Case Report

Simultaneous infection of amoebic liver abscess and hepatitis A infection in a young adult in an endemic region

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Abstract

Concomitant hepatitis A virus (HAV) and amoebic liver abscess are to be considered in patients with clinical signs and symptoms of fever, jaundice, and right upper quadrant pain, especially in endemic areas. Both diseases had similar epidemiology and identical mode of transmission, i.e., the feco-oral route. We report a case of a young female with simultaneous infection of HAV and amoebic liver abscess, emphasizing the role of dual infection and its clinical manifestations.

Keywords: Amoebiasis, Hepatitis A, liver abscess

Introduction

Simultaneous infection of amoebic liver abscess and hepatitis A (HAV) is rarely reported and should be considered in patients with fever, jaundice, and right upper quadrant pain as signs and symptoms, especially in an endemic region for both infections due to identical mode of transmission, i.e., the feco-oral route. We report a case of a 33-year-old female presenting with fever, vomiting, and right upper quadrant pain and diagnosed with amoebic liver abscess and HAV.

Case Report

A 33-year-old female presented to emergency with complaints of high-grade fever, nausea, recurrent vomiting, and loss of appetite from the last six days. On examination, she was conscious and oriented to time, place, and person. Her pulse rate was 98/min, blood pressure 110/80, respiratory rate 20/min, and oxygen saturation 98% on room air. Abdominal examination revealed mild tenderness in the right hypochondrium with a hepatic span of 18 cm, and the rest systemic examination was essentially normal.

Blood investigation revealed normal hemoglobin (Hb 12.4 gm/dl) and platelet count (406×10^9/L), neutrophilic leucocytosis (Total Leukocyte Count (TLC) 14.38×10^9/L). Liver function test revealed mild transaminitis (SGOT 38.8 U/L, SGPT 68.6 U/L, ALP 311 U/L, GGT 197 U/L); renal profile revealed no gross abnormality (urea 23.4 mg/dl, creatinine 0.8 mg/dl). Ultrasound sonography test (USG) od the whole abdomen revealed a well-defined heterogeneous lesion measuring approx. $45 \times 37 \times 42$ mm, volume 37.6 cc seen in the right lobe of the liver suggestive of an abscess [Figure 1]. Chest X-ray posteroanterior (PA) view showed no abnormality. Amoebic serology was sent, which was positive (25.73 NTU). She was

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initiated with intravenous (IV) metronidazole, IV fluid, and USG-guided aspiration of abscess showed anchovy sauce pus.

However, she continued to have a fever, right upper quadrant pain, nausea, and vomiting, so computed tomography (CT) multiphase study shows a solitary well-defined roundish hypodense lesion with thin peripheral smooth enhancing wall with a surrounding rim of edema measuring 47 × 41 × 39 mm in segment VIII of the liver, consistent with liver abscess along with mild thickening of the cecum [Figures 2 and 3]. Repeat blood investigations were done, which revealed markedly elevated liver enzymes (serum glutamic-oxaloacetic transaminase (SGOT) 1028 U/L, serum glutamic pyruvic transaminase (SGPT) 429 U/L, alkaline phosphatase (ALP) 190 U/L, gamma-glutamyl transferase (GGT) 186 U/L), so workup for acute viral hepatitis was done, which revealed Immunoglobulin M (IgM) antibody to hepatitis A virus (anti-HAV) positive [Table 1]. Serial monitoring of liver function along with coagulation profile was done, which was suggestive of acute liver failure. She gradually improved with IV fluids and supportive measures and was discharged after 16 days of admission and was doing well on the last follow-up after one month.

Discussion

Amoebiasis is caused by protozoan Entamoeba histolytica, has a varied spectrum of clinical presentation ranging from asymptomatic to amoebic dysentery and extraintestinal disease, including amoebic liver abscess and pulmonary, cardiac, brain involvement.[1] It is transmitted through the sexual-oral-anal route.[2]

Amoebic liver abscess is the most common extraintestinal manifestation caused due to movement of amebae through the portal venous system.[3] It is clinically presented within 8–20 weeks of exposure,[4] with fever (38.5–39.5°C) and right upper quadrant pain.[5] Hepatomegaly and liver tenderness are commonly observed on physical examination, with less than 10% of cases having jaundice.[5]

Leucocytosis (> 10,000/mm^3) without eosinophilia and mild transaminitis are often seen in blood investigation, commonly with elevated alkaline phosphatase in more than 80% of cases.[6]

The amoebic liver abscess should be suspected in the setting of fever and right upper quadrant pain in endemic areas. Imaging studies include ultrasound, computed tomography (CT), or magnetic resonance imaging (MRI). Abscess appears as a round, well-defined hypoechoic mass on USG,[7] whereas on CT, it appears as a low-density mass with a peripheral enhancing rim. More than 50% of cases have no abnormality in the chest radiograph.[8]

Amoebic serology is negative in the first seven days of infection; however, 35% of uninfected individuals in endemic areas have anti-amoebic antibodies due to the previous infection.[8] So positive serology cannot distinguish between acute and previous

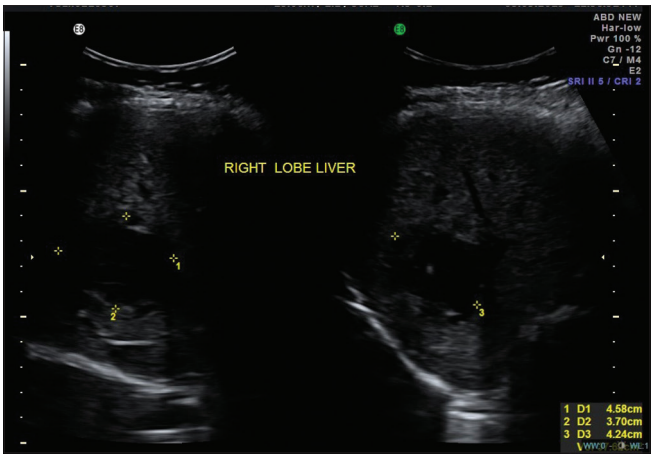

Figure 1: USG of the whole abdomen revealed a well-defined heterogeneous lesion measuring approx. 45 × 37 × 42 mm, volume 37.6 cc seen in the right lobe of liver suggestive of abscess

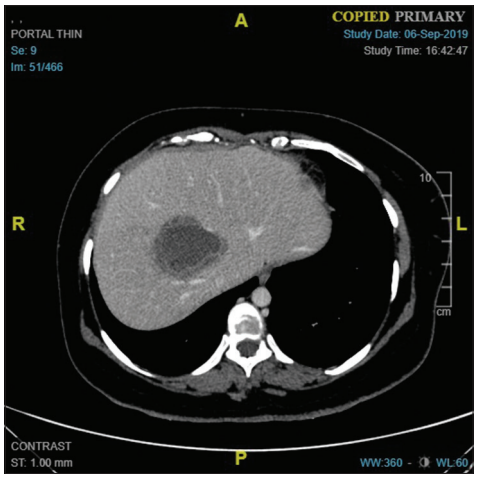

Figure 2: Computed tomography (CT) multiphase study shows a solitary well-defined roundish hypodense lesion with thin peripheral smooth enhancing wall with a surrounding rim of edema measuring 47 × 41 × 39 mm in segment VIII of liver consistent with liver abscess

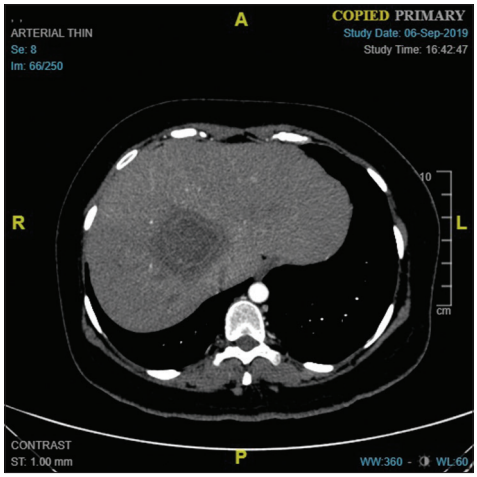

Figure 3: computed tomography (CT) Arterial phase shows solitary well defined roundish hypodense lesion with thin peripheral smooth enhancing wall with surrounding rim of edema measuring 47 x 41 x 39 mm in segment VIII of liver consistent with liver abscess along with mild thickening of cecum

Table 1: Laboratory investigation

Investigation	Reference range	at Admission	Day 5	Day 7	Day 8	Day 10	Day 12	Day 14	Day 30
Hb	g/dl	12.4	12	13	13.1	12.4	11.6	12.4	14.1
TLC	X 10^9/L	14.38	6.2	5.8	5.85	4.64	6.54	5.43	6.88
Platelet Count	X 10^9/L	406	384	447	380	434	532	437	452
Total bilirubin	mg/dl	0.3	0.2	0.5	0.4	1.7	2.6	3.2	0.8
Indirect bilirubin	mg/dl	0.2	0.1	0.2	0.3	0.1	0.2	0.4	0.4
Direct bilirubin	mg/dl	0.1	0.1	0.3	0.1	1.6	2.4	2.8	0.4
SGOT	U/L	38.8	22.6	36.3	1028	5043	3245	2468	45
SGPT	U/L	68.6	17.6	19.4	469	1877	1564	989	38
ALP	U/L	311	131	124	190	202	164	134	30
GGT	U/L	197	93	87	186	186	142	98	24
Urea	mg/dl	23.4	26.2	32	14.2	23	21	34	22
Creatinine	mg/dl	0.8	0.6	0.6	0.8	0.6	0.4	0.7	0.4
PT	Sec	13.8	16	15.6	16.1	23	19	15	12.4
INR		1.21	1.4	1.37	1.41	2.6	2.1	1.7	1.1
Blood culture		Sterile							
Urine culture		Sterile							
Amoebic serology	NTU	25.73							
Amoebic pus stain		Negative							

infection, but negative serology excludes the disease.[9] Treatment includes metronidazole and tinidazole, with a cure rate of more than 90%.[10] Alternative agents include ornidazole and nitazoxanide.[10]

Hepatitis A virus (HAV), identified in 1973, is a member of the Picornaviridae family that causes hepatitis A infection.[11] It is transmitted through the feco-oral route,[12] the incubation period ranging from 15–50 days.[13] Usually, a self-limited illness, but it can present with abrupt onset of nausea, vomiting, anorexia, fever, malaise, and abdominal pain.[14] Dark urine followed by pale stool, jaundice, and pruritus are commonly observed. Jaundice, icterus, hepatomegaly, and tenderness in the right upper quadrant are common physical findings.[15] Laboratory abnormalities are markedly elevated serum aminotransferases (often >1000 IU/dL), serum bilirubin (usually <10 mg/dl), and alkaline phosphatase (up to 400 U/L). Fulminant hepatic failure occurs in less than 1% case, is defined by severe acute liver injury with encephalopathy and impaired synthetic function (INR >1.5),[16] as observed in our patient. Serum aminotransferases peak in one month after exposure, followed by a decline of 75% per week.[17] With full recovery, clinical and biochemical are observed within 2–3 months in 85% of cases, as observed in our case.[17] HAV infection should be suspected in patients in clinical settings of prodrome symptoms with jaundice or elevated serum aminotransferase level. Serum IgM anti-HAV antibodies are done to establish the diagnosis.

Both, amoebic liver abscess and HAV infection have a common mode of transmission, i.e., the feco-oral route, so the coexistence of both diseases should be suspected in endemic areas in a clinical setting of fever, right upper quadrant pain, and jaundice. Jaundice is rarely reported in an amoebic liver abscess, which could be attributed to HAV infection, similar to our case. Previously two cases have been reported with simultaneous amoebic liver abscess and hepatitis A infection, making it an extremely rare infection, especially in endemic areas.[18,19] Among enterically transmitted viruses, i.e., Hepatitis A and E (HEV), HEV is more common compared to HAV in endemic areas as adults are immune to HAV in endemic areas.[20]

Conclusion

Concomitant occurrence of HAV and amoebic liver abscess must not be missed in proper clinical settings, especially in endemic areas. Jaundice is rarely reported in amoebic liver abscess, which could be obstructive jaundice due to bile duct compression from the abscess, so physicians should be cautious before attributing jaundice solely to an amoebic abscess, where the possibility of dual infection is present. One should not miss enterically transmissible Hepatic viruses, i.e., HAV and HEV, especially in endemic areas like India.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

References

1. Haque R, Huston CD, Hughes M, Houpt E, Petri WA Jr. Amebiasis. N Engl J Med 2003;348:1565-73.
2. Billet AC, Salmon Rousseau A, Piroth L, Martins C. An underestimated sexually transmitted infection: Amoebiasis. BMJ Case Rep 2019;12:e228942.
3. Aikat BK, Bhusnurmath SR, Pal AK, Chhuttani PN, Datta DV. The pathology and pathogenesis of fatal hepatic amoebiasis--A study based on 79 autopsy cases. Trans R Soc Trop Med Hyg 1979;73:188-92.
4. Lachish T, Wieder-Finesod A, Schwartz E. Amebic liver abscess in Israeli travelers: A retrospective study. Am J Trop Med Hyg 2016;94:1015-9.
5. Maltz G, Knauer CM. Amebic liver abscess: A 15-year experience. Am J Gastroenterol 1991;86:704-10.
6. Misra SP, Misra V, Dwivedi M. Ileocecal masses in patients with amebic liver abscess: Etiology and management. World J Gastroenterol 2006;12:1933-6.
7. Park MS, Kim KW, Ha HK, Lee DH. Intestinal parasitic infection. Abdom Imaging 2008;33:166-71.
8. Joyce MP, Ravdin JI. Antigens of Entamoeba histolytica recognized by immune sera from liver abscess patients. Am J Trop Med Hyg 1988;38:74-80.
9. Aucott JN, Ravdin JI. Amebiasis and "nonpathogenic" intestinal protozoa. Infect Dis Clin North Am 1993;7:467-85.
10. Lasserre R, Jaroonvesama N, Kurathong S, Soh CT. Single-day drug treatment of amebic liver abscess. Am J Trop Med Hyg 1983;32:723-6.
11. Bradley WH. Homologous serum jaundice. Proc R Soc Med 1946;39:649-54.
12. Barrett CE, Pape BJ, Benedict KM, Foster MA, Roberts VA, Rotert K, *et al*. Impact of public health interventions on drinking water–associated outbreaks of hepatitis A — United States, 1971–2017. MMWR Morb Mortal Wkly Rep 2019;68:766-70.
13. Lemon SM. Type A viral hepatitis. New developments in an old disease. N Engl J Med 1985;313:1059-67.
14. Lednar WM, Lemon SM, Kirkpatrick JW, Redfield RR, Fields ML, Kelley PW. Frequency of illness associated with epidemic hepatitis A virus infections in adults. Am J Epidemiol 1985;122:226-33.
15. Cuthbert JA. Hepatitis A: Old and new. Clin Microbiol Rev 2001;14:38-58.
16. Kemmer NM, Miskovsky EP. Hepatitis A. Infect Dis Clin North Am 2000;14:605-15.
17. Koff RS. Clinical manifestations and diagnosis of hepatitis A virus infection. Vaccine 1992;10(Suppl 1):S15-7.
18. Orenstein WA, Wu E, Wilkins J, Robinson K, Francis DP, Timko N, *et al*. Simultaneous amebic liver abscess and hepatitis A. Am J Gastroenterol 1981;75:52-4.
19. Schwartz E, Piper-Jenks N. Simultaneous amoebic liver abscess and hepatitis A infection. J Travel Med 1998;5:95-6.
20. Bradley DW. Enterically-transmitted non-A, non-B hepatitis. Br Med Bull 1990;46:442-61.