FIG 2-BAGOTA BAG PLASTY over burst abdomen

Case 2

A 65 year old non-alcoholic, non-smoker male patient admitted in surgical ward with complaint of abdominal pain since 10days.

Patient had no complaint of fever, vomiting, diarrhea, constipation. And had no significant past history or comorbidity.

On examination-patient is conscious, cooperative and well oriented to time place and person.

Pulse-86/min BP- 120/80mmhg RR-26/min

On chest examination- AEBE and clear

Per abdomen-soft and mild RHC tenderness.

CXR finding-Right sided minimal pleural effusion noted.

USG abdomen-103 cc and 83 cc minimally liquefied liver abscess in right lobe of liver. Other small developing abscess in left lobe of liver. P/o-multiple minimally liquefied liver abscess with liver parenchymal disease with minimal to mild perihepatic fluid noted.

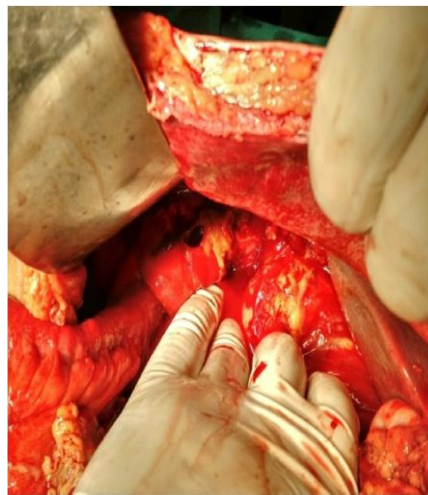


FIG 3-CAECAL PERFORATION

Blood investigation- HB/WBC/PLT-14.5/30.4/42000
SGOT/SGPT/ALP-94/24/193
BILLIRUBIN T/D/I-4.6/3.2/1.4
PROTIEN T/ALB/GLOB-5.3/2.5/2.8
UREA/CREAT-64/0.9
Na⁺/K⁺/Cl⁻ -141/3.8/108.

Procedure-liver abscess (right lobe) tapping done under local anesthesia under USG guidance and 50cc pus aspirated.

4 fresh frozen plasma transfusions.

Repeat blood investigation-HB/WBC/PLT-10.9/21.7/76000.

Repeat USG abdomen-159 cc partially liquefied liver abscess in segment V of right lobe of liver and 24 cc in segment II in left lobe of liver .other one is 60cc ill defined hypoechoic collection in left lobe of liver with multiple internal Echoes and septation suggestive of multiple liver abscess with intraperitoneal rupture.

Segmental dilation of bowel loops noted in right hypochondriac region with reduce peristaltic activity

Multiple fluid pockets noted in entire abdomen which largest present in right and left paracolic gutter with multiple septation. Diffuse omental thickening and mild inflammation of terminal ileum, IC junction, caecum and ascending colon S/O-sequel to infective etiology.

Patient planned for

OT- emergency exploratory laparotomy + right hemicolectomy + ileotransverse anastomosis + proximal loop ileostomy + peritoneal lavage

Operative findings-collection found around perihepatic region and 1×1cm² anterior wall caecal perforation found.

After operation patient remains intubated and shifted to ICU with inotropic support. After 10 hours, patient suddenly deteriorated, pulse and BP non-recordable, CPR given with inotropic support. Despite of all efforts, patient can't reviewed and declared expired.

Case 3

A 50 yr old non-alcoholic, non-smoker male patient admitted in surgical ward with complaint of abdominal pain since 7 days, patient had no history of fever, nausea, vomiting, diarrhoea or constipation , patient had no any significant past history or comorbidity.

On examination- patient is conscious, cooperative and well oriented to time place and person.

Pulse-86/min BP- 120/80mmhg RR-24/min.

On chest examination- Right sided air entry decreased.

Per abdomen- generalized tenderness present.

CXR finding- right sided mild pleural effusion with underlying consolidation

Left sided minimal pleural effusion.

USG abdomen- multiple liver abscess:-96cc in segment VII, 55cc in segment VI, 67cc in segment V.

Moderate inflamed terminal ileum and base of caecum with secondarily inflamed base of appendix and small amount of collection at iliocaecal junction with few septation suggestive of inflammatory/ infective etiology. However Appendicular or Caecal base perforation needs to be ruled out.

Patient resuscitation with fluids and antibiotics given and blood investigation sent.

Blood investigation- HB/WBC/PLT--11.5/34.5/23000

BILLIRUBIN T/D/I-2.0/0.8/1.2

UREA/CREAT-22/1.0

Na⁺/K⁺/Cl⁻ -127/3.3/93

Patient was operated for emergency exploratory laparotomy under a General anaesthesia .

Operative finding- 2 caecal perforation found in in posterior wall of caecum measuring 0.5×0.5cm² with totally sloughed out appendix.

OT-emergency exploratory laparotomy + right hemicolectomy +ileo transverse resection and anastomosis + loop ileostomy + peritoneal lavage.

On HPE examination- inflammatory cells infiltration along intestinal mucosal lining seen with few areas of oedema, necrosis and congestion also present.

Then regular dressing, chest physiotherapy, spirometry, mobilisation was carried out.

Patient then discharged on POD-7 with healthy stitch line and stable general condition and call for regular follow up in hospital for dressing purpose.

Case 4

A 65 year old non-smoker, chronic alcoholic male patient admitted in emergency surgery ward with complaint of pain in abdomen since 10 days associated with continuous fever since 2 days . Patient did not have history of nausea, vomiting, diarrhea, constipation or any other medical comorbidity.

On examination - Patient was conscious cooperative and well oriented to time place and person.

Pulse 100/minute, BP 110 /70mmhg, respiratory rate 30/minute.

On chest examination- right sided minimal crepitation present

Per abdomen -RHC tenderness present.

CXR findings-right sided minimal pleural effusion

USG abdomen-multiple liver abscess with subcapsular rupture largest being noted 430 cc in segment VII of right lobe of liver and multiple partially liquefied liver abscess noted in left lobe of liver with minimal free fluid in RIF, pelvic cavity and perihepatic region

Moderate inflammation of terminal ileum and caecum suggestive of infective or inflammatory etiology

IMPRESSION-multiple liver abscess with largest liquefied abscess in segment VI with subcapsular extension.

Blood Investigation-

HB/WBC/PLT-10.8/6.3/53000

BILLIRUBIN T/D/I-2.9/2.3/0.6

SGOT-152 SGPT-62 ALP-350

UREA/CREAT-109/2.6

PROTIEN T/ALB/GLOB-3.9/2.1/1.8

Na⁺/K⁺/Cl⁻ -138/4.0/99.

Procedure-pigtail insertion in right lobe of liver done under USG guidance under local anaesthesia.50cc stat purulent collection drained.

On next day- patient vitally deteriorates. Pulse -110/min BP-90/60mmhg.so fluid resuscitation and inotropic support started.

Repeat Blood investigations-

HB/WBC/PLT-9.4/70.4/15000

SGPT/SGOT/ALP-673/270/332

BILLIRUBIN T/D/I-2.6/1.4/1.2

PROTIEN T/ALB/GLOB-4.9/2.2/2.0

UREA/CREAT-152/4.0

Na⁺/K⁺/Cl⁻ -130/5.5/104

PT/APTT/INR-26.4 SEC/1.83SEC/>1MIN.

No significant pigtail output noticed on next day.

Procedure-So liver abscess tapping in right lobe of liver done under local anesthesia under USG guidance and 5 CC pus aspirated

4fresh frozen plasma transfused.

After day of this-repeat blood investigations are

HB/WBC/PLT-7.7/51000/75000.

Patient had low blood pressure and tachycardia with tachypnea and on inotropic support. He suddenly grasped and was intubated.

After 6 hours of intubation, pulse and blood pressure was non recordable.

So CPR given with inotropic support ,but despite of all efforts patient cant revived and declared expired.

II. Discussion

Entamoeba Histolytica is primarily an intra-luminal living organism of the large bowel. After consumption of an amoebic cysts via feco-oral route, cysts travel through the small intestine to the large intestine where they spring up the trophozoite stage that invades the bowel wall. Primarily the lesions are located in the large intestine though some may be seen in the terminal ileum. Initial lesions are more commonly localized in those fields where the colonic flow is slow, likely the cecum and recto sigmoid. The initial lesion is pinhead sized, but with rise in mucosal edema central ulceration results. Ulceration is mainly localized to mucosal epithelium and lamina propria. But when the ulcers progress to the muscularis propria they extend laterally along the axis of the intestine undermining the overlying mucosa. The communication of these laterally spreading ulcers with the intestinal lumen through a narrow mucosal defect creates the so called "flask like" ulcers. Adjacent ulcers may coalesce, leading to larger mucosal defects. Sometimes In advance cases, ulcers progresses beyond the muscularis propria and penetration results in a perforation of the intestinal wall. These perforations commonly occur in the cecum. [7]

Amoebic liver abscess is the commonest extra-intestinal complications of amoebiasis. This form takes months to years to develop after the intestinal stage of the infection. The onset of hepatic symptoms may be gradual or rapid. Liver abscess is characterized by pain in the right upper quadrant, fever, anorexia, nausea, hepatomegaly and Liver tenderness etc.

Liver abscesses are most commonly located in the right lobe and may be single or multiple. An early abscess initially presented with a minor area of parenchymal necrosis with greyish-brown cut surface. As far as area of necrosis increases in size, the center liquefies and a true cavity forms. These contents, most of the times sterile and non-pyogenic, become viscid and chocolate-colored ("anchovy sauce"). The incidence of intraperitoneal rupture of amoebic liver abscess is between 6 - 9 percent.

The both of above complications occurring simultaneously are very rare. Mukherjee et al documented single case of amoebic bowel perforation who died on the second post-operative day.[8] Eggleston et al studied 26 cases of bowel perforations of the bowel, out of them 6 cases had un-ruptured liver abscess.[9,10].

From treatment point of view drug of choice for amoebic colitis is metronidazole (given for 5 to 10 days). Treatment is divided into two groups of antiparasitic drugs: luminal and tissue agents. Asymptomatic phase of E. histolytica can be treated with luminal agents alone. Drugs used for the treatment of luminal infections are iodoquinol, diloxanide furoate, and paromomycin (5 to 20 days to eradicate colonization). The addition of broad-spectrum antibiotics to the handling of acute amoebic colitis may be appropriate if perforation is suspected. The possibility of coexisting bacteria causing dysentery must always be considered.

III. Conclusion

Simultaneous presentation of burst amoebic liver abscess with bowel perforation is an extremely rare entity and carries a very poor prognosis with almost 100% mortality.

References

- [1]. Haque, R., C. D. Huston, M. Hughes, E. Houpt, and W. A. Petri, Jr. Current concepts: amoebiasis. N. Engl. J. Med. 2003; 348:1565-1573.
- [2]. Stanley, S. L., Jr. Amoebiasis. Lancet. 2003; 361:1025-1034.
- [3]. World Health Organization. Amoebiasis. Wkly. Epidemiology. Rec. 1997; 72: 97-100.
- [4]. Majeed SK, Ghazanfar A, Ashraf J Caecal amoeboma simulating malignant neoplasia, ileocaecal tuberculosis and Crohns disease. J Coll Physicians Surg Pak. 2013; 13:116-7
- [5]. Rives J D, Heibner W C and Powe U J L. The surgical complications of amoebiasis of the colon. Surg. Clin. N. Amer. 1955; 35, 1421-1426.
- [6]. Radke, R. A. Ameboma of the intestine; an analysis of the disease as presented in 78 collected and 41 previously unreported cases. Ann. Int. Med. 1965; 43:1048-66.
- [7]. Mukerjee Sand Nigam M. Amoebic perforation of the colon. Amer. J. Proctol. 1975; 26(2):57-64.
- [8]. Eggleston F C Verghese M and Handa A K. Amoebic perforation of the bowel: experience with 26 cases. Brit. Surg. 1978; 65:748-751.