Case In Point: Silicone-induced pneumonitis in a transgendered patient

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The authors describe the development of pneumonitis in a patient who had initially presented with edema of the lower extremities. Biopsy results supported the conclusion that the pneumonitis was caused by silicone injections the patient had received 5 years earlier.

The case

A 34-year-old nonsurgical transgendered patient (biologic male) presented with a 1-month history of progressive bilateral lower extremity edema spreading to involve the lower abdomen and scrotum. Five years before admission, the patient had approximately 2 L of liquid silicone injected bilaterally into the thighs and legs for cosmetic purposes. The patient denied any complications from the procedure, had no respiratory complaints after the procedure, and was otherwise healthy until about 1 month before admission.

The patient reported sex with several male partners as well as use of cocaine, "ecstasy," and methamphetamine up to 1 year before admission, with minimal tobacco and alcohol consumption. The surgical history was significant for silicone breast augmentation and rhinoplasty several years earlier.

On presentation, the patient had a temperature of 37.7°C (99.9°F), heart rate of 88 beats per minute, blood pressure of 134/76 mm Hg, respiration rate of 20 breaths per minute, and oxygen saturation of 98% on room air. The patient was not in any acute distress. Decreased breath sounds were heard bilaterally throughout, and rales were heard predominantly over the upper lobes.

Cardiac examination revealed a grade 2/6 systolic murmur best heard at the left upper sternal border. The abdomen was distended, with shifting dullness and pitting edema. Lower extremities exhibited 3+ pitting, brawny, slightly tender edema bilaterally. Right cervical and axillary as well as bilateral inguinal lymphadenopathy was noted.

Laboratory studies indicated the following values: white blood cell count, 13.8 3 10^3/µL; hemoglobin, 9.9 g/dL; platelet count, 645 3 10^3/µL; and lactate dehydrogenase, 964 U/L. A chemistry panel on admission was normal except for a creatinine level of 1.4 mg/dL. A 24-hour urine collection recovered 8400 mg of protein. The albumin level was 1.2 g/dL. Results of tests for anti-double-stranded DNA, anticardiolipin antibody, Coccidioides IgG and IgM, rapid plasma reagin, hepatitis C virus, hepatitis B virus, and HIV were all negative.

The patient was given empiric therapy with clindamycin and rifampin for lower extremity cellulitis. Lower extremity ultrasonography was nondiagnostic for deep venous thrombosis. On hospital day 4, the patient was found to be mildly hypoxic on room air.

A chest radiograph showed subtle evidence of reticular nodular opacities, small bilateral pleural effusions, and increased bilateral lower lung field densities resulting from silicone breast implants (Figure 1). A chest CT scan revealed bilateral, patchy airspace nodules with an upper lobe predominance (Figure 2).

Fiberoptic bronchoscopy revealed normal bronchial mucosa. A bronchoalveolar lavage (BAL) specimen demonstrated acute inflammatory cells and was negative for acid-fast bacilli (AFB). Formal AFB and fungal cultures were negative. Transbronchial biopsy specimens demonstrated polymorphonuclear inflammatory cells, alveolar macrophages, reactive bronchial epithelial cells, and multiple deposits of nonstaining foreign material; translucent filmlike borders strongly suggested the presence of silicone (Figure 3).

A right inguinal lymph node biopsy specimen revealed silicone granulomas and was negative for lymphoma (Figure 4). The diagnosis of silicone diffusion and silicone-induced pneumonitis was made.
The patient was given furosemide for edema and continued to be in stable condition for the remainder of hospitalization. The patient was discharged with recommendations to start corticosteroid therapy for pneumonitis.

**Discussion**

Dermatologists and plastic surgeons first published data on the use of liquid silicone injections for cosmetic purposes in the 1960s. As more experience accumulates, it has become clear that this material is not as inert as initially anticipated. Studies in mice have shown that subcutaneously injected silicone can be engulfed by histiocytes, transported to regional lymph nodes, and distributed throughout the reticular endothelial system, where it forms granulomalike lesions.1 There have been reports of similar complications in humans. Silicone lymphadenopathy; painful local silicone granulomas; and fatal, catastrophic systemic deposition in multiple organs have been reported.2-4 Less commonly, silicone-induced pulmonary complications have been observed; these include fatal pulmonary hemorrhage,5 pulmonary emboli,6 acute and latent pneumonitis,7-10 and pulmonary fibrosis.6 Cases typically involve transgendered persons because they often receive large-volume injections. The results of studies investigating infectious causes have been negative. The clinical spectrum of silicone-induced pulmonary damage and the pathophysiology of these manifestations are poorly understood. Chung and coworkers5 suggested that the variability in pathology may depend on the interplay between the level of immune system activation, host factors, and the amount of injected silicone. Most patients become symptomatic within hours to days after the last injection and present with dyspnea on exertion, cough, fever, hypoxemia, and bilateral pulmonary infiltrates.

Outcomes of acute pneumonitis have ranged from self-resolution within a few weeks to respiratory failure requiring mechanical ventilation.7 Chastre and associates7 also described a latent, mild form of pneumonitis that presented between 6 and 13 months after the last silicone injection. The patients had normal chest radiographic results, slight restrictive changes on pulmonary function testing, and milder signs of alveolitis on BAL analysis.7 To our knowledge, there are no reported cases of silicone-induced pneumonitis occurring as an incidental finding. Because our patient did not have any history of respiratory complaints during the 5 years after injection, it is unclear when the silicone gained access to the lungs. It is possible that the material became fragmented and embolized after being subjected to long periods of mechanical stress or that chronic, repeated microemboli were not substantial enough to cause any symptoms.

The mechanism by which distal silicone injection causes pulmonary pathology has not been clearly determined. We suspect it to be via local lymphatic drainage, ascension through the cisterna chyli, and drainage through the thoracic duct into the left subclavian and jugular veins and into the right side of the heart and pulmonary circulation, where it embolizes in the pulmonary arterial circulation. The lymph nodes gain access to the particles as well; this explains the lymph node and pulmonary pathologic findings in our patient. Our findings support the potential for long-term pulmonary toxicity in asymptomatic patients who have a remote history of subcutaneous silicone injection.

**References:** REFERENCES


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