Case Report

Adult respiratory distress syndrome due to fat embolism in the postoperative period following liposuction and fat grafting*

Síndrome da angústia respiratória do adulto por embolia gordurosa no período pós-operatório de lipoaspiração e lipoenxertia

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Abstract

Fat embolism is defined as mechanical blockage of the vascular lumen by circulating fat globules. Although it primarily affects the lungs, it can also affect the central nervous system, retina, and skin. Fat embolism syndrome is a dysfunction of these organs caused by fat emboli. The most common causes of fat embolism and fat embolism syndrome are long bone fractures, although there are reports of its occurrence after cosmetic procedures. The diagnosis is made clinically, and treatment is still restricted to support measures. We report the case of a female patient who developed adult respiratory distress syndrome due to fat embolism in the postoperative period following liposuction and fat grafting. In this case, the patient responded well to alveolar recruitment maneuvers and protective mechanical ventilation. In addition, we present an epidemiological and pathophysiological analysis of fat embolism syndrome after cosmetic procedures.

Keywords: Respiratory distress syndrome, adult; Embolism, fat; Lipectomy.

Resumo

A embolia gordurosa é definida como a ocorrência de bloqueio mecânico da luz vascular por gotículas circulantes de gordura. Acomete principalmente o pulmão, podendo afetar também o sistema nervoso central, a retina e a pele. A síndrome da embolia gordurosa é uma disfunção desses órgãos causada pelos êmbolos gordurosos. As causas mais comuns de embolia gordurosa e síndrome da embolia gordurosa são as fraturas de ossos longos, mas há relatos de sua ocorrência após procedimentos estéticos. O diagnóstico é clínico, e o tratamento ainda se restringe a medidas de suporte. Apresentamos o caso de uma paciente que evoluiu com síndrome da angústia respiratória do adulto por embolia gordurosa no período pós-operatório de lipoaspiração e lipoenxertia e respondeu bem às manobras de recrutamento alveolar e à ventilação mecânica protetora.Apresentamos também uma análise epidemiológica e fisiopatológica da síndrome da embolia gordurosa após procedimentos estéticos.

Descritores: Síndrome do desconforto respiratório do adulto; Embolia gordurosa; Lipectomia.

Introduction

Fat embolism syndrome (FES) is a severe complication of long bone fractures, and has a mortality rate of up to 36%.[1] More rarely, there are reports of FES after cosmetic procedures, such as liposuction and fat grafting.[2-4] In the lungs, fulminant FES manifests as severe respiratory insufficiency and acute respiratory distress syndrome (ARDS), although the emboli can reach the arterial circulation and affect other sites, such as the central nervous system, retina, and skin.[2,3]

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We report the case of a female patient who developed ARDS due to fat embolism in the postoperative period following liposuction and fat grafting. In this case, the patient responded well to alveolar recruitment maneuvers and protective mechanical ventilation.

Case report

A previously healthy, 53-year-old Caucasian female nonsmoker homemaker, who was a native and resident of São Paulo, underwent bilateral mastopexy, abdominal liposuction, and fat grafting (gluteal augmentation). On postoperative day 3, the patient developed progressive dyspnea and dry cough, with no other symptoms. She was readmitted to the hospital (intensive care unit). Physical examination revealed tachycardia, tachypnea, and hypoxemia on room air. Pulmonary auscultation revealed no alterations. A chest X-ray showed minimal, bilateral interstitial infiltrate, and a high-resolution computed tomography scan of the chest with a protocol for pulmonary thromboembolism, performed 36 h later, revealed bilateral diffuse ground-glass infiltrate and slight bilateral pleural effusion, without thrombi in the pulmonary artery branches (Figures 1 and 2). The blood workup showed anemia, with a hemoglobin level of 7.5 g/dL and a hematocrit of 26%, without other findings. The patient developed hypoxemic respiratory insufficiency on postadmission day 2, and arterial blood gas analysis revealed no metabolic or respiratory acidosis. Orotracheal intubation and mechanical ventilation were necessary, with an arterial oxygen tension/fraction of inspired oxygen ratio of 32 and a lung injury score of 2.6.[5] Inotropic drugs were initiated due to hypotension and oliguria, and the patient developed dialytic renal insufficiency on postadmission day 3. A bedside echocardiogram ruled out the hypothesis of left ventricle dysfunction. A dilated eye exam showed signs of bilateral retinal embolism. Bronchoalveolar lavage was negative for alveolar hemorrhage. Testing and culture for viruses and bacteria were negative, as was testing for macrophages containing fat droplets. Other foci of infection, use of drugs, and inhalation of gases were ruled out through the use of culture and the taking of a history, although empirical antibiotic therapy against gram-positive and gram-negative bacteria, as well as against respiratory viruses, was started and maintained until the final culture and test results became known.

We were able to rule out the most common causes of ARDS, such as pulmonary infection, pulmonary contusion, aspiration of gastric content, and near drowning, as well as indirect triggering factors, such as septic syndrome, septic shock, multiple blood transfusions, polytrauma, pancreatitis, and amniotic fluid embolism. Therefore, we made a diagnosis of ARDS secondary to FES. The patient was treated according to the precepts of protective strategy, using the controlled pressure mode at a tidal volume of 6 mL/kg and alveolar recruitment with titration of the positive end-expiratory pressure in order to improve respiratory system compliance. She presented progressive improvement in the arterial oxygen tension/fraction of inspired oxygen ratio and was extubated on postadmission

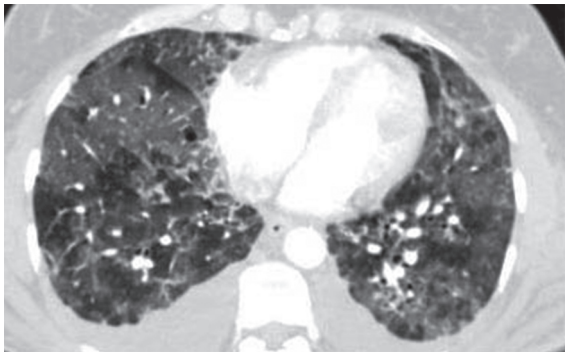

Figure 1 - High-resolution computed tomography scan of the chest, slice 1: ground-glass infiltrate and septal thickening predominantly in the upper thirds, as well as bilateral pleural effusion (atypical finding).

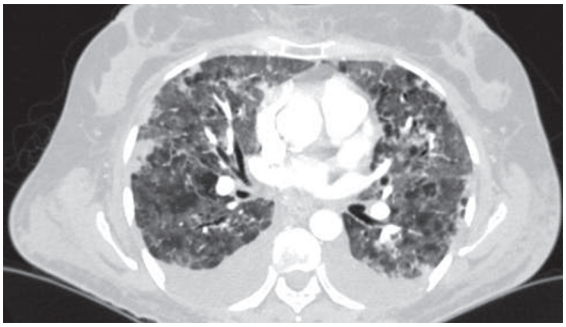

Figure 2 - High-resolution computed tomography scan of the chest, slice 2: ground-glass infiltrate and septal thickening predominantly in the upper thirds, as well as bilateral pleural effusion (atypical finding).

day 8. The patient was discharged from the hospital one month later, with no complaints, normal renal function, and peripheral oxygen saturation of 98% on room air.

Discussion

Since its introduction in the 1970s, liposuction has become more and more popular, to the point of currently being the most common cosmetic surgical procedure.[4] However, reports of adverse consequences, among which are fat embolism and FES, are increasingly common.[2-4,6]

Fat embolism is defined as the occurrence of mechanical blockage of the vascular lumen by circulating fat globules. Although it primarily affects the lungs, it can also affect the central nervous system, retina, and skin. [1,7,8] The definition of FES is fat emboli-induced dysfunction of these organs, and it is a rare, albeit potentially fatal, complication of cosmetic procedures such as liposuction and fat grafting.[2-4] It typically occurs 12 to 72 h after the surgical procedure, presenting as the triad of progressive respiratory insufficiency, altered level of consciousness and petechiae.[3,4,8] Neurological alterations range from mental confusion to altered level of consciousness, and there can be generalized convulsions and focal deficits, the latter being transient and reversible in most cases.[1,8]

During liposuction and fat grafting, there is rupture of small blood vessels and damage to adipocytes, producing lipid microfragments that reach the venous circulation and, consequently, cause lung injury.[1,2,4] Fat emboli can also reach the systemic circulation, affecting other organs, due to the patency of the oral foramen in the interatrial septum, the existence of pulmonary arteriovenous microfistulas, and the deformation of the fat microglobules that cross the pulmonary capillaries.[1,7,9] An animal model study of liposuction has found microparticles of circulating fat and pulmonary lipid deposits in 100% of the animals studied.[4]

Hypothetically, the etiopathogenesis of FES consists of two phases: the mechanical phase and the biochemical phase. The two are not mutually exclusive, and both have been described after major traumas involving long bone fracture, as well as after intramedullary orthopedic procedures.[1,7,8]

Table 1 - Gurd and Wilson criteria.[10]

Major criteria	Minor criteria
Acute respiratory insufficiency	Tachycardia
	Fever
Alterations in the central nervous system	Retinal alterations
	Urinary alterations
Petechiae	Sudden decrease in hematocrit and thrombocytopenia
	Fat in sputum
	Increased erythrocyte sedimentation rate

In the mechanical phase, the fat emboli, after entering the bloodstream, would reach the pulmonary capillary causing mechanical obstruction and could also migrate to distant organs, such as the central nervous system. This theory is corroborated by studies involving intraoperative transesophageal echocardiography in various types of orthopedic surgery in which hypoechogenic intracardiac material is seen during the introduction of intramedullary prostheses.[9] It does not explain, however, the absence of symptoms in the first 12 to 24 h.[1,7,8]

In the biochemical phase, the fat globules, after reaching the pulmonary capillaries, would be hydrolyzed by the lipase produced by the pneumocytes, producing free fatty acids that are toxic to alveolar and endothelial cells. At the onset of the local injury, vasoactive amines and prostaglandins are released. In addition, there is neutrophil recruitment, leading to hemorrhage, as well as to interstitial and alveolar edema. Histopathologically, there is edema, transudate and subsequent alveolar exudate, type II pneumocyte apoptosis, and formation of hyaline membrane.[1-3,7,8] This hypothesis could explain the fact that the onset of symptoms occurs some time after the surgical procedure, since agglutination and degradation of the fat emboli would be necessary in order to trigger the local inflammatory process.[1-3,7,8]

Clinically, post-traumatic FES is classified as "acute fulminant", "subacute", or "subclinical", and there is no standard classification for FES after cosmetic procedures.[1] The acute fulminant form occurs a few hours after the trauma, presenting as respiratory insufficiency, coma, and acute multiple organ failure. The subacute form is the classic triad already described. The subclinical form presents

benign symptoms of mild dyspnea and tachycardia, as well as drowsiness or mild irritability.[1]

In the case presented here, the patient developed respiratory insufficiency, tachycardia, signs of retinal hemorrhage, renal insufficiency, and a decrease in hematocrit without bleeding, allowing the clinical diagnosis according to the criteria by Gurd and Wilson,[10] who proposed that one major criteria and at least four minor criteria are necessary for the diagnosis of FES (Table 1).

Radiologically, FES typically presents as bilateral, symmetrical, alveolar and interstitial infiltrate that are more prominent in the perihilar region and in the lower regions of the lung.[10-12] On tomography scans of the chest, the findings include areas of consolidation, areas of ground-glass opacity (focal or diffuse), and micronodules smaller than 10 mm, these findings being consistent with interstitial and alveolar hemorrhage, as well as with edema and pneumonitis.[1,11,12] A study involving 6 patients with subacute FES evaluated by computed tomography of the chest showed ground-glass opacities in 7 patients and nodular opacities in 2 patients. In 5 of those patients, there was concomitant interlobular septal thickening.[12] In the case presented here, in addition to ground-glass infiltrate and septal thickening predominantly in the upper thirds, we found bilateral pleural effusion (an atypical finding). This finding can be secondary to aggressive fluid resuscitation, since tomography was performed 36 h after symptom onset.

There is as yet no specific treatment for FES. However, it is essential that support measures, such as oxygen therapy or mechanical ventilation, be taken in more severe cases.[1,2,8] In the present case, the patient was treated according to the precepts of the protective strategy, using the controlled pressure mode at a tidal volume of 6 mL/kg and alveolar recruitment with titration of the positive end-expiratory pressure in order to improve respiratory system compliance, as well as dialysis and hemodynamic stabilization in the intensive care unit.

Despite the favorable evolution, the case presented here serves to alert physicians and patients, since it is a severe complication of a cosmetic procedure that is often performed in outpatient clinics.

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