Fat embolism syndrome (FES) is usually described in the setting of major joint replacement, orthopedic injuries, and vertebroplasty, although it has been also reported in cases of severe burns, blunt chest trauma, pancreatitis, and liposuction. Reports on the incidence of FES vary widely. Originally described by Zenker in 1862 and von Bergmann in 1873, the main mechanism of FES pathogenesis involves the passage of bone marrow fat into general circulation. Bone marrow fat often translocates into blood vessels when bones suffer trauma, although only a minority of cases develop clinical manifestations or severe disease. Once the disease is clinically significant, it can affect multiple systems, including the brain, lungs, skin, heart, and eyes.1,2

Aesthetic soft tissue filling is a safe and routine practice, generally causing only local reactions when adverse events do occur.3 We describe the case of a woman who underwent soft tissue injection of a large amount of soft tissue filler (STF) in the gluteal area for aesthetic purposes. She later presented with symptoms of FES. To the best of our knowledge, no one has previously reported an association between STF and FES.

CASE PRESENTATION

A 26-year-old healthy woman with no relevant family or medical history was admitted to the intensive care unit of our hospital. Her symptoms began 24 hours before her presentation, when approximately 200 mL of STF in a semi-liquid state was injected bilaterally into the soft tissue of the gluteal area. The injections were performed in an aesthetic medicine clinic. A close relative described the procedure upon admittance: a catheter was placed in the soft tissue via a small incision and the STF was injected several times. Six hours after discharge from the aesthetic clinic, she presented with chills, low fever, and intense headache. Some hours later, she noticed uncontrollable drowsiness and dysarthria. She was later transferred to our unit. Once in the emergency room, she reported shortness of breath. Her neurological examination revealed confusion and disorientation, with a Glasgow score of 12, but with no other localized neurological signs. Petechiae were seen in the inferior eyelids (Figure 1), chest, and legs.
The patient’s status evolved toward a respiratory failure requiring mechanical ventilation. At that time, her blood gas on room air showed a $P_aO_2$ of 53 mm Hg (pH 7.52), $P_eCO_2$ of 19 mm Hg, 91% $S_aO_2$, $HCO_3$ of 20.3 mmol/L, and $P_aO_2/FIO_2$ ratio of 133. Other lab results showed a leukocyte count of $18.0 \times 10^3/\mu L$, $17.0 \times 10^3/\mu L$ of granulocytes, hemoglobin of 16.6 g/L, platelets at $246 \times 10^3/\mu L$, serum creatinine of 1.04 mg/dL, D-dimer lower than 100 ng/mL, and brain natriuretic peptide of 154 pg/mL. The patient’s cerebrospinal fluid had a protein level of 38.8 mg/dL and a glucose of 87 mg/dL with no cells. Fundoscopy revealed signs of obstruction of the central retinal artery. A computed tomography (CT) pulmonary angiography demonstrated bilateral ground-glass opacities, demonstrated but ruled out pulmonary embolism. Cerebral CT scan showed generalized edema without focal lesions; similarly, another CT scan was performed to identify the source of the suspected fat embolism, which revealed an irregularly distributed foreign body in the soft tissue of the gluteal area (Figure 2). Fat droplets were detected in urinary cytology, bronchial-alveolar lavage, and mixed venous central blood samples. Therefore, the patient presented with three minor and four major components of Gurd and Wilson’s criteria for fat embolism.

Despite developing ventilator-associated pneumonia, the patient’s condition resolved, and she experienced complete recovery of the neurological symptoms and improvement of the visual deficit. She was discharged from the hospital 20 days after admission.

**DISCUSSION**

FES after aesthetic procedures has been described previously, generally in patients who have undergone liposuction or other forms of adipose tissue trauma. FES is identified by the triad of pulmonary dysfunction, neurological signs, and petechiae. The accepted mechanism for FES pathogenesis involves the release of fat droplets into systemic circulation. These droplets are deposited in the lung when fat is physically forced into the venous system as a result of bone trauma.

FES secondary to the use of STF has not been described previously, although we have not found reports of such large amounts of STF as was injected in this case. We ruled out direct injection of the STF into the blood vessels, as the imaging studies did not reveal evidence of direct injection. A possible trigger for the FES observed in this case might be fat trauma during the multiple injections of STF. On the basis of the classical appearance of the diagnostic criteria, the patient was diagnosed with FES.

The diagnosis of silicon embolism syndrome (SES) was also considered in this case, due to the fact that the patient received an injection of an unknown substance and developed the clinical features of FES. It is known that these two syndromes have a clinical presentation and outcome almost identical. Treatment is also similar.

FES presents predominantly with pulmonary and neurological symptoms, in addition to petechiae. The pulmonary manifestations range from asymptomatic hypoxemia to acute respiratory distress syndrome, and the majority of patients require mechanical ventilation. Neurological symptoms include headache, disorientation, seizures, meningeal signs, stupor, and coma. FES is also characterized by a short duration of trunk and neck petechiae, which may also be seen in the mouth and conjunctiva; petechiae may disappear in less than 24 hours. Furthermore, brain CT scans may show hyperdense lesions, interlobar septa thickening, and generalized edema, whereas IRM might reveal fat intensity lesions in the basal ganglia, cerebellar hemispheres, and white matter.

The diagnosis of FES is supported by the presence of at least two major features, or one major plus four minor features, from the criteria described in 1970 by Gurd and
modified by Gurd and Wilson. Nonetheless, we agree with those who claim that clinical judgment and patient evolution are the most important factors in the diagnosis. The presence of fat droplets in the patient’s biological fluids (urine, blood, bronchial-alveolar lavage) may induce a physician toward the diagnosis of FES, but confirmation by the Gurd criteria is still required.

There are no specific treatments for FES. Supportive measures, including mechanical ventilation and brain protection, are of great importance. Fibrates, dextran, heparin, and steroids have all been employed to treat FES, although none of these approaches has been shown conclusively to be effective. FES is considered an autolimited disease, and patients improve when fat droplets and fatty acids are cleared.

CONCLUSIONS

Soft tissue filling is a common cosmetic procedure and we believe that more government regulation should be encouraged to avoid cases such as this one, in which soft tissue fillers were administered improperly with unfavorable consequences.

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