CASE REPORT

Fat attacks!: a case of fat embolisation syndrome postliposuction

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SUMMARY

Liposuction is a procedure commonly performed in the UK usually with a low incidence of serious sequelae; however with larger patients and increased volumes of lipoaspirate, complications have been reported more frequently. One of the rare but very serious complications postliposuction is fat embolism syndrome (FES), a life-threatening condition difficult to diagnose and limited in treatment. The authors present the case of a 45-year-old woman who was admitted to the intensive care unit postelective liposuction for bilateral leg lipoedema. She presented with the triad of respiratory failure, cerebral dysfunction and petechial rash requiring a brief period of organ support. This case highlights that with the recent increase in liposuction procedures worldwide, FES is a differential to always consider. Although still a rare condition this article emphasises the importance of thinking outside the box and how to identify and manage such a life-threatening complication.

BACKGROUND

Liposuction is a procedure commonly performed in the UK usually with a low incidence of serious sequelae; however with larger patients and increased volumes of lipoaspirate, complications have been reported more frequently.^{1–3} Previous reported cases of fat embolism syndrome (FES) following liposuction are rare, but have each described it in the context of liposuction of the abdominal and trochanteric regions with or without abdominoplasty.^{4–5} To the authors' knowledge no case report of FES postliposuction have been reported in the UK.

Fat embolisation describes the pathophysiological phenomenon of fat globules being present within peripheral microcirculation or lung tissue,⁶ common after long bone fractures or major trauma. In a small number of cases however patients go on to develop the uncommon and potentially fatal FES. It is notoriously difficult to diagnose and features include the triad of respiratory failure, cerebral dysfunction and petechial rash.⁷ Usually presenting 24–72 hours after the initial injury,⁷ the underlying mechanisms are still not clear. However current evidence suggests trauma leading to fat globules or free fatty acids causing microvascular occlusion and consequent end organ dysfunction.

CASE PRESENTATION

We present the case of a 45-year-old woman had a background history of morbid obesity (body mass

index (BMI) 65 kg/m²), bilateral leg lipoedema, previous gastric bypass, laparoscopic cholecystectomy, tonsillectomy and depression. The patient was not on any medication which was related to this acute admission.

She had undergone complex bilateral lower leg and knee liposuction 48 hours prior at a local hospital. The surgery had been planned to remove some of the bulk of her lower legs to help her mobilise and subsequently begin the weight loss process. She underwent liposuction via the tumescent technique and approximately 10–13.5 L of lipoaspirate was removed. The intraoperative period was uneventful, she was extubated an hour after the end of the procedure and transferred to the high dependency unit as planned.

Approximately 36–39 hours after the operation the patient became drowsy with a respiratory acidosis and tachycardia as well as a falling urine output. Initial treatment consisted of fluid resuscitation and as she continued to deteriorate she was transferred to intensive care (ITU) with suspected acute respiratory distress syndrome (ARDS).

On arrival initial observations showed a heart rate of 110, blood pressure of 106/45 mmHg, temperature of 36.6°C and SpO₂ of 98% on 15 L O₂. The transferring team had stated prior to transfer she had been desaturating to 80% on removing her face mask. On auscultation heart sounds were normal with no audible murmurs and bilateral inspiratory crackles were heard throughout both lung fields. On neurological examination she was drowsy with an initial Glasgow Coma Score (GCS) of 12, she was profoundly agitated and not complying with therapy, no focal neurology or cranial nerve pathology was found on examination, pupils were equal and reactive to light. No abnormalities were noted on examination of the conjunctiva or sclerae. On examination of the patient's upper arms she had bilateral petechiae. Abdominal examination showed a soft and non-tender abdomen to examination with active bowel sounds. On her lower legs bilateral leg lymphoedema with surgical wounds were noted, these wounds were clean with no signs of infection.

Initial arterial blood gas analysis (ABG) on an FiO₂ of 55% showed pH 7.190, PaCO₂ 29.8 kPa, PaO₂ 14.2 kPa and HCO₃⁻ 22 mmol/L. Initial plain chest film showed bilateral diffuse infiltrates suggestive of ARDS (figure 1). Chest X-ray (CXR) showed indication of bilateral generalised infiltrates (figure 1).

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Figure 1 Chest X-ray on admission showing bilateral infiltrates.

Shortly after arrival she became unresponsive with a sudden drop in heart rate and blood pressure. She was given 800 µg of atropine to which she responded well. The decision was made to intubate her via a rapid sequence induction. Initially she proved difficult to ventilate and as well as respiratory support she required inotropic therapy to maintain her blood pressure.

INVESTIGATIONS

The patient also had blood work completed on admission to the intensive care unit: white cell count $13.4 \times 10^9/L$ (neutrophils $10.38 \times 10^9/L$, lymphocytes $1.75 \times 10^9/L$, monocytes $1.15 \times 10^9/L$), haemoglobin 87 g/L, platelets $172 \times 10^9/L$, C reactive protein 245 mg/L.

She had a slightly deranged coagulation profile and raised troponins—international normal ratio 1.68, prothrombin time 23.9. Initial high sensitivity troponin-T 40 ng/L with a 6-hour repeat showing as 45 ng/L.

Liver function tests showed - alanine aminotransferase 45 U/L, albumin 25 g/L, alkaline phosphatase 87 U/L and bilirubin 14 µmol/L. Her renal function remained normal (creatinine 57 µmol/L and estimated glomerular filtration rate >90).

As part of the patient's routine workup she had blood cultures which grew *Staphylococcus epidermidis*, which was thought to be a contaminant; serial repeat cultures were negative on final testing. The patient continued to have thick frothy secretions suctioned which were cultured; these were negative for growth of an organism. Urine microscopy was negative for any cell casts or free fat globules.

The patient also had a CT study of her head which did not show any acute findings. Initial ECG showed a sinus tachycardia and transthoracic echocardiography (TTE) showed a non-dilated left ventricle with hyperdynamic systolic function and a slightly dilated right ventricle also with hyperdynamic systolic function. No valve abnormalities were identified on TTE.

DIFFERENTIAL DIAGNOSIS

Differentials for respiratory failure in a large patient postoperatively should include pulmonary embolism, pneumothorax, hypoventilation, residual anaesthesia, cardiac failure and mechanical obstruction. Fat embolus is usually quite low down on the list and is usually suggested as being a diagnosis of exclusion.¹ However in patients with comorbidities and risk factors for fat embolism—in this case a high BMI, lymphoedema and

large volume lipoaspirate,² the importance of considering FES as a diagnosis increases.

Currently, there are no gold standard nor validated diagnostic criteria for FES. Diagnosis is usually made via recognition of characteristic clinical criteria and supporting imaging in the context of the presentation. A variety of techniques have been employed to assist in the diagnosis, however none have been specific. Gurd and Wilson⁸ developed diagnostic criteria with particular emphasis on central nervous system signs, chest signs and chest radiographs. Schonfield *et al*⁹ incorporated an arterial blood gas component. They developed set criteria on a points based system (figure 2) that made the diagnosis more or less likely. The diagnosis based on arterial blood gas and pulmonary features has been used by Lindeque *et al*¹⁰ (figure 2).

When faced with a patient at risk of FES clinical examination still remains the most useful tool. The presence of respiratory failure, neurological insult and cutaneous or conjunctival petechiae^{11 12} should raise suspicion. The presentation is normally delayed, occurring after 12 hours with retinal and axillary petechiae and seen up to 72 hours postpresentation. These petechiae occur in up to 50% of patients and are the result of extravasated red blood cells secondary to microemboli.^{12 13} Neurological features such as reduced consciousness, focal deficits and seizures are apparent in about 80% of cases, normally developing after apparent respiratory failure,¹⁴ however this is subject to debate.³

TREATMENT

The patient was initially ventilated and given respiratory support with positive pressure as well as inotropic support with norepinephrine. Sedation was maintained with a propofol and fentanyl infusion. Diuretics were also trialled with limited success in the treatment of the chest. Microbiology suggested antibiotic cover in light of the raised inflammatory markers but these were then stopped in light of the final diagnosis. She also received deep vein thrombosis prophylaxis as well as ranitidine while intubated.

OUTCOME AND FOLLOW-UP

The patient was extubated after 8 days, she was de-escalated to a medical ward after 12 days and discharged from the hospital 2 days later. She was followed up 2 months later and had recovered well. She had no sequelae from her admission to ITU and had made some progress towards weight loss.

DISCUSSION

Recognising FES remains a significant challenge for most physicians even though it was first recognised as early as 1873.¹⁵ Although most commonly associated with major trauma, it is frequently an asymptomatic presentation after long bone fractures and orthopaedic intervention.¹⁶

Fat embolism caused by liposuction is a rare complication with few case reports published worldwide and none to date in the UK. Reviews on the topic discuss the clinical and radiological features of fat embolism;^{3 16 17} currently the diagnosis is clinical but as access to prompt radiological investigations increases, CT findings are becoming an important part of increasing the likelihood of diagnosis as well as ruling out other differential diagnoses.¹⁷

We reached our diagnosis of FES clinically; our patient met all four major Gurd's and Wilson's criteria with respiratory

Figure 2 Diagnostic criteria for FES. FE, fat embolisation; FES, fat embolism syndrome.

failure as she required an FiO_2 of 0.5 on admission; she was also drowsy and confused with notable petechiae. The CXR did indeed show bilateral infiltration consistent with ARDS. According to the Schonfield FEI Score she scored 15/15 on presentation.

As for the pathophysiology it is not fully understood but the current prevailing theories for explaining end organ damage are the mechanical obstruction theory and the biochemical injury theory.⁷ The mechanical obstruction theory proposes that intravascular fat globules are highly proinflammatory generating platelet aggregation and fibrin formation leading to mechanical obstruction of venous beds, and on travelling to the right heart can increase pressures passing through to the arterial system via a patent foramen ovale.^{1 6 18} This is how the characteristic neurological and dermatological features of FES are caused.⁶ Of interest is that the occlusion is usually temporary or partial owing to the fluidity and malleability of the fat globules.

The biochemical injury theory describes neutral fat broken down into free fatty acids which have a directly toxic effect on pneumatocytes, increased vascular permeability and endothelial dysfunction.^{1 6} Infusion of free fatty acid has been shown to induce FES in animal models³ and is used in the creation of animal models to study FES. The various manifestations of the syndrome are usually a combination of these mechanical and biochemical pathways exacerbated by the manifestations occurring in both arterial and venous circulation.⁶

Should one suspect FES, management is non-specific and largely supportive, essentially ensuring good arterial oxygenation. High inspired oxygen concentrations to maintain adequate saturations and arterial oxygen tension in the normal range is paramount.¹⁹ This may need to be achieved with forms of positive pressure ventilation. Fluid control via restriction or diuresis to minimise accumulation in the lungs can help achieve this as long as good circulation is maintained. One however needs to maintain a good intravascular volume in order to provide an adequate circulation and organ perfusion especially since shock is a common complication of FES. This can be achieved with both crystalloid and albumin solutions,²⁰ the latter being particularly useful as it has been shown to bind free fatty acids as well as increase intravascular volume. Specific drugs and surgery in the management of FES have largely proven unsuccessful.¹⁹

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Learning points

- Liposuction is a procedure that is growing more common worldwide and is being done in higher-risk patient groups with more comorbidities.¹ While generally a safe procedure it is important to consider the potential of fat embolism syndrome (FES) as a diagnosis in the postprocedure period. The risk of developing FES can also be reduced by careful patient selection¹ and reduction of lipoaspirate volume.²
- FES is also a difficult condition to initially diagnose especially after a procedure not normally associated with it, such as liposuction. There are many differentials ranging from infection (eg, pneumonia) to pulmonary embolism or even anaesthetic complications. What makes it even harder is the lack of a standardised set of diagnostic criteria to assist the clinician in making the diagnosis. Among these dilemmas is the lack of a specific management protocol with treatment mainly being supportive.
- In this report we have hoped to provide an unusual complication of liposuction and given a brief overview of how to suspect, diagnose and manage the condition.

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