Case Report

Journal of INTERNATIONAL MEDICAL RESEARCH

Journal of International Medical Research
48(8) 1–6

DOI: 10.1177/0300060520949109
journals.sagepub.com/home/imr
SAGE

Severe fat embolism after autologous fat grafting in vaginal tightening and breast augmentation surgery

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Abstract

Autologous fat grafting has become increasingly popular in the field of aesthetic surgery because of its biocompatibility and low donor site morbidity. However, some fatal complications may occur following the surgery. We herein describe a woman who developed severe fat embolism after autologous fat grafting for vaginal tightening and breast augmentation surgery. The patient developed symptoms of dyspnea and hypoxemia. Computed tomography pulmonary angiography showed multiple filling defects in the bilateral pulmonary arteries and branches with a maximum size of approximately 1.2×0.7 cm. Fat embolism was assumed to have occurred by injection of fat directly into the vessels around the vagina. Further research is needed to elucidate the anatomical mechanism underlying this phenomenon.

Keywords

Autologous fat grafting, fat embolism, vaginal tightening surgery, breast augmentation, aesthetic surgery, complication

Date received: 27 March 2020; accepted: 17 July 2020

Introduction

Autologous fat grafting (AFG) has become increasingly popular and widely used in both reconstructive and aesthetic plastic surgery. It has been successfully used in facial contouring, hand rejuvenation, and scar contracture repair.[1] Fat grafting for breast augmentation and reconstruction has shown great aesthetic outcomes with

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high patient satisfaction regarding the size, shape, and texture of the breast mound.[2] The use of AFG for buttock augmentation allows focused augmentation and contouring of the gluteal and thigh region with reduction of adjacent body regions, achieving improvement of the overall body aesthetics.[3] Many physicians have recently begun to expand the application of fat grafting in genital surgery, mainly vaginal tightening and labia majora augmentation.[4,5] AFG is considered to be a safe and minimally invasive procedure with non-fatal complications such as infection, fat necrosis, oil cyst formation, and calcification.[6] However, severe complications have also been reported, including stroke and fat embolism. For example, reflux of fat into the ophthalmic or cerebral artery may occur when transplanting fat into critical areas of the face, resulting in blindness or cerebral infarction.[7,8] Fatal fat embolism may also occur by inadvertent injection of fat into the gluteal vein plexus.[9] However, no cases of fat embolism after AFG in vaginal or breast surgeries have been previously reported. We herein describe a woman who developed severe fat embolism after AFG in vaginal tightening and breast augmentation surgery.

Case Report

A 39-year-old woman presented to the emergency department with dyspnea and hypoxemia. She had undergone liposuction at another hospital, during which 600 mL of fat had been taken from the thighs; 270 mL of fat had been injected into each breast, and 50 mL of fat had been injected into the vagina. Fifteen minutes after the operation, the patient developed sudden chest tightness with cyanosis of the lips. Electrocardiographic monitoring showed hypoxemia and tachycardia (SpO_2 of 48%, blood pressure of 58/40 mmHg, heart rate of 110–130 beats/minute, and respiratory rate of 20–31 breaths/minute). Routine blood examination showed mild leukocytosis (white blood cell count of 28.5×10^9/L with 93.6% neutrophils). Arterial blood gas analysis (ABG) showed a $PaCO_2$ of 37 mmHg, PaO_2 of 86 mmHg, bicarbonate concentration of 15.5 mmol/L, and lactic acid concentration of 2.8 mmol/L with a pH of 7.23. Cardiac injury markers were normal. The patient had no cough, hemoptysis, loss of consciousness, or chest pain. She received dopamine to increase her blood pressure and provide rehydration, and she was then transferred to the emergency department of our hospital. She denied smoking or any drug abuse. The patient had no high-risk factors for thrombosis and no history of spontaneous abortion.

Physical examination showed an SpO_2 of 98% while using a mask for oxygen inhalation at 10 L/min. ABG showed a $PaCO_2$ of 32.9 mmHg, PaO_2 of 74.1 mmHg, bicarbonate concentration of 19.4 mmol/L, and lactic acid concentration of 1.3 mmol/L with a pH of 7.23. Cardiac injury markers were significantly increased (creatine kinase, 442 U/L, D-dimers, 2.37 mg/L FEU; and N-terminal pro-B-type natriuretic peptide, 507 pg/mL). Immune indicators and thrombus indicators were negative. Computed tomography pulmonary angiography (CTPA) revealed multiple filling defects in the bilateral pulmonary arteries and branches, with a maximum size of approximately 1.2×0.7 cm (Figures 1, 2), and multiple patchy shadows in both lower lungs. The main pulmonary artery was slightly widened, reaching about 2.7 cm at its widest point. Echocardiography showed no pathological cardiac structures.

The patient was diagnosed with fat embolism after AFG. She received anticoagulation therapy (low-molecular-weight heparin, 4000 U inhaled every 12 hours), methylprednisolone (40 mg intravenously every 8 hours), and bumetanide (1 mg orally every 24 hours). The patient gradually developed ecchymosis and swelling of

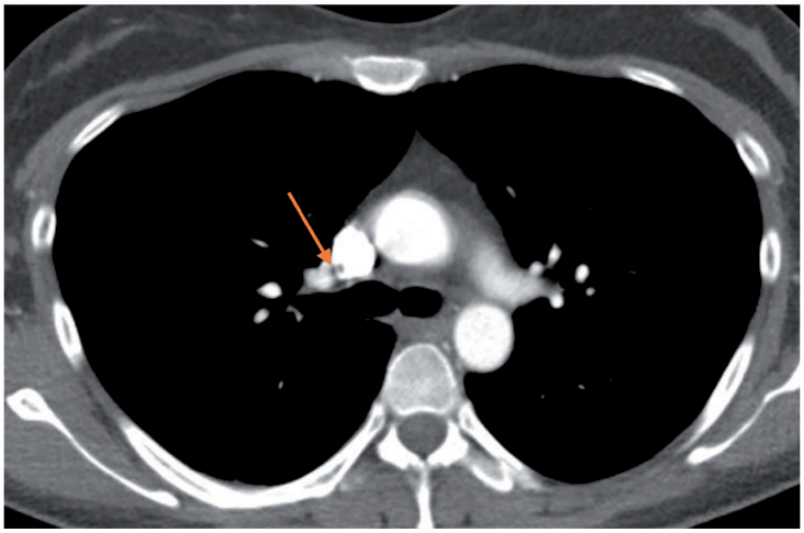

Figure 1. Enhanced computed tomography pulmonary angiography scan showed filling defects in the pulmonary artery (transverse plane).

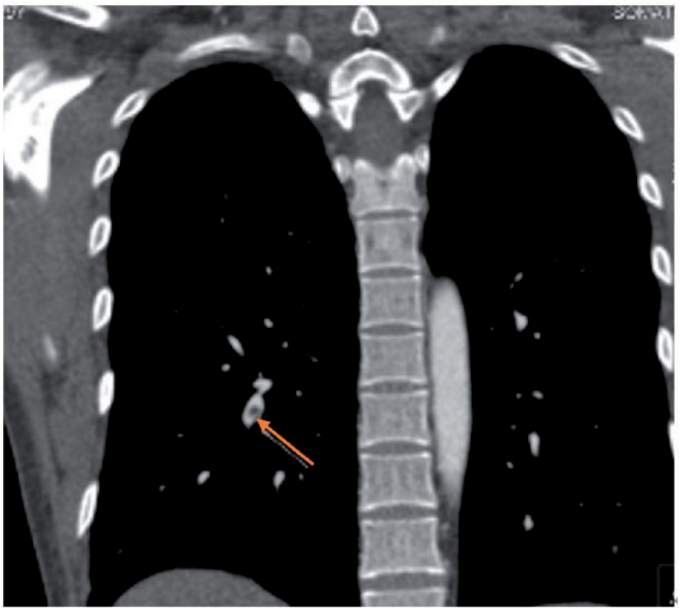

Figure 2. Enhanced computed tomography pulmonary angiography scan showed filling defects in the pulmonary artery with a maximum size of approximately 1.2×0.7 cm (coronal plane).

both lower limbs. Routine blood examination showed a hemoglobin concentration of 72 g/L. The patient was thought to have a hematoma secondary to the anticoagulant therapy; therefore, the anticoagulant therapy was discontinued. She simultaneously underwent pulse magnetic field treatment (once a day for 5 days), blood transfusion,

and continuous oxygen therapy. Her hemoglobin concentration gradually increased and stabilized, and the swelling of her lower limbs gradually improved. The patient's oxygen therapy was gradually changed from an oxygen mask to a venturi mask and then to a nasal cannula, after which the oxygen therapy was discontinued. After 16 days, ABG showed a $PaCO_2$ of 34.8 mmHg, PaO_2 of 84.4 mmHg, bicarbonate concentration of 22.1 mmol/L, and lactic acid concentration of 1.3 mmol/L with a pH of 7.42. CTPA re-examination also showed improved. The patient was discharged 2 days later.

Informed consent was obtained from the patient. This case was reported in accordance with the CARE guidelines. The requirement for approval by an institutional ethics board was waived because no clinical study was performed.

Discussion

The patient described in the present report was a young woman who developed dyspnea and hypoxemia. She had no relevant high-risk factors or thrombus-related factors, excluding the possibility of thrombosis-induced pulmonary embolism. Based on her history of AFG and CTPA findings, she was diagnosed with fat embolism after AFG. Three possible sources of fat emboli in this patient should be considered: liposuction, breast fat grating, or vaginal fat grafting. First, fat embolism caused by liposuction is rare.[10,11] Some reasonable mechanisms have been discussed in previous studies. Fat cells damaged during liposuction release fat droplets. These droplets, rather than fat emboli, may enter the nearby torn veins under pressure; they may then enter the circulation and be transported to terminal organs such as the pulmonary vascular bed, where large fat globules cause a mechanical obstruction and become trapped as emboli in lung capillaries.[12] Large fat droplets may also enter the systemic circulation in patients with preexisting pulmonary precapillary shunts and pathologic venous–arterial communications (such as patent foramen ovale).[13] Our patient had no such signs of pathological cardiac structures. CTPA revealed fat emboli with a maximum size of 1.2×0.7 cm, which was slightly smaller than the diameter of her inferior vena cava (1.4 cm). Considering the above-described mechanism, it is difficult to believe that such a large fat embolus was produced during the liposuction procedure; instead, it was more likely to have been generated by direct injection into vessels. Second, no reports to date have described fat embolism caused by breast fat injection. In most breast augmentation surgeries, fat is injected into the breast tissue, which has few vessels.[6,14] Therefore, we do not believe that the embolism in this case was caused by breast fat grafting.

Finally, we considered the possibility that fat had been injected directly into the vessels around the vagina. Although no reports to date have described fat embolism caused by vaginal fat injection, several cases of embolism caused by vaginal injection of other materials have been reported.[15–17] This process is somewhat similar to the development of embolism caused by buttock augmentation or penis enlargement with fat grafting.[9,18,19] Abundant veins are present in the relatively narrow local space surrounding the vagina. These veins meander and expand, forming a dense venous plexus, and eventually merge into the internal iliac vein and then into the inferior vena cava.[16,17] These anatomical factors may lead to severe vascular injury and injection displacement during fat grafting procedures. Additionally, injection of only a small amount of fat can cause excessive pressure in the narrow space around the vagina.[18,20] To summarize, in the absence of solid evidence obtained by performing

an anatomical examination, the vaginal fat grafting procedure was the most likely cause of the multiple-organ fat embolisms. Further research is needed to elucidate the anatomical mechanism underlying this phenomenon.

Fat embolism not only creates obstructive ischemia, but more importantly, it causes severe systemic allergic reactions. Free fatty acids hydrolyzed by lipoprotein lipase injure the pulmonary capillary endothelium and release local toxic mediators, resulting in alveolar hemorrhage and edema and respiratory failure; this condition is called fat embolism syndrome.[21,22] Respiratory support treatment is vital to avoid further systemic effects of hypoxia.[23] In the present case, anticoagulation treatment interfered with the patient's normal coagulation function and resulted in a secondary hematoma. Therefore, whether anticoagulation therapy is appropriate for patients with pulmonary fat embolism should be carefully weighed.

Conclusion

We have reported a case in which a patient developed severe fat embolism after AFG in vagina tightening and breast augmentation surgery. The fat embolism was confirmed by ABG and CTPA, and the cause was assumed to be direct injection of fat into the vessels around the vagina. Further research is needed regarding the anatomical mechanism.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

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