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References


Rafael Andrade-Alegre, a,∗ Norberto Donoso, b Olivia El-Achtar c

a Sección de Cirugía Torácica, Hospital Santo Tomás, Panamá, Panamá
b Servicio de Cirugía Vascular Periférica, Hospital Santo Tomás, Panamá, Panamá
c Servicio de Cirugía General, Hospital Santo Tomás, Panamá, Panamá

∗Corresponding author.
E-mail addresses: toravasc@cwpanama.net, randradealegre@gmail.com (R. Andrade-Alegre).

Interstitial Lung Disease With Statin-associated Necrotizing Autoimmune Myopathy Responding to Rituximab

Enfermedad pulmonar intersticial con miopatía autoinmune necrosante asociada a estatinas responde al rituximab

To the Editor:

Statins [hydroxyl-methyl-glutaryl-coenzyme-A reductase (HMGCR) inhibitors] are used to treat patients with hypercholesterolemia. One recently described adverse effect of these drugs is necrotizing autoimmune myopathy (NAM). 1–3 In statin-induced NAM, patients present with subacute symmetrical proximal limb weakness and elevated serum levels of the muscle enzyme, creatine kinase (CK). The clinical course is severe, and patients present...
histologically significant necrosis of muscle fibers with minimal or no inflammation.\(^2\textsuperscript{,3}\)

To date, no association between statin-induced NAM and interstitial lung disease (ILD) has been reported in the literature, and only one publication has addressed the use of rituximab in NAM.\(^4\) We report the first case of ILD secondary to statin-induced NAM, correlating muscle disease with pulmonary functional deterioration, responding to rituximab.

A 52-year-old male patient complained of a 3-year history of myalgia, proximal limb muscle weakness (grade 3) and progressive breathlessness (currently grade 2 according to the Modified Medical Research Council dyspnea scale), which started 3 months after taking rosuvastatin for dyslipidemia. He also had diabetes mellitus and hypothyroidism, and was a non-smoker.

Statin use was withdrawn due to elevated CK (3000 U/L, reference values <150 U/L) and aldolase (19.3 U/L, reference values <7.6 U/L) levels; however, the side effects persisted. He was hospitalized due to increasing CK levels (7000 U/L). Physical examination was normal, and oxygen saturation (SpO\(_2\)) was 96% on room air. Electroneuromyography showed mild proximal myopathy. The biceps brachii muscle was biopsied, and histopathology indicated homogeneous muscle size, with moderate necrosis in muscle fibers without significant inflammation (CD4+/CD8+ negative and CD 68+ positive). MHC I on the surface of all muscle fibers was positive. Additional laboratory tests revealed normal serum levels of thyroid hormones, negative autoimmune antibodies, and negative serologies for hepatitis and HIV.

Lung function tests (LFTs) showed a restrictive pattern with reduction of carbon monoxide diffusion capacity (DLCO) (Table 1). High-resolution chest computed tomography (CT) showed a pattern of non-specific interstitial pneumonia (Fig. 1A and B).

<table>
<thead>
<tr>
<th>Drug regimen</th>
<th>Diagnosis</th>
<th>May/13</th>
<th>Aug/13</th>
<th>Dec/13</th>
<th>Apr/14</th>
<th>Jun/14</th>
<th>Aug/14</th>
<th>Nov/14</th>
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<tbody>
<tr>
<td></td>
<td>No drug</td>
<td>Aza150mg</td>
<td>Aza200mg</td>
<td>MMF1g</td>
<td>Cyc IV (5th pulse)</td>
<td>1st month after Rtx</td>
<td>3rd month after Rtx</td>
<td>6th month after Rtx</td>
</tr>
<tr>
<td>FVC</td>
<td>2.24 (53%)</td>
<td>3.28 (71%)</td>
<td>2.71 (59%)</td>
<td>2.65 (60%)</td>
<td>2.73 (59%)</td>
<td>2.63 (58%)</td>
<td>2.78 (63%)</td>
<td>2.81 (63%)</td>
</tr>
<tr>
<td>FEV(_1)</td>
<td>2.02 (60%)</td>
<td>2.86 (77%)</td>
<td>2.29 (62%)</td>
<td>2.27 (64%)</td>
<td>2.34 (63%)</td>
<td>2.19 (61%)</td>
<td>2.40 (67%)</td>
<td>2.43 (68%)</td>
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<tr>
<td>TLC</td>
<td>3.75 (58%)</td>
<td>4.18 (63%)</td>
<td>4.38 (65%)</td>
<td>4.05 (63%)</td>
<td>3.82 (59%)</td>
<td>4.14 (64%)</td>
<td>4.05 (63%)</td>
<td>4.25 (63%)</td>
</tr>
<tr>
<td>RV</td>
<td>1.20 (62%)</td>
<td>0.90 (44%)</td>
<td>1.52 (34%)</td>
<td>1.35 (68%)</td>
<td>1.09 (50%)</td>
<td>1.31 (65%)</td>
<td>1.21 (60%)</td>
<td>1.44 (71%)</td>
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<tr>
<td>DLCO</td>
<td>19.50 (58%)</td>
<td>20.38 (62%)</td>
<td>22.1 (66%)</td>
<td>21.2 (67%)</td>
<td>21.2 (67%)</td>
<td>22 (67%)</td>
<td>18.1 (60%)</td>
<td>20.3 (63%)</td>
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<tr>
<td>MIP</td>
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<td>88 (72%)</td>
<td>60 (49%)</td>
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<td>NA</td>
<td>NA</td>
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<td>MEP</td>
<td>NA</td>
<td>78 (63%)</td>
<td>62 (50%)</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>70 (68%)</td>
<td>NA</td>
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<tr>
<td>CK</td>
<td>&gt;7000</td>
<td>90</td>
<td>193</td>
<td>713</td>
<td>1994</td>
<td>NA</td>
<td>744</td>
<td>148</td>
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<tr>
<td>Aldolase</td>
<td>19.3</td>
<td>4.6</td>
<td>6.3</td>
<td>9.4</td>
<td>27.2</td>
<td>NA</td>
<td>14.7</td>
<td>4.9</td>
</tr>
</tbody>
</table>

Aza: azathioprine (−3 mg/kg/day); Cyc: cyclophosphamide (750 mg/m\(^2\)); MMF: mycophenolate mofetil, Rtx: rituximab (two infusions of 1000 mg, two weeks apart); Pred: prednisolone; FVC: forced vital capacity; FEV\(_1\): forced expiratory volume in 1 second; TLC: total lung capacity; RV: residual volume; DLCO: carbon monoxide diffusion capacity; MIP: maximal inspiratory pressure; MEP: maximal expiratory pressure; CK: creatine kinase; NA: not available.

Fig. 1. (A and B) High-resolution computed tomography showing raised diaphragm, ground glass opacities, interlobular thickening, and traction bronchiectasis predominantly in the lower lobes, a pattern compatible with non-specific interstitial pneumonia. (C and D) CT scans after rituximab treatment showing improvement in ground glass opacities.
Subacute Silicone Pneumonitis After Silent Rupture of Breast Implant

Neumonitis subaguda por silicona tras la rotura silente 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 irritable 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 thoracoscopic. 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

References


Olivia Meira Dias,∗ Bruno Guedes Baldi,∗ André Nathan Costa,∗ Samuel Katsuyuki Shinjo,∗ Renata Miossi,∗ Ronaldo Al dib Kairalla∗

∗División Pulmonar, Heart Institute, University of São Paulo, São Paulo, Brazil

∗División de Reumatología, Hospital das Clínicas, University of São Paulo, São Paulo, Brazil

∗Corresponding author.

E-mail address: meiradias@yahoo.com.br (O.M. Dias).

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