



VV ECMO Rescue of Silicone Syndrome

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Abstract

Silicone Embolism Syndrome can cause an acute life threatening pneumonitis. It is usually seen after illicit injections of silicone. We report a case wherein the silicone use was medically sanctioned but resulted in pneumonitis severe enough to require use of a membrane oxygenator (VV ECMO).

Case Presentation

The patient is a 64 year old woman physician who 30 years prior had had bilateral cosmetic subcutaneous silicone implants performed abroad. Six months prior she started to feel ill and required multiple hospitalizations. Her symptoms included right sided chest pain, capsular contractures from her implants, back discomfort, dyspnea, fatigue and persistent fevers. Implant rupture was diagnosed on the basis of CT scan (Figure 1 and 2). During her work up she was found to have a right coronary stenosis for which a drug eluting stent was placed. A transthoracic ECHO showed a normal ejection fraction of 55-60% - no LVH, normal valve and right ventricle function. A CXR and CT were otherwise unremarkable. She underwent rheumatologic evaluation for fever of unknown origin up to Tmax 102°C. She was anemic, ESR was elevated to 90 mm/hr, and CRP 120 mg/L to 130 mg/L. Her work up was ongoing when three months prior to our encounter she began self-medicating with high dose steroids (prednisone 40 mg). She reportedly felt 'great' and blood assays showed that her ESR and CRP levels returned to normal. She was scheduled for elective implant removal/revision. Her preoperative testing was unremarkable; she had self- tapered to 5 mg prednisone.

At the operation, the silicone and ruptured prostheses were found to be encapsulated allowing for a relatively clean removal of the foreign material. Both implants were removed and a new sub muscular implant was placed on the right. While working to reimplant the left and at this point, approximately three hours after induction, the patient experienced VT/VF arrest then PEA in the setting of extreme hypoxia. Return of spontaneous circulation was achieved after 21 minutes of CPR. The left breast was packed, the surgery terminated and the patient brought to the ICU. In the ICU she experienced continued hypoxia and a second PEA arrest but with relatively quick return of circulation. Her caregivers requested emergent transfer to our institution as part of thrombosis-page for acute myocardial infarction.

Upon arrival patient was on 26mcg/min levophed and 8mcg epinephrine, bedside ECHO was consistent with mild global cardiac stunning, cardiac catheterization was notable for normal coronaries, CVP 20 mmHg, and LVEDP 28 mmHg. CT head negative, CT Chest angiogram revealed no evidence of pulmonary embolism, bilateral alveolar consolidation of the lungs especially at the bases and scattered areas of ground glass opacities. She was in florid pulmonary edema with copious frothy hemorrhagic secretions. Initial ABG on FiO2 1.0 and 16 mmHg PEEP; pH 7.079/pCO2 24.6/pO2 59.6/O2 sat 75.5%/ BE -20.7. Serum lactate 10 (ref. 0.5 mmol/L to 2.2 mmol/L), anion gap 21.7, troponin 2.37 (ref. 0 ng/ml -0.20 ng/ml).

In a hybrid OR she underwent emergent percutaneous placement of 27F Avalon Dual Lumen right internal jugular vein catheter and initiation of VV ECMO at flows of 3L/min. A right common femoral 5F vascular sheathe was placed to allow rapid escalation to V-AV ECMO in the event that it was needed. Heparin was administered to an ACT target of 200-240s. Meanwhile, her metabolic acidosis was treated. Corrective resuscitative measures were taken including the initiation of inhaled nitric oxide, correction of metabolic acidosis, downgrading of vasopressors/inotropes and resetting of ventilator in accordance with a lung protective strategy. The patient left the OR an hour post ECMO initiation with the ABG pH 7.36/pCO2 44.7/pO2 91.8/O2 sat 94.8%. TEE documented unloading of the right ventricle and some early improvement to left ventricle function.

Over the next several days the patient's respiratory status improved, she gained full neurologic

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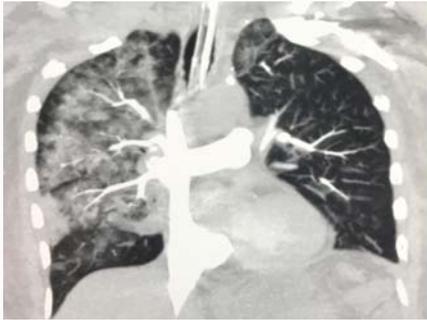


Figure 1: Coronal reconstruction of admission Chest CT scan.

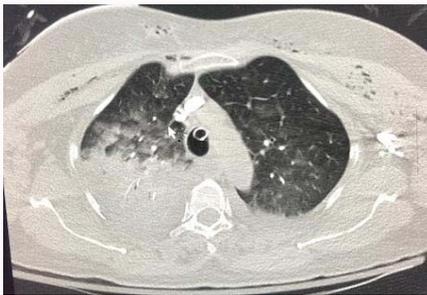


Figure 2: X-section mid lung field of admission Chest CT scan.

and cardiac recovery, underwent bilateral pleural effusions drainage, full explant of her breast prosthesis with closure and was weaned off ECMO on day 9 (Figure 3). She was discharged to rehabilitation on day 14. She was seen in six month follow up earlier this year. The above described events have left her with little long term morbidity.

Discussion

Medical silicones belong to a polymer class characterized by alternating silicon-oxygen atoms. They are chemically and thermally stable, non-immunogenic and, as such, considered biologically inert. As a result they have been used in a number of human implants, most notably breast implants. Anecdotal reports have linked silicone breast implants to a number of recognized rheumatologic diseases (Sjogren's syndrome, Lupus Erythematosus and scleroderma) and to otherwise unexplained symptoms that appear to be auto immune in origin [1]. While a 2000 meta-analysis failed to demonstrate an increased incidence of specific rheumatologic diseases among silicone breast implant patients, serologic studies have linked the HLA-DR53 genotype and the formation of B-cell autoantibodies to symptomatic recipients [2,3]. Our patient during the time that she was discovered to have ruptured implants began to complain of a number of non-specific ailments that were associated with elevated inflammatory markers (ESR and CRP). Rheumatologic evaluation failed to identify a specific rheumatologic illness or explanation for her fever of unknown origin but prednisone did ameliorate the majority of her symptoms. We may speculate that silicone exposure sensitized her immune system but have no confirmation and are unclear as to whether this may have played a role in the severity of her reaction to silicone embolization.

On the day of surgery she experienced the described acute events while under the careful supervision of an operation. That she became severely hypoxic arrested after the manipulation of her ruptured implants and well after anesthetic induction is most consistent with

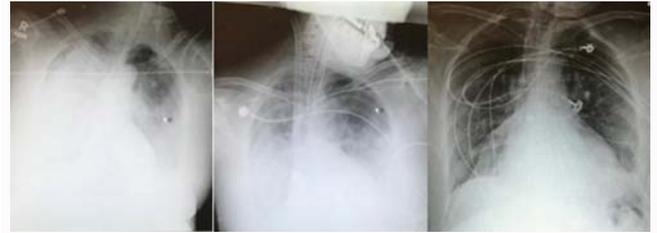


Figure 3: Sequential chest radiographs: day of admission, hospital day (HD) 4 and day of ECMO explants (HD 9).

a diagnosis of Silicone Embolism Syndrome (SES). Moreover, her work up excluded the majority of alternate causes. The migration of silicone, introduced from various perivascular spaces to the pulmonary microcirculation can cause acute inflammation, increase capillary permeability, alveolar edema and alveolar hemorrhage [4]. Lung imaging usually shows bilateral patchy alveolar infiltrates and scattered ground glass opacities. Clinically an acute form occurring within hours or days of inoculation and a more latent form up to six months after injection have been described. Dyspnea, hypoxia and acute respiratory failure are the most common symptoms. Treatment consists of rest, supplemental oxygen and mechanical ventilation if necessary. The role of corticosteroids is unclear. Most patients recovering from the acute phase do so without sequelae.

The patient presented represents a case of severe acute pneumonitis resulting from silicone embolism syndrome. Despite maximal medical therapy within an intensive hospital setting the patient's hypoxia failed to correct and she experienced two PEA arrests. VV ECMO with VA standby was successfully used to rescue, stabilize and recover this patient. In our review of the literature we identified two case reports of patients requiring ECMO support for recovery from silicone embolism syndrome, in each case after the illegal injection of liquid silicone for cosmetic purposes [5,6]. This, to our knowledge, is the first case of ECMO used to successfully recover a patient from silicone embolism syndrome sustained during a medically sanctioned silicone implant/explant procedure.

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