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ORIGINAL ARTICLE FACE AND NECK SURGERY

# Internal Carotid Artery Embolism After Autologous Fat Injection for Temporal Augmentation

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**Abstract**

*Background* There have been several reports of patients experiencing cerebral embolisms following the injection of autologous fat into the face during cosmetic surgery. These embolisms likely resulted from unintentional introduction of fat particles into facial arteries, which then reached the cerebral arteries by retrograde motion.

*Case Presentation* We describe here a patient who developed an internal carotid artery (ICA) embolism after autologous fat injection for temporal augmentation. To our knowledge, this is the first report of a pathologically proven ICA embolism after fat injection into the face.

*Conclusions* Our results suggest that the fat particles reached the cerebral arteries via a previously unknown pathway.

*Level of Evidence V* This journal requires that authors assign a level of evidence to each article. For a full description of these Evidence-Based Medicine ratings, please refer to the Table of Contents or the online Instructions to Authors www.springer.com/00266.

**Keywords** Fat injection · Temporal augmentation · Fat embolism · Cerebral infarction

## Introduction

Autologous fat injection into the face is a commonly performed cosmetic procedure that has been previously reported as safe. Nevertheless, several patients have experienced visual loss or neurologic deficits following injection of fat into the glabellar area, periorbital area, bridge of the nose, lateral aspect of the nose, nasolabial fold, lower lip, chin, upper and lower eyelids, and temporal and frontal regions [1–18]. These complications are thought to result from inadvertent injection of fat into facial arteries followed by retrograde delivery to the orbital and cerebral arteries. We describe here a patient who developed an embolism in the internal carotid artery (ICA) after autologous fat injection for temporal augmentation.

## Case Report

A 42-year-old woman underwent injection of autologous fat, obtained from her abdominal area, for temporal augmentation under local anesthesia (Fig. 1). During the procedure, the patient became unresponsive and lethargic with global aphasia and right-sided hemiplegia. She was immediately transported to a local hospital, and subsequently to the neurologic care unit of our hospital 15 h after the symptom onset. Upon admission, her blood pressure was 110/80 mmHg, and heart and lung auscultation yielded normal findings. Neurologic examination revealed lethargy, normal pupils, global aphasia, and right-

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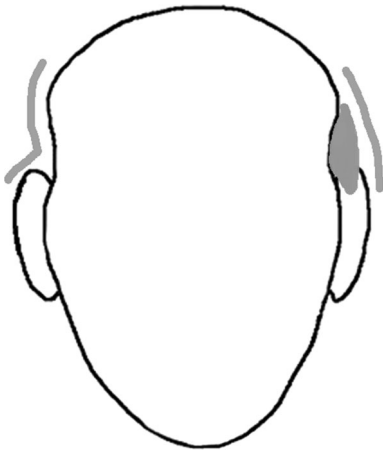

**Fig. 1** The drawing of fat-injected area (temporal region)

sided hemiplegia. A fundoscopic examination showed no fat emboli in her retinal arterioles. Brain computed tomography (CT), performed approximately 16 h after symptom onset, showed acute infarction in the left frontal–temporal–parietal territory of the left ICA, with signals suggesting fat or air in the ICA, anterior cerebral artery (ACA), and middle cerebral artery (MCA) (Fig. 2a–c). Chest radiographs were normal. Laboratory tests showed normal liver and renal function, including normal prothrombin and partial thromboplastin times, fibrinogen level, and fibrin degradation products. Her serum albumin and triglyceride concentrations were slightly elevated, as was her white blood cell count. She had no medical history of hypertension, diabetes mellitus, smoking, heart disease, hypercoagulable state, arterial or venous occlusive disease, hematologic disease, or recent trauma.

Her condition deteriorated rapidly. Twenty hours after the symptom onset, the patient vomited violently and then lapsed into a coma; her right pupil was dilated to approximately 5.0 mm. Because medical management was ineffective, she underwent emergency decompressive craniectomy with necrotic brain tissue resection 24 h after the symptom onset. During preoperative assessment, her left superficial temporal artery (STA) pulses were absent. Examination during surgery showed visible occlusion of the left STA and its branches and the arteries in the left Sylvian cistern. Pathologic analysis of the M2–M3 segment revealed cellular fat tissue in the arteries (Fig. 3a, b). The supraorbital artery was preserved to avoid ischemic necrosis of the temporal flap; however, the incision revealed a $3 \times 3$ $cm^2$ area of black skin necrosis in the temporal area.

Five days after surgery, the patient was discharged from the hospital in an alert state, but with global aphasia and right-sided hemiplegia. She underwent rehabilitation at a local hospital. Two years later, she understands some words but has complete expressive aphasia and right-sided hemiplegia.

## Discussion

Autologous fat transfer to the face was first described in 1893 [19]. Although fat injection was first performed in 1914, its use became more common following the development of liposuction in 1975 [20]. Use of the wet technique [21] and tumescent anesthesia [22] have improved the safety of autologous fat injection and allowed patients to be ambulatory following surgery.

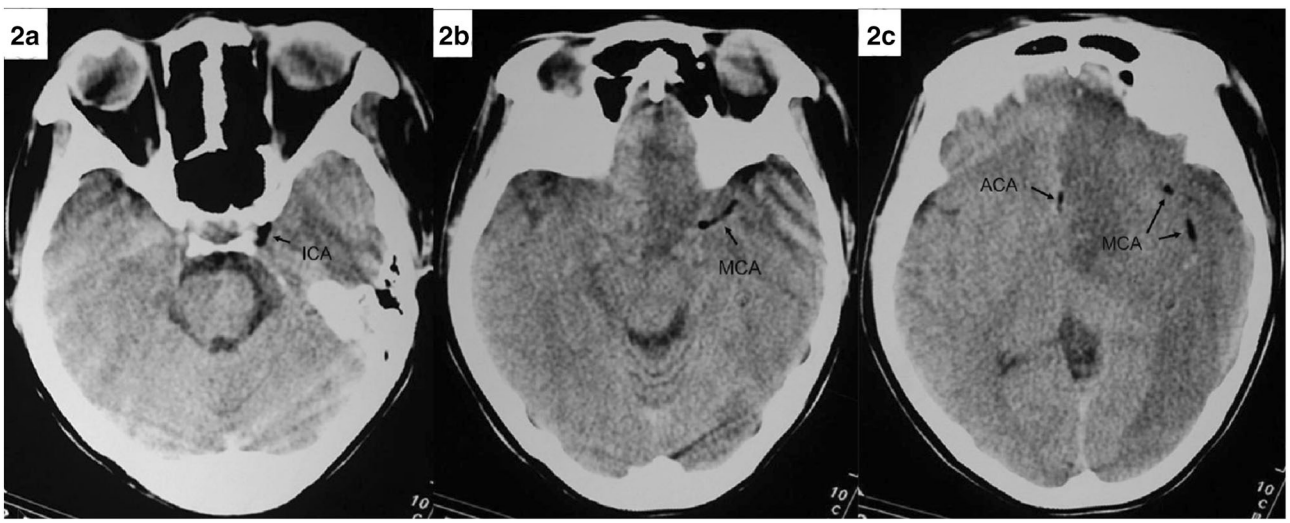


**Fig. 2** **a–c** Brain computed tomography in patient showing acute infarction in the left frontal–temporal–parietal lobes and signals suggesting fat or air in the internal carotid artery (ICA), anterior cerebral artery (ACA) and middle cerebral artery (MCA)

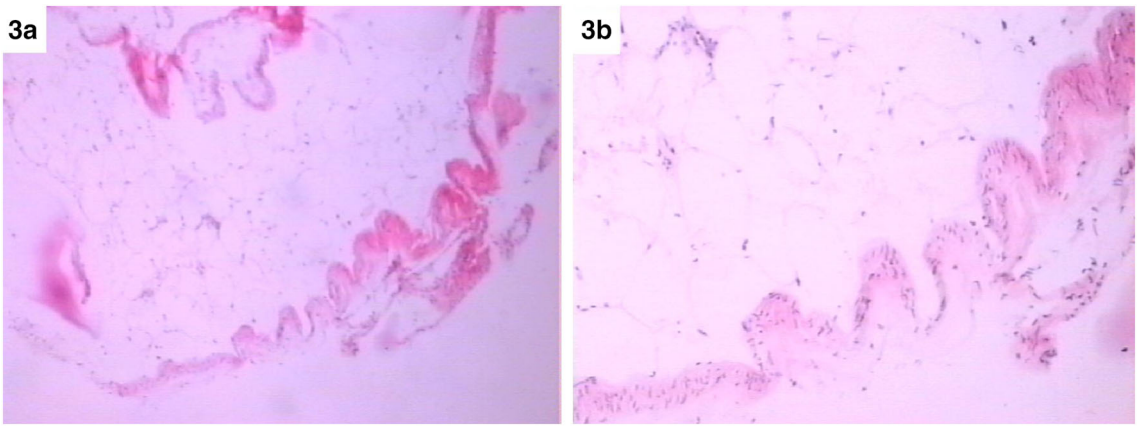


**Fig. 3** **a**, **b** Histologic examination showing part of the middle cerebral artery (MCA) obstructed by fat tissue. Nucleated fat cells were evident (hematoxylin and eosin staining)

Among the risks of both liposuction and lipografting is fat embolism to organs. Intravasation of fat, which results in fat embolism formation, requires three conditions: well-vascularized tissue, fragmentation of the parenchyma, and local pressure increase [23]. The incidence of fat embolism in patients undergoing large abdominal liposuctions has been estimated at 1.3 per 100,000 procedures [24]. Fat particles are thought to enter venous circulation and reach the general circulation via the intervening pulmonary circulation [25]. The incidence of fat embolism in patients undergoing lipografting has not been described.

Cerebral fat embolism is a rare cause of stroke that usually occurs within the context of fat embolism syndrome [26]. A complication of long-bone fractures [27], fat embolism syndrome is a life-threatening disorder characterized by respiratory insufficiency, cerebral involvement, and petechial rash. It has also been reported following surgery, hepatic failure, and acute pancreatitis. Cerebral fat embolism following autologous fat injection into the face has been associated with discoloration of the iris in one patient [7], visual loss in six patients [1, 3, 10, 11, 14, 17], neurologic deficits with visual loss or retinal emboli in nine patients [2, 4, 5, 8, 9, 12, 16, 18], and neurologic deficits without visual loss in three patients (unconfirmed by funduscopic examination) [6, 13, 15].

Blood is supplied to most of the integument of the head by the branches of the external carotid system. However, the masklike area surrounding the eyes, central forehead, and upper two-thirds of the nose is supplied by the ophthalmic branch of the internal carotid system. In this case, it is likely that the patient's autologous fat particles were injected into the peripheral branches of the ophthalmic artery supplying the integument of the head or into branches of the external carotid system. These fat particles could then reach the ocular and cerebral arteries via the ophthalmic artery or the anastomotic branches of the internal and external carotid systems using retrograde motion. The immediate onset of symptoms is an evidence of the direct intravasation of fat particles [2, 4–6, 9–11, 14, 15, 18]. However, some reports show that patients experience delays between fat injection and symptom onset [10, 12, 13, 17]. In patients with delayed symptoms, the amount of fat introduced into the venous system was likely too small to cause immediate vessel occlusion and the fat particles were likely trapped in a distal vessel tree prior to causing clinical signs [23]. Following a delay, fat particles, especially free fatty acids, can cause endothelial damage and delayed vessel occlusion with associated clinical manifestations.

Potential mechanisms for perioperative cerebral infarction associated with nonvascular surgery include diminished perfusion in regions supplied by previously compromised arteries, increased coagulability following surgery, cerebral artery injury, introduction of embolic particles from the heart or aorta into the circulation, or occurrence of air or fat embolisms [28]. Because the woman in this case report was young, had no history of cardiovascular risk factors, experienced symptoms during injection, and had only local anesthesia, her stroke was unlikely to be caused by these mechanisms of perioperative cerebral infarction.

CT findings in the patient showed signals suggesting fat or air in the left ICA, ACA, and MCA, and pathologic examination showed the presence of intravascular fat tissue. The inner diameter of the ICA is 3.5–4.0 mm, so a large volume of fat was probably introduced into the left internal carotid system and then trapped in the ICA, ACA, and MCA.

Pathologic examination of tissue samples from the patient showed intact fat cells, which are typically 67–98 μm in diameter in the subcutaneous area. If fat tissue had reached the cerebral arteries via the anastomotic

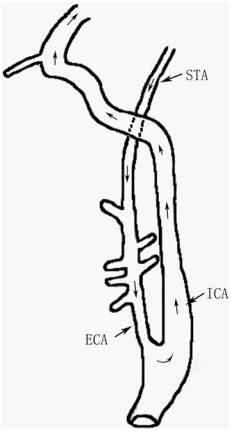


**Fig. 4** Pathway through which fat tissue passes following injection

branches joining the internal and external carotid systems, it should have passed through their capillary network (vessel diameter, 3–9 μm). The maximum diameter of the pulmonary arteriolar network in undamaged lungs is 7 μm [29]. Both of these pathways were blocked in our patient. Although fat tissue reaching the cerebral arteries via the peripheral branches of the ophthalmic artery is likely to cause eye damage, the patient in this report had no evidence of visual loss or retinal emboli; thus, this route was not taken by fat particles in this patient. The presence of fat in the ICA indicates that occlusion did not result from fat-induced secondary biochemical reactions.

Because all other pathways were blocked, the fat tissue in our patient likely reached the cerebral arteries via the external carotid artery (ECA). During surgery, the patient's left STA was found to be occluded. Therefore, the fat tissue was likely introduced into the STA, reached the ECA by retrograde flow, and then entered the ICA at the carotid bifurcation (Fig. 4).

Authors of previous reports have hypothesized that fat particles were introduced into the facial arteries and then arrived in the cerebral arteries by retrograde motion. However, tissue samples from most of those patients were not subject to pathologic examination. Our finding suggests that fat particles reached the cerebral arteries via the ECA. The case warns us that facial fat injection may cause patients devastating or even fatal complications. Therefore, fat injection should be performed vigilantly. The strength applied during injection is a main factor contributing to fat embolism. Fat injection should be performed with gentleness and the lowest pressure. Blunt or large-bore needles are supposed to be the most appropriate instruments for plastic surgeons. Moreover, aspiration should be performed before fat injection to avoid the veins and arteries. Fat injection should not be performed through pre-traumatized soft tissue subjected to other cosmetic procedures, because the risk of fat embolism may be higher.

Treatment of fat embolism is essentially symptomatic. Although plenty of specific treatments have been suggested, the effectiveness of them is not always revealed. Corticosteroid therapy has been proposed as a potential therapy by limiting free fatty acid levels, stabilizing membranes, and inhibiting complement-mediated leukocyte aggregation. However, there is insufficient data to support its effectiveness once fat embolism has been established [30]. Besides, albumin has an additional lipophilic action and could reduce the toxicity of fatty acids. Mechanical thrombectomy could also be a possible solution to realize vascular recanalization [31].

**Compliance with Ethical Standards**

**Conflict of interest** All authors declared there was no conflict of interests involved.

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