Received: 31 January 2022 | Accepted: 19 September 2022

DOI: 10.1002/rcr2.1047

CASE REPORT

Respirology Case Reports OPEN ACCESS APSR WILEY

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

Yong Jie Ding[1,2,3] | Liu Zhang[1,2,3] | Xian Wen Sun[1,2,3] | Ying Ni Lin[1,2,3] | Qing Yun Li[1,2,3]

[1]Department of Respiratory and Critical Care Medicine, Ruijin Hospital, Shanghai Jiao Tong University School of Medicine, Shanghai, China

[2]Institute of Respiratory Diseases, Shanghai Jiao Tong University School of Medicine, Shanghai, China

[3]Shanghai Key Laboratory of Emergency Prevention, Diagnosis and Treatment of Respiratory Infectious Diseases, Shanghai, China

Correspondence
Qing Yun Li, Department of Respiratory and Critical Care Medicine, Ruijin Hospital, Shanghai Jiao Tong University School of Medicine, Shanghai, China.
Email: liqingyun68@hotmail.com

Funding information
Cultivation Project of Shanghai Major Infectious Disease Research Base, Grant/Award Number: 20dz2210500; National Natural Science Foundation of China, Grant/Award Number: 82070089

Associate Editor: Tamera Corte

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 complate 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/m^2, 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 (SpO_2) 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 PaO_2 9.35 kPa (1 mmHg = 0.133Kpa) (On 15 L/min supplemental O_2), $PaCO_2$ 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,

Yong Jie Ding, Liu Zhang, and Xian Wen Sun are co-first authors.

TABLE 1 Clinical variables and laboratory values during the course of illness[a]

Variable	Day of illness									
	1	2	3	4	5	6	7	8	9	10
Clinical variables										
Temperature (°C)	37.8	37.8	37.2	36.9	36.5	36.8	36.5	36.7	36.4	36.6
Respiratory rate (breaths/min)	26	25	22	22	20	20	20	20	18	16
Oxygen saturation (%)	91	95	95	95	96	96	97	96	97	96
Heart rate (beats/min)	114	110	106	100	98	85	82	80	78	76
Oxygen (litres/min)	15	15	10	5	5	5	5	3	2	–
Noninvasive ventilation (h)	20	16	–	–	–	–	–	–	–	–
Laboratory values										
Haemoglobin (g/dl)	14.6	10.1	9.6	–	–	10.9	–	–	–	12.2
Haematocrit (%)	41.3	29.6	28.9	–	–	32.5	–	–	–	37.6
White cells (10^9/L)	5.80	9.81	9.19	–	–	11.32	–	–	–	9.80
Platelets (10^9/L)	255	187	164	–	–	326	–	–	–	350
AST (U/litre)	66	69	78	–	–	40	–	–	–	43
Creatine kinase (ng/ml)	837	1877	3439	–	–	624	–	–	–	360
Albumin (g/L)	36	32	28	–	–	42	–	–	–	45
Lactate dehydrogenase (IU/L)	434	378	344	–	–	311	–	–	–	209
TEG										
R[b] (min)	–	–	2.20	–	–	4.70	–	–	–	–
K[c] (min)	–	–	0.90	–	–	0.90	–	–	–	–
MA[d] (mm)	–	–	72	–	–	77.40	–	–	–	–

[a]Data are for the period starting with the patient's arrival in our emergency and ending on the day before discharge. AST, aspartate aminotransferase.
[b]R reflects the comprehensive action of coagulation factors function (normal range, 5–10 min).
[c]K reflects the comprehensive action of fibrin function(normal range, 1–3 min).
[d]MA reflects the comprehensive action of platelet function (normal range, 51–69 mm); R < 5 min, K < 1 min, and/or MA > 69 mm, indicating a condition of hypercoagulability.

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 haemorrhage 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

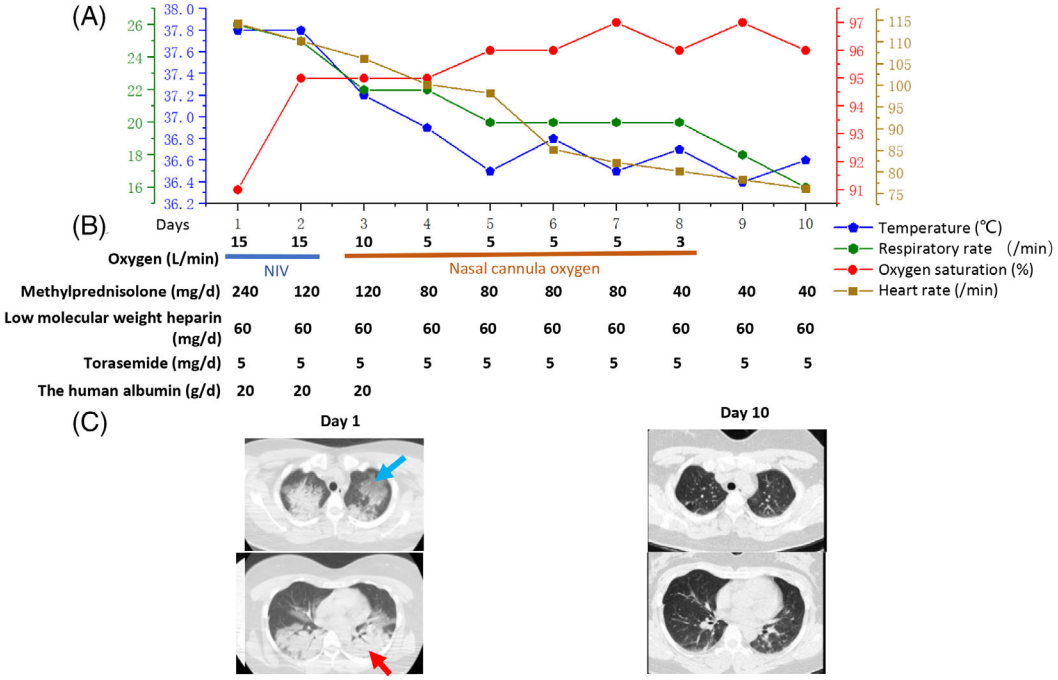

FIGURE 1 (A) All vital signs improved over time. (B) Timelines of NIV, oxygen supplement, and drug therapy. BPAP was used during the first 48 hours with oxygen supplement of 10–20 L/min. On day 3, the BPAP was withdrawn, then nasal cannula oxygen supplement was used and stopped at day8. The administration of methylprednisolone was carried out at 240 mg/d on day 1, 120 mg/d on day 2 and day 3. Along with the obvious improvement of symptoms and oxygenation, the daily dosage of methylprednisolone was decreased to 80 mg on day 4–day 7, then to 40 mg on day 8. Methylprednisolone was withdrawn on day 11 when the infiltrates absorbed significantly in chest CT and the symptoms disappeared. The human albumin was given at a dosage of 20 g/d for the first 3 days (day 1–day 3), combined with diuretics (Torasemide, 10 mg/d) to alleviate pulmonary infiltrates. With the reduction of the moist crackles, albumin was discontinued on day 4, and diuretics (Torasemide, 5 mg/d) were used until day 10. (C) CT scan on day1 showed that lung window settings demonstrate extensive ground-glass opacities with distant areas (blue arrows). Consolidation was seen on the bilateral lower lobe (red arrow). CT scan showed pulmonary parenchymal infiltrates almost absorbed completely on day 10

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.

FUNDING INFORMATION

This work was supported by grants from the National Natural Science Foundation of China (82070089), and Cultivation Project of Shanghai Major Infectious Disease Research Base (20dz2210500).

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 mansucript and accompanying images.

ORCID

Qing Yun Li https://orcid.org/0000-0001-8128-6319

REFERENCES

1. Cárdenas-Camarena L, Bayter JE, Aguirre-Serrano H, Cuenca-Pardo J. Deaths caused by gluteal lipoinjection: what are we doing wrong? Plast Reconstr Surg. 2015;136:58–66.
2. Kainoh T, Iriyama H, Komori A, Saitoh D, Naito T, Abe T. Risk factors of fat embolism syndrome after trauma: a nested case-control study with the use of a nationwide trauma registry in Japan. Chest. 2021;159(3):1064–71.
3. Kosova E, Bergmark B, Piazza G. Fat embolism syndrome. Circulation. 2015;131(3):317–20, 20.
4. Durán H, Cárdenas-Camarena L, Bayter-Marin JE, Ramos-Gallardo G, Robles-Cervantes JA. Microscopic and macroscopic fat embolism: solving the puzzle with case reports. Plast Reconstr Surg. 2018;142:569–77.
5. Christopher CT, Peter VG. Fat embolism syndrome: what have we learned over the years? Dent Traumatol. 2011;13:259–81.
6. Bederman SS, Bhandari M, McKee MD, Schemitsch EH. Do corticosteroids reduce the risk of fat embolism syndrome in patients with long-bone fractures? A meta-analysis. Can J Surg. 2009;52(5):386–93.
7. Mellor A, Soni N. Fat embolism. Anaesthesia. 2001;56(2):145–54.
8. Vignon P, Evrard B, Asfar P, Busana M, Calfee CS, Coppola S, et al. Fluid administration and monitoring in ARDS: which management? Intensive Care Med. 2020;46((12)):2252–64.

How to cite this article: Ding YJ, Zhang L, Sun XW, Lin YN, Li QY. Rapid recovery of fat embolism syndrome with acute respiratory failure due to liposuction. Respirology Case Reports. 2022;10: e01047. https://doi.org/10.1002/rcr2.1047