

Fulminant Emphysematous Hepatitis Mimicking Liver Abscess in a Diabetic Patient

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DOI: 10.31080/ASMS.2023.07.1473

Received: January 13, 2023

Published: February 08, 2023

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Abstract

Emphysematous hepatitis is a rapidly progressive infection of liver which has a high fatality. It is mostly seen in immunocompromised patients with a radiological appearance like emphysematous pyelonephritis but distinguished by histopathology and CT/MRI scans. There is limited data in medical literature regarding the causative organisms, pathogenesis and management of this condition. Despite treatment by broad spectrum antibiotics and surgical care, it may end in fatality as in the present case.

Keywords: Emphysematous Hepatitis; Diabetes; Gas Forming Abscess

Introduction

Diabetic patients have been occasionally diagnosed with intra-abdominal emphysematous infections involving kidney. Few cases have been reported involving emphysematous changes occurring in the liver [1]. Typically seen with gas-forming bacteria, following bowel infarction and abdominal surgeries [2]. Limited case reports have enlisted similar findings on ultrasound scan mimicking liver abscess. Despite aggressive medical and surgical management, emphysematous hepatitis leads to dismal outcomes within 72 hr of admission. We thus report a case of extensive fulminant liver failure with emphysematous hepatitis (EH) and bacteremia, in a patient with type 2 diabetes.

Case Presentation

A 42-year-old diabetic patient was admitted to the casualty in a tertiary care centre in New Delhi, due to complaints of cough, abdominal pain and projectile vomiting. He was having mild cough for the preceding 4 days but presented to us when the symptoms worsened. No significant history of alcohol intake was given. The

patient was a known diabetic on oral antidiabetic drugs with a poor glycemic control. The patient's medical history was not remarkable for chronic obstructive pulmonary disease (COPD), hypertension, or any cardiac ailment or surgery. On presentation, the patient was febrile, well oriented but ill and dehydrated on appearance. Initial examination showed scleral icterus, ascites, and severe right upper quadrant tenderness, and tender hepatomegaly. Biochemistry showed hyperglycemia without ketosis but mild metabolic acidosis with raised lactate levels.

Hemogram showed leucocytosis and severe thrombocytopenia (16000/ul). Blood urea nitrogen was 58 mg/dl and creatinine 1.3 mg/dl which increased to 67.8 mg/dl and 2.17 mg/dl respectively next day. Liver function tests showed aspartate aminotransferase (AST) of 567 U/L, alanine aminotransferase (ALT) of 270 U/L, alkaline phosphatase 1230 U/L, total bilirubin of 10.6 mg/dl, and direct bilirubin of 6.6 mg/dl. Coagulation studies showed a prolonged prothrombin time (PT) of 28 seconds, with International Normalized Ratio (INR) of 2.1. Serum procalcitonin

value was 10.54 ng/ml and CRP values were 27 mg/dl on admission. Reports indicate that patient was in frank sepsis and was thus initiated on broad spectrum antibiotics including carbapenems and parenteral insulin infusion.

Chest Roentgenogram demonstrated bilateral pleural effusion with air shadow under diaphragm (Figure 1). Ultrasound scans demonstrates poorly demarcated space occupying lesion with mixed hypo and hyperechoic echotexture suggesting likely liver abscess. Computed tomography eventually revealed hepatomegaly and parenchymal destruction with air foci within segment 6, 7 and 8 (Figures 2(a) and 2(b)).

Figure 1: Chest roentgenogram reporting gas under right dome of diaphragm.

Figure 2a: CT scan sections showing hepatomegaly and air foci in liver parenchyma.

Figure 2b: CT scan axial sections showing emphysematous hepatitis.

Blood cultures revealed growth of *Klebsiella* spp. and antibiotics modified accordingly. Surgical consultation was sought for possible aspiration and drainage, which was attempted later on and pigtail drainage was kept *in situ*. The cultures from the abdominal drain also revealed similar organism as obtained from blood. Despite aggressive management, the patient's condition deteriorated rapidly and he expired within 72 hours of admission.

Discussion

Emphysematous hepatitis, as the name suggests, is an acute gas-forming, necrotizing infection of the liver which mimics emphysematous pyelonephritis in both radiological and clinical features. It is a rapidly fatal condition in diabetic subjects if not diagnosed and treated timely [3]. The usual pathogens associated with emphysematous infections include gram negative bacilli like *E. coli*, *Enterobacter*, *Pseudomonas*, *Proteus*, and *Klebsiella* [4]. In patients with a *K. pneumoniae* abscess as in our patient, the lesion may appear more solid mimicking a hepatic tumor mass [5].

EH is a very rare entity and only a few cases (less than 10) have been reported in the literature [6]. It Manifests initially as hyperglycemia and sepsis and progress rapidly to multiorgan failure in the absence of therapeutic interventions [7]. Blachar and colleagues first reported a case where a 43-year-old diabetic patient had similar gaseous destruction of the entire liver parenchyma without any evidence of abscess or parenchymal lesion [8]. The pathophysiology of the emphysematous findings on imaging is due to infection caused by acid fermenting bacteria from tissue necrosis that ultimately forms nitrogen (60%), hydrogen (15%), carbon dioxide (5%), and oxygen (5%) and the resultant collection of these gases in necrosed tissue [9].

Other cases with similar radiologic presentations are pneumoperitoneum in a case of cholangiocarcinoma [10] and liver abscess with extensive necrosis [11]. Hepatic infarction is usually caused by intrahepatic gas formation in a transplanted liver secondary to hepatic artery thrombosis and gangrene [12].

EH still remains an extremely rare entity with limited data regarding its pathogenesis, causative factors and management approach. It has significant and poor outcomes in diabetic and immunocompromised patients, since most patients do not improve with routine medical management with or without surgical drainage and progress fatally as evident in this case. Initial presentations are usually with diabetic ketoacidosis masking the underlying fulminate infection, thus high clinical and radiological suspicion is required for salvaging the patient.

Conflicts of Interest

The authors declare that there are no conflicts of interest regarding the publication of this article.

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