Nosocomial Liver Abscess Caused by Extended-Spectrum Beta-Lactamase-Producing Klebsiella pneumoniae

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A nosocomial pyogenic liver abscess caused by an extended-spectrum beta-lactamase-producing Klebsiella pneumoniae isolate presented in a man with adenocarcinoma of the stomach. The K. pneumoniae strain isolated from blood and liver aspirate cultures after antibiotic therapy for recurrent bacteremia was resistant to all extended-spectrum beta-lactams except imipenem and differed from K. pneumoniae strains causing community-acquired liver abscesses.

CASE REPORT

A 48-year-old man with adenocarcinoma of the stomach received a radical gastrectomy in 2003 and then 11 courses of intensive chemotherapy with fluorouracil, calcium folinate, and oxaliplatin for tumor metastasis to the liver and peritoneum. In July 2005, he was admitted to the Tri-Service General Hospital for treatment of oral candidiasis associated with shaking chills, spiking fever, and abdominal fullness. Initially, intravenous therapy was started for treatment of oral candidiasis associated with shaking chills, spiking fever, and abdominal fullness. Initially, intravenous ampicillin-sulbactam and oral fluconazole were given. Escherichia coli, Bacteroides fragilis, and Fusobacterium varium were isolated from two sets of blood cultures. The E. coli strain was susceptible to all cephalosporins but was resistant to ampicillin and trimethoprim-sulfamethoxazole. Cefpirome and metronidazole were administered 5 days later, but the patient’s intermittent spiking fever persisted. A blood culture disclosed Enterococcus faecium and Bacteroides thetaiotaomicron. Gentamicin was added to the patient’s treatment regimen. On day 14, he again developed spiking fever with pain over the right upper quadrant of his abdomen. Computed tomography of the abdomen showed a 4-cm hypodense lesion over segment 6 of the liver (Fig. 1). Percutaneous drainage was performed and pus was aspirated. The liver biopsy revealed a massive necrosis with inflammatory cells in liver tissue. An extended-spectrum beta-lactamase (ESBL)-producing Klebsiella pneumoniae strain, detected by double-disk screening and confirmatory tests based on CLSI recommendations, was isolated from cultures of blood and pus aspirated from the liver. The K. pneumoniae strain was resistant to all cephalosporins and aminoglycosides, but it was sensitive to imipenem. The MICs of the antimicrobial agents were determined by Etest using the interpretive standards based on CLSI recommendations. The MICs were as follows (6): ceftazidime, 256 μg/ml; ceftriaxone, 256 μg/ml; cefepime, 128 μg/ml; cefoxitin, 128 μg/ml; gentamicin, 96 μg/ml; ciprofloxacin, 32 μg/ml; and imipenem, 0.75 μg/ml. The AmpC disk test was negative for the detection of plasmid-mediated AmpC beta-lactamases (data was not shown) (1). The capsular serotype was non-K1/K2 by counter-current immunoelectrophoresis, and the phenotype was nonmucoid (9, 10). PCR for the magA gene was negative (7). In this case, the same antibiogram and pulsed-field gel electrophoresis (PFGE) patterns were demonstrated for isolates from blood and pus aspirated from the liver (data not shown). PFGE demonstrated that the strain had PFGE patterns completely different from those of strains from patients with community-acquired liver abscesses and nosocomially acquired ESBL-producing K. pneumoniae bacteremia (Fig. 2) (20). Although the patient’s clinical symptoms and signs improved after treatment with imipenem for 6 weeks, he died of progressive carcinomatosis with multiple-organ failure 2 months later.

Discussion. This is the first report of a nosocomial liver abscess caused by an ESBL-producing K. pneumoniae strain after intensive chemotherapy for carcinoma of the stomach and prolonged antibiotic treatment for recurrent bacteremia. K. pneumoniae has been emerging as the leading cause of community-acquired pyogenic liver abscess in Taiwan and the United States (2, 3, 8, 11, 14). Different clonal populations of K. pneumoniae cause community-acquired pyogenic liver abscesses (4, 5). Serotype K1/K2 accounts for 78% of isolates, and the non-K1/K2 serotype accounts for 22% (9). ESBL-producing K. pneumoniae strains are one of the most frequent causes of nosocomial pneumonia, intra-abdominal infections, urinary tract infections, and primary bacteremia (13). The prevalence of ESBL-producing K. pneumoniae strains as nosocomial pathogens is increasing worldwide (13, 15, 19). Information regarding K. pneumoniae-associated infections is summarized in Table 1 (3, 9, 12–14, 17). The newly emerged community-acquired K. pneumoniae strains that cause liver abscesses have been associated with more septic metastatic complications and low mortality rates, whereas nosocomial K. pneumoniae strains, including non-ESBL-producing and ESBL-producing strains, have been associated with less septic complications.

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metastatic complications and high rates of mortality. These community-acquired and nosocomial infections have very different presentations.

The availability of extended-spectrum cephalosporins has facilitated the treatment of severe infections caused by gram-negative bacteria. However, the increasing rate of use of these agents has been associated with the emergence of resistant bacterial strains, such as those that produce different SHV- or TEM-derived ESBLs (12, 16).

A well-known virulence factor of *K. pneumoniae* strains associated with liver abscesses is the capsular polysaccharide serotype (such as K1 and K2) (9). However, the ESBL-producing *K. pneumoniae* strain in our case was *magA* negative and non-K1/K2. The PFGE pattern of genomic DNA from the eight *K. pneumoniae* strains isolated from patients with community-acquired liver abscesses (serotype K1, n = 4), nosocomial bacteremias (K1 and non-K1/K2 [i.e., K54 and K55]), and the present case were different; and the genetic dendrogram showed that the strains had low levels of similarity. Further study is needed to determine whether other virulence factors,
TABLE 1. Summary of Klebsiella pneumoniae infection

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Mortality rate (%)</th>
<th>Metastatic complication, proportion of patients</th>
<th>Metastatic complication, proportion of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hepatobiliary-GI tract abnormality</td>
<td>14</td>
<td>Not found except in combination with liver abscess</td>
<td>6</td>
</tr>
<tr>
<td>Metastatic complication, proportion of patients</td>
<td></td>
<td>Endophthalmitis, 10.4%; multiple metastases, 3%</td>
<td>2.5</td>
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<td>DM rate (%)</td>
<td>49</td>
<td>75.4</td>
<td>15.2</td>
</tr>
<tr>
<td>Hepatobiliary-GI tract abnormality</td>
<td></td>
<td>15.2</td>
<td>12</td>
</tr>
<tr>
<td>Mortality rate (%)</td>
<td></td>
<td>Not found</td>
<td>15.2</td>
</tr>
<tr>
<td>Metastatic complication, proportion of patients</td>
<td></td>
<td>Not found</td>
<td>36</td>
</tr>
<tr>
<td>DM rate (%)</td>
<td></td>
<td>15.2</td>
<td>36</td>
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<tr>
<td>Metastatic complication, proportion of patients</td>
<td></td>
<td>36</td>
<td>23.5</td>
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<tr>
<td>Mortality rate (%)</td>
<td></td>
<td>23.5</td>
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</table>

### Notes

- **TABLE 1. Summary of Klebsiella pneumoniae infection**
  - **Hepatobiliary-GI tract abnormality**
  - **Metastatic complication, proportion of patients**
  - **DM rate (%)**
  - **Mortality rate (%)**

### Table Data

- **Community-acquired K. pneumoniae:**
  - Fever: 61.2 (±15.7)
  - Septic shock, pneumonia, UTI: 56.4 (±25–90)
  - Liver abscess, Taiwan: 56.4 (34–78)
  - Liver abscess, United States: 59.2 (±20.7)
  - Other site metastasis: 58 (2.1–90)

- **Nosocomially acquired ESBL-producing K. pneumoniae:**
  - Septic shock, pneumonia, UTI: 43
  - Other site metastasis: 13.1

### References


