

Letters to the Editor

**Pleural and Pericardial Effusion in a Patient
With Polymyalgia Rheumatica[☆]**

Derrame pleural y pericárdico en una paciente con polimialgia reumática

To the Editor:

Rheumatic polymyalgia (RPM) is a relatively common inflammatory disease of unknown origin that presents almost exclusively in adults over the age of 50. It is characterized by morning pain and stiffness in the cervical region and in the pectoral and pelvic girdles and may be associated with giant cell arteritis (GCA).¹ Pleural or pericardial effusion is rare in patients with RPM without GCA and is exceptional as a manifestation of disease onset.²

The case of a 76-year-old woman who presented in the emergency room with fever (38 °C) is reported. She had had episodes of fever and pain in the pectoral girdle radiating to the cervical region over the previous 2 months. On one occasion, she had received oral corticosteroids in tapering schedule, with improvement. Physical examination revealed a systolic murmur in the aortic region and absent breath sounds in the base of the left lung. Clinical laboratory tests showed C-reactive protein 25.5 mg/dl and a small left pleural effusion was observed on the chest X-ray. The initial diagnosis was pneumonia with parapneumonic pleural effusion and the patient was hospitalized with empiric antibiotic therapy. Despite

this, her fever persisted and further examinations were performed. The immunological study revealed positive antinuclear antibodies in a speckled pattern. Tumor marker analysis showed elevated CA-125 levels. The chest-abdominal computed tomography showed a small left pleural effusion and minimal pericardial effusion (Fig. 1). After consultation with the rheumatologist, a diagnosis of RPM with serosal involvement was made and treatment with prednisone (starting dose: 15 mg/day) was initiated. Subsequent clinical course was favorable with resolution of fever and pleural effusion. Three months after discharge the patient remained asymptomatic, so the corticosteroid doses were progressively reduced until discontinuation. The patient has since remained asymptomatic, with no new outbreaks of RPM over a 2-year follow-up period.

Although the elevated CA-125 antigen levels detected in this study may have been caused by other processes, such as Meigs pseudosyndrome secondary to struma ovarii, it was finally interpreted as indicative of serosal involvement. In a review of the literature, only 5 cases of RPM with associated pericardial effusion were identified, of which three also presented pleural effusion. Thoracentesis was performed in one of these cases. The resulting fluid was borderline transudate/exudate, and cytology was negative for malignancy. In another case, pericardiectomy with pericardial biopsy was performed. Analysis of the pericardial fluid showed proteins 5.2 g/dl, lactate dehydrogenase 4562 IU/l and glucose 65 mg/dl. Histological examination found inflammation with fibrosis and areas of interstitial bleeding with fibrin deposits, and

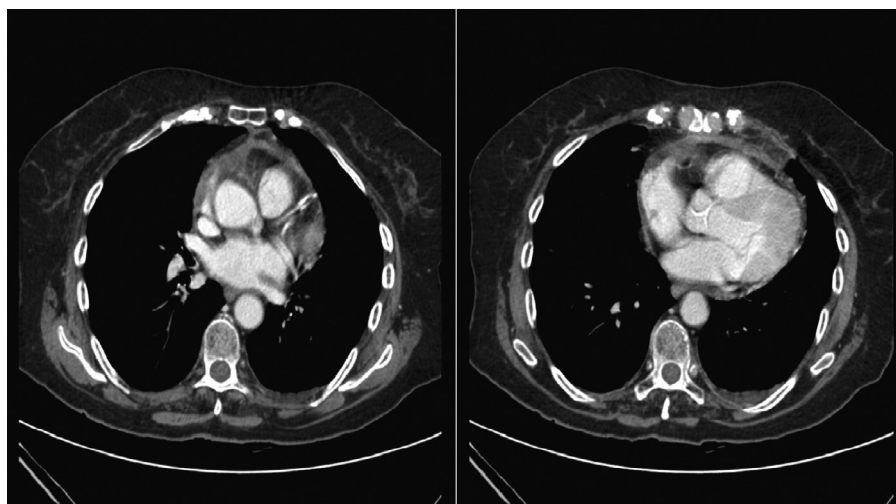


Fig. 1. Chest computed tomography showing small left pleural effusion and minimal pericardial effusion.

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the immunohistochemical analysis revealed lymphocytic infiltrate with predominance of T-cells.³

In conclusion, RPM should be considered in the differential diagnosis of patients over 50 years of age with pleural and/or pericardial effusion.^{4,5} It is essential to identify the cause of effusion in patients with RPM due to the spectacular response to corticoid treatment.

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Leukemic Pleural Effusion: Diagnostic Approach and Controversies in Pleurodesis[☆]



Derrame pleural leucémico: aproximación diagnóstica y controversias en pleurodesis

To the Editor:

The most common causes of pleural effusion in patients with acute myeloid leukemia (AML) are infections (bacterial or viral), other malignancy, chemotherapy and those derived from the malignant process itself. Survival is determined by response to treatment of the hematological disease.¹

A minimum sample volume of 60 mL is required for the cytomorphological diagnosis of malignancy in pleural fluid.²

In cases of pleural effusion refractory to treatment of the underlying disease, pleurodesis must be performed to control respiratory symptoms.

This letter reports the case of a 76-year-old patient with AML diagnosed 2 months previously with compatible bone marrow phenotype and normal cytogenetic results (46,XY[15]) who had received 3 cycles of 5-azacitidine.

He was admitted for dyspnea, 38 °C fever and tachycardia (120 bpm). He had leukocytosis ($45 \times 10^9/L$), anemia (hemoglobin 88 g/L), thrombocytosis ($719 \times 10^9/L$) and serum lactate dehydrogenase (LDH) 1.663 IU/L (normal: 125–220 IU/L). Chest X-ray and computed tomography showed significant left pleural effusion.

A total of 90 mL of pleural fluid were obtained by thoracentesis. This contained 1200 lymphocytes/ μL (normal: $<200/\mu L$), glucose 52 mg/dL (normal: 70–110 mg/dL), LDH 1724 IU/L (normal: 125–220 IU/L) and pH was 7.38. Microbiological cultures were negative. Cyto centrifugation and May-Grünwald/Giemsa staining of the pleural fluid were performed for microscopic examination (Fig. 1). The presence of myeloblasts in pleural fluid was confirmed by flow cytometry immunophenotyping (CD34, CD33, CD13 and CD117, but not CD14 or CD15). Cytogenetic examination with G-banding was normal, consistent with the patient's AML phenotype.

A diagnosis of leukemic pleural effusion was established and pleural drainage was performed, with little response. One week

later, the patient required pleurodesis with bleomycin to control dyspnea derived from worsening pleural effusion. His respiratory syndrome worsened progressively until exitus at 15 days.

In the case of leukemic pleural effusion, the clonal cell line must be confirmed with fluorescence *in situ* hybridization (FISH).³

In the routine screening of these patients for the indication of pleurodesis, there is no clear correlation between pleural fluid pH and survival; clinical status appears to be the best predictor for post-pleurodesis survival.

In patients who have not previously undergone pleurodesis, no significant differences in dyspnea relief have been found between permanent pleural catheter drainage and talc pleurodesis.⁴

Both bleomycin and talc have been shown to be good sclerosing agents, with similar efficacy in pleurodesis for the control of symptomatic malignant pleural effusion. Although bleomycin was used in our patient, it is important to note that talc is cheaper and may have the same or better success rate in the reduction of recurrent malignant pleural effusion than bleomycin and other sclerosing agents, although this difference has not been shown to be statistically significant.⁵

The use of many sclerosing agents in pleurodesis has been reported, including iodized povidone, doxycycline, silver nitrate, interferon alpha-2b and others. Good results have been documented, but disparity in the design of these studies make

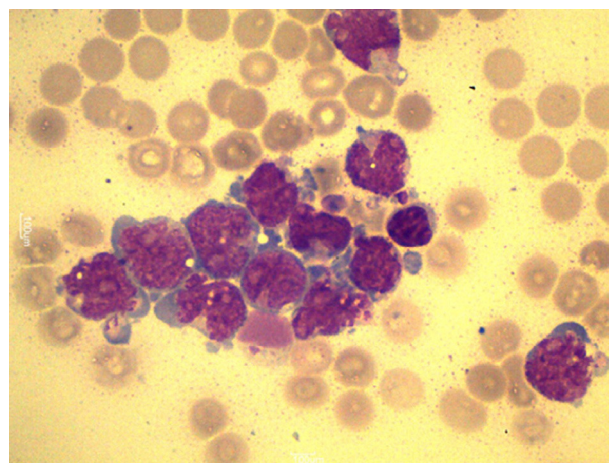


Fig. 1. Microscopic examination of pleural fluid, showing cells with lax and immature chromatin, raised nuclear-cytoplasmic ratio and visible nucleoli, compatible with myeloblasts (May-Grünwald/Giemsa staining, 1000 \times).

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