Yellow Nail Syndrome Associated with Chronic Pericardial Effusion

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A 74-year-old man had typical yellow nail syndrome, associated with pericardial effusion and hyperimmunoglobulinemia (IgG 3,179 mg/dl, IgM 402 mg/dl). A search of the literature fails to reveal a previously reported case of yellow nail syndrome associated with pericardial effusion.

Since Samman and White first described yellow nail syndrome (YNS) in 1964, YNS has been characterized by the triad of nail dystrophy, lymphedema, and exudative pleural effusion. Hypoplasia or aplasia of the lymphatic system is thought to be a possible etiology, but its true etiology remains obscure. In the present communication, we describe the first case of a patient with YNS associated with refractory pericardial effusion.

CASE REPORT

A 74-year-old man consulted a physician for shortness of breath on exertion, nocturnal orthopnea, and pretibial pitting edema. These symptoms first appeared four years earlier and gradually became worse. He had also noticed yellow discoloration of all his nails at the age of 69. At that time it was noted that he had bilateral pleural and pericardial effusions, but no definite diagnosis could be made.

Two months later, he was referred to our hospital for evaluation of increased bilateral pleural and pericardial effusions. He had smoked one pack of cigarettes daily for 50 years. There was no family history of similar symptoms or findings. Physical examination revealed marked swelling of the face, ankles, and pitting edema of lower portions of his legs. The pulse was 70 beats/min and without paradox. Some nails of his fingers and toes were yellow, unevenly pigmented, thickened and showed partial onycholysis (Fig 1). These affected nails grew very slowly during hospitalization (0.21 mm/week).

The serum levels of IgG and IgM were remarkably high (3,179 and 402 mg/dl, respectively). Other laboratory data gave normal results and included ANF, RA, and LE tests and anti-DNA. The chest roentgenogram revealed bilateral pleural effusion. The CT scan of the chest showed both pleural and pericardial effusions (Fig 2). Pulmonary function studies showed a mild combined restrictive and obstructive pattern (vital capacity 50 percent, and the forced expiratory volume in one second, 61 percent of predicted). The bronchoscopic examination did not disclose an endobronchial lesion. Analysis of pleural fluid showed it was an exudate with the protein concentration of 4.4 g/dl, glucose level of 130 mg/dl, and lactic dehydrogenase level of 258 units/dl. Cytologic examination revealed that the fluid was predominantly lymphocytic and negative for malignant cells. Routine culture did not show predominant species of bacteria. A pleural biopsy specimen showed chronic fibrous pleuritis. Perfusion lung scintigraphy and lymphangiogram were performed, but no abnormalities were noted.

Right heart catheterization was performed. Fluoroscopy revealed no calcification of the pericardium. Pressure values in the right heart and pulmonary artery wedge pressure were normal, and there was no tamponade nor effusive constrictive pericarditis. So far, no treatment has been effective in controlling pericardial or pleural effusion.

DISCUSSION

Yellow nail syndrome is not a common disease. About 100 cases have been reported in the literature. The many manifestations associated with this syndrome are well-known. Our patient disclosed high levels of IgG and IgM. These immunologic alterations have been found in a few YNS patients associated with acquired immunodeficiency disease. Others showed various types of immunodeficiencies, notably hypogammaglobulinemia, absence of IgA, and decreased level of IgM. A series of negative findings from laboratory

FIGURE 1. Yellow discoloration of distal one third of fingernails.

FIGURE 2. Whole-body CT scan reveals pericardial and bilateral pleural effusions.

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Yellow Nail Syndrome (Wakasa et al)
tests in our patient excluded the possibility of collagen disease or infections.

It is of interest that pericardial effusion in addition to pleural effusion was found. In some reported cases, measurement of the turnover rate of pleural fluid revealed defective lymphatic drainage. Impaired lymphatic drainage due to hypoplasia or aplasia of the lymphatic system is thought to be a possible etiology of pleural effusion. However, no lymphatic abnormality was found in our patient. The right heart catheterization revealed neither tamponade nor effusive constrictive pericarditis. Thus, we could not find the cause of the pericardial effusion. Pericardial involvement in the YNS has received little attention. We suggest that YNS might be one of the diseases to cause pericardial effusion and should not be missed. Therefore, patients with YNS should be observed more closely and frequently by ultrasound and/or CT scan.

REFERENCES
1 Samman PD, White WF. The "yellow nail" syndrome. Br J Dermatol 1964; 76:153-57

Pulmonary Hemorrhage and Air Embolism Complicating Transbronchial Biopsy in Pulmonary Amyloidosis*

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We describe a fatal complication of transbronchial biopsy in a patient with pulmonary parenchymal amyloidosis. Hemorrhage after biopsy required intubation and positive-pressure ventilation that resulted in massive arterial air embolism. Postmortem findings suggested that the bleeding and air embolism were related to persistent patency of biopsied blood vessels infiltrated with amyloid. Patients with pulmonary amyloidosis may be at increased risk of major complications after transbronchial biopsy.

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Figure 1. Chest roentgenogram showing both parenchymal nodular and alveolar-septal deposition of amyloid.

Amyloidosis localized to the lower respiratory tract is a rare condition that is usually diagnosed by either an open lung biopsy or at autopsy. Recent isolated case reports indicate that transbronchial and percutaneous needle aspiration biopsies can provide sufficient specimens for accurate diagnosis with negligible risks; however, closed biopsy procedures of other organs infiltrated with amyloid are reported to have a high frequency of hemorrhagic complications. We report a fatal complication of pulmonary hemorrhage and arterial air embolism following transbronchial biopsy in a patient with unsuspected pulmonary amyloidosis.

Case Report
An 89-year-old black male farmer presented with a ten-year history of progressive exertional dyspnea. He smoked one pack of cigarettes per day for 69 years, stopping ten months before presentation when he could walk only 20 feet before stopping with shortness of breath. He denied cough, fever, hemoptysis, and weight loss.

Physical examination revealed diffuse inspiratory crackles on auscultation of the chest. The findings from the remainder of his examination were normal. Laboratory findings included arterial blood gas levels on room air as follows: pH, 7.42; arterial carbon dioxide tension, 39 mm Hg; and arterial oxygen pressure, 56 mm Hg. Other data were as follows: hemoglobin, 13.2 g/dl; white blood cell count, 4,300/cu mm with a normal differential; platelet count, 418,000/cu mm; prothrombin time, 12.8 seconds, with a control value of 11.5 seconds; partial thromboplastin time, 28.2 seconds, with a control value of 25 seconds; serum total protein, 8.3 g/dl; and albumin, 3.0 g/dl. Serum electrolyte levels, blood urea nitrogen level, creatinine level, hepatic function profile, and urinary sediment were normal. A test with 5 tuberculin units of purified protein derivative of tuberculin was nonreactive.

The chest roentgenogram (Fig 1) revealed diffuse infiltrates with a mixed acinar and reticulonodular pattern involving all lobes, but less prominent in the apices. A bronchoscopic procedure with transbronchial biopsies under fluoroscopy was performed with topical anesthesia. The findings from examination of the airway were normal. The bronchoscope was wedged in the right lower lobe. Brisk hemorrhage following the second transbronchial biopsy was unresponsive to intrabronchial epinephrine. Despite attempts at local tamponade with the bronchoscope, rapid airway hemorrhage neces-