Community-acquired *Pseudomonas aeruginosa* pneumonia in previously healthy patients

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**Introduction:** *Pseudomonas aeruginosa* community-acquired pneumonia is an extremely rare clinical presentation but has been recognized in anecdotal reports, even in previously healthy patients.

**Case presentation:** We describe a previously healthy man who developed *P. aeruginosa* community-acquired pneumonia (CAP). He died of septic shock with rapidly progressive pulmonary consolidation in the right upper lobe (RUL). We reviewed the literature for *P. aeruginosa* CAP and identified 19 patients of whom 85 % (n = 17) had cavitations and/or consolidations in the RUL. We found that the odds ratio for death of shock at initial presentation was 8.333 (P = 0.046, 95 % confidence interval 1.034–67.142). We also found that *P. aeruginosa* CAP should be considered when individuals present with rapidly expanding cavitary pneumonia and/or consolidations in the RUL accompanied by septic shock, even if they have no known severe underlying disease and were previously healthy.

**Conclusion:** We showed here that radiological findings of *P. aeruginosa* CAP, such as cavitary pneumonia and/or consolidation in the RUL, might be a clinical clue to a diagnosis of CAP as well as the presence of septic shock.

**Keywords:** *Pseudomonas aeruginosa*; cavity; community-acquired pneumonia; septic shock.

**Introduction**

Published studies have indicated that the incidence of community-acquired pneumonia (CAP) caused by *Pseudomonas aeruginosa* ranges from 0.4 % (Torres and Menendez, 2010) to 5 % (Leroy *et al.* 1999) and that the disease has a 30 % mortality rate (Hatchette *et al.*, 2000). These infections usually occur in patients with severe underlying disease, and rarely in healthy individuals. Only 19 patients without any known severe underlying disease have been reported in the literature since 1968. Here, we describe a previously healthy man who developed *P. aeruginosa* CAP and present a review of the literature.

**Case report**

A 61-year-old man was referred to our hospital with a 3 day history of productive cough and right chest pain. He had felt well enough to work full time until the onset of symptoms but appeared ill upon presentation. He had worked as an office worker for 40 years, and denied any dust exposure or illicit drug use, or contact with sick people. He had been administered with oral anti-hyperglycaemic agents to treat controllable type 2 diabetes mellitus for over 15 years. His vital signs were as follows: blood pressure, 104/66 mmHg; heart rate, 128 beats min⁻¹; respiratory rate, 24 min⁻¹; body temperature, 38.7 °C; and 98 % oxygen saturation with ambient air, suggesting systemic inflammatory response syndrome.

Physical findings were normal except for mildly decreased respiratory sounds over the anterior aspect of the right upper lung field. A chest X-ray showed a homogeneous infiltrate in the right upper lobe (RUL) (Fig. 1a), indicating CAP. Thoracic CT (Fig. 1b, c) taken on admission showed consolidation with an air bronchogram in the RUL together with ground-glass opacities. His serum laboratory findings were as follows: normal white blood cell count (8200 μl⁻¹) and remarkably elevated C-reactive protein at 40.0 mg dl⁻¹. There was no detailed medical history on admission, while haemoglobin A1c...
levels showed only a mild elevation (7.2%). This might indicate moderately controlled hyperglycaemia (Nathan et al., 2008). Sputum Gram staining showed a dominant Gram-negative bacillus with other bacteria, and these results were scored as Geckler classification 3. Thus, no apparent pathogens were identified at that time. Based on the data, we immediately treated him with ampicillin/sulbactam (9 g day\(^{-1}\)) upon admission, but he went into shock 7 h later and required aggressive treatment including intubation. In addition, increased serum procalcitonin (71.8 ng ml\(^{-1}\)) and endotoxin (179.9 pg ml\(^{-1}\)) were found, suggesting that he was in septic shock. At that time, the treatment was immediately changed to doripenem hydrate (0.75 g day\(^{-1}\)). Thereafter, P. aeruginosa grew in both sputum and blood cultures on day 2 after hospital admission (hospital day 2). All isolates of P. aeruginosa were non-mucoid type and susceptible to commonly used anti-pseudomonal β-lactam antibiotics. In addition, the PFGE profiles suggested that these isolates were identical (see Fig. S1 available in the online Supplementary Material). On day 3, his general status rapidly deteriorated and he died of septic shock.

Our review of the literature (Table 1) uncovered descriptions of P. aeruginosa CAP arising in 19 previously healthy individuals, as well as the present patient. The ratio of females to males in the total of 20 patients was 7 : 13 [age (median ± sd), 45.9 ± 13.6 years]. The initial symptoms were non-specific, but 15 (75 %), 14 (70 %) and 12 (60 %) of these patients presented with cough, pleuritic pain and pyrexia, respectively. The duration from initial onset to admission ranged from 1 to 30 days (median, 3 days), that from admission to intubation was within 24 h and the mortality rate was 35 % (n=7) over a range of 9 hospital days (median, 1.5 days). Kaplan–Meier analysis confirmed a 61.1 % survival probability at hospital day 9 (Fig. 2). Only five (25 %) of the cultures were positive for P. aeruginosa in sputum, whereas 15 (75 %) sputum plus blood cultures were positive, suggesting a high rate of bacteraemia upon admission. Eight (40 %) of the patients were in shock at initial presentation and seven of them had P. aeruginosa bacteraemia. Univariate analysis identified septic shock at initial presentation (odds ratio, 8.333; 95 % confidence interval, 1.034–67.142; \(P=0.046\)), but not inappropriate initial antimicrobial therapy (50 %, \(n=10\)), as a significant factor for mortality. Importantly, the RUL was involved in 17 (85 %) patients with radiological findings such as cavitations and/or consolidations.

### Discussion

Approximately 4 % of normal adults can harbour P. aeruginosa in their pharynx or colon or on the skin (Rose et al., 1983). Diabetic patients have impaired polymorphonuclear functions including chemotaxis, adherence, phagocytosis and intracellular killing, as well as T-lymphocyte dysfunction (Gupta et al., 2007). This may induce the propagation of pathogens including P. aeruginosa. However, whether the presence of diabetes mellitus influences the infection-related mortality and infection itself is still under debate (Knapp, 2013). For example, a multivariate analysis by Torres and Menendez (2010) showed that independent predictors for CAP due to P. aeruginosa included chronic respiratory disease and enteral tube feeding but not diabetes mellitus, which supports the findings of Pennington et al. (1973). The portal of entry for P. aeruginosa septicaemia is always pneumonia (Ishihara et al., 1995), and this is almost universally fatal (Balch and Griffin, 1977), as in our patient. Our search identified a mortality rate of 35 %, which was similar to that of a previous report (30 %) (Hatchette et al., 2000); we also found that mortality was significantly associated with having septic shock at the time of initial presentation (odds ratio, 8.333). However, empirically appropriate therapy for P. aeruginosa did not contribute to a favourable prognosis. Based on these findings, we speculated that P. aeruginosa CAP occurring in previously healthy persons typically results in bacteraemia, which is sequentially followed by septic shock irrespective of the use of appropriate anti-pseudomonal antibiotics. In accordance with this viewpoint, the increased serum endotoxin level in our patient supported this hypothesis. Although various bacterial factors such as surface components, the type III secretion system, quorum sensing, and scavenging of iron and other extracellular products has been reported (Sadikot et al., 2005; Williams et al., 2010), it is possible that the large amount of endotoxin derived from the pathogen led to excessive inflammation and resulted in systemic inflammatory response syndrome in the present case.

Okada et al. (2012) recently concluded from thin-section CT findings of 29 patients with P. aeruginosa pulmonary infection that ground-glass attenuation or bronchial wall

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**Fig. 1.** Chest X-ray (a) showing massive infiltration in the right upper lung field, and thoracic CT taken on admission (b, c) showing consolidation with an air bronchogram accompanied by ground-glass opacities in the RUL.
Table 1. Clinical and radiological findings of *Pseudomonas aeruginosa* CAP in healthy individuals.

<table>
<thead>
<tr>
<th>Journal</th>
<th>Author</th>
<th>Year</th>
<th>Sex</th>
<th>Age</th>
<th>Initial symptoms</th>
<th>Duration from initial onset to admission</th>
<th>Bacteremia</th>
<th>Shock</th>
<th>Anti-PAB</th>
<th>Prognosis</th>
<th>Elapsed time to death/discharge</th>
<th>Affected area</th>
<th>Radiological findings</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Our patient</em></td>
<td>Tsuji. S</td>
<td>2012</td>
<td>M</td>
<td>61</td>
<td>Fever, cough, right chest pain</td>
<td>3</td>
<td>+</td>
<td>–</td>
<td>–</td>
<td>D</td>
<td>4</td>
<td>RUL</td>
<td>Con</td>
</tr>
<tr>
<td><em>Intern Med</em></td>
<td>Okamoto. M</td>
<td>2012</td>
<td>F</td>
<td>59</td>
<td>Diarrhea, chest pain, cough</td>
<td>3</td>
<td>+</td>
<td>–</td>
<td>–</td>
<td>A</td>
<td>30</td>
<td>RUL</td>
<td>Con</td>
</tr>
<tr>
<td><em>Neth J Med</em></td>
<td>Shaulov. A</td>
<td>2011</td>
<td>M</td>
<td>44</td>
<td>Dyspnea, cough, hemoptysis</td>
<td>4</td>
<td>+</td>
<td>+</td>
<td>–</td>
<td>D</td>
<td>2</td>
<td>RUL/BIL</td>
<td>Cav/Con</td>
</tr>
<tr>
<td><em>Infection</em></td>
<td>Huhulescu. S</td>
<td>2011</td>
<td>F</td>
<td>49</td>
<td>Chest pain, cough</td>
<td>2</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>D</td>
<td>9</td>
<td>Left lobe</td>
<td>Con</td>
</tr>
<tr>
<td><em>Clin Infect Dis</em></td>
<td>Crnich. C.J</td>
<td>2003</td>
<td>M</td>
<td>40</td>
<td>Fever, chills, cough, blood and rust-colored sputum</td>
<td>3</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>A</td>
<td>12</td>
<td>RUL/RML</td>
<td>Con</td>
</tr>
<tr>
<td><em>Clin Infect Dis</em></td>
<td>Hatchette. TF</td>
<td>2000</td>
<td>F</td>
<td>67</td>
<td>Diarrhea, cough, pleuritic chest pain</td>
<td>7</td>
<td>+</td>
<td>–</td>
<td>–</td>
<td>D</td>
<td>2</td>
<td>RUL/RML</td>
<td>Con</td>
</tr>
<tr>
<td><em>Conn Med.</em></td>
<td>Vicram. HR</td>
<td>1999</td>
<td>M</td>
<td>62</td>
<td>Cough, intermittent fevers, weight loss</td>
<td>30</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>A</td>
<td>NA</td>
<td>RUL</td>
<td>Cav/Con</td>
</tr>
<tr>
<td><em>Intensive Care Med.</em></td>
<td>Ishihara. S</td>
<td>1995</td>
<td>F</td>
<td>48</td>
<td>Back pain, dyspnea, fever</td>
<td>1</td>
<td>+</td>
<td>–</td>
<td>–</td>
<td>A</td>
<td>34</td>
<td>RUL</td>
<td>Con</td>
</tr>
<tr>
<td><em>Hosp Pract (Off Ed)</em></td>
<td>Cirigliano. MD</td>
<td>1994</td>
<td>F</td>
<td>20</td>
<td>Fever, dyspnea, right pleuritic pain, chills</td>
<td>2</td>
<td>+</td>
<td>–</td>
<td>–</td>
<td>A</td>
<td>21</td>
<td>RUL</td>
<td>Con</td>
</tr>
<tr>
<td><em>Intensive Care Med.</em></td>
<td>Henderson. A</td>
<td>1992</td>
<td>M</td>
<td>52</td>
<td>Fever, cough, left sided pleurisy</td>
<td>2</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>D</td>
<td>1</td>
<td>RUL/LLL</td>
<td>Con</td>
</tr>
<tr>
<td><em>Aust NZ J Med</em></td>
<td>Quirk. JA</td>
<td>1990</td>
<td>F</td>
<td>40</td>
<td>Fever, mild diarrhea, left sided pleuritic pain</td>
<td>1</td>
<td>+</td>
<td>+</td>
<td>–</td>
<td>A</td>
<td>NA</td>
<td>LUL</td>
<td>Cav/Con</td>
</tr>
<tr>
<td><em>West J Med</em></td>
<td>Harris. AA</td>
<td>1984</td>
<td>M</td>
<td>39</td>
<td>Cough, rigors, drenching sweats, left pleuritic pain</td>
<td>5</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>A</td>
<td>53</td>
<td>LLL</td>
<td>Con</td>
</tr>
<tr>
<td><em>JAMA</em></td>
<td>Rose. HD</td>
<td>1983</td>
<td>M</td>
<td>47</td>
<td>Fever, malaise, pleuritic pain, cough, bloody sputum</td>
<td>3</td>
<td>–</td>
<td>–</td>
<td>+</td>
<td>A</td>
<td>23</td>
<td>RUL</td>
<td>Cav/Con</td>
</tr>
<tr>
<td><em>South Med J</em></td>
<td>Fishman. H</td>
<td>1983</td>
<td>M</td>
<td>29</td>
<td>Right pleuritic pain, chills, cough, dyspnea</td>
<td>2</td>
<td>–</td>
<td>–</td>
<td>+</td>
<td>A</td>
<td>15</td>
<td>RUL</td>
<td>Con</td>
</tr>
<tr>
<td><em>Am Rev Respir Dis</em></td>
<td>Hoogwerf. BJ</td>
<td>1981</td>
<td>M</td>
<td>64</td>
<td>Dyspnea, intermittent fever, chills, cough, weight loss</td>
<td>30</td>
<td>+</td>
<td>–</td>
<td>–</td>
<td>A</td>
<td>35</td>
<td>RUL</td>
<td>Cav/Con</td>
</tr>
<tr>
<td><em>Tex Med</em></td>
<td>Hyslop. IR</td>
<td>1971</td>
<td>M</td>
<td>61</td>
<td>Pleuritic pain, fever, cough</td>
<td>6</td>
<td>+</td>
<td>–</td>
<td>–</td>
<td>A</td>
<td>26</td>
<td>RUL/RLL</td>
<td>Con</td>
</tr>
</tbody>
</table>

A: alive; Anti-P AB, anti-Pseudomonas antibiotics; BLL, bilateral lower lobe; Cav, cavitation; Con, consolidation; D, dead; ES, endotracheal secretion; LLL, left lower lobe; LUL, left upper lobe; N, no; NA, not available; RLL, right lower lobe; RML, right middle lobe; RUL, right upper lobe; Y, yes.
thickening were the main findings, but only nine patients with CAP were included in that study.

The increasing importance of *P. aeruginosa* as a causal agent of CAP has recently been recognized, but radiological findings have not yet been defined. Furthermore, the initial symptoms of *P. aeruginosa* CAP are not specific enough for a precise diagnosis. From this perspective, we showed here that radiological findings of *P. aeruginosa* CAP, such as cavitary pneumonia and/or consolidation in the RUL, might be a clinical clue to a diagnosis of CAP, as well as the presence of septic shock. Further accumulation of such cases would be required for a more precise understanding of the CAP caused by *P. aeruginosa*.

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**References**


