Journal of Liver
ISSN: 2167-0889

Shizuma, J Liver 2017, 6:3
DOI: 10.4172/2167-0889.1000216

Case Report OMICS International

A Case of liver abscess with Portal Vein Thrombosis caused by *α-Hemolytic streptococci*

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Received date: May 31, 2017; **Accepted date:** June 8, 2017; **Published date:** June 10, 2017

Abstract

Cases of liver abscess complicated by portal vein thrombosis are relatively rare. We report a case of liver abscess caused by *α-hemolytic streptococci* and complicated by portal vein thrombosis. A 51-year-old male presented with fever and right hypochondralgia. He had watery diarrhoea and bloody stool for over 1 month before presentation. Laboratory tests showed elevated hepatobiliary enzymes, inflammation findings, and anemia. Abdominal imaging revealed liver abscess in the right lobe and thrombosis of right and left branches of the portal vein; no thrombosis was observed in the extrahepatic portal vein. Colonoscopy revealed nonspecific colitis without diverticula or malignancies, and no possible causes of liver abscess except for enterocolitis were detected. Clinical course and laboratory findings indicated that an amoebic liver abscess was unlikely. Blood and pus cultures were positive for *α-hemolytic streptococci*. A diagnosis of liver abscess caused by *α-hemolytic streptococci* and complicated by portal vein thrombosis was made. The size of the liver abscess reduced, and inflammation findings resolved with 3 weeks of antibiotic treatment and abscess drainage. Portal vein thrombosis disappeared after 2 week anticoagulant therapy.

Keywords: Liver abscess; Portal vein thrombosis; *α-hemolytic streptococci*

Abbreviations: ALT- Alanine aminotransferase; AST- Aspartate transaminase; CRP- C-reactive protein; PVT- Portal vein thrombosis; SMV- Superior mesenteric vein; TIPS- Transjugular intrahepatic portosystemic shunt; WBC- White blood cell

Introduction

Liver abscess is characterized by accumulation of pus with destruction of liver parenchyma. The infection may be transmitted through the portal system, hepatic artery, and biliary system (ascending biliary infections) or directly by infection or inflammation of intra-abdominal organs [1]. Some infections are iatrogenic or of unknown origin [1]. Clinical symptoms include fever, right hypochondralgia, general fatigue, and weight loss. Liver abscess has been classified as pyogenic (bacterial), amoebic (caused by *Entamoeba histolytica*), and fungal from the viewpoint of causative organisms. Major causative organism of pyogenic liver abscess is enterobacteria such as *Klebsiella spp.*, or *Escherichia coli*. *Streptococcus spp.*, which is normal flora of oral or gastrointestinal tract, is also one of the causative organisms of liver abscess [1]. Cases of liver abscess complicated by portal vein thrombosis (PVT) are relatively rare. There have been three reports of liver abscess caused by *Streptococcus spp.* and complicated by PVT [2-4]. Here we report a rare case of liver abscess caused by *α-hemolytic streptococci* and complicated by PVT.

Case

A 51-year-old Japanese man presented to our hospital with fever, right hypochondralgia, and general fatigue. He had watery diarrhoea and bloody stool for over 1 month. The patient had no relevant past medical history and was not taking any medications. His alcohol intake had been 45 g/day for 30 years and smoking history was 20 cigarettes/day for 20 years. His blood pressure was 132/62 mmHg, pulse rate was 96 beats/min, temperature was 38.3°C, and respiratory rate was 19 breaths/min. He was alert and conscious with a slight conjunctival pallor and no icterus.

On auscultation, no signs of cardiovascular or respiratory abnormalities were identified. The patient had mild hepatomegaly with right upper quadrant tenderness; no ascites or peripheral edema was present. Laboratory findings were as follows: white blood cell (WBC) count 14470/μL (neutrophils, 87.5%); red blood cells 331 × 104/μL; hemoglobin, 8.9 g/dL; hematocrit 31.1%; platelets 17.9 × 104/μL; prothrombin time 78%; total proteins 6.1 g/dL; total bilirubin 0.7 mg/dL; aspartate transaminase (AST), 53 U/L; alanine aminotransferase (ALT) 63 U/L; Alp 2007 U/L; γ-GTP 447U/L; LAP 233 U/L; LDH 304 U/L; total cholesterol 133 mg/dL; triglycerides 87 mg/dL; fasting blood glucose 78 mg/dL; and C-reactive protein (CRP) 17.5 mg/dL. Urinalysis results were normal.

Enhanced abdominal computed tomography revealed a low-density, 15-cm diameter lesion in the right lobe of the liver, consistent with a diagnosis of monostotic abscess (Figure 1a) and thrombosis of right (Figure 1b) and left (Figure 1c) branches of the portal vein without thrombosis in the extrahepatic portal vein including the superior mesenteric vein (SMV).

Colonoscopy revealed scattered redness throughout the colon and rectum and absence of diverticula or malignancies. Pathological findings showed nonspecific colitis. Stool culture was negative. We considered that amoebic liver abscess followed by amoebic colitis could not be ruled out and initiated metronidazole (1500 mg/day).

Figure 1a

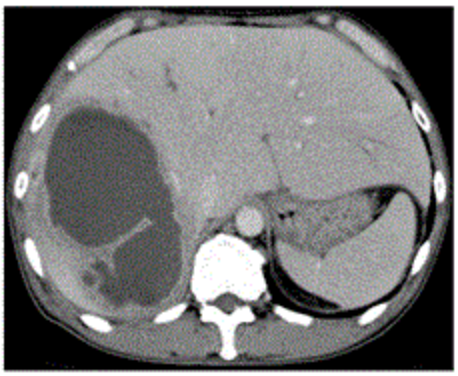

Figure 1b

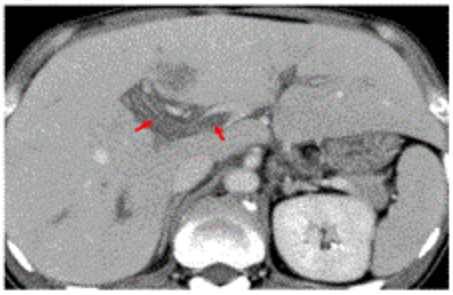

Figure 1c

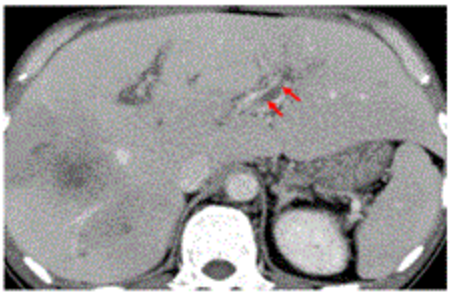

Figure 1a-c: a) Monostotic abscess b) Thrombosis of right c) Portal vein without thrombosis in the extrahepatic portal vein.

The patient was admitted 4 days after the consultation because all beds in our hospital were occupied, and he was diagnosed with liver abscess with PVT. Abdominal ultrasound revealed a mixed echoic mass of 13 cm diameter in the right lobe of the liver and restiform substances in the right and left branches of the portal vein. Two days after admission, the patient developed high fever and worsening of serum inflammatory markers (i.e., WBC and CRP).

Serum immunofluorescence assay for Endamoeba histolytica antibody, which was examined on the consultation day, was negative, indicating that an amoebic liver abscess was unlikely. No possible causes of liver abscess such as biliary infections and intra-abdominal infections except for enterocolitis were detected. Pus obtained following percutaneous transhepatic drainage of the abscess did not have an anchovy paste appearance.

Metronidazole was discontinued and antibacterial therapy (ceftriaxone 2 g/day) was initiated. No findings of portal hypertension such as ascites or esophageal varices were found; anticoagulant therapy (heparin 10,000 unit/day) for PVT was administrated. Cultures of both blood and pus from abscess drainage were positive for *α-hemolytic streptococci*, and antibiotic therapy was switched to piperacillin (2 g/day) following sensitivity testing.

Electrocardiography findings were negative for endocarditis. The size of the liver abscess decreased and inflammation findings resolved with 3 weeks of antibiotic treatment and abscess drainage. PVT had nearly disappeared after 2 weeks of anticoagulant therapy, and heparin was discontinued and replaced with oral warfarin. Anaemia and hypoproteinemia resolved during the hospital stay, and the patient was discharged 5 weeks after admission. The liver abscess disappeared within 3 months of discharge, and no abnormalities were seen on colonoscopy at 6 months after discharge.

Discussion

In this patient, clinical symptoms of enterocolitis such as watery diarrhoea and bloody stool prior to his first consultation and the endoscopic findings were consistent with liver abscess and development of PVT in the portal system followed by enterocolitis. Pus and blood cultures indicated *α-hemolytic streptococci* as the causative organism.

PVT was found in right and left branches of the portal vein and not in the extrahepatic portal veins including SMV. Extension of thrombosis from extrahepatic portal vein may not have been responsible for the development of PVT in our patient [5]. A more likely etiology was injury to the portal vein or venous stenosis because of spread of inflammation, followed by the liver abscess [6,7].

Although pylephlebitis (PVT) is an uncommon complication of liver abscess, it has been reported that liver abscess due to Klebsiella spp. tends to develop the formation of regional thrombophlebitis as compared to other causes of liver abscess [8-10]. *Streptococcus spp.* is normally present in the oral or gastrointestinal flora and can be the cause of liver abscesses [1]. There have been three reports of liver abscess caused by *Streptococcus spp.* and complicated by PVT [2-4].

The clinical characteristics of the 3 reported cases are summarized in Table 1. Liver abscesses were resolved after the combination therapies of antibacterial therapy and abscess drainage in 2 cases [3,4] or antibacterial therapy in other case [2]. The outcomes of PVT were variable; PVT resolved after anticoagulant and antibacterial therapies in one case [2] and without anticoagulant therapy in other case [3]. Moreover, PVT was not resolved after antibacterial therapy, abscess drainage, and anticoagulant therapies in another case [4]. PVT resolved with anticoagulant treatment, which also aimed to resolve complications of established portal thrombosis, such as portal hypertension [11] and extension of the thrombosis to the mesenteric venous arches, which may cause lethal intestinal infarction [12]. However, the indication of anticoagulants for patients with liver abscess and PVT is controversial [9] because there have been no prospective randomized controlled studies on the use of anticoagulants [3]. Moreover, PVTs may respond to the liver abscess treatment [3,8].

On the other hand, previous studies have demonstrated the effectiveness of anticoagulant treatment for PVT or pylephlebitis. Kanellopoulou et al. [13] reported better outcomes in pylephlebitis

patients who received anticoagulants plus antibiotics compared with those who received antibiotics in 100 relevant case reports.

Case (Year)	Age	Sex	Blood culture	Abscess culture	Treatment of liver abscess		Treatment of portal vein thrombosis	Outcome	Reference
					Drainage	Antibiotics			
-2003	59	M	*Streptococcus intermedius*	Not done	(-)	SBT/CPZ	Anticoagulant therapy	Improvement	[2]
-2012	57	F	Negative	*Streptococcus viridans*	(+)	Piperacillin-tazobactam	(-)	Improvement	[3]
-2013	56	M	Negative	*Streptococcus milleri*	(+)	PCG	Anticoagulant therapy	Abscess → improvement PVT → no change	[4]
Our case	51	M	*α-hemolytic streptococcus*	*α-hemolytic streptococcus*	(+)	Piperacillin	Anticoagulant therapy	Cured	
M: Male; F: Female; SBT/CPZ: Sulbactam sodium/Cefoperazon sodium; PCG: Benzylpenicillin potassium; PVT: Portal vein thrombosis									

Table 1: Characteristics of the patients with liver abscess and portal vein thrombosis caused by *Streptococcus spp.*

Plessier et al. [12] recommended prompt anticoagulation in patients with acute PVT of the portal vein or its left or right branches because of the high prevalence of permanent risk factors for venous thrombosis. Hall et al. [14] reported that partial or complete recanalization occurred in 81.8% of patients with acute PVT and liver cirrhosis or malignancies who were treated with anticoagulants and in 37.5% of those who were not. They also reported that spontaneous resolution of acute PVT was uncommon in that patient series [14]. In our patient, PVT resolved without complications with 2 weeks of anticoagulant therapy plus antimicrobial agents and abscess drainage. Therefore, administration of anticoagulant therapy should be considered in patients with liver abscess complicated by PVT.

Although anticoagulant with antimicrobial agents and abscess drainage may be standard therapy for cases with liver abscess and venous thrombosis, it has remained uncertain in regard to the treatments for PVT cases, which were refractory to anticoagulant therapy. However, pericutaneous approach such as transjugular intrahepatic portosystemic shunt (TIPS) [15,16] may be an effective therapeutics option for non-malignant and non-cirrhotic patients with acute or chronic PVT.

Conclusion

Liver abscess caused by *α-hemolytic streptococci* and complicated by PVT resolved after administration of antimicrobial agents and anticoagulants. In such patients, administration of anticoagulants is controversial, but it was beneficial in our presented case.

Conflict of Interest

There was no funding support and there is no conflict of interests to declare.

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