Primary Pulmonary Disease Due to Mycobacterium Avium-Intracellulare

*From the Division of Pulmonary Medicine, Bess Kaiser Medical Center, and Center for Health Research, Kaiser Permanente, Northwest Region, Portland, Oregon.

Reprint requests: Dr. Reich, 3414 North Kaiser Center Drive, Portland, Oregon 97227

Primary pulmonary disease due to Mycobacterium avium-intracellulare (MAI) presented in an immunologically intact child exposed to pet birds. (Chest 1992; 101:1447-48)

The rarity with which MAI leads to primary pulmonary disease in humans is one of the more puzzling aspects of its peculiar epidemiology; only three cases, all involving children, have been reported. In a comprehensive review of the subject of NTM, this pattern was not cited, and in a review of the roentgenographic features of NTM, absence of the primary pattern was listed (along with absence of pleural effusion) as one of the two roentgenographic attributes of MAI which differentiated it from tuberculosis.

**CASE REPORT**

A three-year-old girl, previously in good health, presented in 1990 with complaints of a nonproductive cough and fever of one month's duration. A CR showed atelectasis of the right lower and middle lobe and left hilar adenopathy; the right hilum was obscured by the atelectatic shadows (Fig 1). Complete blood count results were normal; the absolute lymphocyte count was 4,000/cu mm. Primary tuberculosis was suspected on clinical and roentgenographic grounds. A Mantoux test showed no induration to 5 TU at 48 h. The hilar adenopathy diminished over time.

Bronchoscopy revealed extrinsic compression of the bronchus intermedius from the mediastinal aspect and overlying friable ephthematous mucosa. Bronchial washings were negative on smear and culture for MB. At thoracotomy, the two atelectatic lobes were removed along with specimens of enlarged right hilar lymph nodes. The lung and lymph nodes exhibited numerous confluent caseating granulomata. Stains to demonstrate MB and fungi were negative.

**FIGURE 1.** Atelectasis of middle and lower lobe and contralateral hilar adenopathy are evident.
Pleural Effusion Resulting from Metastatic Papillary Carcinoma of the Thyroid*

Andrew N. Vernon, M.D.; Leslie R. Sheeler, M.D.;
Charles V. Bisconti, M.D.; and James K. Stoller, M.D., F.C.C.P.

We present a patient with a symptomatic pleural effusion resulting from papillary carcinoma of the thyroid metastatic to the pleura. Distinctive features include clinically evident pleural metastasis and the presence of psammoma bodies in the pleural fluid, which are characteristic (albeit not pathognomonic) of papillary carcinoma of the thyroid.

(Chest 1992; 101:1449-50)

Although papillary carcinoma of the thyroid generally follows an indolent course characterized by slow growth and the absence of distant metastases, several available reports suggest that metastasis to bone and/or lung parenchyma may occur infrequently.1,2 While pleural metastases have been reported in necropsy series, the clinical occurrence during life of pleural effusions due to metastatic papillary carcinoma of the thyroid has not been recognized.

To extend the clinical and pathologic spectrum of metastatic papillary carcinoma of the thyroid, the present case report is of a patient with dyspnea due to a massive pleural effusion caused by metastatic papillary carcinoma of the thyroid. A distinctive cytologic feature of the pleural fluid is the presence of psammoma bodies, which, to our knowledge, has not been described previously.

CASE REPORT

A 50-year-old woman presented to her local physician with a one-week history of increasing shortness of breath. Associated symptoms included thick yellow sputum, occasional wheezing, paroxysmal nocturnal dyspnea, and two-pillow orthopnea. A chest roentgenogram revealed a large free-flowing right-sided pleural effusion, and the patient was referred to the Cleveland Clinic Department of Pulmonary Disease for further examination.

*From the Departments of Pulmonary Disease (Drs. Vernon and Stoller), Endocrinology (Dr. Sheeler), and Pathology (Dr. Bisconti), Cleveland Clinic Foundation, Cleveland.
†Fellow, Department of Pulmonary Disease.
Reprint requests: Dr. Stoller, Cleveland Clinic Foundation, 9500 Euclid, Cleveland 44195

FIGURE 1. The papillae are lined by tall columnar cells with faintly staining cytoplasm and predominantly basal large nuclei (hematoxylin-eosin, original magnification ×125).

REFERENCES