gery. Antituberculous treatment comprising rifampicin, isoniazid, ethambutol, and pyrazinamide was started. Three months later, he developed a rapid, progressive oliguric renal insufficiency in his right kidney (serum creatinine, 500 μmol/L). Right percutaneous nephrostomy and ureteral catheterization using double J stents were performed, without renal function improvement. He was then referred to the nephrology department. Laboratory findings showed a C-reactive protein level of 133 mg/L, a serum creatinine level of 550 μmol/L, proteinuria of 2.5 g/d, and microscopic hematuria. A transjugular kidney biopsy specimen disclosed a pauci-immune crescentic glomerulonephritis with necrotizing vasculitis. He had also developed anemia and dyspnea. Thoracic computed tomographic scan and bronchic endoscopy evaluation showed an alveolar hemorrhage. A diagnosis of Wegener’s granulomatosis was made. Cytoplasmic antineutrophil cytoplasm antibodies to proteinase 3 were detected in a titer of 1:320. The patient responded to antituberculous therapy and cyclophosphamide pulse of 1000 mg, relayed by prednisone (1 mg/kg/d) associated with a monthly cyclophosphamide pulse of 1 g/d, and monthly intravenous pulse cyclophosphamide therapy (2–6,8,9) (Table). However, a case of Wegener’s granulomatosis that responded to antituberculous therapy has been reported (10), suggesting that antituberculous drugs such as trimethoprim-sulfamethoxazole may eliminate causal or precipitating agents in Wegener’s granulomatosis.

Awareness that retroperitoneal fibrosis can be the sole preliminary symptom of Wegener’s granulomatosis can prevent misdiagnosis, and hence improve therapeutic approaches.

Hassane Izzedine, MD
Aude Servais, MD
Vincent Launay-Vacher, PharmD
Gilbert Deray, MD
Department of Nephrology,
Pitié Salpêtrière Hospital,
47-83 Boulevard de l’Hôpital,
75013 Paris, France


**RECENT-ONSET TUBERCULOUS PLEURISY PRESENTING AS PSEUDOCHYLOTHORAX**

**To the Editor:**

Pseudochyllothorax is an uncommon high-lipid pleural effusion that usually arises from chronic pleurisy and requires several years to develop. Tuberculosis is the most frequent etiology. A rapid course is infrequent and has been described in association with rare infections. We report a patient without a previous history of tuberculosis who presented with pseudochyllothorax of less than 8 months’ development.

A 72-year-old man presented in January 2000 with 4 months of progressive dyspnea on exertion. He smoked, had a history of chronic obstructive lung disease, and had undergone laryngectomy in 1990. He denied having had tuberculosis or contact with *Mycobacterium tuberculosis*. A routine radiograph in January 1999 had revealed a pseudonodular image in the left thorax compatible with nipple shadow (Figure, left), and a control radiograph in April 1999 had showed minimal right pleural effusion (Figure, center).

On admission, diminished breathing sounds were apparent over the right lung. The purified protein derivative was negative. Blood chemical values were normal, except for an erythrocyte sedimentation rate of 99 mm/h. Chest radiograph (Figure, right) and computerized tomography confirmed the presence of a large pleural effusion without pleural thickness. Thoracentesis resulted in a turbid fluid, with 600 x 10⁶ leukocytes/L, 90% lymphocytes; total protein 45 g/L; lactate dehydrogenase 346 U/L; glucose 78 mg/dL; and adenosine deaminase 16.9 U/L (reference
A large amount of cholesterol crystals were observed on microscopic examination. A fiberoptic bronchoscopy was normal. Gram and Ziehl-Neelsen stains, bacteriologic and Lowenstein cultures, and cytology analysis of sputum, pleural fluid, and bronchoalveolar lavage were negative.

Thoracoscopy-guided multiple pleural biopsies, with drainage of 2300 mL of fluid, were performed. All biopsy specimens were nonspecific and showed dense fibrosis, histiocytic inflammation, and cholesterol crystals. A nucleic acid amplification test of pleural fluid (Amplified Mycobacterium Tuberculosis Direct Test, Gen-Probe, San Diego, California) was positive, with a value of 1,777,499 RLU (positive >30,000). Specific treatment for tuberculosis was initiated.

Pseudochylothorax is defined by a cholesterol level >200 mg/dL or the presence of cholesterol crystals (1,2). The pathogenesis of pseudochylothorax is not known. Until 1999, less than 200 cases of pseudochylothorax had been reported in the literature (2). Most patients have long-standing pleural effusion and thickened pleura. The etiology is tuberculosis in 54% of cases and rheumatoid arthritis in 9% (2). A past history of pleuropulmonary tuberculosis, with or without previous drug or collapse therapy, is common in tuberculous pseudochylothorax. Only 9% of patients with pseudochylothorax developed in pleural effusion of less than 5 years’ duration (2); the most frequent cause was parasitic infection (3,4), followed by rheumatoid arthritis (5) and carcinoma of the lung (6). Only one case of tuberculous pseudochylothorax developing in a pleural effusion of less than 5 years’ evolution has been reported (7).

The diagnosis and therapeutic decision may be difficult in tuberculous pseudochylothorax. In the review of the literature by Garcia-Zamalloa et al. (2), Ziehl-Neelsen stain was positive in only two cases, levels of adenosine deaminase correlated with infectious activity in 40% of those tested, and M. tuberculosis was isolated in 14% (11% of pleural fluid cultures and 17% of pleural biopsy cultures). Antituberculous chemotherapy is recommended for patients with pseudochylothorax in which M. tuberculosis is isolated, in culture-negative patients with a history of past tuberculosis, and in purified protein derivative–positive patients with...
progressive effusions (2). The nucleic acid amplification test of pleural fluid, which has a specificity of 100% and a sensitivity of 83% to 87% for organic fluid specimens (8), may be useful in the diagnosis of tuberculous pleurisy and in selecting appropriate treatment for cases of pseudochylothorax in which tuberculous origin is suspected but none of the current indications for treatment are met.

Physicians should be aware that M. tuberculosis may cause rapid-onset pseudochylothorax, even if a previous history of pleuropulmonary tuberculosis is not reported.

Carmen Nogueras, MD
Manuel Montenegro, MD
Maria Vila, MD
Angeles Cabezuelo, MD
Dolores Mariscal, MD
Eugenio Berlanga, MD
Corporació Sanitària Parc Taulí
Sabadell, Spain

PARTIAL ANOMALOUS PULMONARY VENOUS DRAINAGE OF THE SUPERIOR LEFT PULMONARY VEIN INTO THE INNOMINATE VEIN RESULTING IN RIGHT VENTRICULAR FAILURE

To the Editor:
Partial anomalous pulmonary venous return is a congenital defect in which one or more, but not all, of the pulmonary veins fail to drain into the left atrium. We report a patient who presented with progressive right ventricular dysfunction due to a longstanding left-to-right shunt secondary to an anomalous superior left pulmonary vein draining into the innominate vein.

A 49-year-old man with no cardiovascular risk factors was referred to our clinic for progressive exertional dyspnea of 6 years. During the past 3 months, he had worsening shortness of breath, two-pillow orthopnea, and increasing lower-extremity edema. His 12-lead electrocardiogram showed normal sinus rhythm and findings consistent with right ventricular hypertrophy. He had a blood pressure of 130/68 mm Hg, a pulse rate of 92 beats per minute, and a temperature of 97.8°F. His jugular venous pressure was 8 cm. He had a regular rate and rhythm. There was a right ventricular heave without thrills. His S1 and S2 were normal, without extra filling sounds. He had a grade 2/4 holosystolic murmur at the left lower sternal border, which increased with inspiration. His lungs were clear to auscultation. He had full and equal pulses bilaterally, without bipedal edema.

A two-dimensional transthoracic echocardiogram showed normal left ventricular function. The right ventricle was moderately dilated and hypertrophied with moderately reduced systolic function. There was mild-to-moderate tricuspid regurgitation with an estimated peak pulmonary artery systolic pressure of 30 to 40 mm Hg. A transesophageal echocardiogram revealed no evidence of an intracardiac shunt with agitated saline or color-flow Doppler. The left superior pulmonary vein was not well visualized.

The patient subsequently underwent right and left cardiac catheterization, which revealed the following: right atrial mean pressure, 6 mm Hg; right ventricular pressure, 30/14 mm Hg; pulmonary arterial pressure, 20/8 mm Hg (mean, 14 mm Hg); left ventricular pressure, 98/15 mm Hg; and central aortic pressure, 94/46 mm Hg. Selective injection into his left innominate venous system revealed a partial anomalous pulmonary vein (Figure). Oximetry demonstrated an inferior vena cava saturation of 78.1%, right innominate vein saturation of 75.4%, right atrial saturation of 86.5%, right ventricular saturation of 81.4% at the apex, left pulmonary arterial saturation of 82.0%, and systemic arterial saturation of 96.0%. He had a saturation of 97.0% at the junction of the left innominate vein and the anomalous pulmonary vein. His Qp/Qs ratio was 1.4:1.0. His coronary angiogram showed no obstructive lesions. The patient underwent surgical rerouting of the partial anomalous superior pulmonary vein from the innominate vein to the left atrial appendage, with resolution of his symptoms postoperatively.

Anomalous pulmonary venous return is an important diagnosis that is often overlooked in routine clinical practice. In this condition, veins draining an entire lung or specific lobes flow into the innominate vein, right atrium, coronary sinus, superior vena cava, inferior vena cava, or ayzyous vein, rather than the left atrium (1,2). Due to the redirection of blood flow, a left-to-right shunt may result, depending on its duration and mag-