major ventilation/perfusion mismatch. In the right lung, the ventilation/perfusion ratio was very high, whereas in the left lung, this ratio markedly was decreased. Moreover, ventilation/perfusion mismatch may have been worsened by hypoxic vasoconstriction. The lack of response to nitric oxide may be explained by the finding that an increase in left lung perfusion was useless because of poor ventilation and an increase in right lung perfusion did not occur because of right pulmonary artery compression. It is doubtful whether any therapeutic intervention, apart from surgery, would have had any beneficial effect on the patient's gas exchange, hemodynamic status, or both. It is clear that the preoperative assessment of this patient with acute limb ischemia was incomplete and that the diagnosis of aortic dissection should have been considered before surgery; thus, transesophageal echocardiography or tomodensitometry may have been indicated preoperatively.[7] Surgery should have been performed very early on, despite the desperate status of the patient. An earlier and more complete understanding of the mechanisms of hypercapnia, acidosis, and hemodynamic failure might have helped to take the right decision earlier. Physicians should be aware of this rare complication to precisely diagnose the cause of severe hypercapnia and to proceed accordingly.

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Anesthesiology
1998; 89:782–4

Early Fat Embolism after Liposuction

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FAT embolism syndrome (FES) is an uncommon and severe complication that occurs mainly in patients with long-bone fractures. This clinical syndrome includes acute respiratory failure, global neurologic dysfunction, and petechial rash following the precipitating event by 12 to 72 h. Nevertheless, one or more of these findings may be absent, making the diagnosis difficult to establish clinically, particularly after a surgical procedure such as liposuction. The authors report a case of isolated acute dyspnea early after liposuction.

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Received from the Intensive Care Unit and the Department of Pathology, Hôpital Ambroise Paré, Boulogne, France. Submitted for publication October 27, 1997. Accepted for publication May 6, 1998.

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Key words: Bronchoalveolar lavage; computed tomography of the chest; fat embolism syndrome; liposuction.

Case Report

A 29-yr-old woman who was a healthy ballet-dancer without previous significant medical history underwent suction lipectomy of the abdomen, hips, and trocanteric area. The operation was uneventful, removing a total of only 600 ml of fatty tissue. Anesthesia (which lasted 2 h) consisted of fentanyl, midazolam, propofol (loading dose of 2

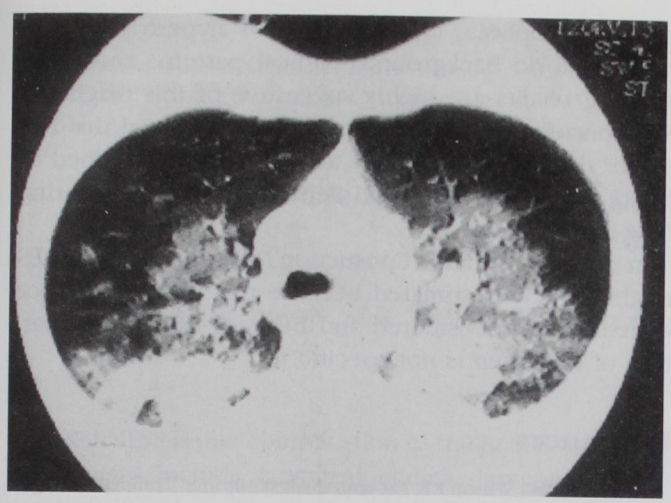

Fig. 1. Computed tomography of the chest performed 3 h after onset of the symptoms showing patchy bilateral ground glasses, macronodular opacities, and alveolar consolidation without pleural effusion.

mg/kg and then 5 $mg \cdot kg^{-1} \cdot h^{-1}$ infusion), and atracurium. There was no blood loss. Total volume of intravenous fluids intraoperatively and postoperatively were 1,500 ml, and urine output was 500 ml. Intraoperative oxygen saturations were 95–99%. One hour postoperatively, while breathing room air, the patient felt suddenly anxious and short of breath, oxygen saturation by pulse oximetry (Sp_{O_2}) decreased from 93% to 82%, without hemodynamic change. A pulmonary embolism was eliminated immediately by pulmonary angiography. The patient was admitted to the intensive care unit for management and diagnosis. At examination, she was dyspneic, with a respiratory rate of 25 breaths/min, a blood pressure of 110/60 mmHg and a heart rate of 95 beats/min. Mental status was normal without focal neurologic signs. No petechiae was found. Lung auscultation revealed diffuse inspiratory rales. Temperature was 38.5°C. Arterial blood gas analysis while the patient was breathing 81 breaths/min of oxygen indicated that the pH was 7.44, arterial oxygen tension (Pa_{O_2}) was 62 mmHg, arterial carbon dioxide tension (Pa_{CO_2}) was 31 mmHg, and bicarbonate (HCO_3-) was 24 mM. Chest roentgenography showed diffuse bilateral pulmonary alveolointerstitial infiltrates. Electrocardiography was normal, with sinus tachycardia (95 beats/min). Doppler echocardiography was normal without right ventricular enlargement. Left ventricular ejection fraction was normal, and systolic pulmonary artery pressure was 27 mmHg. Computed tomography of the chest (fig. 1) revealed patchy bilateral ground glass densities, macronodular opacities, and alveolar consolidation without pleural effusion.

Bronchoalveolar lavage, performed 15 h after (fig. 2), was hemorrhagic with 520 10^3/ml (59% macrophages, 27% neutrophils, 14% lymphocytes); 60% of the macrophages contained red fat droplets after staining with an Oil Red O stain. Protected minibronchoalveolar lavage samples were sterile on the culture.

Serum total calcium and cholesterol concentrations were decreased to 2.02 mM and 3.5 mM, respectively. C reactive protein was increased to 30 mg/l ($N < 10$). Hemoglobin level was the same level before and after the procedure (11 g/dl) and there was no thrombocytopenia. Fundoscopic evaluation results were normal. No fat globules were found in the urine.

The patient's symptoms improved rapidly with trivial fluid support and supplementary oxygen. Results of chest radiography and blood gas analysis returned to normal within 2 days and allowed early hospital discharge on day 3. Subsequent chest computed tomography was normal 10 days after the acute event.

Discussion

Fat embolism syndrome severity can vary from mild adult respiratory distress syndrome to profound refractory hypoxemia leading to coma and death. Rupture of vessels and damage to adipocytes allow microthombi of lipids to enter the venous circulation. After being trapped in the pulmonary capillaries and hydrolyzed by a pulmonary lipase, free fatty acids cause direct toxic damage of the microvascular and alveolocapillary units and subsequently cause release of vasoactive amines and prostaglandins. The histopathology of FES is characterized by interstitial edema, transudate, and later exudate in the alveoli, the death of type II pneumocytes, and hyaline membrane formation.[1]

Fat embolism syndrome is a well-known complication of traumatic injuries (1% to 35% after long-bone fractures[2,3]), cardiopulmonary resuscitation, or lipid infusion for parenteral feeding[4] but seems to be quite rare after liposuction, only five cases have been reported.[5,6,7,8,9] Of these five cases, four were suspected after evidence of adult respiratory distress syndrome with fever was seen, the fifth after transient, focal neurologic signs were noted after surgery. The latter was diagnosed during fundoscopic evaluation, others were diagnosed during autopsy, pulmonary arteriography, or chest radiography without further investigation. Four patients sur-

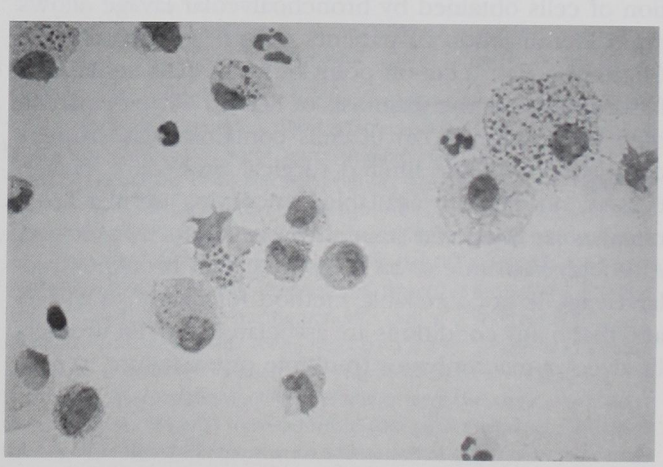

Fig. 2. Light micrograph of cell obtained by bronchoalveolar lavage. Alveolar macrophages with multiple prominent red- or brown-staining cytoplasmic inclusions are shown (old red O; original magnification, ×400).

vived. This patient with FES underwent isolated liposuction without other combined procedures, as described in the other case reports. It does not agree with the assumption that FES after liposuction occurs only after time-consuming, multiple and combined procedures, such as mastopexie or abdominoplasty.[10]

The five reports plus ours emphasized the difficulty of making the diagnosis of FES postoperatively. Many of the diagnostic criteria are insensitive and may reflect response to trauma rather than fat embolism *per se*. The classic clinical triad occurs within 48 h in only 85% of patients, after a free interval of 12–30 h after injury.[11] In our patient, acute respiratory failure was isolated and occurred immediately after the procedure with concomitant infiltrates seen on the chest X-ray. This finding indicates that the inflammatory process might begin earlier than previously reported. It is consistent with experimental data that show that oleic acid-induced pulmonary edema occurs less than 1 h after injection.[12]

In patients with isolated respiratory symptoms, the presence of hypoxemia and pulmonary infiltrates are easily confused with other more commonly encountered problems, such as aspiration pneumonia, left ventricular overload with pulmonary edema, lung contusion, or massive transfusion. In the current case, normal echocardiography excluded heart failure, and sterile, protected minibronchoalveolar lavage eliminated sepsis. Moreover, the outcome was simple without specific therapy. To further establish the diagnosis of FES, bronchoalveolar lavage was performed, and contributed to the diagnosis.

This case report emphasizes that microscopic evaluation of cells obtained by bronchoalveolar lavage allows rapid identification of patients with FES. According to Chastre *et al.*,[13] a cut-off point of 5% of cells containing fat droplets was the diagnosis of FES. In our patient, the high percentage (60%) of cells containing fat droplets contrasted with the limited surgical procedure. Nevertheless, no data are available concerning normal bronchoalveolar lavage fat stain after uneventful liposuction. Although Vedrinne *et al.*[14] reported that bronchoalveolar lavage is not a reliable method for diagnosis of FES and that many conditions are associated with fat droplets in alveolar macrophages (multiple organ failure, sepsis, trauma, neoplasia, lipid infusion, or hypertriglyceridemia), historic background, clinical pattern, and chest imaging results are highly suggestive of this diagnosis. We considered the liposuction to have caused this FES rather than the propofol, which has been described to be an explanation for lipid deposition, but only during long-term infusion.[15]

In conclusion, after liposuction, the incidence of FES might be underestimated because this diagnosis is not systematically considered and because the clinical pattern of FES often is not specific.

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