International Journal of Advances in Medicine
Putri LAMPD et al. Int J Adv Med. 2023 Dec;10(12):852-856
http://www.ijmedicine.com
pISSN 2349-3925 | eISSN 2349-3933
DOI: https://dx.doi.org/10.18203/2349-3933.ijam20233571

Case Report

Liver abscess in patient with uncontrolled type 2 diabetes mellitus: a case report

Luh Ayu Made Paramita Dwisari Putri, A. A. Istri Sri Kumala Dewi*

Department of Internal Medicine, Wangaya General Hospital, Denpasar, Bali, Indonesia

Received: 13 October 2023
Accepted: 09 November 2023

***Correspondence:**
Dr. A. A. Istri Sri Kumala Dewi,
E-mail: agungistrikumala@yahoo.com

ABSTRACT

Liver abscess is a form of infection in the liver, which is characterized by the presence of pus covered by fibrous tissue in the liver parenchyma. This condition is a rare, but potential life-threatening liver infection, especially if not treated properly. DM is one of the most predisposing factors for liver abscess, especially pyogenic liver abscess. Its prevalence reaches more than 25% of all cases of liver abscess. Pathophysiological mechanisms of DM that can cause liver abscess might include harmful effects of hyperglycemia, general diabetic angiopathy, and decreased immunity. We presented the case of an elderly male patient with multiple liver abscess who had a history of type 2 DM with poorly controlled. Through this case will shows multiple liver abscess patients with comorbid type 2 DM and hyperglycemic stage who received antibiotic treatment with good response of therapy.

Keywords: Liver abscess, Pyogenic liver abscess, Diabetes mellitus

INTRODUCTION

Liver abscess is a form of infection in the liver, which is characterized by the presence of pus covered by fibrous tissue in the liver parenchyma. This condition is a rare, but potential life-threatening liver infection, especially if not treated properly. The four most common forms of liver abscess are pyogenic, amoebic, parasites, and fungi liver abscess.[1,2]

Liver abscess is a public health problem in few countries in Asia, Africa, and South America. Liver abscesses have a global distribution, although incidence varies significantly between different countries from more than 900 cases in a 10-year period in Asian countries such as Taiwan, Singapore and South Korea to 23 cases in the same timeframe in non Asian regions. In America are seen in approximately 2.3 cases per 100,000 with higher rates found among men than women. The incidence in Taiwan seems to be very high (17.6 per 100,000). Mortality at one time approached 77%, but newer population-based studies have estimated rates at closer to 6%. People aged 40-60 years are more vulnerable to developing liver abscess that does not result from trauma. The majority of liver abscess is categorized into pyogenic or amoebic, although a minority is caused by parasites and fungi.[1,2] Most amoebic infections were due to *Entamoeba histolytica*, while most of the pyogenics caused by *Klebsiella pneumonia*, *Streptococcus milleri*, *Escherichia coli*, *Staphylococcus aureus* and some more caused by anaerobic bacterial. A Research by Abbas et al. noted that of 67 patients admitted for liver abscesses in the Middle East, 56 were due to pyogenic causes with most cases due to *Klebsiella pneumonia*.[1,3,4]

The common symptoms among the liver abscess are fever, chills, fatigue or malaise, loss of appetite, weight loss, right upper quadrant (RUQ) abdominal pain, jaundice, hepatomegaly and splenomegaly, in a few cases have rare symptoms like coughing, hiccup, pain in low right chest, or pain on the shoulder.[2,5] Laboratory tests may exhibit high count of leucocyte, C-reactive protein, abnormalities in liver function tests, such as increase in alkaline phosphatase. Ultrasonography and computed tomography

(CT) of abdomen are gold standard to confirm the diagnosis of liver abscess.[1,4]

Risk factors predisposing patients to liver abscess range from DM, liver cirrhosis, general immunocompromised state, use of proton pump inhibitor (PPI) medications, male gender, and age >57 years. DM is one of predisposing factor for liver abscess that is well documented in the literature. Studies have found DM as a concomitant disease in 29.3-44.3% of patients with liver abscess.[6]

We presented the case of an elderly male patient with multiple liver abscess who had a history of type 2 diabetes mellitus with poorly controlled. Through this case will shows multiple liver abscess patients with comorbid type 2 DM and hyperglycemic stage who received antibiotic treatment with good response of therapy.

CASE REPORT

A 64-year elderly male patient from Java ethnic was admitted in hospital with major complain epigastric and RUQ abdominal pain for 2 weeks ago before admission. He felt sharp pain and discomfort in RUQ abdomen. The pain did not spread to the other organs of the body. He also complaining that the pain gradually increasing. The pain getting worse since a day before he came to the emergency department. The pain slightly better after he took a pain killer, but in a few hours later, pain would be coming again. The patient also complaining about nausea, vomiting, fatigue and loss of appetite since a week ago but there was no weight loss. The patient also reported having had intermittent fever. Fever usually came in the afternoon until late at night. Then in the morning, his body temperature will be back to the normal without using medication. Besides having fever, the patient also complained of joint pain and general malaise in those fever cycles. The patient had history of type 2 DM and recently started insulin therapy in last 2 weeks. Others medical history like jaundice, hypertension, obesity, heart diseases, kidney and liver diseases were denied. He did not mention any history of chronic diseases in his family and no history of smoking, drunk alcohol, use of intravenous drugs, blood transfusion, or consume herbal or nutritional supplements. He did not have any food or medications allergies. He had no history of contact with pets or wild animal and not travelled outside the country during in the last one year.

Based on physical examination during the admissions we found the general appearance look moderate ill, the patient was alert and conscious with Glasgow coma scale (GCS): 4 for eyes, 5 for verbal, and 6 for motoric. His vital sign included a blood pressure of 110/57mmHg, a pulse 90 beats per minute, respiratory rate 20 times per minute, his temperature is 38°C and oxygen (O_2) saturation was 98% on room air.

Each conjunctiva was pale and sclera was not icteric. There was not lymph nodes enlargement at facial, coli, supraclavicular, and axillary. Heart and lung examination was not remarkable. There was tenderness in RUQ abdomen, liver enlargement, splenomegaly, no signs of peritoneal irritation were observed, no ascites and fluctuation found. Oedema was not found in upper and lower extremity. From complete blood count examination showed markedly suspected by bacterial infections with increased leukocyte count of 15.950 µ/L, haemoglobin was decreased 9.6 g/dL with normal count of MCV and MCH, haematocrit of 39.0%, and platelets of 222 /µl. Blood glucose was very high 405 mg/dl. Electrolyte test showed decreased of sodium level 124 mmol/L and high of potassium level 5.7 mmol/L. An increase AST value was no significant with a value of 45 U/L, while the ALT value was still normal of 48 U/L. Renal function test were normal. Urinalysis showed slightly proteinuria and glucosuria. Widal test showed negative salmonella antibody. An arterial blood pH of 7.41 that indicating no acidosis (Table 1).

Abdominal ultrasonography images were found indeterminate boundaries hypoechoic lesion with internal echo and air components on right lobe of liver. Diameter of the lesion is 8.01×6.51 cm (Figure 1). We assessed the patient as a liver abscess, with type 2 DM, mild anaemia, hyponatremia, and hyperkalemia. Based on clinical sign and symptoms, risk factors and imaging tests we were suspected the patient with a pyogenic liver abscess.

The treatment for this patient consists of antibiotic intravenous ceftazidime 1 gram every 8 hours. Intravenous ondansetron 4 mg every 8 hours was given to the patient for anti-emetic. To control hyperglycaemia during admissions, rapid regulation was done with rapid acting insulin 4 units/hours in the syringe pump. After the blood sugar was controlled, we use combination of 14 units of bolus insulin every meal and 16 units of basal insulin daily. Anaemia was treated by transfusion of 1 bag packed red blood cell (PRC). After transfusion, count of haemoglobin was increased from 9.6 g/dL to 10.5 g/dL. Hyperkalemia and hyponatremia were treated by NaCl 3% drip 12 drop per minute and calcium gluconas 3 times a day.

After 2 weeks of antibiotic treatment, patient's clinical presentation gets a better appearance, no fever, good intake, and decreased of RUQ abdominal pain, but still stiff in palpation. Complete blood count test showed decreased of leucocyte count from 15.950 µ/L to 9.440 µ/L. Abdominal computed tomography with contrast was performed and revealed liver enlargement with size 15 cm. There were multiple cystic lesions in segment 5, 6, 7, and 8 right lobes of the liver. The lesions with thick walls, indeterminate boundaries with air components in it. The largest size of lesion is 6.4×7.1×8.9 cm. Post contrast images show peripheral rim contrast enhancement (Figure 2). The size of lesions was decreased around 2 cm. Surgeon consultation was obtained, because there was progressive improvement from the clinical finding, laboratory test, and abdominal CT, so that interventional surgery therapy was not carried out. It is also because the patient refused any interventional and surgery therapy. The

patient was discharged from hospital and treatment continued with cefixime oral 200 mg 2 times a day for 2 weeks. After 2 weeks, the patient came to the clinic with good condition. There was no abdominal pain and fever were found. In addition, the physical examination revealed no pain it RUQ abdomen and no pain on palpation.

Table 1: Laboratory test result including complete blood count, blood chemistry, electrolyte, urinalysis, and blood gas analysis.

Lab test	Findings	Lab test	Findings
Complete blood count		**Blood gas analysis**	
Leucocyte count	15.950 µ/L (H)	pH	7.41
Erythrocyte count	3.55 µ/L	pCO_2	32 mmHg
Haemoglobin	9.6 g/dL (L)	pO_2	87 mmHg
Haematocrit	39% (L)	$CHCO_3$	21 mmol/L
MCV	87.0 fL	ABE	-4 mmol/L
MCH	27.0 pg	SBC	22 mmol/L
MCHC	31.1 g/L	SO_2	97%
Neutrophil	85.2% (H)	Urinalysis	
Lymphocyte	7.7% (L)	Colour	clear
Monocyte	6.6%	Glucose	+4
Eosinophil	0.3%	Ketones	Negative
Basophil	0.2%	Protein	+1
Platelet count	222.00 µ/L	Blood	Negative
Blood chemistry		Sediment erythrocyte	Negative
Random blood sugar	405 mg/dL	Sediment leucocyte	1-2
Urea	45 mg/dL	Cristal	Negative
Creatinine	1.1 mg/dL	Bacterial	Negative
ALT	48 U/L		
AST	45 U/L		
Albumin	2.8 g/dL		
Electrolyte			
Sodium	124 mmol/L		
Potassium	5.7 mmol/L		
Chloride	83 mmol/L		

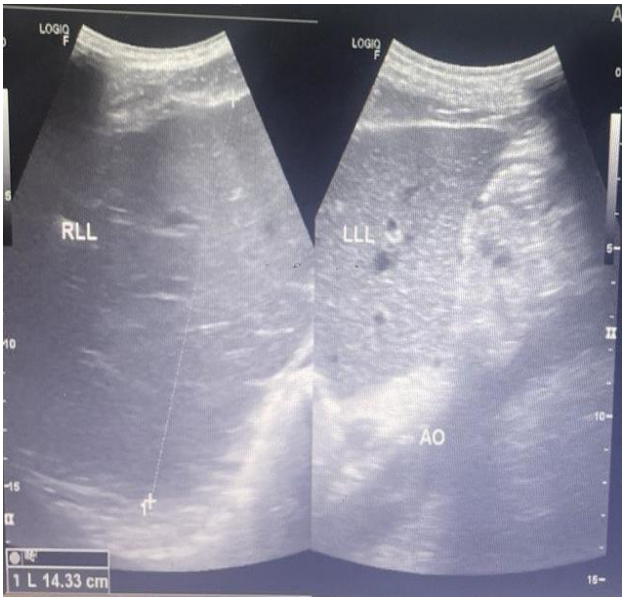

Figure 1: Abdominal ultrasound showed hypoechoic lesion on right lobe of the liver. Diameter of the lesion is 8.01×6.51 cm.

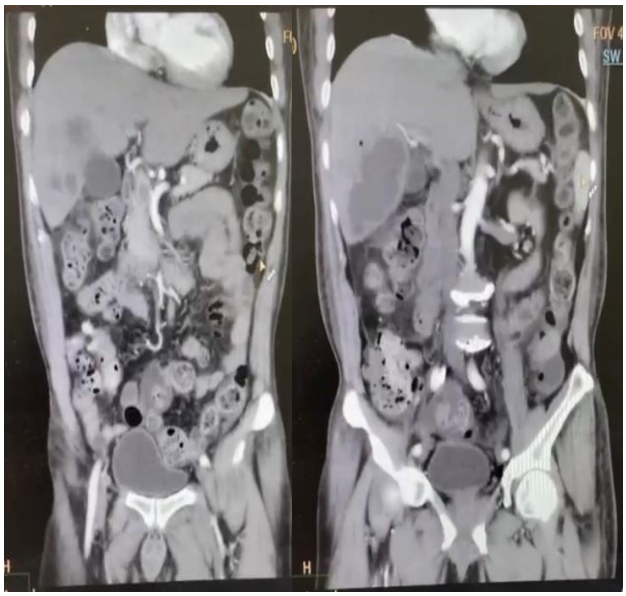

Figure 2: Abdominal computed tomography with contrast revealed liver enlargement with size 15 cm, multiple cystic lesions in segment 5, 6, 7, and 8 right lobes of the liver with the largest size of lesion is 6.4×7.1×8.9 cm.

DISCUSSION

A liver abscess is defined as an encapsulated pus collection of suppurated material within the liver, which maybe caused due to bacterial, parasitic, or fungal infection. The microorganism that causes liver abscess can develop from an intraabdominal infection disseminated from the portal vein, through the bile ducts or can spreading from the infected organs adjacent to the liver, and gastrointestinal tract trauma. According to classification, there are three types of liver abscess: infectious, malignant, and iatrogenic. The majority of these abscesses are categorized as pyogenic or amoebic, although a minority are caused by parasites and fungi. Pyogenic liver abscess can be gram-positive cocci, gram-negative bacilli, or anaerobic organisms. Various fungi can also lead to abscess formation in immunocompromised patients.[1,2]

Risk factors predisposing patients to liver abscess range from DM, liver cirrhosis, general immune-compromised state, use of proton pump inhibitor (PPI) medications, male gender, and old age > 57 years.[6] One of the risk factors of liver abscess that associated with the patient in this case is a history of type II DM that not controlled with drugs. The patient has history of type II DM that just found out and start insulin therapy in 2 weeks before admissions. The patient had very high blood glucose level of 405 g/dl in the first time of admissions. DM is one of predisposing factor for liver abscess that is well documented in the literature. Studies have found DM as a concomitant disease in 29.3-44.3% of patients with liver abscess, especially pyogenic liver abscess. Its prevalence reaches more than 25% of all cases of liver abscess.[6]

Diabetes is associated with increased risk for developing infections from pyogenic liver abscess (PLA) with the most common cause including *Escherichia coli* and *Klebsiella* species. The main mechanism of PLA is hematogenous spread to the liver, either through the portal system or the large circulation, or local spread of infection within the peritoneal cavity. Pathophysiological mechanisms of DM that can cause liver abscess might include harmful effects of hyperglycemia, general diabetic angiopathy, and decreased immunity. The underlying biological mechanisms may include tissue hyperglycemia and a predilection for certain microorganisms.[2,7,8] Previous research studies have shown that DM impairs the innate and adaptive immune system, impairing macrophages, T cells, neutrophils, and NK cells. This immune dysfunction increases the risk of infection including liver abscess. Hyperglycemia also can cause dysregulates the neutrophil phagocytosis and impairs polymorphonuclear leucocyte (PMN) chemotaxis which weakens their immune defense against infections and leaves them more susceptible to abscess formation.[2,7,9]

The clinical presentation of both amoebic and PLA is indistinguishable. From the various case of liver abscess, prolonged and recurrent fever and RUQ abdominal pain are the most usually clinical finding.[2,5] In the studies conducted by Olivier et al in France and Vatan et al in Turkey, RUQ abdominal pain was reported in patients under investigation. In another study, which was conducted in a southern city of Iran, the presented case was reported to have prolonged fever.[10] Their findings in this regard are consistent with the findings of this case. The laboratory tests result in patient such as leukocytosis (predominantly neutrophils), high of inflammatory markers (C-reactive protein), increased of ALT and abnormal liver function tests are often present in liver abscess.[2,8] This is in accordance with what was found in this case, the patient's laboratory test results showed an increase neutrophil count of 85.2%. Abnormal liver function tests were also found in this case, although an increase AST value was no significant with a value of 45 U/L, while the ALT value was still normal of 48 U/L.

Definitive diagnosis requires radiological evidence of an abscess in the liver parenchyma. Abdominal ultrasonography and computed tomography are the gold standard diagnostic modalities. Imaging techniques, such as ultrasonography and CT of abdomen, are useful tools to demonstrate a space occupying lesion and confirm presence or absence of a liver abscess.[5] CT abdomen has a higher sensitivity (97% sensitive) compared to ultrasonography (85% sensitive) for detection of liver abscess.[8] Imaging test of liver abscess is important to determine the location, size, number of abscesses, consistency, and the presence or absence of gas in the abscess.[2] The diagnostic modality that used in this case report was abdominal ultrasonography and CT. According to the literature, ultrasonography and CT abdomen of patient with liver abscess will show cystic lesion. It may show commonly hypoechoic than hyperechoic lesion compared to normal liver parenchyma. Our study found that the abdominal ultrasound found a mass in liver. Diameter of the lesion is 8.01×6.51 cm and Abdominal CT with contrast examination after antibiotic treatment showed multiple cystic lesions in segment 5, 6, 7, and 8 of the liver with the largest size of lesion is 6.4×7.1×8.9 cm.

The symptoms and signs that occur in patients with liver abscess cannot be useful to differentiate between amoebic and pyogenic liver abscess.[5] Most of the patients with liver abscess experience non-specific symptoms such as fever, RUQ abdominal pain, malaise, nausea and vomiting, so it is very difficult to differentiate the etiology just from symptoms and signs. One thing that can differentiate etiology of liver abscess is by looking at characteristics of the lesion. Generally, in amoebic liver abscess (ALA), most commonly occurs as a single lesion in the right lobe but can be present in the left lobe and be multiple. Meanwhile in PLA usually occurs with multiple lesions.[8] CT scan with contract administration in this case showed multiple lesions in the right lobe of the liver, so it is most likely that etiology of the liver abscess in this case is pyogenic. It was also confirmed by the presence of DM, one of risk factors of liver abscess that was existing in this patient, which is in accordance with literature that patients with DM are very vulnerable to experiencing PLA.

Guidelines treatment of liver abscess generally recommend empiric antibiotics therapy. The targeting of antibiotic therapy for liver abscess are both pyogenic and amoebic. Antibiotics should be started immediately to prevent the escalation bacteremia that associated with severe complications, e.g., septic condition.[8] The first line antibiotic therapy includes piperacillin/tazobactam, amoxicillin/clavulanic acid, or 3rd generation cephalosporins (cefotaxime, ceftriaxone, ceftazidime) in monotherapy or in combination with an aminoglycoside (gentamicin). Anaerobic antibiotics, e.g., metronidazole, are used in case with unknown cause or suspicion of amoebic abscess. In the elderly and those with impaired renal function a 3rd generation cephalosporin should be used. Generally, antibiotics treatment should be used for a minimum of 2 to 6 weeks. After the initial intravenous therapy, the oral antibiotics can be used to followed intravenous antibiotic. Therapy for ALA is using metronidazole 500 mg IV every 8 hours for 10 days and followed by luminal agent such as paromomycin for 5-10 days to eradicate any remaining cyst in the intestinal tract.[5,8] Drainage of liver abscess is needed in large size of liver abscess more than 5 cm, or not respond to medication. Other condition e.g., multiple liver abscess, high risk of rupture, secondary infections, or abscess in the left lobe of liver is also need drainage or surgery.[2,8] Antibiotics treatment that given for this patient were ceftazidime (the third generation of cephalosporin), because the patient is classified as elderly, the 3rd generation cephalosporin group was chosen for the patient's therapy in this case. Liver abscess with size more than 5 cm is already indication to perform percutaneous liver aspiration. In this case, consultation with the surgeon already done but percutaneous liver aspiration was not done, because the patient and his family refused any interventional or surgical treatment.

The prognosis of PLA is dependent on the time to diagnosis. Patients with delayed diagnosis are more likely to need medical treatment with drainage procedure. Patients who present with comorbidity such as acute renal failure and acute respiratory failure were likely to have poor outcomes. Complex and ruptured abscesses are associated with increased mortality.[8] The prognosis for patients with PLA who have DM is as poor as the prognosis in another patient who have PLA without DM. Diabetes is a documented risk factor for gram-negative bacteremia, including episodes originating from focal of infection in the abdomen. There is increasing evidence that DM is one of the factors associated with a poor prognosis among patients with gram-negative bacteremia or conditions associated with abdominal sepsis, such as gastrointestinal perforation.[6] Patient in this case had a good prognosis. After 4 weeks antibiotic treatment, there was a good response of antibiotic therapy and patient's condition had been improved.

CONCLUSSION

Liver abscess is a form of infection in the liver, which is characterized by the presence of pus covered by fibrous tissue in the liver parenchyma. This condition is a rare, but potential life-threatening, especially if not treated properly. DM is one of risk factors of liver abscess. We present the case of a 64-year elderly male patient with multiple liver abscess who had a history of type 2 DM with poorly controlled. The patient had given by ceftazidime, the first line therapy for liver abscess and to control hyperglycaemia during admissions, rapid regulation with insulin had given to the patient. After 4 weeks received antibiotic treatment intravenous and orally, the patient showed improvement condition and good response of therapy to antibiotic.

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

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Cite this article as: Putri LAMPD, Dewi AAISK. Liver abscess in patient with uncontrolled type 2 diabetes mellitus: a case report. Int J Adv Med 2023;10:852-6.