Rapid recovery of fat embolism syndrome with acute respiratory failure due to liposuction

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Abstract

Liposuction is not a risk-free procedure and potentially fatal complications may occur, especially liposuction-induced fat embolism syndrome (FES). Here we report the case of a 29-year-old woman who developed FES suddenly during a liposuction operation in a cosmetic medical clinic. She was transferred to the hospital and achieved complete recovery within 11 days by comprehensive therapeutic strategies, including noninvasive ventilation (NIV), corticosteroids, albumin, diuretics and anticoagulation. Liposuction-induced FES is a life-threatening condition, which can be treated with complete recovery by comprehensive therapeutic strategies according to its pathophysiologic mechanism.

KEYWORDS
acute respiratory failure, case report, fat embolism syndrome, liposuction

INTRODUCTION

Liposuction is one potential cause of fat embolism syndrome (FES). Liposuction-induced FES is a rare, but life-threatening condition. The overall mortality is approximately 5%–25%1,2. The terms microscopic fat embolism (MIFE) and macroscopic fat embolism (MAFE) are used to classify the severity and clinical outcome. Herein, we report that a young woman who suffered from liposuction-induced FES an recovered completely as a result of comprehensive supportive treatments, including non-invasive ventilation (NIV), corticosteroids, albumin, diuretics, anticoagulation and so on.

CASE REPORT

A 29-year-old woman, BMI 28.5 kg/m², during a selective liposuction operation on her arms and abdomen, developed sudden onset dyspnea, chest pain, dizziness and violent emesis. Pulse oximetry showed oxygen saturations (SpO₂) of 40% on room air. She was transferred to the emergency room immediately. She had no significant medical history or history of drug use. On admission to our emergency room, she was awake and dysphoric. She had worsening tachypnea and tachycardia with a respiratory rate of 30 breaths per minute and a heart rate of 120 breaths per minute. She was febrile with a temperature of 37.6°C (Table 1, Figure 1A). Physical examination revealed moist crackles in both lungs and subcutaneous ecchymosis on the liposuction sites. An arterial blood gas analysis showed a PaO₂ 9.35 kPa (1 mmHg = 0.133Kpa) (On 15 L/min supplemental O₂), PaCO₂ 4.53Kpa, and pH = 7.40 (Table 1, Figure 1A). Abnormal lab tests included an elevated lactic acid (5.4 mmol/L, normal range, 0.7–2.7), troponin I (0.07 ng/ml, normal range<0.04), creatine kinase (869 IU/L, normal range, 22–269), lactate dehydrogenase (434 IU/L, 0.7–2.7), troponin I (0.07 ng/ml, normal range<0.04), creatine kinase (869 IU/L, normal range, 22–269), lactate dehydrogenase (434 IU/L,
normal range, 98–192), and higher levels of D-dimer and N terminal pro B type natriuretic peptide (3.76 mg/L, and 751.1 pg/ml, respectively). Chest CT scan showed extensive ground-glass opacities and consolidation of pulmonary parenchyma, while no obvious filling defect was demonstrated on computed tomography pulmonary angiogram (CTPA). Brain CT was normal.

A diagnosis of FES was made after exclusion of pulmonary thromboembolism, drug allergy, cardiogenic pulmonary edema, and pneumonia. She developed marked respiratory compromise, without hemodynamic deterioration, or an altered state of consciousness.

NIV was the first-line treatment for respiratory failure in the first 48 h. She was weaned to oxygen via nasal cannula on day 3 and off of supplemental oxygen on day 9. Methylprednisolone was administered for 9 days at a daily dosage of 240 mg on day 1, 120 mg on day 2–3, 80 mg on day 4–7, and 40 mg on day 7–9. Human albumin at a dosage of 20 g per day was administered for the first 3 days (day 1–3), combined with diuretics to reduce pulmonary edema. Low molecular weight heparin (LMWH) was initiated for deep vein thrombosis prophylaxis (1 mg/kg once a day) (Figure 1A, B).

Rapid recovery was achieved with comprehensive supportive treatment. Pulmonary parenchymal infiltrates on CT were reduced significantly at day 10 (Figure 1C). The patient was discharged on day 11 without any symptoms.

**DISCUSSION**

FES can be caused by minimally-traumatic interventions such as liposuction. Early diagnosis and treatment are beneficial in reducing mortality.2 Gurd’s, Lindeque’s, and Schonfeld’s criteria have each been previously proposed for the diagnosis of FES and exclusion of other potential diagnoses. In our case, the patient was diagnosed with FES according to Schonfeld’s criteria. Proposed pathophysiology of most cases of FES combines mechanical and biochemical processes. The mechanical process includes that fat cells activate platelets and accelerate fibrin generation, lodging in the pulmonary capillary, ultimately leading to interstitial hemorrhage and edema, alveolar collapse and reactive hypoxemic vasoconstriction. Massive fat emboli may also cause macrovascular obstruction and shock. The biochemical process suggests that a pro-inflammatory cytokine cascade is caused by toxic free fatty acids, eventually leading to end-organ dysfunction. In the lung, toxic injury to pneumocytes and pulmonary endothelial cells leads to vasogenic and
cytotoxic edema as well as haemorrhage. Ultimately, acute lung injury or acute respiratory distress syndrome develops.\(^3\)

In a case series of 15 patients with liposuction associated FES, 66\% of patients required mechanical ventilation.\(^4\) Invasive ventilation could be avoided with effective NIV in the early stage. The patient in our report was treated with NIV for the first 48 h before being successfully withdrawn. NIV may prevent further catecholamine response and fat mobilization induced by hypoxia, inhibiting the further injury of target organs.

Currently, the management of FES surrounds provision of respiratory and hemodynamic support with a lack of specific evidence-based treatments. Inflammation induced by free fatty acids (FFAs) plays a crucial role in acute respiratory distress syndrome (ARDS). Given the pathogenesis, the early administration of corticosteroids may be effective. A great range has been reported for the effective dosage of methylprednisolone (total 9–90 mg/kg).\(^5,6\) In our case, we empirically used short-term, low-dose methylprednisolone (total 12 mg/kg), and no adverse effects were observed.

Human albumin might diminish further damage through binding to FFAs.\(^7\) Meanwhile, some studies have reported that the combined administration of albumin and diuretics in patients with ALI and ARDS can shorten the ventilation support duration.\(^8\) As such, human albumin (20 g/d, 3 d) and diuretics (5 mg/d, 10 d) were empirically used. Finally, we used LMWH to prevent venous thromboembolism effectively in the case with no hematologic abnormalities.

In summary, liposuction-induced FES may occur in healthy young individuals suddenly during or after liposuction surgery. Rapid recovery can occur with a comprehensive therapeutic strategy.

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**CONFLICT OF INTEREST**

None declared.

**DATA AVAILABILITY STATEMENT**

Data sharing not applicable—no new data generated.

**ETHICS STATEMENT**

The authors declare that appropriate written informed consent was obtained for the publication of this manuscript and accompanying images.
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