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Case Report

Painless Liver Abscess Presenting With Upper GI Bleed

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ABSTRACT

The liver is the organ most subject to the development of abscesses. Only 50% of patients with liver abscesses, however, have hepatomegaly, right upper-quadrant tenderness, or jaundice. Thus one half of patients have no signs or symptoms to direct attention to the liver. We are presenting a unique case of liver abscess which presented with painless jaundice and upper gastrointestinal (G.I) bleed. No case of liver abscess has been reported presenting with upper G.I Bleed in English medical literature yet to the best of our knowledge.

KEYWORDS: Painless liver abscess, Right upper quadrant tenderness, Upper GI bleed.

INTRODUCTION

The annual incidence of liver abscess has been estimated at 2.3 cases per 100,000 populations and is higher among men than women (3.3 versus 1.3 per 100,000) [1].Risk factors include diabetes mellitus, underlying hepatobiliary or pancreatic disease, and liver transplant [2]. Geographic and host factors may also play a role*;* K. pneumoniae is the primary cause of pyogenic liver abscesses in several parts of Asia, and several studies have suggested an association with underlying colorectal cancer [3] . We are presenting a case of liver abscess in chronic alcoholic patient due to Klebsiella pneumonia, presented with painless upper G.I Bleed.

CASE REPORT

A 31 years old alcoholic male, truck driver by occupation presented in the emergency department with h/0 yellow discolouration of eyes for 8 days and one episode of hematemesis. There was no h/o fever with chills and rigors, sweating or loss of appetite. There was no past h/o malena, jaundice, blood transfusion and anemia. Patient had chronic h/o alcohol consumption approximately 100mg/day since 8 years and cigarette smoking 2 packs per/day. He had no h/o hypertension, diabetes and tuberculosis. On general physical examination he had severe icterus even involving skin. There was no pallor, clubbing, cyanosis, oedema of feet and lymphadenopathy. JVP was not raised .Echymosis was present on abdomen and back with minimal ascitis. There were no signs of alcoholic stigma. Liver dullness was obliterated due to pleural effusion, however it was palpable with regular margins up to umbilicus level, there was no tenderness. Spleen was not palpable. Vitals were normal, except for tachycardia. Complete blood examination showed haemoglobin 11gm%,total leucocyte count 11,200 with neutrophils being 80%,lymphocytes 20% and platelets count 1.4 lac/^{mm3} .Peripheral blood film was normocytic and normochromic with toxic granulation.

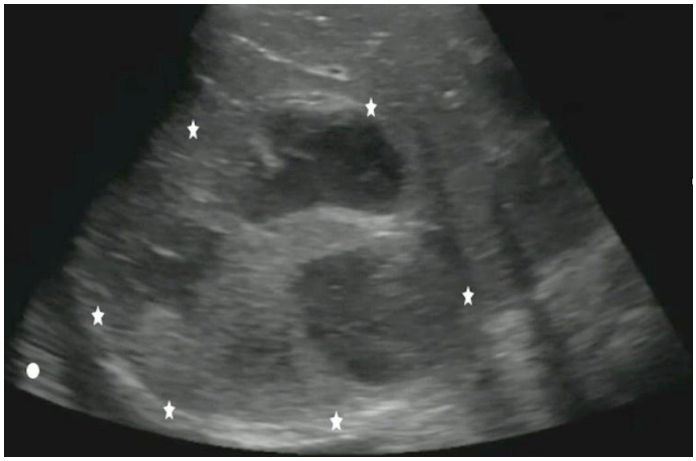
Hepatitis-B, C and HIV was non reactive. Bilirubin was 15gm%, SGOT 268 IU/L, SGPT 182 IU/L, Total serum protein 3.6gm%, Differential serum protein 2.8gm%, albumin globulin ratio was not reversed. Alkaline phosphatase was highly raised 1130 IU/L. PTI was 65%. Renal function tests were normal.

From history and biochemical picture, a provisional diagnosis of alcoholic hepatitis with portal hypertension was made. Conservative treatment was started and endoscopy was planned. Meanwhile ultrasound of abdomen showed enlarged liver approximately 23 cm in size with normal shape and echogenecity. A well defined, predominantly hypo echoic area and irregularly thickened wall was seen in

the left lobe of liver measuring 6.3x3.6 cm in size. Similar lesions were seen in the right lobe, measuring 6.6x6.7 cm and 8.9x 10.1 cm in close proximity to each other(figure 1). Gall bladder was partially distended, visible lumen was clear; however its wall was oedematous and thickened due to ascitis.

Right sided pleural effusion was documented. Endoscopy revealed no variceal rupture. Ultra sound guided aspiration was done which showed pus and sample was sent for culture and sensitivity, which showed Klebsiella pneumonia sensitive for fluoroquinolones and cephalosporins. I.V Metronidazole, third generation cephalosporins and ciprofloxacin with IV fluids along with FFP transfusion was started. With the help of radiological investigation, a final diagnosis of painless pyogenic liver abscess with upper G.I. bleed due to low PTI with right side pleural effusion was made. He responded well to treatment, PTI was raised to 95%, alkaline phosphatase decreased to 582 IU/L after one week of treatment. Patient was discharged in stable conditions after 2 weeks of antibiotic treatment and is now under follow-up.

Figure 1: USG abdomen showing hypoechoic lesions in right lobe of liver, largest measuring approximately 8.9cm



DISCUSSION

No doubt liver is the organ most subject to the development of abscesses. In one study of 540 intra-abdominal abscesses, 26% were visceral. Liver abscesses made up 13% of the total number, or 48% of all visceral abscesses [4]. However, human biology has always remained a science of exceptions; our patient had unusual presentation, having painless jaundice with upper G.I. bleed. Hepatic or liver abscesses are infectious space-occupying lesions in the liver; the two most common abscesses being pyogenic and amoebic. Pyogenic liver abscess (PLA) is a rare but potentially lethal condition, with a reported incidence of 20 per 1,00,000 hospital admissions in a western population [5]. Its severity depends on the source of the infection and the underlying condition of the patient. Amoebic liver abscesses (ALA) are common in tropical regions mainly where 'Entamoeba histolytica' is endemic and is more prevalent in individuals (mostly young males) with suppressed cell mediated immunity.

Fever is most common presenting sign of liver abscess. Some patients, particularly those with associated disease of biliary tract, have symptoms and signs localized to the right upper quadrant, including pain, guarding, punch tenderness, and even rebound tenderness. Only 50% of patients with liver abscesses, however, have hepatomegaly, right upper-quadrant tenderness, or jaundice; thus, one half of patients have no signs or symptoms to direct attention to the liver [6]. Our patient had no fever and tenderness. Fever of unknown origin may be the only manifestation of liver abscess.

The single most reliable laboratory finding is an elevated serum concentration of alkaline phosphatase, which is documented in 70% of patients with liver abscesses [6].Our patient, was having ALP levels 1130 IU/L while other tests of liver function may yield normal results, but 50% of patients have elevated serum bilirubin, and 48% have elevated concentrations of aspartate aminotransferase. Other laboratory findings include leukocytosis in 77% of patients, anemia(usually normochromic, normocytic) in 50% and hypoalbuminemia in 33%.

Ultrasound (USG) is the imaging modality used in the initial evaluation. The appearance on USG varies according to the stage of evolution of the abscess [7].Initially the abscess is hyper echoic and indistinct, but with maturation and pus formation, it becomes hypo echoic with a distinct margin. When the pus is very thick, a fluid-containing lesion may be confused with a solid lesion on USG. USG has a sensitivity of 75% to 95%, but has difficulty in detecting an abscess high in the dome of the right lobe liver and especially multiple small painless liver abscesses (PLAs). By showing gallstones, dilated bile ducts, and hepatolithiasis, USG has the advantage of imaging underlying biliary tract pathology. A computed tomography (CT) scan is more accurate than USG in the differentiation of PLA from other liver lesions and is reported to have a sensitivity of approximately 95%. The portal venous phase using intravenous contrast material gives the best differentiation between the liver and the abscess, with the periphery of the PLA having contrast enhancement as opposed to non-enhancement of the central portion. Magnetic resonance imaging (MRI) does not seem to have any advantage over CT or USG.

Percutaneous drainage combined with antibiotics has become the first line and mainstay of treatment for most

PLAs [8].However, there is growing interest in medical management alone for pyogenic liver abscesses.

Before obtaining positive cultures from blood or pus, broad-spectrum antibiotics should be started to cover Gram-negative and Gram-positive aerobes and anaerobes. Initial therapy with amoxicillin, an amino glycosides, and metronidazole or a third generation cephalosporin and metronidazole generally covers the causative organisms most commonly found, although this regimen may vary according to geographic differences and antimicrobial treatment policies. Initially, antibiotics should be administered parent rally, and after 2 weeks of systemic therapy, appropriate oral agents may be used for a further 4 weeks [9]. In patients with multiple PLAs that are too small to drain, antibiotics may be the only treatment possible. In addition, efforts must be made to identify any underlying biliary obstruction, which needs to be overcome for the antibiotic therapy to succeed.

CONCLUSION

Liver is the organ most subject to the development of abscesses. Its severity depends on the source of the infection and the underlying condition of the patient. One half of patients have no signs or symptoms to direct attention to the liver. Fever of unknown origin may be the only manifestation of liver abscess. The single most reliable laboratory finding is an elevated serum concentration of alkaline phosphatase. Ultrasound (USG) is the imaging modality used in the initial evaluation. A computed tomography (CT) scan is more accurate than USG in the differentiation of PLA from other liver lesions.

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