

Statin-induced NAM was diagnosed and the patient was treated with intravenous methylprednisolone and human immunoglobulin. He was discharged with prednisone (40 mg/day) and azathioprine. Clinical, laboratory and PFT findings improved gradually after 3 months. Prednisone was gradually tapered to 20 mg/day, but muscle strength and dyspnea continued to worsen, while CK and aldolase levels rose. Azathioprine was switched to mycophenolate mofetil but later discontinued due to gastrointestinal intolerance. Cyclophosphamide (CyC) was then introduced monthly (750 mg/m²), but after six CyC doses, a high dose of prednisone had to be maintained (Table 1). Rituximab was then introduced (1000 mg, 2 weeks apart), leading to reduced CK levels and improvements in LFTs and CT results (Fig. 1C and D).

Rituximab, a chimeric monoclonal anti-CD20 antibody, has been used as a rescue drug in the treatment of refractory myositis and ILD associated with antisynthetase syndrome⁵. Our case demonstrated a successful outcome with rituximab, and one possible explanation for this response could be that B cell depletion prevents not only persistent autoantibody production in NAM but also antigen presentation and interaction with other T cells.

A major limitation of our case report is that anti-HMGCR autoantibodies were not analyzed. Nevertheless, the temporal association between the onset of symptoms and statin use, the presence of significant necrosis without inflammation on muscle biopsy, and the requirement of intense immunosuppression allowed a confident diagnosis of statin-related NAM.

We emphasize the importance of considering statins as potential etiologic factors of ILD. Patients with statin-related NAM should

be actively tested for ILD, and rituximab seems to have a role in refractory cases.

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Subacute Silicone Pneumonitis After Silent Rupture of Breast Implant[☆]



Neumonitis subaguda por silicona tras la rotura silenciosa de un implante mamario

To the Editor:

Silicones are a group of polydimethylsiloxane polymers with differing viscosity, depending on their chain length. They are widely used in cosmetic and reconstructive surgery due to their supposed physical stability and lack of immunogenicity. However, these compounds are not inert, and numerous local and systemic complications associated with their use have been reported.^{1–3}

Most cases of pulmonary toxicity described in the literature are associated with subcutaneous injections of liquid silicone, and this practice is currently banned by the FDA. In contrast, systemic complications due to silicone gel prostheses are exceptionally rare.¹ We report the case of subacute pneumonitis caused by silicone in a patient with breast implants.

A 55-year-old woman, non-smoker, with a history of primary biliary cirrhosis, who had received bilateral breast implants 10 years previously. She presented in the pulmonology clinic with a 3-month history of symptoms including irritative cough, low-grade fever, pleuritic chest pain, dyspnea on moderate exertion, asthenia, and loss of appetite. Of note on physical examination were tachypnea, 24 breaths/min and crackles in upper left fields on auscultation.

Arterial blood gases, complete blood count and serum biochemistry results were normal.

On chest radiograph, ground glass opacities and airspace consolidation in both lung bases and periphery were observed. The initial diagnosis was pneumonia, and the patient began antibiotic treatment with moxifloxacin. However, her progress was slow, so she was admitted for further tests. A fiberoptic bronchoscopy was performed, which revealed no pathological findings. Chest computed tomography revealed new areas of parenchymal consolidation in the upper right lobe (Fig. 1A). Finally, the patient underwent a surgical lung biopsy by videoassisted thoracoscopy. The pathology study gave a diagnosis of foreign body giant cell reaction, with macrophages containing lipid vacuoles (Fig. 1B). Magnetic resonance imaging of the breast confirmed the intra- and extracapsular rupture of the right breast prosthesis. The prosthesis was removed surgically and oral corticosteroid treatment was initiated, after which the patient's progress was favorable.

Silicone implants are increasingly used in breast surgery both for reconstructive and cosmetic reasons. Migration of silicone after transplant generally occurs after rupture of the prosthesis, although silicone can also seep through an intact shell.⁴

The first case of silicone pneumonitis was described in 1975, and since then similar case series have been reported, mostly due to subcutaneous injections of liquid silicone. The pathogenesis of this disease is unknown, but the most accepted hypotheses suggest hematogenous or lymphatic dissemination of the silicone. Two clinical courses have been described: the acute form, which occurs with sudden-onset dyspnea, fever and chest pain; and the latent form, with onset 6 months after the application of the biopolymer, that occurs with more simmering symptoms.⁵

The definitive diagnosis can be achieved with transbronchial or open biopsy, although the presence of macrophages with

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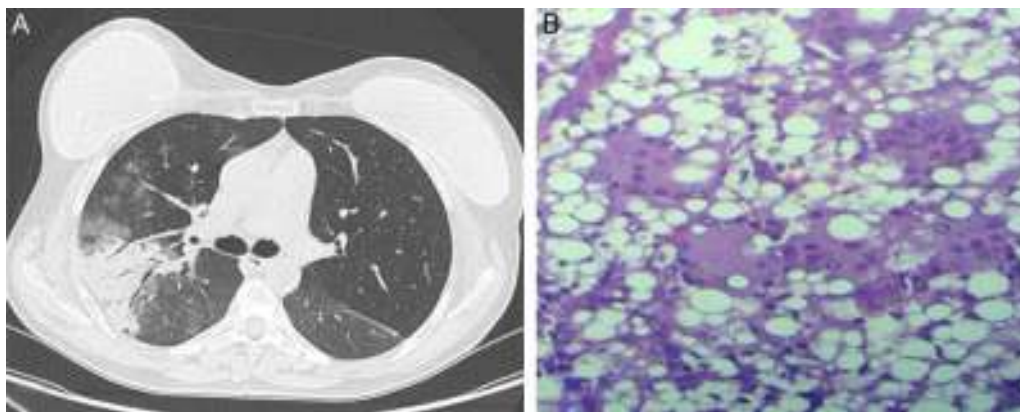


Fig. 1. (A) Chest computed axial tomography slice, showing areas of parenchymal consolidation with air bronchogram and ground glass opacities in the upper right lobe. (B) Histology study ($\times 40$) of the lung biopsy showing multinucleated macrophages with foreign body vacuoles (silicone).

intracytoplasmic lipid inclusions in the bronchoalveolar lavage is characteristic of silicone pneumonitis, and may obviate the need for a biopsy when clinical suspicion is well-founded. Acute phase treatment entails respiratory support, with administration of high-flow oxygen and mechanical ventilation in more severe cases.

In conclusion, silicone pneumonitis is a rare and potentially severe complication that may occur after the application of silicone for cosmetic purposes. This complication occurring in association with a gel prosthesis is even rarer, although it is important to include it in the differential diagnosis of patients with breast implants who present inflammatory pulmonary processes.

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Dehydration in Adult Cystic Fibrosis Patients*



Deshidratación en pacientes adultos con fibrosis quística

To the Editor:

Cystic fibrosis (CF) affects the eccrine and exocrine epithelial cells, causing pulmonary disorders, abnormally concentrated sweat, and pancreatic failure. The genetic alteration responsible for this disease is a mutation of the cystic fibrosis transmembrane conductance regulator (CFTR) protein, which acts as a chloride channel, controlling ion transport through the apical membrane.¹ Hot weather, intense physical exercise, stress, fever, vomiting, diarrhea or overdressing² can cause patients to present chloride, sodium and potassium depletion, due to a failure to replace loss of salts.³ Since survival of patients with this disease has increased in recent decades, CF is no longer an exclusively pediatric condition, so it is important to know that other non-respiratory complications exist that may present as a medical emergency.²

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We report 3 cases of adults with CF, who, despite the usual recommendations, presented severe dehydration syndrome during the summer.

A 19-year-old man, transport driver, presented in the emergency room with a clinical picture of intense asthenia and some vomiting. He had been working under exposure to high temperatures for several hours. He presented in the emergency room with blood pressure 137/71 mmHg, normal body temperature, mucocutaneous pallor, and sunken eyes.

A 25-year-old woman with CF, summer camp monitor, presented in the emergency room with a 24-h history of vomiting (about 20 episodes) and inability to take anything by mouth. She also reported reduced urine output. On arrival, she had arterial hypotension (85/52 mmHg).

A 28-year-old man, employee in a mechanical workshop, presented in the emergency room with cramps, generalized muscle pain, and reduced urine output. The day before, he had been working in the sun for a long period.

The patients' laboratory tests results on admission and discharge are shown in Table 1. All patients received serum replacement therapy, leading to improved biochemical parameters.

Under normal conditions, chloride and sodium are reabsorbed from sweat via the CFTR channel in the sweat glands. When