

Community-Acquired Nonbacteremic Acinetobacter Pneumonia

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A *Acinetobacter calcoaceticus* variety *anitratus* is an encapsulated, non-spore-forming gram-negative bacillus that may be found as the normal flora of the skin, conjunctiva, and perineal area. *Acinetobacter* is a well-documented source of nosocomial infections and is a less common source of primary community-acquired pneumonias.¹ Eighteen cases of community-acquired *Acinetobacter* pneumonia have been previously reported with eight of these cases being fatal and all 18 having blood cultures positive for the organism.²⁻⁶ This report will describe a case of documented community-acquired *Acinetobacter* pneumonia with pleural effusion and negative blood cultures.

CASE REPORT

A 54-year-old male truck driver presented to the emergency room of Memorial Hospital of Laramie County in Cheyenne, Wyoming, with a chief complaint of severe left-sided chest pain and shortness of breath. Onset of the pain was sudden and was not associated with activity; the pain was located over the left precordium, extending along the left lower rib cage and into the axilla. The pain was sharp and pleuritic. Past medical history was unremarkable. Family history was remarkable for type II diabetes mellitus in the patient's mother. The patient admitted to a greater than 60-pack-year smoking history and the consumption of 8 to 16 ounces of vodka per week.

Initial physical examination revealed a temperature of 38 °C (100.4 °F), pulse of 117 beats/min, blood pressure 140/110 mmHg, and respiratory rate of 32 to 38/min. The patient appeared dusky, apprehensive, dyspneic, and had the odor of alcohol. Examination of the chest revealed bibasilar rales with absent breath sounds in the left base.

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An expiratory rub was heard periodically over the left lateral chest. Cardiac examination was unremarkable except for tachycardia. The abdomen was distended, rigid, and tender to palpation with absent bowel sounds. No masses could be palpated because of the distention. The remainder of the examination was otherwise unremarkable.

Initial laboratory data included a leukocyte count of $14.7 \times 10^9/L$ ($14,700/mm^3$) with 0.45 neutrophils, 0.16 band cells, 0.30 lymphocytes, 0.07 monocytes, 0.01 eosinophils, 0.01 basophils; random glucose 24.4 mmol/L (440 mg/dL). Renal studies, hepatic enzymes, amylase, and electrolytes were within normal limits. Urinalysis revealed trace protein, glucose 0.3 mmol/L (5.0 mg/dL) and acetone 860 μ mol/L (5.0 mg/dL). Arterial blood gas revealed pH 7.40, oxygen 6.3 kPa (47 mmHg), carbon dioxide 5.3 kPa (40 mmHg), bicarbonate 24 mmol/L (24 mEq/L), total bicarbonate 24 mmol/L (24 mEq/L), base excess -0.5, and an 0.81 oxygen saturation on 6 L of oxygen. Radiography of the chest revealed an infiltrate in the left lung base. Abdominal films were consistent with an ileus. Electrocardiogram showed a sinus tachycardia with a marked R wave in chest leads V_2 and minor ST segment depression in leads V_2 , V_3 and V_4 . Serial electrocardiograms and cardiac enzymes were normal, as was a ventilation perfusion scan done shortly after admission.

After admission, three blood cultures were obtained with temperature elevations. Two sputum samples were obtained and were suggestive of *Streptococcus pneumoniae* on Gram stain. Treatment was initiated with intravenous ampicillin (2 g initial dose, then 1 g every four hours), vigorous pulmonary therapy, and oxygen supplementation.

A repeat chest x-ray examination on the second day of admission showed further progression of the inflammatory process in the left base with the development of a significant pleural effusion. Based on the culture report of a gram-negative bacillus, tobramycin (100 mg every eight hours) was added. At 72 hours, a thoracentesis was performed. No bacteria were identified on the fluid Gram stain, and subsequent routine and acid-fast cultures had no growth.

Preliminary sputum culture identification showed few α -hemolytic streptococci, few *Neisseria* organisms, and many *Enterobacter* organisms. Final culture identification of both sputum samples revealed the presumed *Enterobacter* organism to be *Acinetobacter calcoaceticus* variety *anitratum*. Sensitivity studies confirmed the organism was sensitive to the tobramycin.

The patient's clinical course improved at day 6. The leukocyte count peaked at $20.6 \times 10^9/L$ ($20,600/mm^3$) on day 5 and returned to normal $10.9 \times 10^9/L$ ($10,900/mm^3$) at the time of discharge. The patient's hypoxia also gradually improved during this time, and oxygen supplementation was decreased from 6 L to room air. Following ten days of intravenous antibiotics, the patient was converted to oral trimethoprim-sulfamethoxazole (160 mg/1800 mg every 12 hours) for the completion of a 21-day course.

The patient's ileus, felt to be secondary to the pneumonia, was treated with nasogastric tube and suction, and resolved during the first five to six days of hospitalization. The patient's elevated blood glucose was initially treated with regular subcutaneous insulin. Split dosage of NPH and regular insulin were instituted once full caloric intake was obtained. The patient's blood pressure remained elevated, and he was started on methyl dopa (250 mg twice daily).

Following discharge, his chest x-ray findings and arterial blood gas levels returned to normal. His diabetes mellitus is controlled with glyburide (7.5 mg daily) and diet. The hypertension continues to be well controlled with the methyl dopa.

DISCUSSION

Gram-negative bacillary pneumonias are a major cause of community-acquired pneumonias. Lerner⁷ has defined the following criteria for definitive diagnosis of gram-negative bacillary pneumonia: (1) the gram-negative bacillus that can be cultured is the same as the predominate organism from two or more consecutive sputum specimens, (2) the same gram-negative bacillus can be isolated from both blood and sputum in close temporal proximity, (3) gram-negative bacilli can be isolated from pleural fluid, and, (4) the clinical course and response to antibiotics must be compatible with the diagnosis.

Acinetobacter must always be considered in a gram-negative pneumonia because there is significant morbidity and mortality from this disease. The typical patient is older (median age of 50 years), male, and has a chronic underlying disease. Alcoholism is a common co-existing problem. Cases have also been reported with non-Hodgkins

lymphoma, chronic renal failure, alveolar proteinosis, and chronic bronchitis.

The usual presentation of the disease is shortness of breath, productive cough, pleuritic chest pain, and acute onset of fever. Typically, the patient will have evidence of severe respiratory distress, hypoxemia, and leukopenia. The patient's condition may progress rapidly to shock within 24 hours. The chest x-ray film shows a lobar or bronchopneumonia pattern that often becomes bilateral within 24 hours of admission. Pleural effusions occur in approximately one half the reported cases and may develop into empyemas. Blood cultures have been positive in all previously reported cases. Organisms found on sputum Gram stain may be misinterpreted as *Neisseria* or *Hemophilus*.⁸ Carbohydrate metabolism analysis is essential to differentiate *Acinetobacter* from *Enterobacter*.

Antimicrobial therapy must be initiated quickly and appropriately; two-drug therapy of an aminoglycoside and an antipseudomonal penicillin is recommended. Delay in treatment has been shown to increase the likelihood of mortality. Trimethoprim-sulfamethoxazole is the drug of choice for oral therapy because of its degree of anti-*acinetobacter* activity. The organism has little or no response to first- and second-generation cephalosporins, penicillins, erythromycin, clindamycin, or chloramphenicol. Recommended treatment time is 14 to 21 days.

The above case supports the fact that gram-negative bacillary pneumonias do occur in community settings. Because of the severity and the high mortality rate (44 percent) from pneumonia caused by *Acinetobacter calcoaceticus* variety *anitratus*, prompt and proper treatment, including adequate antibiotic coverage, oxygen therapy, and treatment of shock, need to be initiated for favorable outcome in these patients.

References

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