some of the diagnoses that should be entertained when a physician encounters a patient with the triad of airway obstruction, eosinophilia, and systemic disease. While some patients may require lung biopsy, diagnosis can often be made by clinical criteria alone. It is best to tailor the workup for each patient on an individual basis, but a workup to establish the diagnosis with certainty is usually appropriate.

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Epoprostenol for Treatment of Pulmonary Hypertension in Patients With Systemic Lupus Erythematosus

To the Editor:

As reported by Robbins et al (January 2000), although IV epoprostenol therapy improves symptoms, exercise tolerance, and hemodynamics in patients with pulmonary hypertension associated with systemic lupus erythematosus (SLE), the overall improvement may not necessarily be as great as that seen with patients who have pulmonary hypertension without associated collagen vascular disorders. In addition, although thrombocytopenia has been reported with long-term epoprostenol therapy, the degree to which this is attributable to the epoprostenol therapy as opposed to other etiologies is unclear. Other causes of thrombocytopenia in pulmonary hypertension patients may include the following: pulmonary vascular sequestration due to the underlying pulmonary vascular disease; leukopenia with portal hypertension and splenic sequestration; an autoimmune-mediated “idiopathic thrombocytopenia purpura (ITP)-like” syndrome; and as a consequence of the presence of antiphospholipid antibodies. Furthermore, the degree to which epoprostenol therapy may exacerbate the predisposition for thrombocytopenia in patients with pulmonary hypertension associated with SLE is unknown.

In reviewing our experience with six pulmonary hypertension patients with a history of SLE who were treated with long-term epoprostenol, four of the six patients (who all subsequently died receiving long-term epoprostenol treatment) also had life-threatening thrombocytopenia despite other aspects of their lupus considered quiescent (Table 1). Three of these four patients required long-term aggressive steroid therapy as well as repeated administration of IV γ-globulin in an attempt to improve their thrombocytopenia. Despite a previously documented “ITP-like” syndrome requiring treatment prior to institution of long-term epoprostenol complicating the picture in two of these patients, with treatment for the ITP-like syndrome their platelet counts were maintained >75,000 10^9/L prior to starting long-term epoprostenol therapy.

The most recent death occurred in patient 4 following splenectomy performed for refractory thrombocytopenia (platelet count, 10,000 to 20,000/mm^3) despite prednisone, 60 mg qd, repeated pulse-dose IV steroids, and IV γ-globulin. Because she had no manifestations of active lupus, she was not treated with cyclophosphamide; consideration had also been given to vincristine for refractory thrombocytopenia. Although she had clinically improved from a pulmonary hypertension standpoint with long-term epoprostenol use, she had not improved hemodynamically (by repeat cardiac catheterization at 12 months and 19 months). In addition, she unfortunately was not a candidate for lung transplantation due to her progressive thrombocytopenia. Splenectomy was therefore performed. She was treated perioperatively with inotropic support, low-dose vasopressin, and inhaled nitric oxide. She had no surgical bleeding complications, although her platelet count only responded to perioperative platelet transfusions. She died 6 days postoperatively with an acute pulmonary hypertensive crisis. At autopsy, there was evidence of diffuse microemboli in her lungs only, without an inflammatory component (Fig 1). The surgically removed spleen was remarkable and without evidence of platelet sequestration.

This case (patient 4) raises the possibility that the localization of platelet destruction in SLE patients with pulmonary hypertension is the pulmonary vascular bed, suggesting the need to strongly consider lung transplantation despite its high risk in patients with thrombocytopenia, as opposed to consideration of palliative splenectomy. We remain concerned that long-term epoprostenol use may exacerbate thrombocytopenia in this high-risk patient population, which may prove fatal. Overall, this adverse effect of epoprostenol therapy (eg, thrombocytopenia), may outweigh the potential benefits of epoprostenol treatment in some pulmonary hypertension patients with a history of SLE. Serial reevaluations of risk/benefit considerations for therapeutic options are needed for all pulmonary hyper-

FIGURE 1. Organized pulmonary thrombus (top; original × 100) and high-power magnification of small vessel thrombus with platelet consumption (bottom; original × 200).
In the July 2000 issue, the article “Beliefs Among Pulmonologists and Thoracic Surgeons in the Therapeutic Approach to Non-small Cell Lung Cancer” (CHEST 2000; 118:129–137), by Schroen and co-workers, the abstract contained several typographical errors in percentages due to misplaced decimals. Specifically, the differences in lung cancer survival expectations should be as follows: between pulmonologists and cardiothoracic surgeons, 22% vs 10%; between physicians trained before 1980 and after 1980, 29% vs 10%; between physicians seeing 10 lung cancer patients annually and those seeing 25, 57% vs 77%; and between underestimated and correctly estimated survival in early-stage disease, 58% vs 72%. The percentages were shown correctly in the text of the article.