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Case report

A 20-year-old woman with rapidly progressive dyspnea and diffuse pulmonary infiltrates

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ABSTRACT

Silicone is a liquid polymer previously considered to be immunologically inert and favored in cosmetic procedures. Increasing evidence shows a multisystemic inflammatory reaction to its administration constituting the silicone embolism syndrome (SES). The majority of adverse effects are seen in the pulmonary system resulting in extensive diffuse alveolar damage and ultimately ARDS. Neurologic involvement occurs frequently and is uniformly fatal. Large volume injections, high pressure infiltrations and prior exposure to silicone have been implicated, with an IgG polydimethylsiloxane antibody described. Most patients meet Schonfield criteria for fat embolism syndrome and treatment is largely supportive. As the illicit use of injectable silicone rises worldwide, so does the incidence of related morbidities and fatalities, necessitating a high index of suspicion for SES in patients with neurologic or pulmonary symptoms and recent exposure to liquid silicone. We report an unusual case of multi-organ dysfunction following silicone injection.

1. Case report

A previously healthy 20-year-old female from England had flown into the US with friends for a “pumping party”. She arrived with the intention of injecting 3000 ccs of hospital grade silicone into her thighs and buttocks, with lesser quantities for her friends who had previously received silicone injections without complications. Approximately 4 h after administration of the injections she began to experience chest tightness with mild dyspnea and was taken to the ER.

On physical examination, the patient was in no distress while breathing room air. Vital signs were normal. The lungs, heart, and abdominal examinations revealed no abnormalities. The extremities demonstrated extensive bilateral greater trochanteric swelling without erythema with a palpable doughy consistency. Neurologic examination revealed no focal deficits.

Laboratory data including complete blood count, serum chemistry, cardiac enzymes and urine for toxicology screening were all negative. Initial electrocardiogram was normal and chest radiographs showed diffuse interstitial infiltrates and minimal pulmonary vascular congestion (Fig. 1).

Ninety minutes later, she became lethargic, markedly dyspneic and diaphoretic. Arterial blood gas analysis on 100% oxygen were pH 7.29, pCO2 37 mmHg, pO2 53 mmHg, and oxygen saturation 82%. She was intubated and transferred to the ICU. Chest CT revealed subcentimeter non-calcified pulmonary nodules, peripheral ground-glass opacities and interlobular septal thickening in all lung lobes (Fig. 2).

What is the diagnosis?

2. Discussion

Silicone embolism syndrome (SES) is a potentially fatal, multisystemic complication that results from the illegal cosmetic injection of liquid silicone (polydimethylsiloxane). Although silicone polymers were favored for use in cosmetic procedures (Fig. 3) as they were previously believed to be immunologically inert compounds with high thermal stability and minimal tissue reaction,[1] there is increasing evidence showing a widespread inflammatory reaction to its administration.[2]

Beyond the occurrence of direct intravascular injection which frequently occurs in illicit cosmetic silicone administration, embolic phenomena can also occur as a result of silicone penetration into the microvasculature in the setting of increased perivascular tissue pressure. The majority of adverse effects are seen in the pulmonary system with a spectrum of events ranging from acute silicone pneumonitis (usually characterized by fever, respiratory insufficiency and bilateral interstitial infiltrates on chest radiographs) to silicone-induced embolic phenomena. These are associated with vascular congestion and hypersensitivity pneumonitis resulting in

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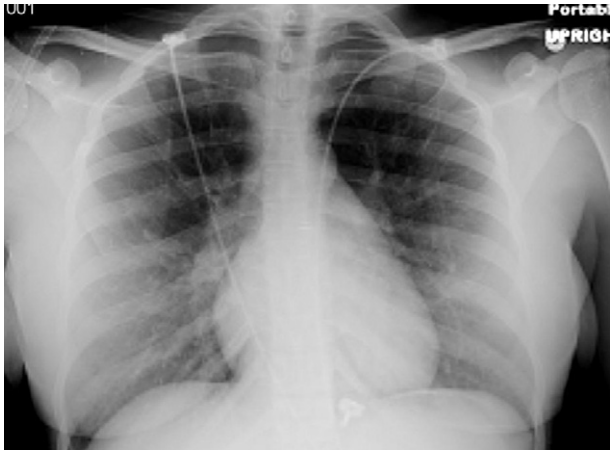

Fig. 1. Initial chest radiograph showing diffuse interstitial infiltrates and minimal pulmonary vascular congestion.

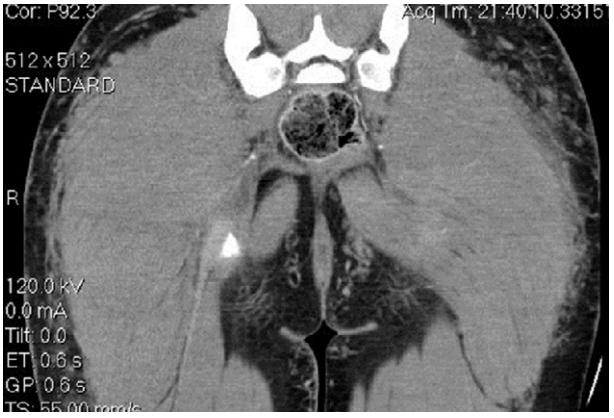

Fig. 3. A Pelvic CT coronal slice depicting areas of silicone injection into the gluteal and trochanteric areas bilaterally.

extensive diffuse alveolar damage and ultimately ARDS.[3] Most patients develop clinical signs within the first 24 h of silicone administration and the onset of symptoms has been linked to a higher mortality rate (20%).[4] The most frequent symptoms include hypoxemia, dyspnea, fever, chest pain, cough and hemoptysis.[1] Bronchoalveolar lavage (BAL) commonly reveals alveolar hemorrhage, and a restrictive pattern is usually observed on pulmonary function studies. While the acute presentation is typical for the majority of patients, delayed-onset pneumonitis and injection-site inflammation occurring years after silicone administration has been described. Migration of micro-droplets of silicone could also assume a delayed presentation in the form of pulmonary fibrosis.[4] Occasionally, pulmonary toxicity has been described to lag behind CNS manifestations especially when initial chest radiography and pulmonary examinations are benign in the presence of lethargy. Increasing release of silicone emboli from the source results in a slow progression to ARDS similar to the manifestation of heroin induced pulmonary edema.[5] Neurologic sequelae of silicone embolism vary from mild alteration in levels of consciousness to frank coma. Interestingly, the absence of underlying cardiac septal defects does not preclude the occurrence of neurologic manifestations, as microinfarcts in white matter following cerebral silicone embolism has been described in these individuals and observed to be uniformly fatal.[2] Large volume injections, high pressure infiltrations and prior exposure to silicone have been associated with a worse prognosis and increased rapidity of symptom onset.[2] The presence of an IgG polydimethylsiloxane antibody which selectively binds to the silicone polymers has been implicated in this inflammatory process. Histology typically reveals multi-organ involvement with granulomas diffusely dispersed within the cardiopulmonary, renal, hepatic and gastrointestinal organ systems. Histopathologic analysis with the aid of infrared spectrophotometry and atomic absorption reveals these granulomas to consist of silicone vacuoles, tissue macrophages, neutrophils, eosinophils and fibrin deposits. Pulmonary silicone embolism characteristically results in a consistent chest CT imaging pattern of bilateral peripheral ground-glass opacities and interlobular septal thickening as portrayed by the present patient.[4]

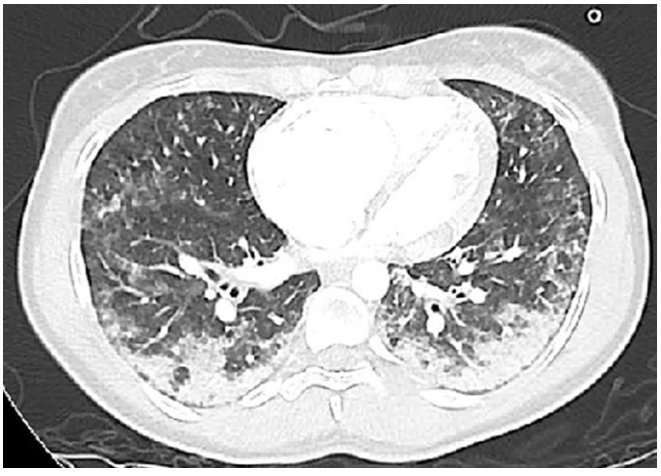

Fig. 2. Chest CT showing subcentimeter pulmonary nodules, peripheral ground-glass opacities and interlobular septal thickening in all lung lobes.

The mechanism of silicone injury to pulmonary capillaries closely mimics fat embolism with the occurrence of bilateral alveolar hemorrhages and diffuse presence of silicone droplets in pulmonary alveolar macrophages and lung capillaries. Similar to fat embolism syndrome (FES) in which inappropriate activation of the coagulation cascade has been demonstrated in experimental and bone marrow models, spontaneous thrombosis of the internal jugular vein has been described in pulmonary silicone embolism. Ingestion of silicone and fat by these alveolar macrophages has also been postulated to result in modulation of pulmonary immunoregulatory mechanisms and provoke an exaggerated inflammatory response. These suggest a common pathophysiologic mechanism involving the coagulation system in both FES and SES. Notably, most patients with SES meet Schonfield criteria for fat embolism syndrome where the presence of petechial hemorrhages, chest x-ray changes, hypoxemia, tachycardia, tachypnea, confusion and fever are used to determine a cumulative score.[1]

While treatment is largely supportive with supplemental oxygen and high dose steroid administration constituting the mainstay of therapy, the use of adjunct salvage mechanical ventilation techniques, and recruitment maneuvers like prone ventilation have been suggested to improve oxygenation.[2]

The present patient appeared to be in ARDS from pulmonary silicone embolism and presented issues of futility of care exacerbated by unprecedented high doses of silicone injection. These facilitated a progressively rapid decline in her clinical course. She rapidly deteriorated despite application of evidence based protocols for treatment of ARDS, and lapsed into pulseless electrical activity, expiring 3 h post intubation. Illicit use of injectable silicone is on the rise in the United States and abroad. With this comes an increasing incidence of related morbidities and fatalities. A high

index of suspicion for SES should be triggered in patients with neurologic or pulmonary symptoms and recent exposure to liquid silicone.

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Contributors

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Conflict of interest

The authors have no conflict of interest.

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