Thrombocytosis in Chronic Eosinophilic Pneumonia

Meir Brezis, M.D.;** and Joel Lafair, M.D.†

Thrombocytosis has not yet been described in chronic eosinophilic pneumonia. A typical patient with chronic eosinophilic pneumonia is described. The patient's thrombocyte count was up to 900,000 per cu mm, which returned to normal under steroid therapy. Thrombocytosis may be associated with chronic inflammation and should not require further investigations such as lung biopsy, in an otherwise usual case of chronic eosinophilic pneumonia.

Persistent marked thrombocytosis has been associated with various myeloproliferative disorders, malignancy, and chronic inflammatory diseases. A reactive thrombocytosis, generally of less than 700,000/ cu mm, has been reported in chronic or slowly resolving pneumonitis. Thrombocytosis has neither been described in association with pulmonary eosinophilia nor in the 35 cases of chronic eosinophilic pneumonia (CEP) thus far reported in the literature. The present report suggests that such an association may exist.

Case Report

A 39-year-old man was hospitalized because of cough, dyspnea, fever, night sweats, and weight loss that had increased in severity for several months prior to admission. The patient appeared to be in mild respiratory distress breathing 20 breaths per minute; his temperature was 38.5°C (101.3°F). Some scattered rales and bronchial breath sounds were heard in the left axillary area.

The chest film demonstrated dense infiltrates scattered in both lungs which were markedly increased over the next few days (Fig 1). The erythrocyte sedimentation rate was 100 mm/hr. Hemoglobin was 11 gm percent, white blood cell count 10,300/cu mm, with 40 percent eosinophils with a total eosinophilic count varying from 3,500 to 4,500/cu mm. The sputum was loaded with eosinophils. The IGE was 2,600 ng/ml (normal < 300). The thrombocyte count was 900,000/cu mm. Bleeding time and clot retraction were within normal range. The bone marrow displayed marked eosinophilia and slight plasmacytosis. Liver biopsy disclosed mild eosinophilic triaditis. Several sputum cultures grew C albicans, but precipitins for neither this nor A fumigatus could be demonstrated in multiple serologic studies.

Percutaneous lung biopsy demonstrated a dense infiltration by histiocytes, plasma cells, lymphocytes and numerous eosinophils. Immunofluorescence showed the presence of IGE.

After the administration of prednisone, 40 mg daily, the pulmonary infiltrates cleared completely within a few days. Concomitantly, eosinophil as well as thrombocyte counts rapidly returned to normal range.

During a 30-month follow-up period, the patient has remained disease-free and thrombocytopathy is now stable. Our patient exhibits the expected spectrum of defects of cardiac conduction, fibrosis and degeneration of the connective tissue; to our knowledge, there are no previous reports in the English literature of atrial standstill attributed to systemic lupus erythematosus. Our patient manifests both atrial standstill, with an inability to pace the atrium, and also complete heart block. It is reasonable to speculate that the patient has diffuse atrial disease and disease of the conduction-bundle system secondary to recurrent systemic lupus erythematosus, pericarditis, myocarditis, and myocardial arteritis. Over the past five years the patient has been treated with prednisone and implantation of a permanent pacemaker, and his condition is now stable. Our patient exhibits the expected spectrum of defects of cardiac conduction (sinus nodal arrest, bradycardia-tachycardia syndrome, fascicular block, asystole, and atrial standstill) in systemic lupus erythematosus theoretically possible on the basis of the pathophysiology of pericarditis, cardiomyopathy, or abnormalities of connective tissue.

References


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During a 30-month follow-up period, the patient has re-
mained asymptomatic on the alternate-day regimen of 20 mg prednisone. Several attempts to reduce steroid dose further have been followed by relapse.

**DISCUSSION**

Greater familiarity with chronic eosinophilic pneumonia\(^4,5\) today permits a diagnosis of the condition simply based upon a typical clinical and radiologic picture. The etiology remains unknown and the present report cannot confirm *C albicans* as a causative allergen as has been suggested.\(^6\)

The presently advocated policy is to perform lung biopsy only in the atypical case where some uncertainty exists despite thorough clinical evaluation.\(^4,7,8\) Thrombocytopenia, for instance, may be a clue to a hematologic malignancy presenting with pulmonary eosinophilia, as was recently seen in our institution.\(^9\) Likewise, thrombocytosis is associated\(^1\) with conditions that may present with pulmonary eosinophilia such as lymphomas, carcinomas, collagen diseases, tuberculosis and sarcoidosis.\(^2\) Marked thrombocytosis in CEP seems to require further investigation such as lung biopsy. It is important, therefore, to recognize that such an association is not necessarily a clue to another lung biopsy. The thrombocytosis may be reactive to the chronic inflammation present in CEP and will disappear after the appropriate corticosteroid therapy.

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**Co-existing Aortic Stenosis and Secondary Hypertrophic Cardiomyopathy Manifested by an Hourglass Left Ventricle* **

**Successful Treatment with Verapamil**

Yonathan Hasin, M.D.; Joe Leon Rod, M.B., B.Ch., and Gideon Friedman, M.D.

A patient with moderate aortic stenosis had severe hypertrophy and a typical hourglass appearance of the left ventricle. His effort-induced angina and dyspnea responded to treatment with verapamil. We suggest that the aortic stenosis resulted in secondary hypertrophic cardiomyopathy which may be treated by calcium antagonists.

Asymmetric left ventricular hypertrophy may occur in patients with increased afterload\(^1\) or fixed LV outflow tract obstruction.\(^2-5\) It has led to speculation about the existence of a secondary type of hypertrophic cardiomyopathy.\(^2,4\) The recent description of obstruction at the midventricular level (the hourglass left ventricle) in hypertrophic cardiomyopathy\(^6-8\) has added a further anatomic pattern to the obstructive form of hypertrophic cardiomyopathy. Successful treatment of hypertrophic cardiomyopathy by calcium antagonists have been described.\(^9\)

This report describes the co-existence of severe left ventricular hypertrophy manifesting a typical hourglass appearance with moderate valvular aortic stenosis, treated with verapamil.

**CASE REPORT**

A 60-year-old man presented with prolonged chest pain.

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