Community-acquired Klebsiella pneumoniae liver abscesses — an “emerging disease” in Australia

James R Anstey, Timothy N Fazio, David L Gordon, Geoff Hogg, Adam W Jenney, Matthias Maiwald and Jonathan J Wilksch

Liver abscess due to Klebsiella pneumoniae infection has been widely reported in Asia, but rarely reported in Australia until now. We describe four previously well Asian-born patients who presented across Australia with community-acquired K. pneumoniae liver abscesses. With prompt recognition, appropriate antibiotics and early drainage, outcome is significantly improved, although vigilance for metastatic complications is essential. (MJA 2010; 193: 543-545)

Clinical records
During 2008 and 2009, four patients (two men, two women) presented around Australia with community-acquired Klebsiella pneumoniae liver abscesses (KPLAs). All four patients were previously well and did not have diabetes. Patients 1, 2 and 3 were Australian residents who were born in Asia and had recently visited there; Patient 4 was visiting from China.

All patients presented to hospital after several days of gastrointestinal and other symptoms. Liver abscess was shown on computed tomography scans, and K. pneumoniae infection was

1 Clinical and microbiological details of four patients with community-acquired Klebsiella pneumoniae liver abscesses

<table>
<thead>
<tr>
<th>Patient 1</th>
<th>Patient 2</th>
<th>Patient 3</th>
<th>Patient 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Demographics</td>
<td>M; 33y; Filipino-born; Victorian resident for 2 y</td>
<td>F; 52y; Malaysian-born; long-term Victorian resident</td>
<td>F; 67y; Malaysian-born; long-term SA resident</td>
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<tr>
<td>Recent travel/contacts</td>
<td>Lived with Filipino friends. Trip to Middle East via India 1 month prior</td>
<td>Trip to Malaysia 6 weeks prior</td>
<td>Trip to Malaysia between presentations</td>
</tr>
<tr>
<td>Features on presentation</td>
<td>2 days of vomiting, myalgias, fevers and rigors; hypotension, mild epigastric tenderness, RUQ tenderness</td>
<td>Several days of aches, rigors, diarrhoea, hypotensive, febrile</td>
<td>2008: 3 days of epigastric pain, low-grade fevers; 2009: 5 days of malaise, vomiting, RUQ pain</td>
</tr>
<tr>
<td>Notable investigations</td>
<td>First abdominal U/S normal; CT of abdomen: 5 cm septate liver lesion (Box 2)</td>
<td>Abdominal U/S: 9 cm multiloculated liver abscess; confirmed on CT of abdomen</td>
<td>2008: CT of abdomen: 3 cm liver lesion, near-resolved after 2 months; 2009: CT of abdomen: 6 cm liver abscess, progressed to 7.5 cm with central necrosis 1 week later</td>
</tr>
<tr>
<td>Microbiology</td>
<td>K. pneumoniae cultured on three sets of BC and abscess fluid; string test positive (Box 3)</td>
<td>K. pneumoniae BC and abscess fluid</td>
<td>2008 and 2009: K. pneumoniae BC</td>
</tr>
<tr>
<td></td>
<td>Resistant to ampicillin; sensitive to amoxicillin/clavulanic acid, ciprofloxacin, gentamicin</td>
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</tr>
<tr>
<td>Serotype</td>
<td>K1</td>
<td>K1</td>
<td>2008: isolate not serotyped; 2009: K2</td>
</tr>
<tr>
<td>Drain/surgery</td>
<td>Pigtail catheter</td>
<td>Pigtail catheter</td>
<td>Pigtail catheter, then laparotomy and chest drain</td>
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<tr>
<td>Antibiotics</td>
<td>Rationalised to ceftriaxone for 2 weeks; discharged on oral amoxicillin/clavulanic acid for 2 months</td>
<td>Rationalised to ceftriaxone for 1 month; discharged on oral cotrimoxazole for 11 weeks (rash with ciprofloxacin)</td>
<td>2008: 1 week ceftriaxone then 5 days of amoxicillin/clavulanic acid; 2009: 3 weeks ceftriaxone then 1 week amoxicillin/clavulanic acid</td>
</tr>
<tr>
<td>Complications</td>
<td>Brief acute renal impairment (creatinine to 180 μmol/L), 24 hours of septic shock requiring ICU</td>
<td>24 hours of septic shock requiring ICU</td>
<td>2009: 48 hours of septic shock requiring ICU</td>
</tr>
</tbody>
</table>

BC = blood culture. CT = computed tomography. ICU = intensive care unit. RUQ = right upper quadrant. U/S = ultrasound.
diagnosed following culture of abscess fluid or blood. Patients were treated with appropriate antibiotics and pigtail catheters for drainage; Patients 3 and 4 required surgical treatment.

Patients 1, 2 and 3 were well at follow-up; Patient 4 returned to China and was lost to follow-up. Patient 3 suffered a recurrence about 10 months after her first presentation, but this was too remote to be clearly attributable to her short course of antibiotics (only 10 days).

Box 1 summarises the clinical and microbiological details of the four patients.

Discussion

A community-acquired Klebsiella pneumoniae primary invasive liver abscess syndrome has been recognised in Asia for more than 20 years, with almost 1000 reported presentations published by 2008; it has been reported less frequently in other regions. K. pneumoniae infection accounted for over 80% of primary liver abscesses reported from Taiwan in the 1990s. Increasingly, cases have been seen outside Asia, primarily among patients of Asian ethnicity, including in the United States. It has only rarely been reported in Australia until now. Of interest are an absence of prior hepatobiliary disease, an association with diabetes, and a risk of metastatic spread. Community-acquired KPLA has been associated with severe metastatic complications, including endophthalmitis.

The geographical distribution of KPLA may be explained by the finding that K. pneumoniae isolates from Taiwan were far more likely to have a hypermucoviscous phenotype and to belong to K1 or K2 serotypes than those in other countries except South Africa, where invasive disease is also seen. Indeed, of the many K. pneumoniae capsular serotypes isolated from patients in an Australian tertiary hospital inpatient setting, K1 and K2 accounted for only 10 of 293 (3.5%) presentations.

All our patients had recently been in Asia, which raises the possibility of exposure to these virulent strains of the organism. However, case reports from the US have involved emigrants from Vietnam and Korea who had not travelled home for some years. A third-generation cephalosporin such as ceftriaxone is usually an effective treatment, with good penetration of vitreous fluid and cerebrospinal fluid, allowing it to reach metastatic lesions in these locations. In the case of endophthalmitis, systemic antibiotics should be combined with intravitreal injections. Treatment is required until clinical state, biochemistry and radiology indicate resolution, often requiring antibiotics for 4 to 6 weeks.
Another mainstay of therapy is computed tomography- or ultrasound-guided percutaneous abscess drainage. Surgical drainage may be necessary when percutaneous techniques have failed, as was seen in our Patients 3 and 4.

Metastatic spread not uncommonly complicates KPLA; reports from Taiwan estimate the frequency of this at between 3.5% and 20%. Endophthalmitis, lung abscesses and meningitis are the more common complications. Ophthalmological and other organ review is therefore indicated when KPLA is diagnosed. Visual recovery in patients with endophthalmitis is often poor; a high index of suspicion and early intervention before visual changes are noted may improve outcome.

Response to antibiotics and drainage is generally good. In contrast with patients with underlying biliary tract disease, long-term recurrence rates in patients with spontaneously occurring liver abscess appear to be low. Given the emerging global trend of $K. pneumoniae$ liver abscesses, Australian clinicians should be mindful of this condition, particularly, but not exclusively, in patients of Asian origin with abdominal infection or whose cultures reveal this organism. In this setting, a hypermucoviscous isolate of $K. pneumoniae$ may belong to serotype K1 or K2, and be associated with metastatic infection, particularly endophthalmitis, lung abscess and meningitis.

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Competing interests

None identified.

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