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Case Report

Global Journal of Health Sciences and Research



Pyogenic liver abscess due to *Escherichia coli* in a case of chronic alcoholism

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Received : 22 August 2022 Accepted : 20 December 2022 Published : 20 February 2023

DOI 10.25259/GJHSR_4_2022

Quick Response Code:



ABSTRACT

Pyogenic liver abscess (PLA) is an intra-abdominal visceral abscess. Most often the etiological agent is a bacteria or parasite. Even though the incidence is low, it is important to establish the etiological agent for targeted therapy and in recent times, there is an increase in mortality rate. We report herewith a case of PLA due to *Escherichia coli* in a 60-year-old chronic alcoholic patient without any other evidence of infective foci who responded well to targeted antibiotic therapy and percutaneous drainage. This case report highlights the importance of microbiological work up in every case of PLA. Early diagnosis, percutaneous drainage, and targeted antibiotic therapy remain the mainstay of treatment.

Keywords: Pyogenic liver abscess, Escherichia coli, Alcoholism

INTRODUCTION

Pyogenic liver abscess (PLA) is an intra-abdominal visceral abscess commonly occurring secondary to an infection elsewhere in the abdomen, the most common being a biliary tract infection.^[1] The most common etiologic agents among bacteria are *Klebsiella pneumoniae*, *Escherichia coli*, and among parasites, *Entamoeba histolytica* and *Echinococcus granulosus*.^[2] In recent times, there is a steady increase in the associated mortality rate (26%) which can be ascribed to the underlying comorbid condition of the patient, virulence of infecting pathogen, etc.^[3] Due to the emergence of multidrug-resistant bacterial pathogens, it is essential to identify the etiological agent and the drug susceptibility pattern which guides the clinician to initiate appropriate antibiotic therapy.

CASE REPORT

The patient is a 60-year-old man who presented with complaints of intermittent fever and upper abdominal pain of 1-month duration. The patient reported bilateral swelling of the feet of 4 days duration and passing blood-stained stools for 1 month. He had a history of dengue and typhoid fever in the recent past. He is not a known case of diabetes mellitus or hypertension. The patient is an alcoholic and smoker for the past 40 years, consuming 90 mL/day on alternate days and has a pack year of 1. The initial clinical examination revealed a body temperature of 102°F and a pulse rate of 106/min. Upon general examination, he appeared pale, and bilateral pitting pedal edema was present. Per abdominal examination revealed abdominal pain with

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tenderness on palpation in the epigastric region and right hypochondriac region. There was no intercostal tenderness or guarding rigidity. Rectal examination revealed a pile mass at 7 o' clock position with no active bleeding. Upper gastrointestinal endoscopy revealed esophageal candidiasis for which oral fluconazole therapy was started. Ultrasound of abdomen revealed multiple large hypoechoic regions in the right lobe of the liver, largest measuring around 12 cm length 8.2 cm breadth and 8.6 cm depth with peripheral vascularity suggestive of an organizing abscess and no other abnormality. A percutaneous pigtail catheter was introduced under ultrasound guidance for drainage of pus. He was provisionally diagnosed as a case of the right lobe liver abscess with the upper gastrointestinal bleed for evaluation and Grade II hemorrhoids. The patient was put on intravenous piperacillin-tazobactam 4.5 g and metronidazole 750 mg 8th hourly as empirical treatment. Peripheral blood smear examination showed the following results: White blood cell count, 21300 cells/mm³ (neutrophils 87%), and hemoglobin level, 5.9 g/dL. His serum alkaline phosphatase (222 IU/L) and total bilirubin (3.1 mg/dL) levels were elevated and serum albumin level was reduced (2.5 g/dL). Other liver and renal parameters were within normal limits. Serology for HBsAg, antibodies to HCV, and HIV were negative. The aspirated pus was sent for bacterial culture and sensitivity. Direct microscopic examination of the pus sample showed plenty of pus cells and Gram-negative bacilli. The sample was inoculated onto 5% of sheep blood agar, MacConkey agar, brain heart infusion broth, and thioglycollate broth. Blood agar showed non-hemolytic grey moist colonies and MacConkey agar showed lactose fermenting colonies after 24 h of aerobic incubation at 37°C. No anaerobic culture on plates was done. Gram staining of the colony revealed Gram-negative bacilli with parallel sides and rounded ends, motile, catalase test positive, and oxidase test negative. Further biochemical testing revealed the following reaction, Indole was produced, citrate was not utilized, urea was not hydrolyzed, triple sugar iron agar showed acidic slant/acidic butt with gas, methyl red test was positive. Phenotypically, the organism was identified as E. coli. Subcultures of broth on 5% sheep blood agar and MacConkey agar yielded the same organism. Antimicrobial susceptibility testing was performed by Kirby-Bauer disc diffusion method. The organism was sensitive to tobramycin (10 µg), gentamicin $(10 \,\mu g)$, ciprofloxacin $(5 \,\mu g)$, meropenem $(10 \,\mu g)$, imipenem (10 µg), piperacillin tazobactam (100/10 µg), and resistant to, ceftriaxone (30 µg), cefotaxime (30 µg), and ceftazidime (30 µg). The patient was switched over to intravenous gentamicin 80 mg 12 hourly after the susceptibility report. Cytopathological examination of the pus aspirate suggested features consistent with acute inflammatory pathology. On frequent drainage of pus and targeted antibiotic therapy, the patient improved clinically and was discharged in a healthy

state. The patient was asymptomatic at follow-up after 2 weeks.

DISCUSSION

A liver abscess is a cavitary accumulation of pus within the parenchyma of the liver. The etiology could be diverse which include aerobic and anaerobic bacteria, protozoan, and helminthic parasites, trauma, foreign body, and idiopathic. The most common bacterial agents include E. coli, K. pneumoniae, Streptococcus, and Enterococcus,^[2] with up to 33% of abscesses being polymicrobial.^[3] The bacteria usually originate from the intestinal tract. In our case, the etiological agent is E. coli which is similar to other established studies.^[4,5] An increased incidence of PLA is found in patients with comorbid conditions such as diabetes, malnutrition, and immunosuppression.^[6] About 50% of PLA is due to biliary tract infection and found in the right hepatic lobe due to preferential portal venous flow.^[7] The most common laboratory abnormalities are hypoalbuminemia, leukocytosis, and elevated liver enzymes.^[8] Blood cultures are positive in approximately 50% of the cases only. Age of the patient in our case is similar to other studies conducted by Mohsen et al. where he observed PLA frequently in patients aged ≥ 60 years and a study by Kaplan et al. observed PLA frequently in association with advancing age of >65 years.^[3,8] Blood culture was negative in our case probably because of the initial empirical antibiotic therapy for about 1 week. The diagnosis of liver abscess rests on imaging studies and the etiology on microbiological studies. Identifying the causative agent of PLA helps in predicting the complication and initiating appropriate treatment to prevent the same. Empirical treatment should include broad-spectrum antibacterials like third-generation cephalosporins aminoglycosides or piperacillincombined with tazobactam and metronidazole. Our patient was on empirical treatment with piperacillin-tazobactam and metronidazole. Very few cases of E. coli liver abscess have been reported from India. Study conducted by Malik et al. and Ghosh et al. in Northern India reported 43%; and 39% of E. coli in liver abscess.^[4,5] E. coli is a motile Gram negative bacilli belonging to the family Enterobacterales. Virulence factors of this organism include Fimbrial adhesins, heatstable and heat-labile enterotoxins, Cytotoxic Necrotizing Factors, siderophores, resistance to complement, Type III secretion system, the RTX pore-forming toxins, etc.^[9] An Ireland study conducted in the year 2020 established a high mortality rate of 28% among patients with E. coli PLA and another study by Chen et al. in the year 2018 reported a mortality rate of 26%.^[9,10] This figure is relatively higher than other published studies in the year 1999 and 2002 which is 8% and 12.8%.^[3,11] Further studies are needed to

explore the upcoming novel virulence factors and genes responsible for the worst outcome in these patients. This study highlights the importance of a microbiological workup to find the possible etiological agent and its susceptibility report, due to the emergence of multidrugresistant pathogens. The possible attributing factor for PLA in our case is chronic alcoholism and old age. Chronic alcoholism is one of the risk factors for developing PLA as established in many other studies.^[7,12] Alcohol causes disruption of the gut integrity and flora, which may be the possible reason for the gut microbial etiology of PLA.^[13] Ultrasound-guided percutaneous drainage of the pus along with initiation of targeted antibiotic improved the clinical outcome of the patient described herein.

CONCLUSION

PLA due to *E. coli* is increasingly being reported in recent times. It is also associated with a higher mortality rate as established in a few studies. It is important to establish the etiological agent as there is an emergence of highly virulent and multidrug-resistant bacteria. Early clinical diagnosis, appropriate antimicrobial therapy along with frequent percutaneous drainage of the pus will improve the clinical outcome.

Acknowledgment

We acknowledge the contribution of the Department of General Surgery, Sri Manakula Vinayagar Medical College and Hospital for providing the case details.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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How to cite this article: Sugumaran R, Ranganathan US, Thiyagarajan M, Shivekar SS, Rangasamy G. Pyogenic liver abscess due to *Escherichia coli* in a case of chronic alcoholism. Glob J Health Sci Res 2023;1:45-7.