A rare case of fat embolism syndrome secondary to abdominal liposuction and gluteal fat infiltration

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Abstract

Fat embolism syndrome (FES) is a rare, life-threatening condition habitually associated with traumatic events such as fractures and, less commonly, burns, liposuction and bone marrow harvesting and transplant [1]. The biochemical theory for this condition suggests that fat droplets embolize and convert into fatty acids, eventually leading to toxic injury and inflammation, which results in increased vascular permeability, edema and hemorrhage [2]. FES may have an asymptomatic interval lasting 12–72 hours after the insult; however, in some cases, signs have also been seen intraoperatively. Pulmonary signs and symptoms are customarily the earliest and manifest in 75% of patients. Nevertheless, neurologic and dermatologic manifestations are also characteristic, and most severe cases could perhaps present with disseminated intravascular coagulation, right ventricular dysfunction, shock or death. The following case consists of a 37-year-old patient that presented with fat embolism syndrome during liposuction and gluteal fat infiltration.

INTRODUCTION

Fat embolism syndrome (FES) is a multisystemic disorder whereby fat emboli disseminate within the systemic circulation to various organ systems leading to characteristic clinical manifestations. The most severely affected organs are the skin, lungs, eyes and central nervous system [1–3]. In 1873, Bergman first described FES as a triad of confusion, dyspnoea and petechiae following long bone fractures. Epidemiologic data suggest that this disorder occurs in 3–4% of patients with long bone fractures and rarely arises secondary to intraosseous fluid administration, intravenous hyperalimentation, long-term steroid administration, bone marrow harvesting, lipid-soluble radiocast and liposuction [1, 4]. Signs and symptoms are vague, which makes the diagnosis challenging. Gurd and Wilson proposed diagnostic criteria where two primary criteria or one major criterion and four of the minor criteria are required to diagnose FES. Later, Murry and Racz, Lindegue et al., and Schonfeld proposed different FES criteria. However, Gurd and Wilson’s criteria are generally used [3, 4]. Organ support therapy is the mainstay treatment for this syndrome [4].

CASE PRESENTATION

The present clinical case consists of a 37-year-old male patient with a BMI of 34. One, who underwent liposuction, ultimately extracting 5000 ml of adipose tissue with serosanguineous fluid. Subsequently, the surgery proceeded with the infiltration of 400 ml of adipose tissue into the superficial and deep compartments of each gluteus. Minutes after the procedure concluded, the patient exhibited a sudden bradycardia of 40 bpm. Atropine was administered, displaying slight improvement; however, bradycardia recurred along with low blood pressure and abnormal cardiac rhythm. The patient’s oxygen saturation dropped to 72% while being mechanically ventilated with a...
FiO\textsubscript{2} of 75%. An arterial blood gas analysis was ordered, which revealed a pH of 7.25, PaCO\textsubscript{2} of 55 mm Hg, and PaO\textsubscript{2} of 96 mm Hg. The PaO\textsubscript{2}/FiO\textsubscript{2} calculation was utilized, obtaining a result of 128 mm Hg. Epinephrine and lidocaine were administered accordingly. Nevertheless, symptoms did not improve, and the patient became asystolic. After 10 minutes of cardiopulmonary resuscitation, the patient’s blood pressure and heart rate were restored, and treating physicians transferred the patient to the intensive care unit (ICU), where he received assisted ventilation. An electroencephalogram (EEG) evidenced slow theta range activity in both frontoparietal regions consistent with hypoxic–ischemic encephalopathy. While in the ICU, the patient presented with a persistent fever of 38.6°C resistant to antipyretic treatment, which was suspected to be secondary to the hypoxic–ischemic encephalopathy. Physicians managed the patient with fluid support and supplementary oxygen in order to maintain adequate hemodynamic stability. According to Gurd and Wilson’s criteria, the patient was diagnosed with FES. After a few months, the patient presented a satisfactory clinical outcome and was discharged.

**DISCUSSION**

FES is a clinical entity arising from the embolization of fat droplets into capillary beds, associated with trauma and rarely nontraumatic interventions, resulting in pulmonary, neurological and cutaneous manifestations. Nontraumatic etiologies encompass sickle crisis, pancreatitis, osteomyelitis and liposuction \([4, 5]\). This syndrome has an incidence of ~0.9% \([5]\). FES has no pathognomonic features, which makes this heterogenous disease challenging to diagnose. However, the most significant clinical feature of FES is respiratory insufficiency. Gurd and Wilson criteria, seen in Table 1, are frequently adopted to establish the diagnosis. Laboratory samples and radiological images are nonspecific; however, results may provide clues to support the diagnosis \([5, 6]\). In general, a thoracic computed tomography scan usually displays vascular congestion and pulmonary edema. The only proven treatment for FES is supportive care and supplemental oxygen if needed. Maintenance of intravascular volume is a crucial factor in preventing lung injury. Anticoagulation has been useful in animal models but is not commonly used with patients since benefits remain unproven, and there is an evident risk of bleeding \([6]\).

**CONCLUSION**

Literature shows a low incidence of FES; nevertheless, clinicians must have a high degree of clinical suspicion to detect this diagnosis when patients present with sudden respiratory insufficiency and neurological manifestations. Early identification is essential since this pathology is associated with a high mortality risk if not appropriately treated with supportive measures. Symptoms and manifestations make the diagnosis challenging since there are no pathognomonic features related to this syndrome; hence, clinical history is essential in establishing the cause.

**CONFLICT OF INTEREST STATEMENT**

None declared.

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