Neisseria sicca Pneumonia

To the Editor:

Acute pneumonia caused by species of Neisseria are uncommon. Most of the reported cases are due to *Neisseria meningitidis* group Y1 and *Branhamella catarrhalis*2 (formerly known as *Neisseria catarrhalis*). This report describes the first case due to *Neisseria sicca*.

CASE REPORT

An 80-year-old white man was admitted with a one-day history of cough, chills and fever. Three weeks previously he had been hospitalized and was diagnosed as having "pneumococcal" pneumonia, treated with 1.2 million units of procaine penicillin intramuscularly daily for five days, and responded well. One week later the patient started coughing and produced purulent sputum. His family physician prescribed ampicillin 250 mg four times a day for two weeks. Three days after ampicillin was discontinued, he developed cough, shacking chills and fever, and was subsequently admitted. Past medical history indicated the patient has chronic atrial fibrillation and atherosclerotic heart disease.

Physical examination revealed an elderly male in acute respiratory distress. His temperature was 102°F (38.9°C), pulse rate 110 per minute, blood pressure 110/70 mm Hg, respiratory rate 28 per minute. Pertinent physical findings were limited to the chest; crepant rales and bronchial breathing were heard over the left mid and lower lung fields.

Laboratory data: white blood cell count 12,000/cc mm with 71 percent polys, 12 percent bands, 11 percent lymphocytes, 6 percent monocytes; hemoglobin 12.3 gm percent; hematocrit 37 percent; BUN 34 mg percent; creatinine 2.8 mg percent; urine specific gravity 1.015; pH 6.0; WBC 100,000/high power field, RBC 10-15 per high power field. Arterial blood gas levels: Pco2 58 mm Hg; pH 7.42; O2 saturation 100 percent, Pco2 41 mm Hg, HCO3 25 mEq/liter. Chest x-ray film (Fig 1) revealed fluffy alveolar infiltrates involving the left lower lobe.

Because of recent antibiotic therapy and the patient's inability to cough effectively, a transtracheal aspiration was done. Purulent sputum was obtained and cultured aerobically and anaerobically. Gram stain showed numerous polymorphonuclear and gram-negative diplococci. The patient was started on gentamicin 80 mg every 18 hours and aqueous penicillin G 6 million units daily. The patient improved over the next two days. Gentamicin was stopped on the second day when sputum culture grew *Neisseria sicca* in pure culture. On the seventh hospital day, penicillin was stopped. The patient remained afebrile and was discharged. Six months after discharge he continued to be well.

DISCUSSION

*Neisseria sicca* is a member of the family Neisseriaceae. It is a normal inhabitant of the nasopharynx and is rarely pathogenic. A search of the literature reveals only a single case of meningitis and septicemia due to this agent.3

Pulmonary infections due to Neisseria species are uncommon. In the normal host, most Neisseria species infections are due to *Neisseria meningitidis* group Y.1 *Branhamella catarrhalis* has been described among coal miners with chronic lung disease2 and in the immunodeficient host.4 Clinically and radiologically, pulmonary infections due to Neisseria species cannot be distinguished from other causes of bacterial pneumonias. The diagnosis of pneumonia due to Neisseria species remains difficult. Since Neisseria species are part of the normal flora of the upper respiratory tract and considered as "nonpathogenic," routine culture of sputum is of little value. However, the use of transtracheal aspiration may aid in diagnosis.

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Figure 1. Chest roentgenogram reveals an extensive left lower lobe infiltrate.
MB Isoenzyme of Creatine Phosphokinase and Exercise Stress Tests

To the Editor:

The article by Marmor et al1 entitled “MB isoenzyme of Creatine Phosphokinase: Indicator of Ischemia in Coronary Arterial Disease” once again calls attention to the difficulty of interpreting an elevated concentration of the MB isoenzyme of creatine phosphokinase following an abnormal exercise stress test in the presence of normal levels of myoglobin and total creatine phosphokinase in the serum and in the absence of diagnostic electrocardiographic changes. Marmor et al indicated that “in severe hypoxia, metabolic changes occur in the membrane of the myocardial cell, changes which are responsible for the release of the MB isoenzyme of creatine phosphokinase.” These investigators concluded that the patient had severe hypoxia, and supporting laboratory data “ruled out acute myocardial infarction.”

An equally tenable argument might be that the patient suffered a small myocardial infarction. Exercise-induced myocardial ischemia was not associated with an increase in the MB isoenzyme of creatine phosphokinase, as measured by a very sensitive method, in our laboratory. In another report, neither catecholamine stimulation nor atrial pacing to angina resulted in release of enzyme from the heart. In our laboratory, atrial pacing to angina did not result in an increase in the level of the MB isoenzyme of creatine phosphokinase either in the coronary sinus or systemic circulation. Thus, it seems very unlikely that myocardial ischemia would result in an elevation of the concentration of the isoenzyme of creatine phosphokinase. On the other hand, the MB isoenzyme of creatine phosphokinase is very sensitive, and an increase in the level may reflect acute myocardial infarction even in the presence of a normal level of total creatine phosphokinase. This determination of the level of the isoenzyme is considerably more sensitive to the presence of myocardial necrosis than are serum levels of myoglobin or diagnostic electrocardiographic changes. When properly measured, the level of the MB isoenzyme of creatine phosphokinase may be the most sensitive method currently avail-

![ECG Figure](image)

**Table 1—Levels of Enzymes in 42-Year-Old Man after Direct-Current Shock**

<table>
<thead>
<tr>
<th>Level, international units</th>
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<tbody>
<tr>
<td>Serum Glutamic-Oxaloacetic Transaminase</td>
</tr>
<tr>
<td>Time</td>
</tr>
<tr>
<td>Control</td>
</tr>
<tr>
<td>8 hr</td>
</tr>
<tr>
<td>24 hr</td>
</tr>
<tr>
<td>48 hr</td>
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</tbody>
</table>

**SGOT (IU)**

<table>
<thead>
<tr>
<th>Control</th>
<th>8hrs</th>
<th>24hrs</th>
<th>48hrs</th>
</tr>
</thead>
<tbody>
<tr>
<td>7</td>
<td>6</td>
<td>4</td>
<td>9</td>
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</table>

**CPK-MB (IU)**

<table>
<thead>
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<th>Control</th>
<th>8hrs</th>
<th>24hrs</th>
<th>48hrs</th>
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<tr>
<td>0.3</td>
<td>0.2</td>
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Figure 1. Exercise ECG from 42-year-old man.

124 COMMUNICATIONS TO THE EDITOR

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