

Scientific Letters

Pseudochylothorax without Pleural Thickening Associated with Rheumatoid Arthritis[☆]



Seudoquilotórax sin engrosamiento pleural asociado a artritis reumatoide

To the Editor,

Pseudochylothorax is a very uncommon type of pleural effusion characterized by a high cholesterol content, and usually occurs in chronic effusions with pleural thickening.¹ The most common causes are tuberculous effusion, sequelae from therapeutic pneumothorax, and rheumatoid pleurisy, although it has been seen in other clinical situations.^{1,2} While pleural effusion is a relatively common finding in rheumatoid arthritis, only around 20 cases of pseudochylothorax have been reported in these patients, so it is difficult to determine the real prevalence of this process.³

Cholesterol in the pleural space was thought to be due to erythrocyte and neutrophil degradation within a thickened pleura, but it seems more likely to originate from serum lipoproteins than from cell lysis.¹ Pleural thickening with fibrotic scar tissue preventing fluid absorption was considered a key element in the etiopathogenesis of pseudochylothorax.¹ However, in recent years, some cases of pseudochylothorax have been reported in patients with rheumatoid arthritis who have not shown pleural thickening, bringing the generally accepted pathogenic mechanism into question.^{4,5} We have diagnosed a new case of pseudochylothorax in a patient with rheumatoid arthritis who did not present pleural thickening. A 53-year-old woman, smoker (25 pack-years) with no previous respiratory symptoms, was referred to the respiratory medicine specialist after a small pleural effusion with no associated symptoms was detected on the study performed before an intervention for abdominal hernia. A chest radiograph obtained 7 months previously from the same patient did not show pleural effusion. She had been diagnosed with rheumatoid arthritis 2 years previously and was receiving treatment with corticosteroids, methotrexate and certolizumab. On chest CT (Fig. 1) pleural effusion was observed with no signs of pleural thickening. A thoracocentesis was performed, yielding a sterile, opalescent fluid with no malignant cells, pH 7.03, glucose <20 mg/dl, proteins 7 g/dl, LDH 14,691 U/l, ADA 137 U/l, rheumatoid factor <9 U/ml, triglycerides 17 mg/dl and cholesterol 255 mg/dl (in serum 186 mg/dl).

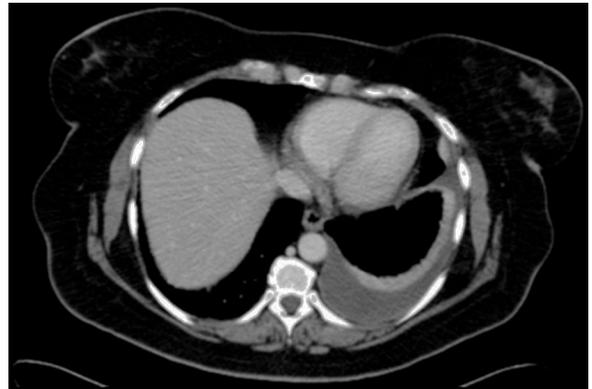


Fig. 1. CT image showing pleural effusion with no signs of pleural thickening.

Our case showed characteristics of pleural effusion due to rheumatoid arthritis (very low pH and glucose and high LDL levels) and pseudochylothorax (low triglycerides and high cholesterol concentration in pleural fluid), and adds to the few previous observations of pseudochylothorax associated with rheumatoid arthritis in the absence of pleural thickening. This finding suggests that pathogenic mechanisms must exist for the formation of this type of pleural effusion other than the chronic process of fluid within a fibrotic pleura,⁴ and confirms that the absence of pleural thickening does not rule out pseudochylothorax. Pseudochylothorax, if asymptomatic, does not require any specific treatment. However, when it occurs in association with rheumatoid arthritis, cases have been described in which intensive treatment of the underlying disease leads to control or resolution of the pseudochylothorax.

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***Pneumocystis jirovecii* Pneumonia Complicating the Progress of a Patient with Glioblastoma Multiforme Receiving Temozolomide[☆]**



Neumonía por Pneumocystis jirovecii complicando la evolución de una paciente con glioblastoma multiforme en tratamiento con temozolomida

To the Editor,

Pneumocystis jirovecii pneumonia (PJP) is an opportunistic infection that is particularly common in patients with human immunodeficiency virus (HIV), although it is also encountered in patients receiving corticosteroids, immunosuppressants and

anticancer drugs. Temozolomide is a relatively new alkylating anticancer agent used as a first-line drug in the treatment of glioblastoma multiforme (GM).¹ A rare but serious complication of temozolomide is the appearance of PJP. The pathogenic mechanism is associated with the induction of lymphocytopenia and selective T cell dysfunction.² Few references are available in the literature that describe the development of PJP in patients receiving temozolomide treatment.³

We report the case of a 69-year-old woman with a diagnosis of multifocal GM (Fig. 1A), who presented with a clinical picture of fever and rapidly progressing respiratory failure, 4 weeks after starting temozolomide and high-dose corticosteroids. Chest radiograph (Fig. 1B) showed increased ground glass

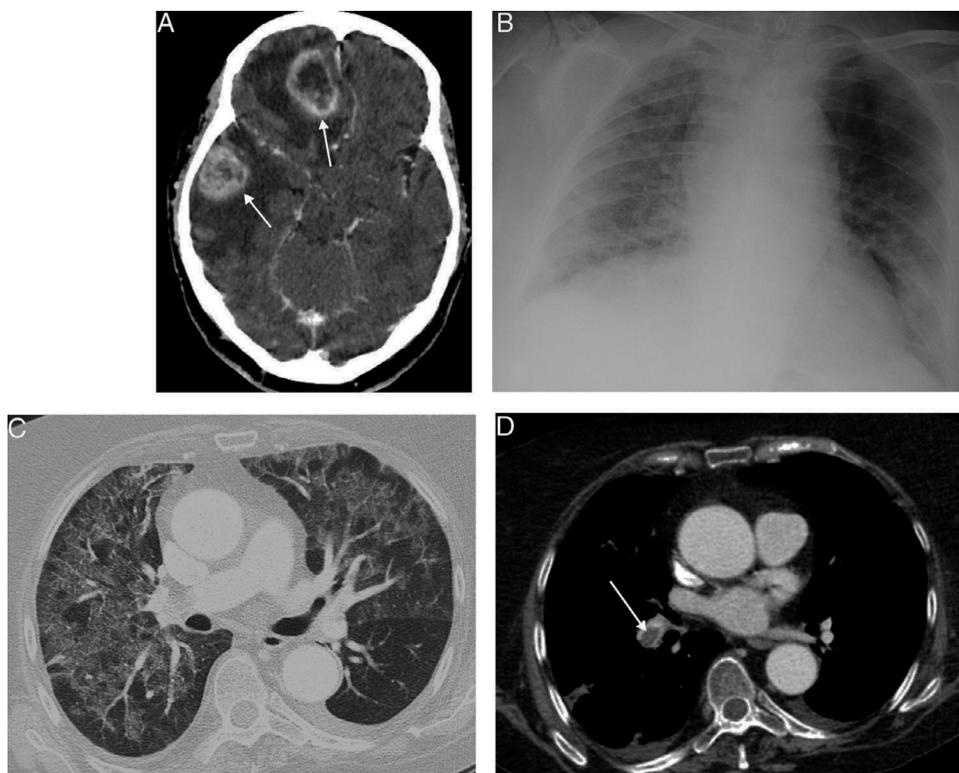


Fig. 1. (A) Axial CT image of the head, after administration of intravenous contrast medium, showing 2 intra-axial ring enhancing lesions (arrows) in the left cerebral hemisphere, with a significant mass effect and associated perilesional edema. (B) Anteroposterior chest radiograph showing ground glass opacities in both lungs. (C) Axial image of chest CT (lung window), showing a noteworthy mosaic pattern in the pulmonary parenchyma, with areas of ground glass attenuation alternating with others of less density, typical of *Pneumocystis jirovecii* infection. (D) Axial image of chest CT (mediastinum window) with intravenous contrast (obtained at the same level as image C), showing central filling defect in the right lower lobe artery (arrow), associated with pulmonary thromboembolism.

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