of the fibroblastic and histiocytic variety. Thus, the demonstration of polymorphonuclear leukocytes in our case strongly supports the diagnosis of acute rheumatic endocarditis.

The late development of mitral insufficiency was undoubtedly a factor in the patient's clinical deterioration. It would appear that the three main factors producing recurrent mitral stenosis, namely: inadequate operations, mitral insufficiency and rheumatic activity may occur in different combinations or may occur together. In our patient, they all probably contributed to the unfavorable result.

REFERENCES
5. SOULIÉ, P., JOLY, F., CARLOTTI, J., and SERVILLE, M.:

Mesothelioma and Tuberculosis Caused by Atypical Mycobacteria*

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A case of mesothelioma associated with pulmonary tuberculosis, caused by atypical organisms, is reported. Both diseases were present at the time of discovery; there was no history of exposure to benzene or irradiation. Antimicrobial therapy had no effect on the mesothelioma, and the mycobacterial disease process showed stability, with no evidence of involvement of the spleen or lymph glands, or terminal military spread. This case, to our knowledge, is the first one in which coexistence of mesothelioma and mycobacteriosis caused by atypical (Group 1) organisms has been reported. Causal relationship between these two diseases could not be established in this patient.

In most cases, the etiology of mesothelioma is unknown, although in some patients, it may be secondary to chronic benzene intoxication, exposure to ionizing radiation, metastatic malignancy of the bone marrow or tuberculosis. More recently, in a few cases, chromosome abnormalities were reported.1 In various reports concerning the association of myeloproliferative disorders with tuberculosis, the organism isolated in sputum, secretions, or tissues, was Mycobacterium tuberculosis. This report records a case of mesothelioma associated with pulmonary mycobacteriosis caused by group 1 (photochromogen) organisms.

CASE REPORT

A 70-year-old retired Negro janitor was transferred on July 25, 1962 to the Excelsior Springs Division of the Veterans Administration Center, Wadsworth, Kan., from another hospital. He had been admitted to the other hospital on March 13, 1962, with a long history of dyspnea, increasing for the past two years, associated with a weight loss of 50 pounds, poor appetite, moderate productive cough, and weakness. Chest x-ray films showed right apical infiltration and a small, fairly well-delineated confluent density at the right subapical region. Tomograms disclosed a small cavity at the subapical region on the right. Sputum smears were positive for acid-fast bacilli, and the cultures yielded photochromogen mycobacteria (Buxton group I). The result of bronchoscopy was negative, and no malignant cells were found in the bronchial aspirations and sputa; group 1 mycobacteria grew on the washings from the right bronchial tree. Antimicrobial therapy, consisting of isoniazid (INH) and para-aminosalicylic acid (PAS), was started on April 23, 1962. White blood cell count (WBC) at that time was 19,000 with many immature granulocytes and three erythroblasts per 100 WBCs; hematocrit reading 34 per cent; hemoglobin level, 10.3 g.

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MYELOFIBROSIS AND TUBERCULOSIS

Figure 1. Infiltration at the right upper lung field.

Figure 2. Vertebral body. Marrow fibrotic with spicules of recently formed bone.

Interstitial pneumonitis, fibrosis, and emphysema of the lungs, myeloid metaplasia involving ribs, vertebrae, right ilium, and left tibia, splenomegaly (1,000 gm), advanced extramedullary hematopoiesis in spleen, liver, and adrenals, and recent coagulation necrosis of the cortices of both adrenals. In the liver, the sinusoids and portal spaces contained numerous immature blood cells, including myelocytes, clusters of erythroblastic cells, and occasional megakaryocytes. Similar cells infiltrated the capsule in one section. In the adrenals, multiple sections showed almost complete obliteration of the cortex by coagulative necrosis. The medulla did not appear necroto, but was heavily infiltrated and replaced by immature blood cells similar to those noted in the liver; such cells also formed nodular infiltrates in the adrenal capsule. Megakaryocytes were fairly numerous. Special stains failed to show fungi or acid-fast organisms. In the spleen, the pulp was infiltrated and expanded by large numbers of immature blood cells similar to those seen in the liver. Sinusoids also contained clusters of similar cells. Scattered foci of hemorrhage and necrosis were noted in the pulp, especially beneath the capsule, surrounded by bands of hemosiderin-laden macrophages. The lymphoid follicles were widely separated, but appeared normal, although inactive. No granulomas were seen.

Comment

The diagnosis of myelofibrosis with myeloid metaplasia and tuberculosis caused by atypical mycobacteria seems unequivocal in the present case. There was no history of exposure to benzene or irradiation. Coexistence of tuberculosis and myeloproliferative disorder, according to André et al,2...
may be due to: (1) mere coincidence; (2) enhancing of tuberculous infection by blood disease; (3) treatment of the hematologic disorder, leading to superimposed tuberculosis; (4) tuberculosis giving rise to the blood condition; or (5) an interplay of the above. The impression of these authors, based upon their own and previously reported cases, was that the combination of the two diseases was something more than fortuitous. It is noteworthy that out of their three cases, one had tuberculosis of the spleen, and another lymph gland tuberculosis. Of three cases of coexistent tuberculosis and myeloproliferative disease reported by Corr et al., one had cervical lymph gland tuberculosis. Borchers et al. reported a patient who was observed without treatment for ten years as a case of chronic myelogenous leukemia before tuberculosis of the spleen was diagnosed. Samuelson et al. found no less than four cases of coexistent cervical lymph gland tuberculosis among ten cases of myelobiosis in Sweden, an apparently significant finding in a country in which both diseases have been relatively rare. The possibility of development of impaired resistance to tuberculous of the lymphoid tissue due to the myelobiotic disease process is brought up by the authors. Specific antimicrobial therapy had a favorable, sometimes dramatic, effect on the coexisting myeloproliferative disease in some cases; in most others, no such improvement was noted, although the tuberculous disease process responded well to the treatment. No spleen or lymph gland tuberculosis was noted in our case, although M. kansasi is not infre-