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Case Report

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Pyogenic Liver Abscess Mimicking Liver Neoplasm on Computed Tomography Scan in a Patient With Elevated CA 19-9

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Abstract

Pyogenic liver abscess (PLA) presentation on computer tomography (CT) scan depends mainly on the degree of maturation and internal contents, but rarely presents similarly to primary liver cancer (PLC) or hepatic metastasis. However, distinguishing PLA from PLC can be a challenge, and several cases were reported previously where tumor markers helped in this differentiation. We present a rare case of PLA with radiological findings similar to PLC or liver metastasis on CT scan in a patient with elevated tumor marker, carbohydrate antigen (CA) 19-9. This case illustrates that a combination of risk factors and clinical findings including localizing symptoms, fever, leukocytosis and tumor markers has to accompany image findings to reach an appropriate differentiation in a query case of hepatic lesion.

Keywords: Liver abscess; Liver cancer; Computed tomography; Tumor marker

Introduction

Pyogenic liver abscess (PLA) was found to be associated with cancers of different origins. Although inflammation-mediated carcinogenesis can be carried out over a considerable time, certain cancers can masquerade as liver abscesses either through spontaneous tumor necrosis or biliary obstruction superimposed by bacterial infection caused by tumor thrombi [1, 2]. As previously published in the American Association for the Study of Liver Diseases (AASLD) and the Asian Pacific Association for the Study of the Liver (APASL) guidelines, computed tomography (CT) imaging is sufficient to confirm the diagnosis of hepatocellular carcinoma (HCC) without the need for histopathologic confirmation [3, 4]. We present a rare case of PLAs caused by *Streptococcus constellatus* with similar radiological features to liver neoplasm on contrast enhanced CT scan in a patient found to have elevated tumor marker carbohydrate antigen (CA) 19-9.

Case Report

An 88-year-old female presented to the emergency department for evaluation of upper respiratory tract infection non-specific symptoms which started 4 days ago. The patient had a medical history significant for diabetes mellitus, colonic diverticulosis and depression.

The patient was found to have systemic inflammatory response syndrome including fever and respiratory distress. Electrocardiogram showed new onset atrial fibrillation with rapid ventricular response.

On admission, laboratory results revealed high anion gap metabolic acidosis and leukocytosis as shown (Table 1). Urine analysis was normal. Chest and abdomen X-rays were unremarkable.

An ultrasound of the abdomen showed abnormal echotexture related to hepatic parenchymal disease with two hypodense heterogeneous mass lesions identified in the right hepatic lobe measuring 3 × 3.8 cm and 5.3 × 3.6 cm (Fig. 1A). Non-specific gallbladder wall thickening (5 mm) without gallstones, intrahepatic biliary ductal dilatation and normal common bile duct caliber were also noted in the ultrasound. The patient was admitted to the medical intensive care unit (ICU) for management of diabetic ketoacidosis (DKA) and sepsis.

Blood cultures taken on admission revealed *Streptococcus constellatus* growth in a total of four bottles. Because the nature of the organism was suggestive of disseminated abscesses formation, a CT scan of the chest, abdomen and pelvis was performed. CT scans with contrast revealed low density lesions involving both the right and left lobes of the liver with enhancement of septae (Fig. 1B). Differential diagnosis included cholangiocarcinoma, metastatic liver lesions or multi-centric HCC. It is believed that certain cancers can masquerade as liver abscesses either through spontaneous tumor necrosis or biliary obstruction superimposed by bacterial infection caused by tumor thrombi. Based on the radiological interpretation and elevated CA 19-9, impression of primary liver cancer (PLC)

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Table 1. Laboratory Data on Admission (in SI Units)

Parameter	Value	Reference range
White blood count ($\times 10^9$/L)	12.6	4.5 - 11.0
Neutrophils (proportion of 1.0)	0.08	0.56
AST (µkat/L),	7.98	0.17 - 0.51
ALT (µkat/L),	6.5	0.17 - 0.68
Total bilirubin (µmol/L)	10.26	5.0 - 21.0
Alkaline phosphatase (µkat/L)	1.35	0.5 - 2.0
Glucose (mmol/L)	26.25	3.9 - 6.1
Acetone (mmol/L)	0.34	< 0.17
Beta hydroxybutyrate (µmol/L)	1,189.22	< 300
Serum osmolality (mmol/kg)	316	275 - 295
Creatinine (µmol/L)	146.74	53 - 106
BUN (mmol/L)	19.99	2.9 - 8.2
Lactate dehydrogenase (µkat/L)	7.26	1.7 - 3.4
Prothrombin time (second)	14.8	Control 10.0 - 13.3 s
Activated partial thromboplastin time (second)	26.4	Control 23 - 33 s
Lactic acid (mmol/L)	6.8	0.6 - 1.7
Anion gap (mmol/L)	19	8 - 16
Hemoglobin A1C (proportion of 1.0)	0.08	0.02 - 0.03
Hepatitis C Ab	Non-reactive	Non-reactive
Hepatitis Ag index	Non-reactive	Non-reactive
CA 19-9 (kU/L)	163	< 35
Alpha-fetoprotein (µg/L)	< 1.3	< 10
Arterial blood gas		
PH	7.341	7.35 - 7.45
PCO_2 (kPa)	4.83	4.7 - 5.9
CO_2 (mmol/L)	19	21 - 28

with superimposed bacteremia was pursued.

An initial liver biopsy was done and the result was inconclusive; however, the sample showed benign hepatic tissue with no evidence of malignant cells. A repeat liver biopsy aspirated pus-like material following 2 weeks of intravenous antibiotics. Gram staining showed many gram positive cocci with no growth at that time. A diagnosis of liver abscesses caused by *Streptococcus constellatus* resulted from portal pyemia secondary to perforated micro-abscess of diverticulae was concluded.

The patient was started empirically on broad spectrum antibiotics (vancomycin, pipracillin-tozabactam and metronidazole) while in the ICU. She was also started on anticoagulation therapy for atrial fibrillation. After a total of 4 weeks of intravenous antibiotics, a CT scan of the abdomen with contrast showed improvement in the size and number of hepatic lesions (Fig. 1C). Unfortunately, the patient's condition was further complicated with *Clostridium difficile* colitis, and she expired in the hospital during the treatment.

Discussion

Differentiating PLC from PLA can be a real challenge. Criteria including male gender, hepatitis B and/or hepatitis C infection, and cirrhosis were provided to be a meaningful tool in distinguishing the two diagnoses [1]. Non-invasive radiological findings in CT or MRI of wash-in and wash-out were deemed to have a high yield specificity and acceptable sensitivity in diagnosing HCC [4]. However, in our case, the PLAs caused by *Streptococcus constellatus* presented with similar radiological features to liver neoplasm on contrast enhanced CT scan in a patient found to have elevated tumor marker CA 19-9.

Incidences of PLA mimicking PLC, including HCC in 80-90% of all cases, and intrahepatic cholangiocarcinoma (ICC), were reported in the literature. Such mimicry led to, in many cases, incorrect diagnosis and treatment including unnecessary surgery [5-9].

In one report, the absence of liver cirrhosis on CT and

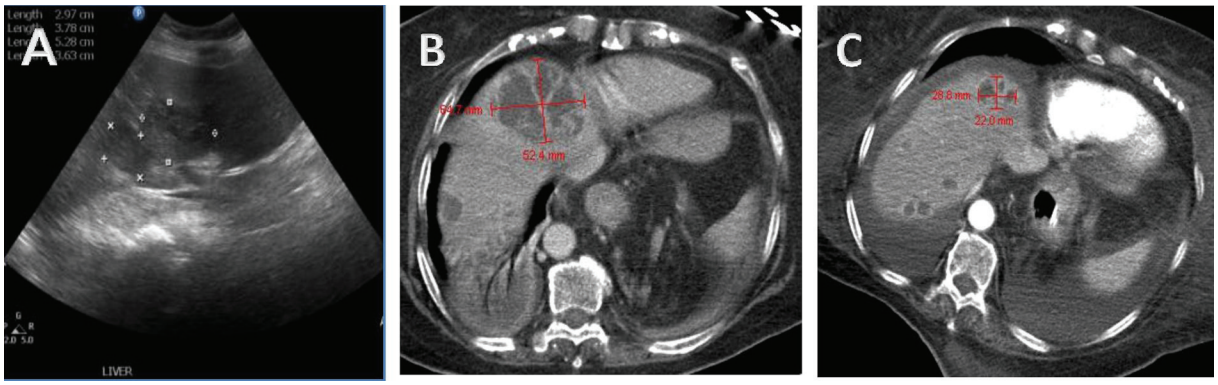

Figure 1. Hepatic abscess caused by *Streptococcus constellatus* mimicking HCC or hepatic metastasis. (A) Abdomen US demonstrating two hypodense heterogenous right hepatic lobe lesions measuring 3 × 3.8 cm and 5.3 × 3.6 cm. (B) Initial contrast enhancing CT scan illustrating multifocal multi-septated enhancing lesions. (C) Contrast enhanced CT showing improvement in hepatic multi-septated abscesses following 4 weeks of antibiotic therapy.

MRI along with normal liver functions was not enough evidence to exclude HCC as a diagnosis. The absence of fever and leukocytosis resulted in hepatic abscess exclusion, and the patient was exposed to unnecessary surgery [6]. In a contrary case, PLA was reported in a cirrhotic patient who presented with sepsis of unclear etiology [9].

Radiological presentation of hepatic abscesses ranges from hypodense rim-enhancing well-circumscribed cystic lesion to a heterogeneously mosaic enhancing mass [7, 10]. Enhancing contrast CT is considered one of the gold standards to differentiate PLA from PLC or hepatic metastasis [7, 11, 12]. While most liver hypervascular liver tumors (as HCC) show greatest background conspicuity on late arterial phase, hypovascular tumors (as liver metastasis) are usually seen well on the portal venous or equilibrium/delayed phase of contrast [13].

HCC usually presents with dynamic radiological changes such as early arterial enhancement and delayed wash-out, well-defined capsule with delayed enhancement or even a mosaic pattern of enhancement [11, 12]. Application of dynamic imaging criteria to diagnose HCC was suggested only in cirrhotic patients regardless of etiology and patients with chronic hepatopathy [3]. Imaging diagnosis of HCC is a worldwide accepted guideline for HCC diagnosis in cirrhotic patients [4]. Pseudo-capsule appearance of HCC on portal venous phase can mimic unilocular abscess [14]. Cystic HCC (unilocular and multifocal), although rare, was described previously in the literature in non-cirrhotic patients [8, 15]. Cystic hepatic lesions in the setting of pyrexia and leukocytosis should always raise suspicion for abscess [5].

Contrast enhanced ultrasounds were dropped from AASLD recommendations to diagnose PLC due to the potential risk of misdiagnosis of ICC as HCC [3]. Additionally, biopsies were also excluded from HCC diagnosis guidelines by the AASLD and APASL for small, atypical lesions in view of high false-negative rate and should be used after careful evaluation in selected cases only without wash-in or wash-out findings on two imaging modalities [4].

ICC can present as periductal, intraductal or intrahepatic mass forming [13]. Mass forming ICC usually appears homogeneous, low-attenuated mass with irregular peripheral enhancement or a retracted capsule on CT [16]. There are currently no guidelines posted for ICC screening in high risk groups, although hepatobiliary MRI and CA 19-9 are frequently used without proven benefit [16, 17].

CT scan was shown to be effective in staging and following liver metastases and is utilized in many cancer centers [18].

Interestingly, HCC can present as PLA initially which predicts a worse prognosis [19]. In a case series study, elevated alpha fetoprotein was used to diagnose HCC when symptoms including fever, chills, right upper quadrant abdominal pain, nausea, vomiting, weight loss and diarrhea were associated with PLC in patients that presented initially as PLA [20]. A combination of elevated serum CA 19-9 with polysomy identified by fluorescence *in situ* hybridization (FISH) raise a high index of suspicion of malignancy in patients with underlying primary sclerosing cholangitis even without mass on imaging [21]. Fever and/or leukocytosis, even though not common, were previously reported to be associated with tumor-associated infections [22].

Conclusion

Hepatic abscess presentation on CT scan depends mainly on the degree of maturation and internal contents, but rarely present similarly to PLC or hepatic metastasis [23]. According to AASLD and APASL guidelines, CT imaging is sufficient to confirm diagnosis of HCC, as a PLC, without need for histopathological confirmation [4]. However, clinical presentation and laboratory findings should always be taken into consideration when interpreting radiological data. CT or ultrasonography-guided biopsy should always be used in rare instances of any doubt between the two entities.

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Author Contributions

All authors listed have contributed sufficiently to the project to be included as authors, and all those who are qualified to be authors are listed in the author byline.

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Conflict of Interest

The authors declare that they do not have a conflict of interest.

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