

ORIGINAL RESEARCH

Clinical and Biochemical Profile in Patients of Liver Abscess: A Study from Tertiary Care Centre in Bihar

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ABSTRACT

Aim: Clinical and Biochemical Profile in Patients of Liver Abscess: A Study from Tertiary Care Centre in Bihar.

Methods: This cross-sectional research was conducted. Patients who met the inclusion criteria and provided written informed consent were chosen for the research after authorization from the institutional ethics committee was obtained. All consenting people above the age of 18. This research comprised patients who presented with consistent symptoms of liver abscess and patients who were confirmed with liver abscess radiologically (Ultrasonography and CT Scan, if necessary). Patients who refused to provide permission for the research were omitted from the trial.

Results: Alcohol use was the most prevalent risk factor in our research, accounting for 50(50%) of the cases. Diabetes mellitus (DM) was shown to be another substantial risk factor, accounting for 22% of all cases in the current investigation. Both risk factors were present in 14% of the cases. The most prevalent symptom was abdominal pain (97 percent), followed by fever (95 percent). The most prevalent per abdominal examination finding was hepatomegaly (81%), followed by abdominal pain (73 percent). Anorexia was discovered in 51% of the patients. As a result of the problems, 13% of patients had right sided pleural effusion and 7% developed ascitis. Based on biochemical measures, 81 (81 percent) of 100 patients exhibited TLC counts more than 11000/mm³. ALP levels were elevated in 97 percent of patients. In 75 individuals (75 percent), the INR was raised.

Conclusion: If a patient has a protracted fever and discomfort in the upper abdomen, a liver abscess should be considered, especially if the patient is an alcoholic or has diabetes mellitus. Ultrasonography is a simple and inexpensive way to identify a liver abscess. To avoid complications, morbidity, and death, early and urgent treatment is essential.

Keywords: Liver Abscess, Clinical and Biochemical Profile

INTRODUCTION

Liver abscess is linked with up to 20% mortality ¹ and is classified into many categories depending on aetiology, the most common of which are amoebic (ALA) and pyogenic (PLA) liver abscess. Surprisingly, ALA is more frequent in underdeveloped countries. In wealthy countries, ¹ PLA accounts for the majority of hepatic abscesses. PLA is caused by ascending biliary tract infection, hematogenous spread through the portal venous system, septicemia

with liver involvement via the hepatic artery circulation, and secondary dissemination by intraperitoneal infection. PLA is most often caused by *Escherichia coli*, *Klebsiella*, and *Streptococcus*.²

Although there are no clear clinical criteria for separating ALA from PLA, the following factors may be used to make a differential diagnosis: younger age, residence in or recent travel to locations with endemic amoebiasis, diarrhoea, and severe stomach discomfort increase clinical suspicion of ALA. Ultrasonography (USG), serological tests such as indirect hemagglutination test, reddish brown (anchovy paste like substance) aspirate from the abscess, negative gramme stain, and quick clearance following metronidazole therapy are used to confirm the diagnosis. The picket fence arrangement of the temperature chart, nausea, vomiting, anorexia, haematological analysis of leukocytosis, anaemia, and positive blood or aspirate culture for bacterial aetiology are used to make the diagnosis of PLA. The treatment of liver abscesses has progressed significantly, with minimally invasive drainage taking centre stage. Radiological imaging has increased diagnostic competence and revolutionised therapy strategy by enabling percutaneous approaches employing needle aspiration or catheter drainage. While open surgery should be reserved for the most difficult instances.^{2,3}

In the absence of considerable alcohol usage, NAFLD comprises a histological spectrum ranging from steatosis through steatohepatitis, severe fibrosis, and cirrhosis. In the Western world, NAFLD is the most frequent cause of liver disease.⁴

In this paper, we report a research of clinical, lab parameters, and management of suspected liver abscess patients in order to make an early diagnosis, initiate timely treatment, and avoid sequelae.

METHODS AND MATERIALS

This cross-sectional research was conducted. Patients who met the inclusion criteria and provided written informed consent were chosen for the research after authorization from the institutional ethics committee was obtained. All consenting people above the age of 18. This research comprised patients who presented with consistent symptoms of liver abscess and patients who were confirmed with liver abscess radiologically (Ultrasonography and CT Scan, if necessary). Patients who refused to provide permission for the research were omitted from the trial.

METHODOLOGY

The investigation was done on 100 patients with liver abscess who came to medicine OPD/Casualty during a one-year period. A full history was gathered, as well as a thorough physical examination. All patients had a full hemogram, a liver function test, a kidney function test, a coagulation profile (PT/INR), and an ultrasound of the abdomen. The hospital laboratory's reference ranges were used to create the reference ranges for these examinations. Urine and blood cultures were submitted. Serologies were also performed for *Entamoeba histolytica*, HIV, hepatitis B, and hepatitis C viruses. Whenever pus was aspirated, cultures were performed. When the abscess was liquefied, the pus was aspirated, and the patients were put on antibiotics on an empirical basis.

RESULTS

The research included 100 patients, 85 (85%) of whom were men and 15 (15%) of whom were girls. The male-female ratio was 5.67:1. The age range of 30-40 years had the greatest number of instances. The patients' average age was 40.58 years. (See Table 1). The aetiology of liver abscess (based on amoebic serology and pus culture) indicated that 80 percent were amoebic (n=80) and 20 percent were pyogenic (n=20). Alcohol use was the most prevalent risk factor in our research, accounting for 50(50%) of the cases. Diabetes mellitus (DM) was

shown to be another substantial risk factor, accounting for 22% of all cases in the current investigation. Both risk factors were present in 14% of the cases (Table 2).

The most prevalent symptom was abdominal pain (97 percent), followed by fever (95 percent). The most prevalent per abdominal examination finding was hepatomegaly (81%), followed by abdominal pain (73 percent). Anorexia was discovered in 51% of the patients. As a result of the problems, 13% of patients had right sided pleural effusion and 7% developed ascitis (Table 3). On ultrasonography, the right lobe was affected in 77% of the patients, the left lobe in 15%, and multiple abscesses (both lobes) in 11% of the cases.

Based on biochemical measures, 81 (81 percent) of 100 patients exhibited TLC counts more than 11000/mm³. ALP levels were elevated in 97 percent of patients. In 75 individuals (75 percent), the INR was raised (Table 4). In the current investigation, we only aspirated the pus once the abscess had been liquefied. As a result, pus cultures were negative in 98 of 100 patients, despite the fact that the patients had been put on empirical antibiotics prior to pus aspiration. In the current series, there was no mortality.

Table 1: Demographic profile of liver abscess patients

| Gender | Number | % |
|----------|--------|----|
| Male | 85 | 85 |
| Female | 15 | 15 |
| Age | | |
| Below 20 | 7 | 7 |
| 20-30 | 21 | 21 |
| 30-40 | 44 | 44 |
| 40-50 | 15 | 15 |
| Above 50 | 13 | 13 |

Table 2: Risk factor profile of liver abscess patients

| Risk factors | Number | % |
|--------------|--------|----|
| Alcoholic | 50 | 50 |
| DM | 22 | 22 |
| ALC+DM | 14 | 14 |

Table 3: Clinical features of liver abscess patients

| Signs and symptoms | ALA=80 | PLA=20 | Total | % |
|----------------------|--------|--------|-------|----|
| Fever | 75 | 20 | 95 | 95 |
| Pain abdomen | 77 | 20 | 97 | 97 |
| Vomiting | 15 | 6 | 21 | 21 |
| Nausea | 43 | 6 | 49 | 49 |
| Jaundice | 19 | 0 | 19 | 19 |
| Cough | 17 | 6 | 23 | 23 |
| Abdominal tenderness | 59 | 14 | 73 | 73 |
| Hepatomegaly | 69 | 12 | 81 | 81 |
| Anorexia | 45 | 6 | 51 | 51 |
| Pleural effusion | 10 | 3 | 13 | 13 |
| Ascitis | 4 | 3 | 7 | 7 |

Table 4: Biochemical parameters of liver abscess patients

| | ALA=80 | PLA=20 | Total=100 | % |
|---------------------|--------|--------|-----------|----|
| TLC >11000/ μ L | 67 | 14 | 81 | 81 |
| S. Bil >1.2mg/Dl | 30 | 3 | 33 | 33 |

| | | | | |
|-----------------|----|----|----|----|
| SGOT >35 | 53 | 14 | 67 | 67 |
| SGPT >35 | 57 | 12 | 69 | 69 |
| SAP (IU/L) >100 | 77 | 20 | 97 | 97 |
| S. albumin <3.5 | 70 | 19 | 89 | 89 |
| INR >1 | 56 | 19 | 75 | 75 |

DISCUSSION

Tropical nations have a higher prevalence of liver abscess. ^{5,6} *E. histolytica* (amoebic) and bacteria are the most prevalent etiological agents causing liver abscess (pyogenic). The majority of liver abscess cases in underdeveloped countries are caused by amoebic bacteria. In the current research, ALA accounted for more than three-fourths of the cases, with the majority of them being isolated right lobe abscesses. This pattern of participation has been observed before in earlier ALA series by Sharma et al, Mukhopadhyay et al, and Ghosh et al. ⁷⁻⁹

The mean age of the patients in our research was 40.58 years, which was consistent with Indian studies, Ghosh et al, Sharma et al, and Mukhopadhyay et al, which reported 41, 40.5, and 43.64 years, respectively.

⁷⁻⁹ The most common age group for liver abscess was 40-50 years of age, with comparable findings in ALA patients, although PLA was found in all age groups with equal frequency. However, research from the west, where PLA are more numerous, show that the average age is beyond 60 years. ¹⁰⁻¹²

In terms of gender predilection, just 15 patients were female after recruiting 100 consecutive patients. Male participation is more prevalent in Indian data; Sharma et al, Mukhopadhyay et al, and Ghosh et al observed male to female ratios of 14.4:1, 7:1, and 11:1, respectively. ⁷⁻⁹ Pang et al and Heneghan et al, on the other hand, reported it to be 2:1 and 1.22:1, respectively. ^{10,11}

Alcohol intake was identified as a key risk factor in the current investigation, with 60 percent of ALA patients exposed to it, whereas only 15 percent of PLA cases confirmed to be alcoholic. It contradicts the findings of Islam QT et al, who discovered a relationship between indigenous alcohol and the development of pyogenic liver abscess. ¹³ Ghosh et al's research included 72 percent alcoholic patients. ⁹ Alcohol inhibits the action of Kupffer cells (specialised macrophages) in the liver, which play a vital role in amoeba clearance. Furthermore, it seems that invasive amoebiasis is reliant on the availability of free iron. A high iron level in the diet, commonly gained from country liquor in chronic drinkers, predisposes to invasive amoebiasis, as does a carbohydrate-rich diet. ¹⁴

Diabetes mellitus was another risk factor noted in 22% of instances, with drunkenness being a risk factor in 14% of patients. Ghosh et al reported diabetes in 9% of patients, whereas Das et al recorded diabetes in 70% of patients. When compared to control participants, diabetic patients had a 3.6-fold greater chance of acquiring PLA, according to Thomsen et al. ¹⁵

The most prevalent symptoms in the current research are abdominal discomfort and fever, with a frequency of 97 percent and 95 percent, respectively. Ghosh et al found it to be prevalent in 99 percent and 94 percent, respectively, although other studies find it to be present in 62-94 percent and 67-87 percent, respectively. ⁷⁻⁹

Other major effects were nausea and anorexia, which were seen with 49 percent and 51 percent of patients, respectively, with a higher prevalence in ALA. In their research, Ghosh et al found that 93% of patients had anorexia and 54% had nausea and vomiting. ⁹ Cough was another rare symptom that occurred in 23% of individuals.

Jaundice was seen in only 19% of patients, which is consistent with the trend. Ghosh et al discovered it in 26% of patients. ⁹ It was observed in 45-50 percent of patients in previous Indian investigations. ¹⁶ However, with the development of effective antibiotic treatment, it

has becoming less prevalent. Sharma et al found it in just 12.7% of cases.⁷ Yoo et al. examined data from patients during the 1970s and 1980s and found that the incidence of jaundice decreased from 25% to 7% throughout this time period.¹⁷ Diarrhoea was seen in 25% of patients in the current research, which was previously described by Ghosh et al in 23% and varying from 4% to 33% in prior investigations.^{9-10, 18-20}

We found hepatomegaly in 81% of the patients. Ghosh et al found it in 89 percent of instances, which agrees with the current research, however Das et al found it in only 40 percent of cases, which contradicts the current study.^{9,21}

In the current research, leukocytosis was identified in 81 percent of patients, with 83.75 percent and 70 percent frequency in ALA and PLA, respectively. As a result, leucocytosis is not an absolute sign of PLA and cannot be used to distinguish ALA from PLA. Ghosh et al. found similar results with leucocytosis in 82 percent of patients.⁹ In a research conducted by Malik and his colleagues, leucocytosis was found in 68 percent of PLA patients.²² Khan and colleagues discovered 26.7 percent polymorphonuclearleucocytosis.²³

Serum alkaline phosphatase was raised in 97 percent of patients in the current research, with 96.25 percent and 100 percent frequency from ALA and PLA, respectively, which is consistent with prior investigations.

SGOT and SGPT levels were elevated in 64% and 66% of patients, respectively. 89 percent of patients had albumin levels of <3.5g/dL or above. Ghosh et al. observed a similar result. In 85.7 percent of PLA patients and 74% of overall liver abscess cases, the PT >INR was more than one.

In terms of radiological findings, 67 percent of patients had solitary abscesses, whereas 35 percent had numerous abscesses. Similarly, Ghosh et al. discovered 65 percent of single abscesses.⁹ In accordance with prior investigations, the right lobe was mostly affected.^{8,9,21,24,26} The average volume of the abscess was 210cc, with involvement of either the right, left, or both sides.

The three problems seen in this research were pleural effusion, ascites, and ascites. Pleural effusion was reported in 13% of the patients, whereas ascites was seen in 7% of the cases. Siddiqui et al. observed a similar result.⁶ Other problems such as sepsis, multi-organ failure, and mortality were not seen in this research, which might be due to the disease's early detection and rapid and vigorous treatment. Another factor is that the current study's sample size is tiny.

CONCLUSION

If a patient has a protracted fever and discomfort in the upper abdomen, a liver abscess should be considered, especially if the patient is an alcoholic or has diabetes mellitus. Ultrasonography is a simple and inexpensive way to identify a liver abscess. To avoid complications, morbidity, and death, early and urgent treatment is essential.

REFERENCES

1. Perez JY Jr. Amoebic liver abscess: Revisited. *Philip J Gastroenterol* 2006;2:11-3.
2. Pillai DR, Keystone JS, Sheppard DC, MacLean JD, MacPherson DW, Kain KC. *Entamoeba histolytica* and *Entamoeba dispar*: Epidemiology and comparison of diagnostic methods in a setting of nonendemicity. *Clin Infect Dis* 1999;29:1315-8.
3. Makkar RP, Sachdev GK, Malhotra V. Alcohol consumption, hepatic iron load and the risk of amoebic liver abscess: A case-control study. *Intern Med* 2003;42:644-9
4. Farrell GC, Larter CZ, Nonalcoholic fatty liver disease: from steatosis to cirrhosis. *Hepatology*, 2006;43:S99-S112.
5. Ochsner A, DeBakey M, Murray S. Pyogenic abscess of the liver: II. An analysis of forty-seven cases with review of the literature. *Am J Surg.* 1938;40(1):292-319.

6. Siddiqui MA, Ahad MA, Ekram AS, Islam QT, Hoque MA, Masum QA. Clinico-pathological profile of liver abscess in a teaching hospital. TAJ: J Teachers Assoc. 2008;21(1):44-9.
7. Sharma N, Sharma A, Varma S, Lal A, Singh V. Amoebic liver abscess in the medical emergency of a North Indian hospital. BMC research notes. 2010;3(1):21.
8. Mukhopadhyay M, Saha AK, Sarkar A, Mukherjee S. Amoebic liver abscess: presentation and complications. Indian J Surg. 2010;72(1):37-41.
9. Ghosh S, Sharma S, Gadpayle AK, Gupta HK, Mahajan RK, Sahoo R, et al. Clinical, Laboratory, and Management Profile in Patients of Liver Abscess from Northern India. J Trop Med. 2014;2014:1423-82
10. Pang TC, Fung T, Samra J, Hugh TJ, Smith RC. Pyogenic liver abscess: An audit of 10 years' experience. World J Gastroenterol. 2011;17:1622-30.
11. Heneghan HM, Healy NA, Martin ST, Ryan RS, Nolan N, Traynor O, et al. Modern management of pyogenic hepatic abscess: a case series and review of the literature. BMC Res Notes. 2011;4(1):80.
12. Mohsen AH, Green ST, Read RC, McKendrick MW. Liver abscess in adults: ten years experience in a UK centre. QJM. 2002;95(12):797-802.
13. Islam N. The poor access to under land for housing. In urban land management in Bangladesh. Ministry of Land, Government of Bangladesh; 1992:131-40.
14. Makkar RP, Sachdev GK, Malhotra V. Alcohol consumption, hepatic iron load and the risk of amoebic liver abscess: a case-control study. Intern Med. 2003;42(8):644-9.
15. Thomsen RW, Jepsen P, Sørensen HT. Diabetes mellitus and pyogenic liver abscess: risk and prognosis. Clin Infect Dis. 2007;44(9):1194-201.
16. Aikat BK, Bhusnurmath SR, Pal AK, Chhuttani PN, Datta DV. Amoebic liver abscess-a clinicopathological study. Indian J Med Res. 1978;67:381-91.
17. Yeah HM, Kim WH, Shin SK, Chun WH, Kong JK, Park IS. The changing patterns of liver abscess during the past 20 years- a study of 482 cases. Yeosec Med J. 1993;34(0):340-50.
18. Reed SL. Amebiasis: an update. Clin Infect Dis. 1993;14(2):385-93.
19. Maltz G, Knauer CM. Amebic liver abscess: a 15- year experience. Am J Gastroenterol. 1991;86(6).
20. Chuah SK, Chang-Chien CS, Sheen IS, Lin HH, Chiou SS, Chiu CT, Kuo CH, Chen JJ, Chiu KW. The prognostic factors of severe amebic liver abscess: a retrospective study of 125 cases. The Am J Trop Med Hygiene. 1992;46(4):398-402.
21. Das AK, moni Saikia A, moyee Saikia A, Dutta N. Clinico-epidemiological Profile of Patients with Liver Abscess: A Hospital Based Study. Indian J Basic Applied Med Res. 2015;5(1):17-25.
22. Malik AA, Bari SU, Rouf KA, Wani KA. Pyogenic liver abscess: Changing patterns in approach. World J Gastrointest Surg. 2010;2:395-401.
23. Khan M, Akhter A, Mamun AA, Mahmud TAK, Ahmad KU. Amoebic liver abscess: Clinical profile and therapeutic response. Bang J Med. 1991;2:32-8.
24. Hathila TN, Patel CJ, Rupani MP. A cross-sectional study of clinical features and management of liver abscesses in a tertiary care hospital, Ahmedabad, Gujarat. Nat J Med Res. 2014;4:249-52.
25. Ramani A, Ramani R, Shivananda PG. Amoebic liver abscess. a prospective study of 200 cases in a rural referral hospital in South India. Bahrain Med Bull. 1995;17(4).
26. Jha AK, Das A, Chowdhury F, Biswas MR, Prasad SK, Chattopadhyay S. Clinicopathological study and management of liver abscess in a tertiary care center. J Nat Sci Biol Med. 2015;6:71-5