

# Fat Embolism Syndromes Following Liposuction

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Received: 11 March 2008 / Accepted: 29 April 2008 / Published online: 29 May 2008  
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**Abstract** Fat embolism syndrome (FES) after liposuction is likely a life-threatening disorder, though its incidence is low. The three chief clinical manifestations include respiratory insufficiency, cerebral involvement, and petechial rash. Although FES is a multisystem disorder, the most seriously affected organs are the lungs, brain, cardiovascular system, and skin. Many laboratory findings are characteristic but nonspecific. The pathogenesis of FES after liposuction has been looked at both mechanically and biochemically. Diagnosis is difficult; Gurd and Wilson's diagnostic criteria based on clinical examination is still extensively used in clinics at present. There is no specific therapy for FES after liposuction for the moment, so prevention, early diagnosis, and supportive therapies are important. In this article we discuss the clinical presentation, pathogenesis, and current methods to prevent FES and, if possible, ways to treat this complication.

**Keywords** Fat embolism · Fat embolism syndrome · Complications · Liposuction

Nowadays, liposuction has become popular and commonly performed with few side effects. However, with lipoaspirate volumes increasing in clinical practice, complications have occurred more frequently. Among these complications, fat embolism syndrome (FES) after liposuction, as after other traumatic or nontraumatic injuries, is severe and the mortality rate is high. Separate cases of FES following

liposuction have been reported but there has been no systematic review of it to date.

## Concept of FES and Liposuction

Liposuction is a procedure that allows the surgical removal of excess adipose tissue in healthy individuals. It is popular in many countries and usually performed with few clinical side effects. However, with increased lipoaspirate volumes, complications have been reported. FES is a serious but relatively uncommon clinical disorder seen especially in patients with nontraumatic injuries, including liposuction. Although the incidence of FES after liposuction is very low, it is likely life-threatening.

## Incidence and Mortality

Overall clinical complications of liposuction occur in about 5–10% of the cases but are usually minor, consisting primarily of anesthesia, seroma, edema, pigmentation, pain, and hematoma [1]. The overall mortality from FES after liposuction is approximately 10–15% [1, 2], with higher mortality associated with fulminant FES due to severe right heart failure compared with FES in which the mortality relates largely to underlying respiratory failure (or rarely cerebral edema causing brain death) [3].

## Clinical Presentation

### Respiratory System

The lung is usually the first organ impaired by fat emboli in patients with FES. This localization can be explained by

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the pressure differences in the various vascular beds. It is known that mean capillary pressures are significantly lower in the lung and the liver than in the brain, kidney, and peripheral vascular beds. Respiratory system dysfunction occurs frequently, and its severity may vary, ranging from mild, manifested only with dyspnea and/or tachypnea, to severe, characterized by symptoms indistinguishable from acute respiratory distress syndrome (ARDS) [4].

#### Central Nervous System

Neurologic manifestations appear in as many as 80% of patients with FES [5] and usually precede the development of respiratory symptoms by 6–12 h [6]. CNS dysfunction ranges from mild disorientation to coma. Cerebral edema caused by fat emboli may contribute to neurologic dysfunction [7].

#### Skin

Petechia on the upper body are found in about 50% of patients [8]. The characteristic petechial rash is usually observed on the head, neck, anterior thorax, subconjunctiva, and axilla [9, 10]. This is believed to be the only pathognomonic feature of FES and usually appears on the second or third day after injury [3].

#### Cardiovascular System

The most frequent cardiovascular sign that occurs in patients with FES is tachycardia, but it is not specific and sensitive in the diagnosis of FES.

#### Eyes

Fifty percent of patients with FES experienced retinal manifestation [11]. The changes include Macular edema, frequently found as an isolated sign at first and then along with other fundoscopic changes with the progression of the disease; cotton-wool spots, directly related to systemic flow; and retinal hemorrhage, observed as punctate or flame-like formations found near a blood vessel or elsewhere on the retina [12].

### Laboratory and Imaging Findings

Laboratory findings include a sudden decrease in the hematocrit level, a rise in the serum lipase level, hypoxemia, thrombocytopenia, anemia, and hypocalcemia. These findings are usually present during the first 24–72 h. Many of these laboratory findings have been considered characteristic of FES, but they are nonspecific and possibly occur in patients both with and without the syndrome [1, 3, 13].

#### Detection of Fat Droplets

Fat globules are frequently observed in urine [1, 14], but it is too sensitive to be of value in the diagnosis of FES [15]. Chastre et al. [13] proposed that the bronchoalveolar lavage that can be used to identify fat droplets within cells may be a rapid and specific method for establishing the diagnosis of FES, but the results were inconclusive.

#### Plasma Biochemistry and Hematology

An animal study [16] reported recently that serum aspartate aminotransferase and alanine aminotransferase enzyme levels were elevated significantly postoperatively (10–48 h) in animals subjected to lipoplasty compared with controls, which is in accordance with the previous study [17]. These findings indicate that there is hepatic impairment in the course of FES.

#### Arterial Gas Analysis

Arterial blood gas analysis of patients after liposuction usually indicates hypoxia and hypocapnia, clinical features of respiratory distress. This is important because hypoxia is usually the first sign of the development of FES [3] and the development of clinical syndromes can cause the PaO<sub>2</sub> to fall to 50 mmHg or lower [1, 8, 14].

#### Chest Radiography

Chest X-ray usually appears normal on admission but develops signs of generalized pulmonary interstitial and alveolar opacification over 1–3 days. The typical display is multiple flocculent shadows, defined as the “snowstorm appearance,” caused by diffuse bilateral alveolar infiltration [12]. The radiologic signs may remain for up to 3 weeks [18].

#### Cerebral CT Scan and Cerebral MRI

Computed tomography (CT) and magnetic resonance imaging (MRI) of the brain are used to confirm the extent of organ involvement and exclude alternative pathologies [12]. The high-resolution CT (HRCT) findings of a mild fat embolism consist of bilateral ground-glass opacities and thickening of the interlobular septa. Centrilobular nodular opacities are present in some patients [19].

### Pathophysiology and Pathogenesis

Histopathologic micrographs revealed pathologic changes in the lung, kidney, and brain. In the lung, H&E staining showed alveolar hemorrhagic edema with fat droplet

depositions and fibrin thrombi. Multiple fat deposits were found in the glomeruli of the kidney and in the cerebral capillaries. However, pathologic involvement, including fat droplets in tissues and arterioles, was only 25.0% with renal lesions and 62.5% in the brain compared with all subjects ( $n = 8$ ; 100%) with lesions in the lung [20].

PLA<sub>2</sub>, NO, free radicals, and proinflammatory cytokines (TNF- $\alpha$ , IL-1 $\beta$ , and IL-10) play a role in the pathogenesis of FES-induced ARDS [20]. According to Rae et al. [21], the PLA<sub>2</sub> propeptide was involved in patients with acute injury, which caused neutrophil sequestration and activation in the pulmonary vasculature and interstitium. Based on recent studies in patients with FES, PAF and PLA<sub>2</sub> levels increased in bronchoalveolar lavage fluid [22], and plasma PLA<sub>2</sub> concentrations increased significantly as well [20]. This result supports the involvement of PLA<sub>2</sub> in the pathogenesis of ARDS associated with FES. Hsu et al. [23] suggested that NO may also be involved. NO produced via iNOS (inducible nitric oxide synthase) in alveolar macrophages in FES is toxic to the lung [20, 24].

Studies found that plasma levels of NO<sub>x</sub> and MG, as well as proinflammatory cytokines such as TNF- $\alpha$ , IL-1 $\beta$ , and IL-10, were markedly increased. The formation of MG has been considered a biochemical marker of hydroxyl radicals [25], and free radicals accompanied with proinflammatory cytokines participate in ARDS and other changes associated with FES [20]. On the basis of these findings, FES was considered a systemic inflammatory reaction, which is consistent with Fabian's [26] opinion that the action of lipases results in the release of nonesterified fatty acids into the general blood circulation that induce an inflammatory response.

### The Mechanical Theory

The mechanical theory of FES after liposuction supposes that the fat in the area of trauma or injured tissue is the origin of the pulmonary emboli [1, 18]. The fat droplets enter into veins near the trauma or injured tissue when rupture of the vessels and damage to adipocytes occur, and then are transported to the pulmonary vascular beds and systematic circulation, depositing as fat emboli [27]. Blood vessels blocked by intravascular fat globules range from 10 to 40  $\mu\text{m}$  in diameter [1, 3]. The pulmonary arterial pressure increases following obstruction due to fat emboli and hypoxia and the increased pressure can push the fat emboli into general circulation by which they can be deposited in other organs [28].

### The Biochemical Theory

The biochemical theory proposes that free fatty acids such as chylomicrons are the chief products of FES and they are toxic to pneumocytes and the capillary endothelium [14,

18]. Free fatty acids cause interstitial and alveolar hemorrhage, edema, and chemical pneumonitis by producing endothelial damage, inactivating lung surfactant, and increasing permeability [1, 29]. These events often induce ARDS [30]. Hormone changes secondary to trauma or tissue injury induce a systematic increase of free fatty acids. The release of catecholamines after trauma and tissue injury could mobilize free fatty acids and decrease utilization of fat in the body [18, 28, 29]. The increase in the levels of lipase and lipoprotein lipase and the increase of activity of these lipases release circulating free fatty acids [29]. Other hormonal factors may also be responsible for lung changes. The vasoactive amines released from injured tissue and the histamine released from lung parenchyma could cause pulmonary vasospasm, bronchospasm, and endothelial injury [1]. Besides emboli, products of protein degradation induced by increased adrenocortical hormone secretion and C-reactive protein may also be responsible for aggregation of the chylomicrons [1], which could cause obstruction.

Fulminant fat embolism syndrome and fat embolism syndrome are not caused by the same pathophysiologic effect associated with the systemic liberation of fat. Fulminant fat embolism syndrome is a severe manifestation of acute cardiovascular pulmonary obstruction by fat produced by a sudden intravascular liberation of a large amount of fat. The subsequent platelet aggregation and release of vasoactive and thrombogenic substances contribute to pulmonary hypertension and edema [3] which exacerbate the acute pulmonary vascular obstruction by fat. Severe right heart failure, shock, and even death often occur within the first 1–12 h of injury [31]. Fat embolism syndrome is believed to be caused by the toxic effects of free fatty acids liberated at the endothelial layer which cause capillary disruption, perivascular hemorrhage, and edema [3].

Based on the theories discussed above, mechanical and biochemical mechanisms contribute to both fat embolism syndrome and fulminant fat embolism syndrome. A large amount of fat that escaped from the site of the injured tissue via torn venules to the circulatory system and were produced as a result of blood biochemical changes emerges in the circulatory system and causes an acute pulmonary vascular obstruction, then the fulminant fat embolism syndrome appears. The obstruction and edema in the pulmonary vascular beds produce arterial hypertension, which can lead to severe right heart failure, shock, and often death during the first 12 h of injury.

### Diagnosis

Several approaches have been suggested to confirm the diagnosis of fat embolism syndrome [3], but clinical

**Table 1** Gurd's criteria for the diagnosis of fat embolism syndrome [32]

<i>Major criteria</i>	
1.	Axillary or subconjunctival petechia. This occurs transiently over 4–6 h in 50–60% of patients
2.	Hypoxemia (PaO <sub>2</sub> 60 mmHg; FiO <sub>2</sub> 0.4)
3.	Central nervous system depression disproportionate to hypoxemia and pulmonary edema
<i>Minor criteria</i>	
1.	Tachycardia (110 beats/min)
2.	Pyrexia (38.5°C)
3.	Emboli in the retina on fundoscopic examination
4.	Fat present in urine
5.	Sudden unexplained drop in hematocrit or platelet values
6.	Increasing erythrocyte sedimentation rate
7.	Fat globules in the sputum
8.	Symptoms within 72 h of skeletal trauma
9.	Shortness of breath
10.	Altered mental status
11.	Occasional long tract signs and posturing
12.	Urinary incontinence

examination remains the gold standard [5]. The manifestations of FES were first described by Gurd [32] in 1970, and then refined by Gurd and Wilson [33] in 1974. They stated that at least two major symptoms or signs or one major and four minor symptoms or signs must be present to diagnose the syndrome (Tables 1 and 2). The three classic symptoms of FES, respiratory distress, cerebral dysfunction, and petechial rash, usually appear within 24–72 h after the procedure [27]. Symptoms of fat embolism include tachycardia, tachypnea, fever, hypoxemia,

**Table 2** Criteria for the diagnosis of fat embolism syndrome according to Gurd and Wilson [33]

<i>Major criteria</i>	
1.	Respiratory insufficiency
2.	Cerebral involvement
3.	Petechial rashes
<i>Minor criteria</i>	
1.	Pyrexia (usually 39°C)
2.	Tachycardia (120 beats/min)
3.	Retinal changes (fat or petechia)
4.	Jaundice
5.	Renal changes (anuria or oliguria)
6.	Anemia (a drop of more than 20% of the admission hemoglobin value)
7.	Thrombocytopenia (a drop of 50% of the admission thrombocyte value)
8.	High erythrocyte sedimentation rate (ESR 71 mm/h)
9.	Fat macroglobulinemia

**Table 3** Fat embolism index [36]

Presentations	Points
Diffuse petechia	5
Alveolar infiltrates	4
Hypoxemia (<70 mmHg)	3
Confusion	1
Fever > 38°C	1
Heart rate > 120 beats/min	1
Respiratory rate > 30/min	1
FES = 5 or more points	

hypocalcemia, hypocapnia, thrombocytopenia, and occasionally mild neurologic symptoms.

Another diagnostic system [34] has been suggested. Schonfeld et al. [35] proposed a fat embolism index that gives points for different diagnostic criteria (Table 3). A diagnosis of FES is made with a score of 5 or more.

Arterial blood gas analysis is important because the development of clinical syndromes can cause PaO<sub>2</sub> to drop to 50 mmHg or lower [1, 8, 14]. Detection of fat globules in blood, urine, and sputum is not particularly helpful in confirming the diagnosis. Almost all patients with fractures and tissue injury have fat emboli [3], but few develop FES [9, 36]. Chastre et al. [13] suggested that use of bronchoalveolar lavage (BAL) for diagnosing FES was rapid and specific. Globules in cells from BAL permit rapid identification of abnormal fat globules in the lungs of patients with FES [1, 13]. Unexplained anemia and thrombocytopenia are often found. Hypocalcemia and elevated lipase are also reported [22].

Imaging findings play an important role in confirming the diagnosis of FES. Chest radiography, noncontrast CT scan, high-resolution CT, ventilation-perfusion imaging, MRI, transcranial Doppler sonography, and intraoperative transesophageal echocardiography are included. However, Georgopoulos and Bouros [4] suggest that all these laboratory investigations still lack the specificity to diagnose FES, but can be helpful in excluding some cases.

## Prevention and Treatment

There is no specific therapy for FES at present, so prevention, early diagnosis, and supportive therapies are very important [3]. Prevention of FES should include the following:

- (1) Careful selection of patients and techniques. Liposuction is generally indicated for localized areas of fat in relatively young patients with good skin [14].
- (2) Differentiation of fat emboli from thrombotic emboli. The differential diagnosis is very important because they are treated differently [14].

- (3) Reduction of surgical time and the amount of fat aspirated. Long anesthesia time, hypotension, high concentrations of oxygen, blood transfusions, and large fluid shifts may set up the lung for injury from fat emboli [1].
- (4) Appropriate and aggressive attendance. Prognosis will be favorable if there is appropriate and aggressive management [37, 38].
- (5) Fluid treatment for a minimum of 24 h postoperatively. The fat particle could enter into the circulation system and then initiate FES during the liposuction procedure, therefore, fluid administration intravenously to clear the globules is essential during the first 24 h after the operation [6].
- (6) Careful attendance and patient postoperative monitoring. We should not release the patient from the hospital the day of the procedure [38].

Fat embolism syndrome is a self-limiting disease [3, 18], correlating with respiratory distress, so therapeutic measures are aimed mainly at improving respiratory conditions during the disease [3, 8] and which include (1) spontaneous ventilation when hypoxia appears [39], (2) continuous positive airway pressure or CPAP, and (3) mechanical ventilation and positive end expiratory pressure (PEEP). When  $\text{FiO}_2 > 60\%$  and  $\text{CPAP} > 10 \text{ cmH}_2\text{O}$ , endotracheal intubation, mechanical ventilation, and PEEP can be used to achieve a  $\text{PaO}_2 > 60 \text{ mmHg}$  [40]. However, both mechanical ventilation and PEEP have no intrinsic beneficial value in dealing with a pulmonary fat embolism and may even induce acute damage; therefore, the purpose of mechanical ventilation and PEEP is to acquire adequate gas exchange without further pulmonary injury [3].

Pharmaceutical therapy is also considered in dealing with FES. Many agents have been used to treat FES over the years, including 2% sodium bicarbonate solution, choline, Trasylol, and clofibrate [41, 42]. Ethyl alcohol and heparin are of no significant benefit and are seldom used. Lipoprotein lipase, considered a clearing factor, is released after intravenous injection of heparin; this is called the “heparin effect” [29]. Low-molecular-weight dextran is helpful for decreased blood viscosity, reducing platelet adhesion, reversing thrombocytopenia, and reducing cell aggregation [26]. Based on the study by Korhan and Hakan [14], human albumin should be given for fluid replacement because it has the ability to bind FFAs (free fatty acids). However, it has been suggested that there is no evidence that infusing albumin reduces the effects of FFAs, or hyperoncotic albumin increases plasma oncotic pressure [43]. The infused albumin may even accumulate in the pulmonary interstitial compartment and worsen the respiratory failure [44]. Besides, nitric oxide and prostacyclin are considered beneficial in pulmonary supportive therapy [3].

Finally, steroids are used extensively in clinics when FES occurs. The hydrolysis of the fat into FFAs and glycerol results in a significant rise in blood FFAs. The FFAs acting locally lead to an increase in the permeability of the capillary bed, destruction of the alveolar architecture, and damage to lung surfactant. It is at this point that the use of hydrocortisone in massive doses may be of value [15]. Methylprednisolone limits the increase of FFAs, diminishes the inflammatory response, inhibits complement-mediated leukocyte aggregation, protects capillary integrity, stabilizes lysosomal membranes, and minimizes interstitial edema accumulation [18, 27, 29]. It is used frequently to treat FES in clinics, but the dose and optimal timing of administration have not been established and should be further studied to make certain.

In summary, FES occurs after many procedures, including liposuction. Preventive measures include careful selection of patients and techniques, patient attendance and postoperative monitoring, reduction of surgical time, and attention to the amount of fat aspiration. Treatment is largely symptomatic and should be mainly supportive.

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