International Surgery Journal
Altwiher R et al. Int Surg J. 2024 Nov;11(11):1861-1865
http://www.ijsurgery.com
pISSN 2349-3305 | eISSN 2349-2902

DOI: https://dx.doi.org/10.18203/2349-2902.isj20243244

Case Report

Asymptomatic presentation of giant liver abscess: simultaneous *Klebsiella pneumoniae* and amoebic infection in an immunocompetent comorbid patient

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Received: 31 August 2024
Revised: 03 October 2024
Accepted: 12 October 2024

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ABSTRACT

Liver abscess is a rare clinical entity, giant abscess is also rare and asymptomatic presentation in immune competency is even rare. Simultaneous *Klebsiella pneumoniae* and amoebic liver abscess is rarely reported in immunocompetent patients, just few articles. Our case, a 69-year-old man was discovered to have hypoechoic liver mass during a scan of the abdomen for evaluation of dysuria. He was hospitalized through ER for advanced assessment. Systemic inquiries of the patient revealed generalized fatigue, constipation, but no fever, chills, abdominal pain or hypotension. Physical examination revealed normal liver span and no right upper quadrant tenderness. Tumor markers were not strikingly high and an enhanced abdominal computed tomography showed a large area of low attenuation with some liquefaction in the liver. Echo-guided aspiration revealed 220 ml of pus, which grew *Klebsiella pneumoniae*, and the same organism was isolated from the blood. Cytology examination of the pus showed scattered amoeba. The patient gradually improved over 3 weeks on treatment with meropenem and metronidazole, along with sustained drainage of the abscess cavity. No parasite ova or amoeba were found in the stool. He had no evidence of immunocompromise. Parasitic diseases may be a predisposing factor for bacterial infections, including pyogenic liver abscess. Giant size, possible coexistence of amoebae and bacteria in a liver abscess and asymptomatic presentation should not be discounted.

Keywords: Asymptomatic liver abscess, Giant liver abscess, Amoebic liver abscess, Pyogenic hepatic abscess, *Klebsiella pneumoniae*

INTRODUCTION

The recognized presenting symptoms of pyogenic hepatic abscess (PHA) are fever, right upper quadrant (RUQ) pain, jaundice and symptoms of septic shock. Some of described risk factors are diabetes mellitus (DM), intravenous drug use, proton pump inhibitor use, and other miscellaneous.[1] PHAs are rare with global annual incidence rate is about 2.3 cases per 100,000 people and with an occurrence rate of about 3.6 in every 100,000 people in USA.[1] The giant liver abscess is that more than 5 cm in size, and this is even rare. Here a combination of drainage and antibiotic treatment is needed to decrease the risk of serious complications. The background risk factors for PHA include male sex and elderly. Some clinical entities are more prone to PHAs like blunt

trauma, hemorrhoidectomy, inflammatory bowel disease, diverticulitis, malignancy, appendicitis, perforated bowel, dental extraction, acupuncture, and cirrhosis.[1-4] Gram-negative bacilli microbes such as Klebsiella, *E. coli*, Enterococcus, streptococcus and staphylococcus are the most common known causative organisms of PHA.[5] Rarely isolated bacteria from PHAs are some of normal flora which include *Streptococcus constellatus*, gram-positive and catalase-negative cocci.[2,6,7]

In this report, we present a 69-year-old gentleman with multiple comorbidities who was accidentally discovered have hypoechoic liver mass during us scanning of his abdomen for evaluation of burning micturition at the urgent care department with no specific abdominal and general signs of liver abscess or sepsis. He was noted to have hypoechoic liver lesion thus he was admitted for advanced workup. Ultimately, he was found to have a silent giant poly microbial PHA. Pus cultures obtained by percutaneous drainage (PTD) revealed simultaneous klebsiella pneumonia and amoebic infection. In contradistinction to a similar single of giant PHA case that reported in the literature, our case was asymptomatic.[8]

CASE REPORT

A 69-year-old Saudi male, known for hypertension and diabetes mellitus on insulin therapy, presented to the urgent care department with dysuria. The Attending general practitioner ordered a urine analysis and ultrasonic scanning of the abdomen. Radiologist noted hypoechoic mass to which on-call surgical registrar was consulted. The clinical history didn't declare RUQ pain, fever, and jaundice only some general fatigue & chronic constipation. Moreover, he denied a history of abdominal pain of any kind, bleeding per rectum, nausea or vomiting, trauma or weight loss.

Upon examination, he looked somewhat unwell, but he is conscious, alert and oriented, dry mucous membranes, his temperature was 36.8℃ and other vital indices were normal. His abdomen was soft, lax and non-distended.

There was no area of tenderness. There were no hepatosplenomegaly and no palpable masses, and the liver span was 8.5 cm. The initial laboratory surveillance revealed mild leukocytosis of 12.3 with normal neutrophils, high INR (1.65), normal urea, high serum creatinine (175), normal liver function profile, elevated CEA (48.96) and CA 19.9 (88.14).

The basic radiological assessment betrays normal findings of CXR. Ultrasonic scanning of the abdomen revealed Mildly enlarged liver with a complex hypoechoic lesion seen in the right lobe measuring about 8x6cm & no IHBR dilatation. A decision to admit the patient was taken. He was kept in NPO and on intravenous fluids. As a multidisciplinary team, urgent consultation to ID physician and clinical pharmacist was done and empirical intravenous cefuroxime sodium and metronidazole were initiated.

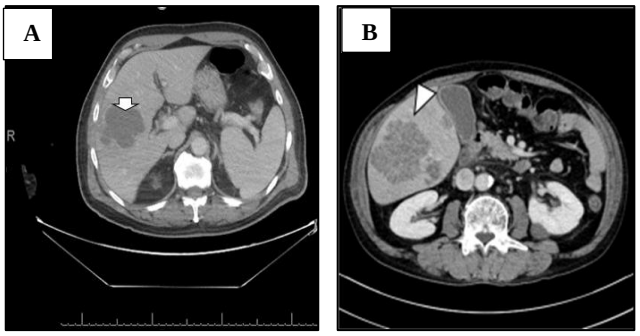

Figure 1: Contrast enhanced CT liver before PCD.

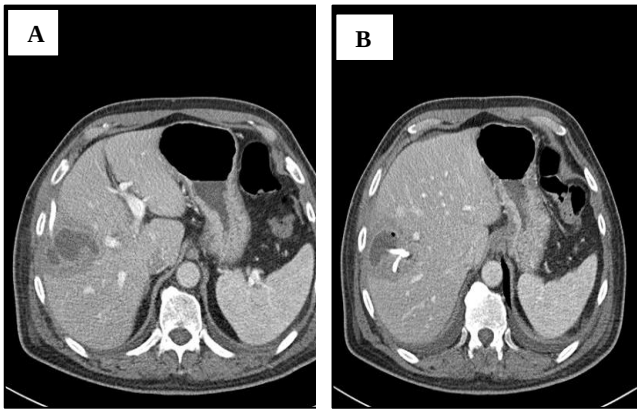

Figure 2: Contrast enhanced CT liver after PCD.

After good hydration over first 24 hours, his serum creatinine reverted to normal 112, then Triphasic CT scan of abdomen was safely applied and revealed ill-defined irregular fluid-density lesions in right lobe measuring about 9.5×6.4×5 cm (CC X TS XAP) with faint marginal enhancement and surrounding perifocal hepatic edema (Figure 1 and compare with Figure 2 post drainage). On the second day post hospitalization, the patient had disturbed liver enzymes and a significant rise in prothrombin time from 19.1 to 23.2. The patient was planned and consented for image guided percutaneous drainage of his giant liver abscess by the interventional radiologist.

After the first 24 hours of hospitalization, the patient started to be ill and exhibited coagulation abnormalities and laboratory changes (Table 1). Therefore, urgent correction of coagulopathy status was performed by transfusion of six units FFP. Samples for cytology, microbiology and biochemical analysis were subjected for examination. klebsiella pneumonia and entamoeba histolytica trophozoites and cysts were isolated from exudate culture. There were no malignant cells detected and negative PCR for echinococcus antibodies. After drainage of large abscess cavities, post admission fever

disappeared, and hepatic enzymes derangement and prothrombin time were optimized. The patient discharges safely, symptom-free, vitally and biochemically stable and less than 5 cm hypo attenuated area. Post discharge and rehabilitation care included prevention by continuation of antibiotics, checking blood count, renal function, bilirubin, aminotransferases and abdomen and re-evaluated at OPD with no remarks and finally discharged from outpatient service after 8 weeks was directed toward the Saudi cancer colon screening program pathway.

Table 1: Summary of key test results.

Test	Normal value	Results			
		Initial	Week 1	Week 2	Last
Haemoglobin	g/dL [13.8-17.2]	14.3	12.8	13.9	14
White cell count	X10^3/MICR[3.5 [10-	13.39	12.93	11.4	7.82
Neutrophils	% [75-40]	72.2	87.2	69.7	46.9
Creatinine	umol/L [115-40]	175	128	112	110
Glucose fasting - FBS	mmol/L [6.1-4.2]	14.71	15.61	13.74	8.11
C-reactive protein	mg/dL [0.3-1]	2.3	2.2	1.4	0.9
Sodium serum	mmol/L -132] [145	130.18	122.57	126.18	131.49
Potassium serum	mmol/L [5.1-3.2]	4.15	4.53	3.5	4.18
Uric acid serum	umol/L -202] [416	278	254	248	214
Calcium serum	mmol/L -2.12] [2.52	2	1.67	1.83	1.91
Chloride serum	mmol/L [98- [107	96.54	92.85	95.88	99.14
Lipase serum	U/L [60-13]	24.40	-	-	-
Amylase serum	U/L [100-0]	40	-	-	-
AST(SGOT)	U/L [37-0]	24.2	20	16.8	17.6
ALT(SGPT)	U/L [19-33]	41.7	36.8	31.1	16.5
GGT SERUM	U/L [61-5]	81	96	109	71.1
Total Bilirubin	umol/L [21-0]	8.3	7.9	5.8	4.9
Direct Bilirubin	umol/L [5-0]	5.7	4.2	2.7	2.7
Albumin	g/L [34 -54]	30.7	24	27.4	32.7
INR	[1.2-0.8]	1.58	1.91	1.72 1.36 [1.2-0.8]	1.58

Initial assessment/ Tumor Marker State			Viral and parasitic serology	
AFP	ng/mL [0-10]	420	HIV 1 and 2 antibodies and p24 antigen	Negative
CEA	ng/ml [5.2-0]	48.96	Hepatitis B	Negative
CA 19.9	U/ml [39-0]	88.14	Hepatitis C	Negative
CA 125	U/ml [0-35]	33.08	Leptospirosis antibodies	Negative
			Widal test	Negative
			Amoebic indirect fluorescent antibody test	Positive at a titre of 1:80
			ACP test	Negative

AST(SGOT) = aspartate aminotransferase
CRP = C-reactive protein
ALT= The alanine aminotransferase
CEA: Carcinoembryonic antigen
ACP = Amoebic cellulose precipitation

Table 2: Pus culture sensitivity results.

Culture sensitivity result		Pus culture	Growth
		Isolated microorganisms	*Klebsiella pneumoniae*
		Direct microscopy	Many *Entamoeba histolytica* cysts
Antimicrobial agent	**Sensitivity**	**Antimicrobial agent**	**Sensitivity**
Amikanamikacin	Sensitive	Ertapenem	Sensitive
Amoxicillin-clavulanate	Sensitive	Gentamycin	Sensitive
Ampicillin	Resistance	Imipenem	Sensitive
Cefazolin	Sensitive	Meropenem	Sensitive
Cefepime	Sensitive	Nitrofurantoin	Sensitive
Ceftazidime	Sensitive	Piperacillin-tazobactan	Sensitive
Ceftolozane-tazobactam	Sensitive	Tigecycline	Sensitive
Ceftriaxone	Sensitive	Trimethoprime /sulfamethoxazol	Sensitive
Cefuroxime sodium	Sensitive		

DISCUSSION

Liver abscesses are rare with an incidence rate of 1.07 to 3.59/100,000 in the West and up to 17.59/100,000 in the East.[10] Their life-threatening complications of bacterial, fungal, protozoal and worm infections are so serious. The patients' age and comorbidities (diabetes, cirrhosis, malnutrition) increase the risk of their occurrence. Immune compromised people during chemotherapy, immunosuppressive treatment, inherited or acquired immunodeficiency syndrome have a higher risk of liver abscesses caused by fungi and opportunistic microorganisms.[11] Depending on the etiology, abscesses can be classified into either bacterial, protozoal, parasitic and fungal types. Our scenario is a combined bacterial and protozoal.

Primary *K. pneumoniae* liver abscess is a prevalent infection in Taiwan. In the 1990s, this infection was first described as an emerging disease in Taiwan, although since then, it is increasingly reported elsewhere, including the United States.[12] In the literature, a case report of simultaneous *Klebsiella pneumoniae* and amoebic liver abscess is reported in an immunocompetent patient but he was symptomatic and septic.[8] Therefore, our case scenario was extremely rare, interesting and message full. A pyogenic hepatic abscess (PHA) most commonly presents with symptoms of fever, right upper quadrant (RUQ) pain, and jaundice. Asymptomatic liver abscess in immunocompetent patients is extremely rare. Common risk factors include, but are not limited to, intravenous drug use, diabetes mellitus (DM), proton pump inhibitor use, advancing age, blunt trauma, malignancy, inflammatory bowel disease, diverticulitis, cirrhosis, appendicitis, perforated bowel, dental extraction, acupuncture, and hemorrhoidectomy.[11] However, our patient's only co-morbidity was advancing age and DM, both of which can affect immune function.[12,13] He had lacked all potential risk factors for developing PHA as well as the typical physical symptoms that are commonly associated with PHAs. Due to this, it was clinically challenging to determine if the hepatic abscess caused the patient's bacteremia or if the bacteremia resulted in asymptomatic hepatic seeding.

Mortality rate of pyogenic liver abscess is as high as 15%. Our patient was recognized, evaluated & properly managed in a reasonable time without any adverse events or complication despite the giant presentation of his abscess, asymptomatic state, presence of multiple co-morbidities and mixed nature of abscess.[14] It in the context of giant liver (i.e >5 cm in size) in the literature, it is reported that multimodal management is needed.[15] However, studies including cases of a 10 cm giant pyogenic liver abscess treated with antimicrobials alone has been reported in the past.[16,17] Our case, was treated as being globally recommended by antibiotic treatment and drainage. Not a single asymptomatic giant liver abscess in immunocompetent patient was encountered in the literature. In this case, we treat aggressively by both broad-spectrum parenteral antibiotics and urgent image guided drainage. We started empirically with meropenem, then every effort to correct electrolyte and metabolic derangement to make interventional radiological drainage possible. Fortunately, obtained pus culture results exhibited a wide range of sensitivity.

However, we continued on meropnem as the most potent due to giant abscess nature, atypical presentation and multi co-morbidities of the patient. As aforementioned, the possible coexistence of amoebae and bacteria in a liver abscess should not be discounted. The published case of simultaneous klebsiella and amoeba in 2008, the patient was symptomatic septic and furthermore, the abscess was not giant.[8] To the best of our knowledge, this is the first case report of a giant liver abscess in an immunocompetent patient caused by simultaneous klebsiella and amoeba that unexpectedly presented asymptomatically.

CONCLUSION

An extremely rare case of an asymptomatic, incidentally discovered, PHA in a comorbid patient with advanced age, HTN and DM. We aim to enlighten clinicians on such a case to ensure a comprehensive differential workup to be done to rule out PHA through admission pathway even with a lack of typical symptoms such as RUQ pain, fever, and jaundice. This case demonstrates that liver abscesses that are polymicrobial in nature may be difficult to diagnose if entirely asymptomatic.

Assuming hypoechoic liver lesion on US abdomen in elderly as metastatic deposit and dispose the patient from emergency service, is unwise because a possibility of asymptomatic liver abscess is there. The wisest decision that provided patient safety is admission and advanced workup. A possible etiology for liver abscesses should be sought, with investigations guided by the microbiological diagnosis. Multiple infections can be present even in immunocompetent patients, especially in presence of comorbidities.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Altwiher R, Alzaid A, Osman A, Alhabeeb Y, Alrdaian D, Almesmar N. Asymptomatic presentation of giant liver abscess: simultaneous *Klebsiella pneumoniae* and amoebic infection in an immunocompetent comorbid patient. Int Surg J 2024;11:1861-5.