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CLINICAL INFORMATION

Severe fat embolism in perioperative abdominal liposuction and fat grafting☆

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Abstract
Background and objectives: Fat embolism syndrome may occur in patients suffering from multiple trauma (long bone fractures) or plastic surgery (liposuction), compromising the circulatory, respiratory and/or central nervous systems. This report shows the evolution of severe fat embolism syndrome after liposuction and fat grafting.
Case report: SSS, 42 years old, ASA 1, no risk factors for thrombosis, candidate for abdominal liposuction and breast implant prosthesis. Subjected to balanced general anesthesia with basic monitoring and controlled ventilation. After 45 min of procedure, there was a sudden and gradual decrease of capnometry, severe hypoxemia and hypotension. The patient was immediately monitored for MAP and central catheter, treated with vasopressors, inotropes, and crystalloid infusion, stabilizing her condition. Arterial blood sample showed pH = 7.21; PCO_2 = 51 mmHg; PO_2 = 52 mmHg; BE = −8; HCO_3 = 18 mEq L^{-1}, and lactate = 6.0 mmol L^{-1}. Transthoracic echocardiogram showed PASP = 55 mmHg, hypocontractile VD and LVEF = 60%. Diagnosis of pulmonary embolism. After 24 h of intensive treatment, the patient developed anisocoria and coma (Glasgow coma scale = 3). A brain CT was performed which showed severe cerebral hemispheric ischemia with signs of fat emboli in right middle cerebral artery; transesophageal echocardiography showed a patent foramen ovale. Finally, after 72 h of evolution, the patient progressed to brain death.
Conclusion: Fat embolism syndrome usually occurs in young people. Treatment is based mainly on the infusion of fluids and vasoactive drugs, mechanical ventilation, and triggering

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factor correction (early fixation of fractures or suspension of liposuction). The multiorgânico involvement indicates a worse prognosis.

PALAVRAS-CHAVE
Embolia gordurosa;
Peroperatório;
Lipoaspiração

Embolia gordurosa grave no peroperatório de lipoaspiração abdominal e lipoenxertia

Resumo
Justificativa e objetivos: A Síndrome da Embolia Gordurosa (SEG) pode acontecer em pacientes vítimas de politrauma (fratura de ossos longos) ou operações plásticas (lipoaspiração), comprometendo circulação, respiração e/ou sistema nervoso central. O presente relato mostra evolução de SEG grave após lipoaspiração e lipoenxertia.
Relato do caso: SSS, 42 anos, ASA 1, sem fatores de risco para trombose, candidata a lipoaspiração abdominal e implante de prótese mamária. Submetida à anestesia geral balanceada com monitorização básica e ventilação controlada. Após 45 minutos de procedimento, houve queda súbita e progressiva da capnometria, hipoxemia e hipotensão grave. Imediatamente foi monitorizada com PAM e cateter central, tratada com vasopressores, inotrópicos e infusão de cristaloides, obtendo estabilização do quadro. Amostra sanguínea arterial mostrou pH = 7,21; PCO_2 = 51 mmHg; PO_2 = 52 mmHg; BE = −8; HCO_3 = 18 mEQ/l e lactato = 6,0 mmol/l. Ecocardiograma transtorácico mostrou PSAP = 55 mmHg, VD hipocontrátil e FEVE = 60%. Diagnóstico de embolia pulmonar. Após24 h de tratamento intensivo, a paciente evoluiu com anisocoria e coma com escala de glasgow 3. Realizada TC de encéfalo que evidenciou isquemia cerebral grave, hemisférica, com sinais de êmbolos de gordura em A. cerebral média D; o ecocardiograma transesofágico mostrou forame oval patente. Finalmente, após 72 h de evolução, a paciente evoluiu para morte encefálica.
Conclusão: A SEG ocorre geralmente em jovens. O tratamento baseia-se principalmente na infusão de líquidos e drogas vasoativas, ventilação mecânica e correção do fator desencadeante (fixação precoce de fraturas ou suspensão da lipoaspiração). O comprometimento multiorgânico indica pior prognóstico.

Introduction

The presence of fat emboli or free fatty acids in the pulmonary or systemic circulation can trigger the fat embolism syndrome (FES), often originated from long bone fracture of the lower limbs and pelvis. On a smaller scale, it can result from cosmetic surgery such as liposuction and/or fat grafting, cardiopulmonary bypass, pancreatitis, joint repair, severe burns, sickle cell anemia, diabetes mellitus, and lipid parenteral infusion.[1,2]

FES is a relatively rare condition (0.3–5.0%), but extremely severe, with mortality rates ranging from 10% to 36%.[1,2]

Historically, the first description of FES happened in the mid-nineteenth century on autopsy findings by Zenker.[3] Later, still in the same century, Von Bergman made the first clinical diagnosis of FES,[4] describing the classic triad characterized by acute respiratory failure with diffuse pulmonary infiltrate, neurological dysfunction and skin manifestations (petechiae). However, this triad occurs in only 0.5–2.0% of cases.[5] The passage of emboli into the systemic circulation and the severe neurological involvement (coma), as well as other organs, are indicative of a poor prognosis, especially the possibility of interatrial communication (persistence of patent foramen ovale).[6]

This report shows the fatal outcome of a massive fat embolism case during the intraoperative period of an abdominal liposuction and fat grafting due to the presence of patent foramen ovale, culminating with severe embolic stroke and brain death.

Case report

SSS, 42 years old, ASA 1, with no risk factors for deep vein thrombosis, candidate for abdominal liposuction and breast prosthesis implant. The patient was anxious and fearful about the possibility of complication and death. Therefore, general anesthesia was the option, a less common practice for this type of procedure at the Service (thoracic epidural anesthesia with sedation is the standard practice). The general anesthesia was balanced with propofol, remifentanil, atracurium, sevoflurane, and controlled ventilation with 90% end-tidal oxygen. Basic monitoring was performed (ECG, capnography, oximetry,

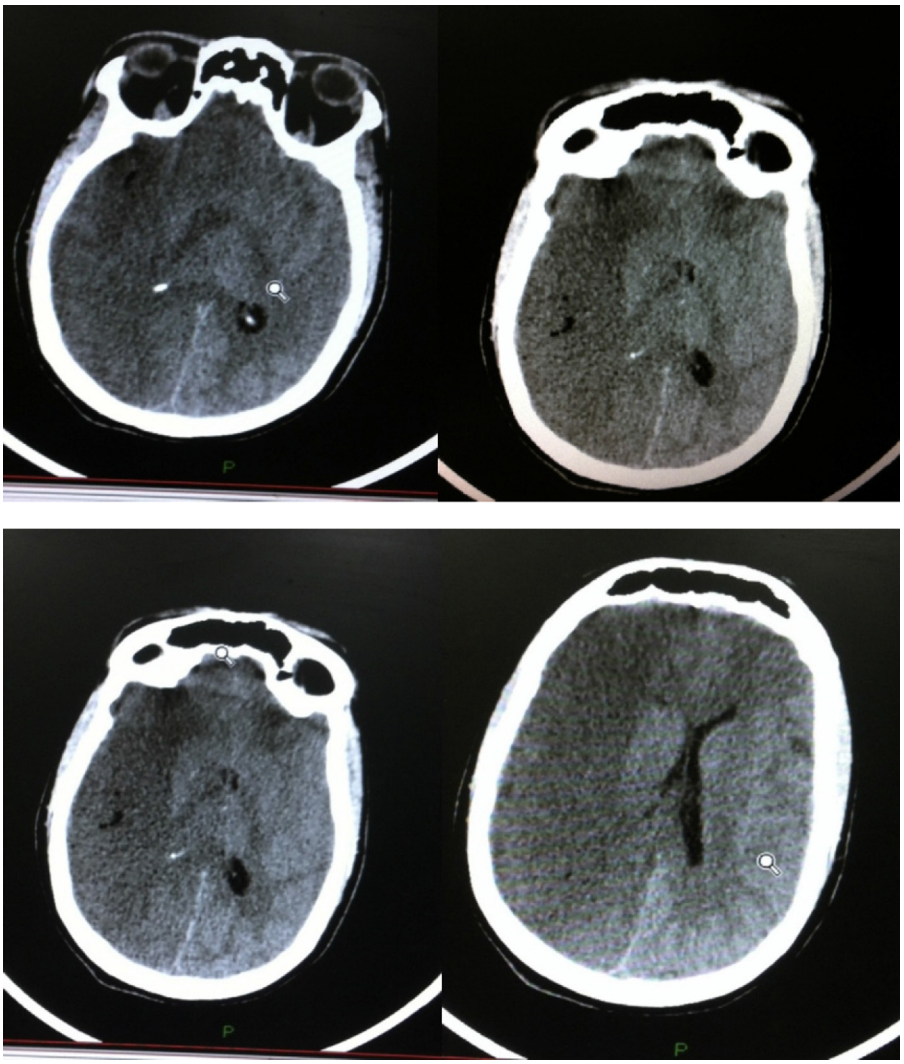

Figure 1 CT with fat emboli in middle cerebral artery and signs of severe ischemia.

and NIBP). After 45 min of unchanged procedure, there was a sudden and progressive fall in capnometry, hypoxemia, and severe hypotension, coinciding with the time of gluteal fat grafting in the prone position. Immediately, the patient was placed in the supine position and monitored with MAP and central catheter. She was treated with vasopressors, inotropes, and infusion of crystalloid, obtaining the stabilization of the clinical picture. Arterial blood sample showed pH = 7.21; PCO_2 = 52 mmHg; PO_2 = 51 mmHg; BE = −8; HCO_3 = 18 mEq L^{-1}, lactate = 6.0 mmol L^{-1}, hemoglobin = 11.8 g dL^{-1}, platelet count = 120,000, sodium = 139 mmol L^{-1}, potassium = 5.9 mmol L^{-1}, and glucose = 254 mg%. Transthoracic echocardiography performed in the operating room showed PASP = 55 mmHg, hypocontractile RV, and LVEF = 60%. The hypothesis of pulmonary fat embolism was strengthened and it was decided to refer the patient to the intensive care unit (ICU) for supportive treatment. After 24 h of intensive care, the patient developed anisocoria and coma with Glasgow scale 3. The brain CT performed showed severe cerebral ischemia, hemispherical, with signs of fat emboli in right middle cerebral artery (Fig. 1); transesophageal echocardiography showed patent foramen ovale. Unfortunately, after 72 h of evolution, the patient developed brain death.

Discussion

FES is clinically underdiagnosed due to the low specificity and sensitivity of laboratory tests and physical examination. Moreover, the diagnostic confusion with other syndromes (e.g., thromboembolism, myocardial infarction, acute respiratory distress syndrome, among others) often delay the diagnosis. The classic triad involving acute respiratory failure, neurological dysfunction, and petechiae is infrequent and is manifested after 24–72 h, usually after long bone trauma.[7] The increased serum concentration of lipase or presence of lipiduria may assist in diagnosis, as well as imaging tests (MRI).

Schaikh et al. highlighted the importance of brain MRI, especially in those patients who develop sensory disturbances or neurological deficits.[8]

Gurd and Wilson, in the 1970s, established major and minor diagnostic criteria for FES requiring at least one major

Table 1 Gurd's diagnostic criteria.[1]

Major criteria: acute respiratory failure, central nervous system depression, skin and mucosal disorders (petechiae)
Minor criteria: tachycardia, fever, retinal changes, low hematocrit (unexplained anemia), increased erythrocyte sedimentation rate (ESR) and thrombocytopenia, fat globules in the urine or in the secretion of bronchoalveolar lavage

and three minor criteria or two major and two minor criteria to confirm the syndrome (Table 1).[1] The adoption of these criteria is still useful for the diagnosis of FES in current clinical practice.

Under general anesthesia, respiratory symptoms, especially hypoxemia, may be masked by mechanical ventilation with high fraction of inspired oxygen. In our case, the fall in capnography was the first change, indicating low pulmonary perfusion, followed by oxygen desaturation around 90%. The early detection of the complication allowed therapeutic measures to be taken in a timely manner, before a possible cardiac arrest. The latter could occur if the anesthetic technique chosen was epidural, because of the chance of poor response to vasopressors due to installed vasoplegia and relative hypovolemia.

FES's pathophysiology involves endothelial dysfunction by the release of fatty acids from fat emboli, leading to vasculitis with activation of platelet aggregation and consumption of clotting factors. This process may be perpetuated causing microcirculation occlusion, thrombocytopenia, disseminated intravascular coagulation, and bleeding, the latter being rarer.[8] The central nervous system and other organs involvement indicates the passage of fatty microemboli and/or free fatty acids into the systemic circulation through anatomical pulmonary shunts and/or communication between the right and left heart chambers.[6,8] The foramen ovale, which is an opening (communication) between the two atria present in the fetal circulation, is closed right after birth, but in about 10–25% of the adult population it may become patent by any increased pressure in the right chambers of the heart.[6,8]

In the present case, the choice of general anesthesia and the ventilation mode with positive expiratory pressure may have contributed to open the patient's interatrial communication. The positive end-expiratory pressure used in mechanical ventilation may lead to hemodynamic changes, such as increased cardiac work, and pressure changes in the right heart chambers, with consequent right-left shunt, if there is any communication between the chambers.[9]

Clinically, it is not possible to screen patients for the presence of a patent foramen ovale in the pre-anesthetic evaluation, as the functional capacity and auscultation are normal in most cases. The gold standard for the diagnosis of patent foramen ovale is transesophageal echocardiography, which should be performed in all patients eligible for liposuction or fat grafting, a fact that would make the majority of these surgeries unfeasible.[3,6] Mueller et al. reported a case of paradoxical cerebral embolism due to persistent foramen ovale. The patient developed quadriplegia and cognitive impairment.[10] On the other hand, Folador et al. reported a successful case after liposuction, which progressed to massive pulmonary fat embolism (without cerebral embolization), and the patient survived without sequelae only with supportive treatment.[11]

FES treatment is supportive and includes the management of respiratory dysfunction, hemodynamic, and early fixation of long-bone fractures. Corticosteroids may be useful for prevention, but it has not been proven to be effective for overt syndrome. In theory, these drugs limit the endothelial damage caused by the free fatty acids.[12,13]

Performing esthetic procedures in tertiary hospitals refers to the security required for anesthesiologists to exercise their function comfortably, which allows offering the patient the best technique indication and the best diagnostic and therapeutic resources. Platt et al. reported deaths after liposuction operations due to pulmonary fat embolism, in addition to other deaths by lidocaine poisoning and fluid overload.[14]

In this report, the FES diagnosis was done while in the operating room, through clinical suspicion and the transthoracic echocardiography performed. The intensive care unit received the patient within minutes, already with invasive monitoring and vasoactive amine support, a fact that may not occur in a non-hospital environment.

Conclusion

FES can occur both in critically ill patients, victims of long-bone or multiple traumas, and in candidates for surgical liposuction, a fact that should alert anesthesiologists for early diagnosis and treatment, as well as for the minimum conditions of work required to provide security. Fat grafting is potentially dangerous because it increases the risk of FES due to accidental intravascular injection of fat emboli. Neurological impairment and a higher number of affected organs indicate a worse prognosis of FES.[15,16]

Conflicts of interest

The authors declare no conflicts of interest.

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