Pneumonia due to a resistant *Acinetobacter* organism

A case report from the respiratory intensive care unit

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**Summary**

A severe pneumonia due to *Acinetobacter calcoaceticus* occurring in a patient after chest trauma is described. *Acinetobacter* was isolated on 69 occasions during 1979 in the Respiratory Intensive Care Unit at Groote Schuur Hospital. Attention is drawn to the widespread resistance to antibiotics, including aminoglycosides, that this organism has developed.

*Acinetobacter calcoaceticus* is a non-fermentive Gram-negative bacillus widely distributed in nature. It can readily be isolated from healthy people and is frequently recovered from patients in intensive care units. Until recently the organism has been regarded as either a colonizer or a commensal. It now appears that active infection with this organism may be more common than was previously thought, especially among seriously ill patients. The problem is compounded because the organism has been found to develop resistance readily to commonly used antibiotics, including the aminoglycosides.

In this report, which describes a case of pneumonia thought to have been caused by *Acinetobacter*, the severity of the illness and some of the problems encountered in its treatment are emphasized.

**Case report**

A 39-year-old man was admitted to the Intensive Care Unit at Groote Schuur Hospital after a motor vehicle accident. He had previously been in good health, but had been a smoker. His only injuries were five fractured ribs, Nos. 6-11 on the right side in the anterior axillary line, which caused a significant flail segment on inspiration (Fig. 1). He was fully conscious and not distressed or cyanosed. The blood pressure was 110/70 mmHg, the pulse rate 80/min, and the respiratory rate 24/min. His temperature was 36.5°C. The only abnormal physical sign was the paradoxical movement over the affected area of the chest cage.

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It was decided to ventilate him electively until the segment stabilized. A volume-cycled ventilator was used with 40% entrained oxygen and 5 cm positive end-expiratory pressure. Penicillin was the only antibiotic given. An uncomplicated stay in the Intensive Care Unit for the next 10 days was anticipated. However, the day after admission he became pyrexial with a temperature of 38°C. A chest radiograph showed pneumonic changes and a small right-sided pleural effusion under the injured ribs. Copious secretions were suctioned from his endotracheal tube. These grew *Streptococcus pneumoniae* sensitive to penicillin and an *Acinetobacter* species sensitive to tobramycin and co-trimoxazole and insensitive to gentamicin.

As the *Acinetobacter* species was considered at the time to be of no clinical significance, it was not specifically treated. Gentamicin was added to the penicillin as a general Gram-negative cover. This has been our practice for a number of years in the intensive care units. In spite of this the pneumonia extended, becoming almost confluent on the left side as well as the right (Fig. 2). The patient became severely hypoxic with a Po2 of 6 kPa and was extremely difficult to ventilate, even after sedatives and muscle relaxants. He had a respiratory arrest following a tension pneumothorax. *Acinetobacter* species were repeatedly grown from his sputum in pure culture. Gram-negative organisms and numerous pus cells were seen on the Gram stains. A constant feature was the excessive bronchial secretion throughout the illness.

On the 10th day the antibiotic regimen was changed to tobramycin and co-trimoxazole because it was felt that the *Acinetobacter* had by this stage become an active pathogen. For a further 2 weeks he remained extremely ill, with high-grade pyrexia, bilateral patchy consolidation on chest radiography and low arterial Po2 with a large alveolar-arterial gradient. Gradually, over the ensuing 3 weeks, he
TABLE I. RESULTS OF RESPIRATORY FUNCTION TEST BEFORE AND AFTER BRONCHODILATATION

<table>
<thead>
<tr>
<th></th>
<th>2 mo. after injury</th>
<th>3 mo. after injury</th>
<th>8 mo. after injury</th>
<th>Predicted</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
<td>Before</td>
<td>After</td>
</tr>
<tr>
<td>FVC (ml)</td>
<td>700</td>
<td>700</td>
<td>1 650</td>
<td>1 830</td>
</tr>
<tr>
<td>FEV₁ (ml)</td>
<td>690</td>
<td>700</td>
<td>1 780</td>
<td>1 730</td>
</tr>
<tr>
<td>FEV₁/FVC (%)</td>
<td>98</td>
<td>103</td>
<td>94</td>
<td>94</td>
</tr>
<tr>
<td>TLC (ml/min mmHg)</td>
<td>6</td>
<td>10</td>
<td>14</td>
<td>31</td>
</tr>
</tbody>
</table>

FVC = forced vital capacity; FEV₁ = forced expiratory volume per second; TLC = total lung capacity.

Colonization studies have shown that *Acinetobacter* may be isolated from the skin from as many as 25%, and from the throats of 7% of healthy people and that it also forms part of the normal flora of the lower gastrointestinal tract. In one survey the organism was found in 20% of samples taken from dry hospital floors. Other established sources of this organism include various parts of mechanical ventilators, humidifiers and blood-collecting tubes when cleaning and sterilization are inadequate. Patients with tracheostomies, catheters and intravenous lines and those on ventilators, especially if broad-spectrum antibiotics are being used, are the usual sources of the organism. Most isolates in hospital are thus obtained from intensive care units, both from the patients and from the environment. Even in these situations only a few isolates represent active infection. A wide variety of infections due to *A. calcoaceticus* have been described, including septicaemia, endocarditis, meningitis, brain abscesses, tracheobronchitis, pneumonia, lung abscesses, urinary tract infections, skin abscesses, intra-abdominal infections and cellulitis. The criteria for deciding whether these infections are due to *Acinetobacter* are not clear-cut. In the case of pneumonia, which is the most common infection, Glew and Moellering suggested that three of the following criteria must be met for the diagnosis to be made: (i) production of copious amounts of thick sputum; (ii) demonstration in the Gram-stained sputum smear of both Gram-negative cocci and polymorphonuclear leucocytes in large numbers; (iii) repeated isolation of moderate growths of *A. calcoaceticus* from sputum and tracheal aspirate cultures; (iv) isolation of the organism from blood and sputum simultaneously; (v) isolation of *A. calcoae­ ticus* from the lung at autopsy.

In our patient three of these criteria were fulfilled. Although blood cultures were performed on numerous occasions the patient was continually on antibiotics and hence no growth was obtained.

Bilateral bronchopneumonia is the most common pattern of pneumonia but lobar pneumonia with cavitation, effusion and empyema may occur. Although *Acinetobacter* causes pneumonia more frequently in the compromised hospital patient, it may also be a cause of infection in the community, especially in middle-aged persons with a chronic underlying disease or a history of alcoholism. In this setting it causes a particularly fulminant infection with a high mortality rate.

The antibiotic resistance of the organism is causing increasing concern since it is showing resistance to the aminoglycosides, particularly gentamicin, and more recently tobramycin and amikacin. Aminoglyco­side resistance has increased considerably in recent years. In one study 25% of organisms were found to be resistant to gentamicin and 16% of these were also resistant.
to tobramycin. Our experience has been similar. During 1979 Acinetobacter was isolated on 69 occasions from 53 patients in our respiratory intensive care ward. We found 72% to be resistant to gentamicin and 22% to tobramycin. The detailed results of sensitivity tests are shown in Table II. The high degree of resistance to all commonly used antibiotics is shown.

It is important to appreciate the role that this organism may play in causing pneumonia in patients receiving intensive care. The fact that it is so frequently found in this environment stresses again the need for strict attention to aseptic techniques, adequate and repeated hand washing and the use of bacteriologically clean respiratory apparatus in an effort to halt cross-infection.

### Table II. Results of Antibiotic Sensitivity Tests on 69 Isolates from 53 Patients in the Respiratory Intensive Care Unit

<table>
<thead>
<tr>
<th>Drug</th>
<th>Resistant</th>
<th>Sensitive</th>
<th>Unrecorded</th>
<th>% resistance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cephalosporin</td>
<td>61</td>
<td>3</td>
<td>5</td>
<td>88</td>
</tr>
<tr>
<td>Chloromycin</td>
<td>58</td>
<td>5</td>
<td>6</td>
<td>84</td>
</tr>
<tr>
<td>Gentamicin</td>
<td>50</td>
<td>17</td>
<td>2</td>
<td>72</td>
</tr>
<tr>
<td>Amoxycillin</td>
<td>47</td>
<td>20</td>
<td>3</td>
<td>68</td>
</tr>
<tr>
<td>Tetracycline</td>
<td>44</td>
<td>20</td>
<td>5</td>
<td>63</td>
</tr>
<tr>
<td>Chlorotrimoxazole</td>
<td>24</td>
<td>41</td>
<td>4</td>
<td>35</td>
</tr>
<tr>
<td>Amikacin</td>
<td>21</td>
<td>37</td>
<td>11</td>
<td>30</td>
</tr>
<tr>
<td>Tobramycin</td>
<td>15</td>
<td>40</td>
<td>14</td>
<td>22</td>
</tr>
</tbody>
</table>

**REFERENCES**


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**Narkose-aspekte van harttamponade na amebiese lewerabse**

'n Gevalbespreking

**A. BOSMAN, A. J. P. LESSING**

**Summary**

The problem of tamponade due to rupture of an amoebic abscess, although rare, is extremely complex, and in the planning of the anaesthetic technique the following factors should receive attention: (i) the low cardiac output in which bradycardia, vasoconstriction, high inflation pressures and excessive fluid load must be avoided; (ii) pulmonary complications in which a double-lumen endotracheal tube should be considered; and (iii) sepsis.

**References**