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CASE REPORT

Recurrent Serotype K1 *Klebsiella pneumoniae* Liver Abscess: A Single or Different Pathogen?

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We report a case of an 81-year-old woman who had a history of type 2 diabetes mellitus with the presentation of a recurrent community-acquired liver abscess caused by capsular serotype K1 *Klebsiella pneumoniae* after a previous liver abscess had been cured. With regards to the serotype K1 *K. pneumoniae* strains, the molecular genome of the recurrent strain differed completely from the strain that had caused the primary community-acquired liver abscess even though the antibiogram was the same. This case attempts to highlight that capsular serotype K1 could be an important factor influencing liver abscess formation and its subsequent recurrence.

Key words: *Klebsiella pneumoniae,* recurrence, liver abscess, serotype K1

INTRODUCTION

The epidemiology, management, and mortality in patients with pyogenic liver abscess, a potentially life-threatening intra-abdominal infection, have changed dramatically over the last two decades. In the past, *Escherichia coli* was the predominant causative agent. However, recent reports indicate that the incidence of *Klebsiella pneumoniae* has surpassed that of *E. coli* in Asia and western countries. The early use of broad-spectrum antibiotics plus drainage is the gold standard to control morbidity and mortality.[1-4]

A recurrent *K. pneumoniae* liver abscess (KLA) has seldom been reported,[5,6] and a recurrent liver abscess with the same pathogen, but different molecular pattern even less so. Herein, we describe a case of a liver abscess caused first by *K. pneumoniae*, and then unusually a recurrent liver abscess 4 months later caused again by *K. pneumonia* in which the strain shared the same capsular serotype and antibiogram with the first *K. pneumoniae* strain.

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CASE REPORT

An 81-year-old woman presented with a history of type 2 diabetes mellitus and hypertension, which had been controlled by regular oral medications for >10 years. Her first admission to our department was on May 30, 2010, because of poor appetite and general malaise for 1-week. At the emergency department, laboratory examinations showed leukocytosis and elevated C-reactive protein. Accordingly, abdominal ultrasonography was performed, which disclosed multiple hypoechoic lesions over the inferior portion of the lateral segment (S3), medial segment of the left hepatic lobe (S4) and inferior portion of the anterior segment of the right hepatic lobe (S5), which were seen as multi-lobulated cystic lesions on abdominal computed tomography, with the largest being about 5 cm × 2 cm in size over segment 4 [Figure 1]. Mild anemia and positive stool occult blood test were also noted. A liver abscess was confirmed by fine needle aspiration with pinkish pus from the hepatic lesions. She was treated with percutaneous transhepatic abscess drainage and parenteral antibiotics with third generation cephalosporin during hospitalization. The drained abscess culture showed serotype K1 *K. pneumoniae* with positive *magA* and *rmpA* genes on June 6, 2010, which was susceptible to the antibiotics we had administered. After discharge, an oral antibiotic regimen was employed for a total of 4 weeks. One month after discharge, follow-up abdominal sonography showed no focal lesions of the liver parenchyma [Figure 2].

Unexpectedly, the patient developed another episode of a liver abscess in the following 3 months. She presented with intermittent fever with chills and fatigue for 5 days, and visited

our emergency department again on October 3, 2010. Complete blood count showed more severe leukocytosis and a higher level of C-reactive protein than during the previous admission, and a positive stool occult blood test was also noted. Abdominal ultrasonography and noncontrast abdominal computed tomography both disclosed an irregular hypoechoic lesion (size: About 4.2 cm) over the superior portion of the anterior segment of the right hepatic lobe (S8), which was different from the first episode [Figure 3]. A liver abscess was diagnosed by fine needle aspiration of the S8 lesion. An aspirated pus culture showed *E. coli*, however blood cultures revealed ampicillin-resistant serotype K1 *K. pneumoniae* positive for the *magA* and *rmpA* genes, but with a different type of pulse-field gel electrophoresis [Figure 4]. The patient then received 4 weeks of antibiotic treatment, after which no complications including metastatic foci infection of the endophthalmitis or brain abscess were noted from the recurrent liver abscess. The characteristics and demographic data of the primary and recurrent KLA are shown in Table 1.

DISCUSSION

A pyogenic liver abscess may be caused by hematogenous dissemination or via the portal vein from a gastrointestinal infection by disseminated sepsis.[7] The disease can also arise from surgical or penetrating wounds and occasionally tissue super-infections. The pathogenesis leading to a major liver abscess is polymicrobial, and mixed enteric facultative and anaerobic pathogens are the most common. The typical clinical manifestations are fever, abdominal pain, nausea, vomiting, anorexia, and malaise, however these are nonspecific. In the current report, the antibiogram of the recurrent K1 KLA strain remained uniquely resistant to ampicillin, which was the same as the strain of the primary K1 KLA. From the view point of the genome, a different molecular pattern was demonstrated between the primary KLA and the recurrent strain. The most probable explanation why *K. pneumoniae* lead to the recurrent liver abscess is that it had colonized the intestinal tract or previously been seeded on the portal venous system.

Community-acquired pyogenic liver abscesses caused by *K. pneumoniae* has become an important health issue in

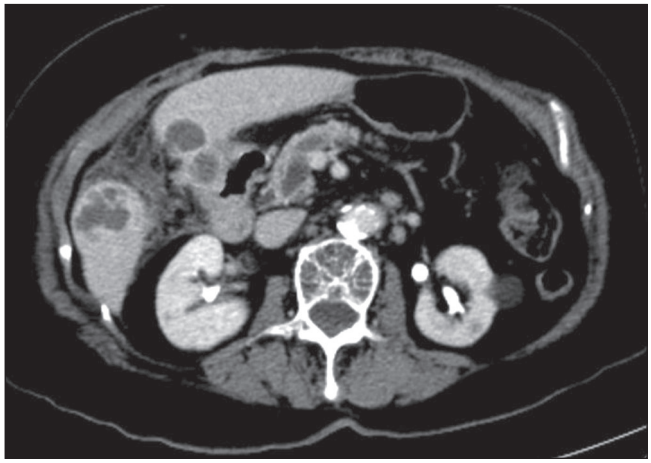

Figure 1. Abdominal computed tomography at the first admission revealed multiple multi-loculated cystic lesions over S3, S4 and S5

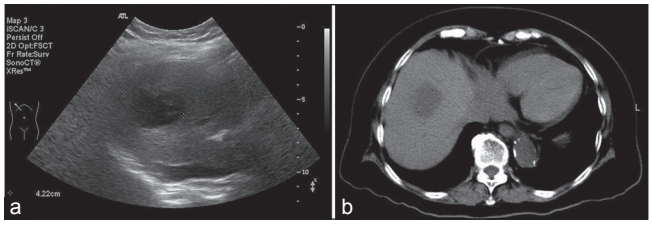

Figure 3. The secondary admission showed an irregular hypoechoic lesion over the S8 segment by abdominal ultrasonography (a) and abdominal computed tomography (b)

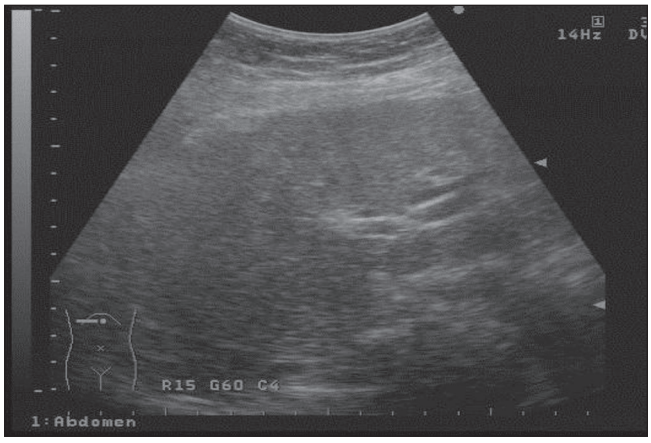

Figure 2. Following-up abdominal sonography showed no focal lesions of the liver parenchyma

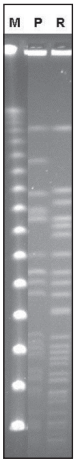

Figure 4. Strains of capsular serotype K1 primary ***Klebsiella pneumoniae*** liver abscess (KLA) and recurrent KLA showed different molecular patterns in pulsed field gel electrophoresis (P: first primary KLA, R: recurrent KLA, M: Marker)

Table 1. The characteristics and demographic data of primary and recurrent *Klebsiella pneumoniae* liver abscess

Characteristics	Time of diagnosis	
	May 30, 2010	October 03, 2010
Clinical manifestations	General malaise, poor appetite	Intermittent fever with chills and fatigue
HbAlc level (%)	7.4	6.4
Liver abscess		
Size (cm)	5 cm (S4, the largest)	4.22 cm
Location	S3, S4 and S5	S8
Pus culture	*Klebsiella pneumoniae* (AMP resistant only)	*Escherichia coli* (AMP resistant only)
Bacteremia	No growth	*Klebsiella pneumoniae* (AMP resistant only)
Stool occult blood	Negative	No data
Treatment	Percutaneous drainage, third generation cephalosporin	Percutaneous drainage, third generation cephalosporin

Drugs selected for susceptibility tests include the following: AMP, cefazolin, gentamicin, amikacin, AMP plus sulbactam, piperacillin plus tazobactam, ceftriaxone, ceftazidime, cefepime, ertapenem, imipenem and ciprofloxacin. AMP = ampicillin; HbA1c = glycosylated hemoglobin A1c

Taiwan and the USA. One report indicated that the annual incidence of pyogenic liver abscesses has increased steadily from 11.15/100,000 population in 1996 to 17.59/100,000 in 2004.[8] Risk factors include diabetes mellitus, gastrointestinal malignancy, hepato-biliary pathology and renal diseases. In one case–control study, patients with diabetes had a 3.6-fold increased risk of experiencing a pyogenic liver abscess.[9] Abscesses caused by *K. pneumoniae* have been reported to be more strongly associated with diabetes or impaired fasting glucose than liver abscesses caused by non-*K. pneumoniae*.[10]

Poor glycemic control with a higher level of glycosylated hemoglobin A1c leads to a higher risk of KLA and recurrent liver abscesses. We assume that diabetes mellitus is also a risk factor for recurrence. However, recurrent liver abscesses have been reported to occur in different hepatic segments resulting from common pathogens with *K. pneumoniae*.[11] Good glycemic control can increase the ability of neutrophil phagocytosis and prevent recurrent pyogenic liver abscesses.

CONCLUSION

Serotype K1 *K. pneumoniae* more easily causes liver abscesses and thus recurrence, especially in patients with diabetes and poor glycemic control.

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DISCLOSURE

The authors have no financial conflicts of interest.

REFERENCES

1. Rahimian J, Wilson T, Oram V, Holzman RS. Pyogenic liver abscess: Recent trends in etiology and mortality. Clin Infect Dis 2004;39:1654-9.
2. Kim JK, Chung DR, Wie SH, Yoo JH, Park SW, Korean Study Group for Liver Abscess. Risk factor analysis of invasive liver abscess caused by the K1 serotype *Klebsiella pneumoniae.* Eur J Clin Microbiol Infect Dis 2009;28:109-11.
3. Jepsen P, Vilstrup H, Schønheyder HC, Sørensen HT. A nationwide study of the incidence and 30-day mortality rate of pyogenic liver abscess in Denmark, 1977-2002. Aliment Pharmacol Ther 2005;21:1185-8.
4. Mohsen AH, Green ST, Read RC, McKendrick MW. Liver abscess in adults: Ten years experience in a UK centre. QJM 2002;95:797-802.
5. Yang YS, Siu LK, Yeh KM, Fung CP, Huang SJ, Hung HC, *et al*. Recurrent *Klebsiella pneumoniae* liver abscess: Clinical and microbiological characteristics. J Clin Microbiol 2009;47:3336-9.
6. Hsu WH, Yu FJ, Chuang CH, Chen CF, Lee CT, Lu CY. Occult colon cancer in a patient with diabetes and recurrent *Klebsiella pneumoniae* liver abscess. Kaohsiung J Med Sci 2009;25:98-103.
7. Clarençon F, Scatton O, Bruguière E, Silvera S, Afanou G, Soubrane O, *et al*. Recurrent liver abscess secondary to ingested fish bone migration: Report of a case. Surg Today 2008;38:572-5.
8. Tsai FC, Huang YT, Chang LY, Wang JT. Pyogenic liver abscess as endemic disease, Taiwan. Emerg Infect Dis 2008;14:1592-600.
9. Keynan Y, Rubinstein E. Diabetes mellitus and pyogenic liver abscess: Risk and prognosis. Clin Infect Dis 2007;45:801.

10. Yang CC, Yen CH, Ho MW, Wang JH. Comparison of pyogenic liver abscess caused by non-*Klebsiella pneumoniae* and *Klebsiella pneumoniae*. J Microbiol Immunol Infect 2004;37:176-84.

11. Tsai SS, Huang JC, Chen ST, Sun JH, Wang CC, Lin SF, *et al*. Characteristics of *Klebsiella pneumoniae* bacteremia in community-acquired and nosocomial infections in diabetic patients. Chang Gung Med J 2010;33:532-9.