Print ISSN: 2321-6379
Online ISSN: 2321-595X

Original Article

Prospective Observational Study to Review the Contemporary Management of Amoebic Liver Abscess

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Abstract

Introduction: Amoebic liver abscess is an inflammatory space-occupying lesion of the liver caused by *Entamoeba histolytica*. Trophozoites after lodging in sufficient numbers in venules lead to thrombosis and infarction and microabscesses formation and thence amoebic abscess. Fever and pain right upper quadrant are very common.

Aims and Objectives: The aim of the study was to review the contemporary management of amoebic liver abscess.

Materials and Methods: This hospital based observational study for 100 patients was performed in the Surgery Department in SGT Medical College, Budhera, Gurugram, Haryana. A detailed history and focussed clinical examination and required investigations were done. Patients were divided into three categories according to sizes of abscesses, <5 cm, 5–8 cm, and >8 cm. In Category I, patients were treated with metronidazole 40 mg/kg body weight in three divided doses. If toxemia was there, then metronidazole 100 ml, IV 8 hourly was given for 3–4 days and switched onto oral metronidazole. In Category II, if a trial of conservative treatment for 3 days did not relieve the symptoms, and in Category III, ultrasound-guided pigtail catheterization was done.

Results: In our study, maximum patients were between 21 and 60 years of age. Male:female ratio was 4.55:1. Patients presented with fever, pain right hypochondrium, and hepatomegaly. Liver enzymes were elevated. Total lymphocyte counts were raised and serum albumin was decreased in some patients.

Conclusion: Due to our concrete planning and meticulous care at every step, there was lack of infection at the port site or deep-seated, and there has been no failure of resolution, no hemorrhage, recurrence, pleural complications, and fever and there has been no death in our series.

Key words: Abscess, Amoebic, Complications, Drainage, Liver, Ultrasound

INTRODUCTION

Amoebic liver abscess is an inflammatory space-occupying lesion of the liver caused by *Entamoeba histolytica*. Sushruta described "atissar" but did not give an account of hepatic involvement. Hippocrates (460–377 BC) described two types of large hepatic abscesses. In one the pus was "pure and white." In other the pus was like oil. Alaxender – the Great was born after the death of Hippocrates, died at the age of 33 years, possibly of amoebic liver abscess. Amoebic liver abscess by that time had reached as far as Indus and must have slipped in the endemic area. Napoleon contracted tropical hepatitis, died in 1921 due to Ruptured left lobe abscess into stomach (contrary to the common belief that he died of carcinoma stomach). Dr. Charles Morehead, Professor of Medicine, Grant Medical College Bombay reported 1st case of hepatic abscess in 1848. Prof. Morehead concluded that there is the coexistence of amoebic liver abscess and ulceration of mucosa of the large intestine.

Access this article online

www.ijss-sn.com

Month of Submission : 02-2019
Month of Peer Review : 03-2019
Month of Acceptance : 03-2019
Month of Publishing : 04-2019

Amoebiasis is defined as harboring of *E. histolytica* with or without clinical manifestation. *Entamoeba dispar* is a separate species, which is non-pathogenic. It is morphologically

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identical to *E. histolytica* but differ in the surface antigen, DNA markers, and RNA sequence.[1,2] Incidence of disease is as follows. Of all amoebic infections, 10% cause intestinal amoebiasis. Of intestinal amoebiasis, about 6% cause an amoebic liver abscess.[3] Of amoebic liver abscess, 10% occurs in left lobe and 10% occurs in both lobes. 80% are single and 20% are multiple.

Mechanism of virulence for *E. histolytica* is like this: Trophozoites first attach to colonic mucosa by their N acetyl lectine. Then, there is the release of enzymes phosphorylase a and pore-forming peptides and cause cytolytic effects. Further virulence is due to their ability to resist reactive oxygen species and nitrogen species. Cell injury caused results in the different lesion. In invasive amoebiasis, there may be minute superficial ulcerations, hemorrhage, perforation, amoebic appendicitis, and ameboma. In the extra-intestinal mode, the trophozoites enter mesenteric venule, lymphatics and carried to liver, and lung and other organs.

Trophozoites after lodging in sufficient numbers in venules lead to thrombosis and infarction of small areas of hepatic parenchyma. At this stage, if resistance is good, healing occurs. If host nutrition and immune status is poor, then further destruction by the cytolytic activity of amoeba occurs leading to amoebic hepatitis. There is microabscesses formation, which coalesce, leading to the formation of an amoebic liver abscess. Fluid of amoebic liver abscess is usually anchovy sauce like but maybe creamy white even though there is no secondary infection[4] or it may be reddish brown. It is odorless and sterile. It is a mixture of blood and destroyed liver cells. An abscess is mostly, solitary, in the right lobe of liver due to streaming of blood in the portal vein, because superior mesenteric vein flow goes to right lobe. Inferior mesenteric vein and splenic vein flow go to left lobe.

Clinical presentation is as below. Most common age involved is 21–60 years. Fever and pain right upper quadrant occurs in >90% patients. Concurrent diarrhea occurs in a few patients. There may be cough, shortness of breath, and pleural pain. Hepatomegaly may be present. Pointed intercostal tenderness may be present. Amoebic liver abscess must be thought of in differential diagnosis of pyrexia of unknown origin. Jaundice may be found.

An abscess may rupture into the lung, pleural cavity, peritoneal cavity, skin, intestine, and pericardial cavity. Conservative treatment is done by oral or IV metronidazole. In patients where indicated percutaneous catheter drainage by pigtail catheter is done. Resolution is said to occur when there is clinical relief such as relief in pain, tenderness, guarding, toxemia, anorexia, and hepatomegaly. Sonography resolution is indicated by a decrease in the size of the abscess. Surgery is done when there is a failure of percutaneous catheter drainage, or there is thick pus or the abscess is multiloculated, or there are multiple abscesses.

MATERIALS AND METHODS

Study Site

The study was performed in the Surgery Department at SGT Medical College, SGT University, Budhera, Gurugram, Haryana, over a period of 2 years and 6 months from September 2015 to March 2018.

Study Design

This was a hospital-based prospective observational study.

Selection of Subjects (Cases)

A total of 100 patients were studied. Informed consent was taken for examination and investigations, giving due respect to maintain the patient's privacy and keep them comfortable.

Ethical Considerations

The Institutional Ethics Committee's approval for research on human subjects was taken. Throughout the study, strict ethical norms were maintained. Written informed consent was taken from the patient in their local language (mother tongue).

Statistical Analysis

The data were collected properly, and entries were made, and statistical analysis was carried out using simple mathematical expressions like percentage. The data were subjected to appropriate statistical test wherever applicable.

Data Collection

The patients were taken from the outpatient department and emergency unit of general surgery department. A detailed history and focused clinical examination were done. Investigations done were, hemoglobin, bleeding time, clotting time, total lymphocyte counts (TLC), differential leukocyte count, platelet count, blood sugar, liver function test, blood urea, serum creatinine, hepatitis C virus, HIV, hepatitis B virus surface antigen, ultrasonography, X-ray chest posteroanterior view, and electrocardiogram. Serological investigations were not done. Pyogenic liver abscesses were not considered in this study.

Procedure

On the basis of clinical examination and investigations and ultrasonography patients were divided into three categories. In Category I, amoebic liver abscesses of size <5 cm, small abscesses in the liquefaction stage without any complications were considered. These were treated with

oral metronidazole 40 mg/kg body weight in three divided doses. If toxemia was there, then metronidazole 100 ml, IV 8 hourly was given for 3–4 days and then switched onto oral metronidazole. Those patients who responded within 48 h were given a full treatment of 10 days.

In Category II, abscesses of size 5–8 cm were taken. They were given a trial of conservative treatment of 3 days. If there was no response clinically, they were taken for ultrasound-guided (USG) pigtail catheter drainage.

In Category III, abscesses of size >8 cm were taken. They were straightway treated by USG pigtail catheter drainage. The pus from abscess was sent for culture and sensitivity. The pigtail catheter was removed when drainage was <10 ml/24 h for 2 consecutive days.

Time taken for resolution of abscess cavity was noted, complications if any were recorded. Hospital stay was recorded. Patients were followed up weekly for a month, monthly for 3 months and 3 monthly for a year.

RESULTS

In our study maximum patients, 92 (92%) were between 21 and 60 years of age [Table 1]. The age ranged from 11 to 70 years. The mean age in our study was 33 years. 82 (82%) were male patients and 18 (18%) were female patients. Male:female ratio was 4.55:1 [Table 2]. Most of the patients, 66 (66%) belong to low socioeconomic status. 4 (4%) patients belonged to a higher status [Table 3]. 90 (90%) patients presented with fever, 92 (92%) patients presented with pain right hypochondrium, rigor and chills were present in 42 (42%), nausea/vomiting in 44 (44%), weight loss in 40 (40%), diarrhea in 22 (22%), cough in 28 (28%), and shortness of breath present in 18 (18%) patients [Table 4]. Jaundice was present in only 20 (20%) patients. Tenderness in right hypochondrium was present in most, 60 (60%) patients. Hepatomegaly was found in 44 (44%) patients. In a few patients ascites, pleural effusion or toxemia was present [Table 5]. Most of the patients, 52 (52%) were anemic, with a hemoglobin level <10 gm. Raised serum bilirubin was present in 18 (18%) patients. Serum glutamic oxaloacetic transaminase (SGOT) and serum glutamic pyruvic transaminase (SGPT) were elevated in 22 (22%) patients. Serum alkaline PO_4 was raised in 33 (33%) patients. TLC was raised in 58 (58%) patients. Serum albumin was decreased by 34 (34%) patients [Table 6]. In our series, the size of abscess cavity was <5 cm in 26 (26%) patients, 5–8 cm in 42 (42%) patients and 9–12 cm in 32 (32%) patients [Table 7]. The time taken for resolution of abscess cavity of size <5 cm was about 6 weeks. For resolution of abscess cavity of size 5–8 cm was 10 weeks and for 9–12 cm, it was about 8 months [Table 8]. In our series, due to our concrete planning and meticulous care at every step, there was no infection at port site and deep-seated, no failure of resolution, no hemorrhage,

Table 1: Age incidence of amoebic liver abscess patients

Age group in years	Number of patients (%)
0–10	0 (0)
11–20	2 (2)
21–30	14 (14.5)
31–40	22 (22.5)
41–50	32 (32)
51–60	24 (24)
61–70	6 (6.5)
71–80	0 (0)
81–90	0 (0)
91–100	0 (0)
Total	100 (100)

Table 2: Sex incidence of amoebic liver abscess patients

Number of male patients	Number of patients (%)
82	18 (4.55:1)

Table 3: Socioeconomic status of amoebic liver abscess patients

Socioeconomic status	Number of patients (%)
Low	66 (66)
Middle	30 (30)
High	4 (4)
Total	100 (100)

Table 4: Symptoms of amoebic liver abscess in patients

Symptoms	Number of patients (%)
Fever	90 (90)
Pain right hypochondrium	92 (92)
Rigor, chills	42 (42)
Nausea/vomiting	44 (44)
Weight loss	40 (40)
Diarrhea	22 (22)
Cough	28 (28)
Shortness of breath	18 (18)

Table 5: Signs of amoebic liver abscess in patients

Signs	Number of patients (%)
Jaundice	20 (20)
Tenderness in right hypochondrium	60 (60)
Hepatomegaly	44 (44)
Ascites	6 (6)
Pleural effusion	5 (5)
Toxemia	4 (4)

recurrence, pleural complication, and fever. There was no death in our series [Table 9]. Median hospital stay of the patient was 7 days with a range of 3–18 days [Table 10]. About 60% of patients turned up for follow-up. Rest were lost after 3 or 4 weeks of follow-up.

DISCUSSION

In our study, most significant clinical features are upper right quadrant pain and fever. Bhat describes pain right hypochondrium present only in 30% of patients.[5] Das describes the presence of pain in about 90% of patients.[4] We feel that if the chief complaints of a patient are a pain in the right hypochondrium, tender hepatomegaly, and fever, then this entity should always come to our mind. Sonography remains an important tool in the diagnosis.[6] In our study, the incidence of the disease is maximum between 31 and 60 years. Male:female ratio is 4.55:1. Bhat describes male to female ratio as 10:1.[5] Most of the patients suffering from this disease in our study are from poor socioeconomic group. This is true also because the disease is spread by fecal-oral route.[7] The disease is caused by ingestion of food contaminated with *E. histolytica* cysts. This occurs where there is no proper disposal of excreta and water supply is not safe, which is found mostly in poor socioeconomic societies. Tenderness in the right hypochondrium and hepatomegaly are most common features. Mild jaundice is also found in 20 (20%) patients. Jaundice is severe if the abscess is big or multiple, situated at porta hepatis.[8] Hepatic encephalopathy may occur in multiple liver absceses.[9] Rupture of the liver abscess with peritonitis is seen occasionally. The incidence of the sepsis reported is around 25%.[9-11] More than half of patients were anemic. TLC was raised in 58 (58%) patients. SGOT, SGPT, and serum alkaline PO_4 were also raised in many patients. Das also supports this finding.[4] Decreased serum albumin is found in 34 (34%) patients. This being a prognostic marker if its value is <2 g%. Other prognostic markers are bilirubin >3.5 mg%, encephalopathy, huge volume of abscess, and delay in the exploration of abscess or burst in the peritoneal cavity and burst into the pericardium. Sharma *et al.*[12] have also mentioned similar prognostic markers. We have divided our abscesses into three categories. In Category I, the size of the abscess taken was <5 cm diameter. This was present in 26 (26%) patients. These were treated with oral metronidazole 40 mg/kg body weight in three divided doses. If toxemia was there, then metronidazole 100 ml, IV 8 hourly was given for 3–4 days and then switched onto oral metronidazole. Those patients who responded within 48 h were given a full treatment of 10 days. Irusen *et al.*[13] also treated amoebic liver abscess with oral or intravenous metronidazole with good results. Antiamoebic therapy alone is very effective, and there is no need for its combination with routine needle aspiration.[14,15]

In Category II, the size of the abscess was 5–8 cm diameter and it was in 42 (42%) patients. In Category III, the size of the abscess was >9–12 cm diameter and it was in 32 (32%) patients. In Category II, if a trial of conservative treatment for 3 days did not relieve the symptoms, and in Category III, USG pigtail catheterization was done. de la Ray Nel *et al.*[16] did aspiration in abscesses in which there was no clinical improvement after 48–72 h, in left lobe abscess, when there was very thin liver tissue around the abscess and seronegative abscesses. The abscess cavities of Category I, II, and III

Table 6: Findings of investigations

Investigations	Number of patients (%)
Hb <10 g	52 (52)
Raised serum bilirubin	18 (18)
Raised SGOT, PT	22 (22)
Raised serum alkaline PO4ase	33 (33)
Raised TLC	58 (58)
Decreased serum albumin	34 79 (34 79)
ELISA test for amoebiasis	

SGOT: Serum glutamic oxaloacetic transaminase, Hb: Hemoglobin, TLC: Total lymphocyte counts, PT: Pyruvic transaminase

Table 7: Size of abscess cavities in patients

Size of abscess cavity (cm)	Number of patients (%)
<5	26 (26)
5–8	42 (42)
9–12	32 (32)
Total	100 (100)

Table 8: Time taken for resolution of abscess cavities

Size of abscess (cm)	No of days
<5	6 weeks
5–8	10 weeks
9–12	8 months

Table 9: Hospital stay (days)

Median hospital stay	Range
07	3–18

Table 10: Complications of treatment (pig tail catheterization)

Complications	Number of patients (%)
Infection (port site and deep-seated)	Nil
Failure of resolution	Nil
Hemorrhage	Nil
Recurrence	Nil
Pleural complication	Nil
Fever	Nil
Mortality	Nil

resolved in 6 weeks, 10 weeks, and 8 months, respectively. Usually, sonographic resolution lags behind clinical/hematological resolution. This is very significant because a patient of amoebic liver abscess who has become alright with proper treatment may still show abscess cavity on USG and an errant clinician may think that the person is still suffering from an amoebic liver abscess. Average median hospital stay in our study was 7 days with a range of 3–18 days.

Most important aspect of our series is the lack of infection at the port site or deep-seated, after drainage. There has been no failure of resolution, no hemorrhage, recurrence, pleural complications, and fever. There has been no death in series due to our concrete planning and meticulous care at every step. I also lay stress on community prevention, for which following measures should be taken. Safe disposal of human excreta, protection of water supply, washing of hands before taking food and after defecation, washing of vegetables before use, care of food handler, and counseling to avoid consumption of alcohol.

CONCLUSION

Due to our concrete planning and meticulous care at every step, there was lack of infection at the port site or deep-seated, and there has been no failure of resolution, no hemorrhage, recurrence, pleural complications, and fever and there has been no death in our series.

ACKNOWLEDGMENTS

I would like to acknowledge the books, SRB's Manual of Surgery 5th edition by Sriram Bhat M, Bailey, Love's Short Practice of Surgery 26th Edition by Norman S Williams, Norman S. Williams, Christopher J K Bulstrode, P Ronan O'Connell, and A Concise Textbook of Surgery, 5th edition by S Das.

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How to cite this article: Goel KS, Sinha AR. Prospective Observational Study to Review the Contemporary Management of Amoebic Liver Abscess. Int J Sci Stud 2019;7(1):36-40.

Source of Support: Nil, **Conflict of Interest:** None declared.