Rapid Appearance of Rheumatoid Pleural Effusion*

Kenneth G. Torrington, MAJ, MC

A patient with longstanding rheumatoid arthritis presented with an acute febrile illness and rapidly accumulating pleural effusions. The characteristics of rheumatoid effusions are reviewed and the rapid onset of her rheumatoid effusions is documented. The differential diagnosis of an acute febrile illness associated with a pleural effusion must include rheumatoid pleuritis, especially in the patient with known rheumatoid disease.

Rheumatoid arthritis is a common rheumatologic disease, often associated with systemic manifestations. Pleuropulmonary involvement is well recognized and has been seen to occur in five primary roentgenographic patterns.1 These include (1) pleural disease characterized by pleural effusions; (2) diffuse pulmonary disease characterized by a coarse reticulonodular pattern (chronic fibrosing pneumonitis); (3) large discrete nodules, pathologically similar to subcutaneous nodules; (4) Caplan's syndrome, ie, rheumatoid nodules plus pneumoconiosis; and (5) pulmonary arteritis and hypertension. The rheumatoid pleural effusion is remarkable for its predominant occurrence in men,2 usually of middle age. The effusion may antedate the systemic disease. It is usually unilateral3 and occurs most often on the right side.3 This report describes several unusual features of rheumatoid pleural effusion and, in addition, documents the remarkable rapidity with which the effusion may develop.

CASE REPORT

A 61-year-old white woman with longstanding rheumatoid arthritis was admitted to Walter Reed Army Medical Center on August 10, 1976 with a ten-day history of shaking chills, fever, anorexia, and malaise. Evaluation as an outpatient five days prior to admission had been nondiagnostic, and her chest x-ray film had shown no acute disease. On the date of admission, she had experienced the rather sudden onset of severe pleuritic, right, anterior chest pain which was also exacerbated by palpation. She denied cough or significant shortness of breath.

In 1949, she was hospitalized for one year in a tuberculosis sanitarium for presumed pulmonary tuberculosis. She had never received antituberculosis chemotherapy. For ten years she had had severe, seropositive rheumatoid arthritis. In June, 1976, the rheumatoid factor by Bentonite flocculation was reactive at 1:2048.

Physical examination revealed a thin, debilitated white woman whose respiratory rate was 32 per minute. Her temperature was 101.8°F (38.7°C) orally. Her blood pressure was 106/50 mm Hg and pulse rate was 120 per minute. Examination of the chest revealed generally decreased breath sounds, especially in the right base posteriorly, where there was accompanying dullness to percussion. Multiple jolts revealed classic rheumatoid deformity and synovial thickening, and multiple rheumatoid nodules were present over extensor surfaces. The remainder of the examination was noncontributory. Complete blood count was normal except for a hematocrit of 34.6 percent. Supine chest x-ray film revealed a large right pleural effusion (Fig 1). The results of the three thoracenteses performed appear in Table 1.

On the basis of the characteristics of the pleural fluid, the patient was suspected of having a rheumatoid effusion. She gradually defervesced on treatment with aspirin-Maalox (Ascriptin). Subsequent laboratory reports on the pleural fluid revealed no evidence for tuberculous or septic empyema. By the fourth hospital day, a left pleural effusion had also appeared (Fig 2). Four weeks after admission, small bilateral pleural effusions were all that remained. The patient was clinically stable and left the hospital.

DISCUSSION

Pleuropulmonary disease in rheumatoid arthritis was suggested more than 100 years ago. Bagenstoss and Rosenberg were the first to document rheumatoid pleuritis in 22 of 30 patients in an autopsy series in 1943. It is now generally agreed that pleuritis is the most common manifestation of rheumatoid lung disease.2,5

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Figure 1. Anteroposterior chest roentgenogram taken on August 10, 1976, with the patient supine, demonstrates a right pleural effusion.
Table 1— Characteristics of Pleural Fluid and Concurrent Blood Glucose Determinations

<table>
<thead>
<tr>
<th></th>
<th>August 11</th>
<th>August 12 Following intravenous administration of 500 ml of 10% glucose in water over one hour</th>
<th>August 27</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cell count (eu mm)</td>
<td>600 WBC, 45 RBC</td>
<td>270 WBC, 12,400 RBC</td>
<td>4,300 WBC, no RBCs</td>
</tr>
<tr>
<td>Differential cell count</td>
<td>80% polys, 20% lymphs</td>
<td>92% polys, 8% lymphs</td>
<td>89% polys, 11% lymphs</td>
</tr>
<tr>
<td>Specific gravity</td>
<td>1.008</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total protein</td>
<td>4.6 gm%</td>
<td></td>
<td>5.1 gm%</td>
</tr>
<tr>
<td>LDH</td>
<td>1900 mU/ml</td>
<td>2420 mU/ml</td>
<td>1700 mU/ml</td>
</tr>
<tr>
<td>Pleural fluid glucose</td>
<td>12 mg%</td>
<td>43 mg%</td>
<td>10 mg%</td>
</tr>
<tr>
<td>Blood glucose</td>
<td>95 mg%</td>
<td>124 mg%</td>
<td>93 mg%</td>
</tr>
<tr>
<td>Amylase</td>
<td>92 Units/100 ml</td>
<td>86 Units/100 ml</td>
<td></td>
</tr>
<tr>
<td>Rheumatoid factor</td>
<td>Negative</td>
<td>Negative</td>
<td>Negative</td>
</tr>
<tr>
<td>(Betonite)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gram stain and culture (aerobic and anaerobic)</td>
<td>Negative</td>
<td>Negative</td>
<td>Negative</td>
</tr>
<tr>
<td>Fungal and tuberculous smears and cultures</td>
<td>Negative</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pleural fluid cytology</td>
<td>Acute and chronic inflammation; negative for malignancy</td>
<td></td>
<td>Acute and chronic inflammation; negative for malignancy</td>
</tr>
<tr>
<td>Pleural biopsy</td>
<td>Nonspecific fibrinous pleuritis</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

This patient was presumed to have a rheumatoid pleural effusion on the basis of characteristic pleural fluid findings. Classically, the rheumatoid effusion is described as a greenish-yellow exudate. This appearance is likely due to the presence of cholesterol crystals, which may be noted in longstanding effusions of various etiologies. It is of interest that abnormal systemic cholesterol metabolism has never been associated with a cholesterol-rich pleural effusion. Prior investigators have described yellow pleural fluid, as was seen in this case, and this finding probably reflects the acute nature of the effusion.

The white blood cell count in rheumatoid pleural effusion usually ranges between 1,500 and 5,000 and is composed of a predominance of lymphocytes. The predominance of polymorphonuclear leukocytes in this case, and noted previously by others, probably reflects the acuteness of the process. The pleural fluid LDH is often strikingly elevated in rheumatoid effusions, a finding of significant diagnostic importance in this case as well. The mechanism for the elevation of LDH is not understood.

Pleural fluid glucose has been noted to be strikingly depressed in most patients with rheumatoid effusions. In 1966, Dodson and Hollingsworth demonstrated in two patients that the infusion of 500 ml of 10 percent dextrose in water over one hour failed to raise the glucose of the rheumatoid effusion. The mechanism of action of the low pleural fluid glucose was not proven, although decreased trans-

![Anteroposterior chest roentgenogram taken on August 13, 1976, with the patient erect, shows bilateral pleural effusions. Incidentally noted are loculated right pneumothoraces which represent a complication of thoracentesis with pleural biopsy.](Image)

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port of glucose into the pleural space was postulated. In the reported patient, the pleural fluid glucose rose from 12 mg percent to 43 mg percent following glucose infusion. The significance of this rise cannot be determined since no standard responses to intravenous glucose infusion have ever been set up. In addition, Russakoff et al. found an unpredictable response of pleural fluid glucose following oral glucose tolerance tests in patients with a variety of pleural diseases. In this patient, other well-recognized causes of low pleural fluid glucose such as bacterial empyema, tuberculous empyema, or malignancy were ruled out by subsequent negative smears, cultures, cytology, and microscopic examination.

In the two patients reported by Dodson and Hollingsworth, low pleural fluid rheumatoid factor serology was noted. Other investigators have described a high titer of rheumatoid factor in the pleural fluid similar to that seen in the present case. Many authors have found a high titer of rheumatoid factor in the serum.

Pleural biopsy has been reported to be of help in making the diagnosis of rheumatoid pleuritis. When pleural biopsy yields lesions similar to peripheral rheumatoid nodules, the diagnosis is quite certain. More often, however, the biopsy will show only nonspecific pleuritis, which should not deter the clinician from making the diagnosis of rheumatoid pleuritis. Pleural fluid cytology is helpful only by ruling out the presence of a malignant effusion.

This case report is unusual from several viewpoints: its occurrence in a woman, that it was bilateral, and the acute onset. The most striking aspect is the rapidity with which the pleural effusion appeared. This was documented by the progression from normal chest x-ray film findings on August 6, 1976 to a unilateral right pleural effusion on August 10, 1976 to bilateral effusions on August 13, 1976.

Although Carr and McGurkin described a patient hospitalized with rheumatoid arthritis in whom a pleural effusion developed over the course of a few days, this is the first report of rheumatoid disease as the probable cause of an acute febrile illness associated with acute pleurisy. Rheumatoid pleuropulmonary disease can occur before, during, or after the clinical appearance of rheumatoid arthritis. This report points out the need to consider rheumatoid effusion as part of the differential diagnosis of acute pleurisy with effusion, particularly in the patient with known rheumatoid disease.

REFERENCES

11. Mays EE: Rheumatoid pleuritis: Observations in eight cases and suggestions for making the diagnosis in patients without the typical findings. Dis Chest 53:202, 1968

Ventricular Ectopic Rhythms due to Rapid Runaway Pacemaker*

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A patient initially had syncope due to a runaway pacemaker firing at an unusually rapid rate (30 impulses per second). The ventricular arrhythmia was characterized by numerous ectopic beats, with coupling intervals related to the length of the preceding cycle and runs of ventricular tachycardia with slight variations in the intervals between beats. This case demonstrates the clinical characteristics of a very rapidly firing, low-intensity, ventricular parasystolic focus.

Local myocardial excitation with exit block (parasytole) has been postulated as the underlying mechanism in some patients with ventricular ectopic activity. Initially, parasystole characteristically occurs with ectopic beats whose coupling interval varies; the interectopic intervals are mathematically related to each other, and the parasystolic focus is "protected" from the normally conducted beats so that the ectopic cycle cannot be reset. We have seen a patient with a runaway ventricular pacemaker firing at an unusually rapid rate.

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