Community-Acquired Liver Abscess Caused by Serotype K1 *Klebsiella pneumoniae* with CTX-M-15-Type Extended-Spectrum β-Lactamase

*Klebsiella pneumoniae* has emerged as the leading cause of community-acquired pyogenic liver abscess in many countries. Community-acquired pyogenic liver abscess due to *K. pneumoniae* with a unique antibiogram indicative of resistance only to ampicillin has been demonstrated. We reported the first case on community-acquired pyogenic liver abscess caused by CTX-M-15-type extended-spectrum β-lactamase (ESBL)-producing *K. pneumoniae* in a man without comorbidity or history of hospitalization.

In August 2006, a previously healthy 39-year-old man experienced a fever with shaking chills for 3 days. Abdominal computed tomography with contrast showed a low-density lesion with a central linear enhancement of about 4.5 cm in liver segment 8. The initial therapy was daily intravenous administration of 2 g ceftriaxone for empirical therapy. Sonography-guided percutaneous drainage of the liver abscess was performed. However, even after 1 week of the aforementioned therapy, a fever with shaking chills remained. Ultimately, *Klebsiella pneumoniae* was identified in the liver aspirate; susceptibility testing revealed resistance to cefazolin, ceftriaxone, cefotaxime, and cefepime but susceptibility to trimethoprim-sulfamethoxazole, gentamicin, ciprofloxacin, imipenem, and ertapenem. More refined identification confirmed the presence of CTX-M-15-type ESBL-producing *K. pneumoniae*. Ertapenem (1 g daily) was prescribed. The patient’s general condition immediately improved, and the fever abated. The patient was discharged after a 2-week regimen of ertapenem. Oral trimethoprim-sulfamethoxazole for sequential treatment was prescribed according to the antibiogram of the *K. pneumoniae*. He remained well at a 6-month follow-up.

This strain of serotype K1 CTX-M-15 ESBL-producing *K. pneumoniae* differed from strains causing community-acquired liver abscess with respect to the antibiogram pattern, the pulsed-field gel electrophoresis pattern (Fig. 1), and treatment (1, 3). The capsular serotype was determined with a capsular swelling test and countercurrent immunoelectrophoresis (3). The ESBL CTX-15 was detected by PCR amplification with a previous method (2). The primers used for the *bla*<sub>CTX-M-15</sub> gene were CTX-F (5′-GGTTAAAAAATC ACTGCCGT-C′-3′) and CTX-R (5′-TTGTGTGACGGATTTA GCGC-3′). Sequencing was done with corresponding primers specific for the *bla*<sub>CTX-M-15</sub> gene. The sequence was compared with that in the GenBank nucleotide database under accession no. AY044436 at http://www.ncbi.nlm.nih.gov/BLAST/. Multidrug-resistant strains of *K. pneumoniae* including ESBL have not been identified as the cause of community-acquired liver abscesses.

ESBL strains of *K. pneumoniae* are common causes of nosocomial bacteremia, pneumonia, and urinary tract infection but are rarely associated with liver abscess. Infections caused by ESBL-producing *Enterobacteriaceae* are particularly notorious for precipitating treatment failure and death. ESBL-producing *Escherichia coli*, particularly that producing the CTX-M type of ESBL, is an emerging pathogen and has become widespread since 2001 in both the United Kingdom and Europe (4, 5). The spread of *E. coli*-producing CTX-M-15 has involved β-lactamase transfer with the dissemination of *bla*<sub>CTX-M</sub> genes in both the general community and the hospital environment. In Asia, evolution of CTX-M-type β-lactamase genes from *bla*<sub>CTX-M-5</sub> to *bla*<sub>CTX-M-15</sub> under the selective pressure of antimicrobial therapy has occurred (6).

The pathogenesis of *K. pneumoniae* liver abscess is uncertain, although the capsular serotype K1/K2 seems likely involved, given the resistance of this serotype to phagocytosis and killing by neutrophils. Further studies are ongoing to elucidate the relationship between the virulence factors and resistant genes.

In conclusion, the presently reported case has shown that the strain of type CTX-M-15 ESBL-producing *K. pneumoniae* causing liver abscess differed from strains causing community-acquired liver abscess. Control of the spread of antibiotic-resistant bacteria such as ESBL-producing *Enterobacteriaceae* from the hospital environment to the general community is an important clinical and epidemiological concern. *K. pneumoniae* strains with ESBL-producing and virulence serotype K1 characteristics may be increasing in community-acquired liver abscess, complicating the management of the malady.

This study was supported by grants from the Tri-Service General Hospital (TSGH-C95-50 and C95-51) and the National Science Council (NSC 95-2314-B-016-013).

REFERENCES


![Dendrogram based on pulsed-field gel electrophoresis of nine clinical *K. pneumoniae* isolates. No. 12, 13, 21, 25, 27, 71, and 72 were isolated from community-acquired liver abscess of serotype K1; no. 10 was isolated from nosocomial liver abscess of ESBL-producing non-K1/K2; no. 27-2 is the present strain isolated from community-acquired liver abscess of ESBL-producing serotype K1.](http://aac.asm.org/Downloadedfrom)


Sheng-Chiang Su
Division of Infectious Diseases and Tropical Medicine
Department of Internal Medicine
Tri-Service General Hospital
National Defense Medical Center
Taipei, Taiwan

L. K. Siu
Ling Ma
Division of Clinical Research
National Health Research Institutes
Taipei, Taiwan

Kuo-Ming Yeh
Division of Infectious Diseases and Tropical Medicine
Department of Internal Medicine
Tri-Service General Hospital
National Defense Medical Center
Taipei, Taiwan

Chang-Phone Fung
National Yang-Ming University
Taipei, Taiwan

Jung-Chung Lin *
Feng-Yee Chang
Division of Infectious Diseases and Tropical Medicine
Department of Internal Medicine
Tri-Service General Hospital
National Defense Medical Center
Taipei, Taiwan

*Phone: 886 2 87927257
Fax: 886 2 87927258
Email: linjungchung1@yahoo.com.tw

Published ahead of print on 3 December 2007.