Consolidation of Both Upper Lobes*

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A 30-year-old woman was admitted to the intensive care department because of imminent respiratory failure (PO₂, 7.0 kPa, PCO₂, 4.6 kPa). During the preceding two weeks, she had complained of fatigue, headache, thirst, vertigo, and a nonproductive cough with subfebrile temperature. Her medical history was unremarkable except for a history of back pain and proximal arthralgias during the last year.

Physical examination revealed shortness of breath at rest, hypotension, hepatomegaly, and normal breath sounds with expiratory crackles.

Initial laboratory results showed an ESR of 88 mm, leukocytosis (20.6×10⁹/L with 92 percent segments, 4 percent bands), hyponatremia (123 mmol/L), hyperkalemia (6.1 mmol/L), severely elevated urea (58.8 mmol/L) and creatinine (489 μmol/L) values. Calcium value was normal (2.59 mmol/L), whereas phosphate (2.09 mmol/L) and alkaline phosphatase (262 U/L) values were elevated. No liver function abnormalities were found. Urinalysis showed normal sediment, and the excretion of sodium was 4 mmol/L and of creatinine 7.0 mmol/L.

An AP chest roentgenogram showed diffuse symmetrical bilateral shadowing of the upper lung fields (Fig 1).

Because of the presumptive diagnosis of pneumonia with septic shock, prerenal uremia and salt depletion, therapy consisting of fluid supplementation, inotropic drugs, and erythromycin was started. Despite this, the situation deteriorated, and within a few hours, mechanical ventilation was necessary. A second chest roentgenogram showed the same abnormalities in both upper lung fields with signs of pulmonary edema.

Bronchoscopy showed some sanguinous fluid without other abnormalities. Gram-stains and cultures of the bronchial fluid revealed no microorganisms. After further rapid clinical worsening, the antibiotic regimen was extended with aztreonam and rifampicin, and later with penicillin, streptomycin, and doxycyclin. In spite of all these measures, shock was irreversible with signs of capillary leakage, diffuse intravascular coagulation, and anuria. All cultures and autoimmune and viral serologic test results were negative. Four days after admission, she died. An autopsy was performed.

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Diagnosis: Diffuse pulmonary calcification

Post-mortem examination showed conspicuous pathologic changes in the lungs. Both lungs were enlarged and the upper parts of the solid, dry mass were remarkably airless. There was no edema. Microscopic examination of several slides taken from various parts of the lungs showed, apart from deposition of hyalin material, diffuse calcium deposition along the alveolar and vessel walls as confirmed by von Kossa staining, while inflammatory cells or fibrosis were strikingly absent (Fig 2). Calcium deposition was not found in organs other than the lungs. Other pathologic findings during post-mortem examination consisted of marranic endocarditis of the tricuspid and pulmonary valves, hepatic enlargement with steatosis and congestion, and a thrombus in the wall of the right renal vein. Thyroid and parathyroid glands, digestive and urogenital tracts, and the bone marrow were all normal, except for a moderate degree of tubular necrosis.

Diffuse pulmonary calcification was first described by Virchow. Microscopically, it is characterized by calcium deposition in the alveolar and bronchiolar basement membranes. There are two different forms of calcification: the dystrophic and the metastatic form. Roentgenologically, these two forms usually have distinctive patterns. Examples of the dystrophic form are the multiple nodular calcifications which can occur after long-standing elevated left atrial pressure, or in already damaged lung tissue, for instance, after infectious diseases such as varicella, or in combination with silicosis. A second and rare example is interstitial ossification which is characterized by branching shadows of calcific density extending along the bronchovascular distribution of the interstitial space. It has been found in fibrosing alveolitis, after long-term busulfan therapy, and in association with long-term chronic pulmonary congestion.

Microscopically widespread pulmonary calcification secondary to disturbances in the calcium/phosphorus metabolism such as hyperparathyroidism and renal failure, is rather frequently found. This metastatic form of calcification is always associated with calcium deposition in other tissues. However, detection of the calcium in the lungs on the chest roentgenogram is extremely unusual in these patients.

In the reported patient, the pattern of the calcification was comparable to what is found in metastatic calcification. The major difference was the lack of extrapulmonary deposition of calcium. There were no convincing signs of chronic disturbances of the calcium/phosphorus metabolism secondary to hyperparathyroidism, chronic renal disease, or primary bone disease. Also, hypervitaminosis D was very unlikely because the levels of 250H-calciferol and 6,25-dihydroxycalciferol, which were determined afterwards, were within the normal range. The roentgenologic abnormalities are more or less the same as found in a patient reported by Mootz et al. In this patient, extensive pulmonary calcification was the cause of respiratory failure. Although unproven, this might also have been the cause in our patient.

REFERENCES
1 Virchow R. Kalk-metastasen. Virchows Arch Path Anat 1855; 8:103