A 44-year-old white man clerk was admitted on March 9, 1953, with a six-month history of dyspnea, low grade fever, slight weight loss, and chronic productive cough. Chest roentgenogram revealed right upper lobe infiltration with a cavity, and several sputum concentrates and cultures were heavily positive for tubercle bacilli. Treatment included streptomycin, PAS, and INH. The chest lesion regressed.

All sputum and gastric cultures after April 21, 1953, were negative for tubercle bacilli. Streptomycin, INH, and PAS were discontinued in July, 1954. Serial chest films revealed no change until April, 1956. The patient was entirely asymptomatic, but streptomycin, INH, and PAS were re-instituted in April, 1956, because of marked worsening of the roentgen findings (Fig. 1). He was re-hospitalized on September 25, 1956, with new complaints of dyspnea and 10 pound weight loss. Physical examination was essentially normal.

PPD No. 2 and histoplasmin skin tests were positive, and the coccidiodin was negative. Serologic studies for coccidioidomycosis, histoplasmosis, and blastomycosis...
were repeatedly negative, and numerous sputum and gastric cultures and guinea pig examinations were negative for tubercle bacilli. The roentgen findings did not regress. On May 16, 1957, he developed severe right-sided chest pain, went into shock, and expired within several hours.

**Diagnosis: Pulmonary Alveolar Proteinosis**

At necropsy, the lungs were considerably heavier than normal. Cut surface revealed loss of normal architecture with replacement by grayish-white, firm tissue. There was no significant intrathoracic lymphadenopathy. Microscopic examination revealed many alveoli partially or completely filled with granular amorphous eosinophilic material. Inflammatory cells were conspicuously absent, and no granulomas were seen. The intra-alveolar material was strongly positive to PAS stains. With oil red-O stains, minimal lipid was seen in most alveoli, although some contained greater collections of lipid. The alveolar material failed to stain with mucicarmine stains. Differential stains suggested that the eosinophilic material was probably a mucoprotein.

Special stains for bacteria, parasites, and fungi were negative. Spectrographic studies revealed no evidence of beryllium, cadmium, nickel, zinc, or mercury. Fixed lung tissue was examined for radioactivity, but none was found. Formalin-fixed lung tissue was analyzed and compared to the normal:

<table>
<thead>
<tr>
<th>Per Cent Dry Weight</th>
<th>Normal Lung</th>
<th>Present Case</th>
</tr>
</thead>
<tbody>
<tr>
<td>% Protein</td>
<td>82.4</td>
<td>64.0</td>
</tr>
<tr>
<td>% Lipid</td>
<td>3.4</td>
<td>16.1</td>
</tr>
<tr>
<td>% Carbohydrates</td>
<td>0.9</td>
<td>1.3</td>
</tr>
</tbody>
</table>

As indicated in the above table, the lipid content was high, and much greater than one would suspect from the fat-stained sections.

The classic article by Rosen, Castleman, and Liebow, summarized 27 patients. They noted that PAS-positive granular and floccular proteinaceous material, rich in lipids, was deposited within the lumens of distal air spaces, where little, if any, inflammatory reaction was produced.

Radiographically, the appearance simulates that of pulmonary edema, but does not show rapid clearing. Clinically, there is dyspnea, often with cough, increasing fatigability, and weight loss. Death may occur as a result of progressive filling of alveoli or superimposed infection. The etiology is unknown.

**References**


**Occlusive Juvenile Aortitis**

A clinicopathologic review in an acquired disease of the aorta and its branches in young adults was presented by Dr. A. K. Basu, Calcutta. Twenty-two cases studied between 1953 and 1960 were discussed. Twelve of these were seen during the past two years. It was suggested that the disease is not such a rare entity as is generally believed. The possibility of rheumatic affection as the etiologic agent was suggested. This belief is based partly on the clinical features of the cases (the age, incidence, sex, febrile episodes, occasional joint pains and anemia) or partly on the histologic features. The histology of the affected arteries shows predominantly features of arteritis affecting the medial coat associated with intimal thickening. The nature and intensity of the arteritis is variable. Inflammatory cellular exudate, often focal in distribution and associated with focal disruption, is the most characteristic feature. In most cases, there are changes in the group substance with excess of acid mucopolysaccharide. Presence of hematophytic substance in the media which histochemically is akin to a similar substance associated with rheumatic arteritis was the outstanding feature in two cases. Giant cell arteritis is a very characteristic feature in two cases. The atheromatous changes appeared to be secondary to the medial lesion. The author emphasized that the disease constitutes a progressive lesion and that the process may be rapidly downhill. Once the obstructive factor is established, efforts must be made to remedy it by operation. Fortunately, in most cases this can be accomplished and the patients can be greatly benefited.

Presented at the 7th International Congress on Diseases of the Chest, New Delhi, India, February 20-24, 1963.