

## Case Report

# Internal and External Carotid Artery Embolism Following Facial Injection of Autologous Fat

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**Abstract**

Autologous fat injection is a common aesthetic procedure for soft-tissue augmentation of the face. Although this procedure is generally regarded as safe, several patients have experienced acute visual loss or cerebral infarction after these injections. We describe a case of internal and external carotid artery fat embolism that occurred following injection of autologous fat into the face. It appeared that the injected fat entered a branch of the left external carotid artery and that the embolus likely migrated into the left internal carotid artery and distally into the left ophthalmic artery, left anterior artery, and middle cerebral artery.

**Level of Evidence: 5****Keywords**

cosmetic medicine, autologous fat injection, facial injection, fat embolism, internal carotid artery, external carotid artery



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In the hands of experienced cosmetic surgeons, injection of autologous fat into the face is generally considered safe. However, cases of sudden visual loss or cerebral infarction have occurred after this procedure.<sup>1,2</sup> In those cases, it was surmised that the fat had been injected into facial arteries and reached the orbital and cerebral arteries retrogradely.<sup>1,2</sup> In this report, we describe a patient who experienced acute fatal cerebral infarction after injection of autologous fat into the face. We also discuss the pathogenesis and the potential risks of injecting fat into the face.

**CASE PRESENTATION**

Under general anesthesia, a 22-year-old right-handed woman with no significant medical history had undergone injection of autologous fat into the temporal and frontal regions of her face for aesthetic purposes. The procedure was performed by a plastic surgeon at another institution. There was no evidence of scarring (or other previous injury) in the recipient tissues.

According to the operative note, the procedure consisted of harvesting autologous fat from both thighs and injecting the fat into the frontotemporal depression. The fat was injected into each left temporal (25 mL each) and frontal (24 mL) region. A blunt cannula, measuring 2.5 mm in diameter, was attached to a 10-mL syringe previously washed with sterile saline. After insertion of the cannula beneath the skin, negative pressure was applied to the syringe by retracting the

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plunger mechanically with help from the thumb and fingers. After aspiration, the syringes were placed upside down into a syringe rack. The syringes were kept in the rack for 5 to 10 minutes to ensure separation of fat cells. Following this, the syringes were drained such that only fat was left behind. The fat cells were washed with normal saline to remove leftover blood and oil, which failed to separate. The general injection site was behind the frontotemporal hairline. Injections were administered at the subcutaneous level, with autologous fat placed at the suprapariosteal level of the temporal area. A blunt cannula (2.0 mm in diameter) with a 1-mL syringe was used for initial and subsequent injections. Only a minute amount of fat was injected during each subsequent pass, which was done as the cannula was being withdrawn. The precise locations and levels of injection were not communicated to us.

Vital signs were monitored closely during the procedure, and there was no indication that anything had gone wrong. However, for the first 4 hours posttreatment, the patient's physical status and vital signs were not monitored closely. Five hours after the procedure, the patient had not emerged from the anesthesia. Right-sided hemiparesis and a positive right-sided Babinski reflex were noted at this time. The patient's left eye was midline fixed, and the pupil was dilated and unresponsive to direct light stimulation. One hour later, diffusion-weighted magnetic resonance imaging showed recent ischemic lesions in the left anterior and middle cerebral arteries (Figure 1A). T2-weighted imaging showed high signal intensity in the left internal carotid artery (Figure 1B). Magnetic resonance angiography showed occlusion of the left intracranial internal carotid artery, as well as the left anterior and middle cerebral arteries (Figure 1C). The patient underwent immediate decompressive craniectomy and was transferred to the intensive care unit of our hospital.

The following day, she regained consciousness but developed aphasia, vision loss in the left eye, and right hemiplegia. On posttreatment day 3, skin necrosis developed in her left temple. Repetitive transcranial duplex sonography showed occlusion followed by recanalization of the left internal carotid artery, left external carotid artery, and left ophthalmic artery. Computed tomography (CT) scans of the brain demonstrated an infarct that included the left anterior and middle cerebral arteries (Figure 2). A focal area of low attenuation in the proximal left middle cerebral artery (MCA) was consistent with a fat embolism based on Hounsfield unit measurement. Despite diagnosis of the fat embolism, findings from chest CT scans were normal. Transthoracic echocardiography did not show a patent foramen ovale or right-to-left intracardiac shunt. Disorders of blood vessels, coagulation disturbance, and

immune system were excluded by intensive diagnostic procedures. Similarly, other rare causes of fat embolism were ruled out, such as infectious disease, inflammation of bone or soft tissue, and other metabolic disorders.

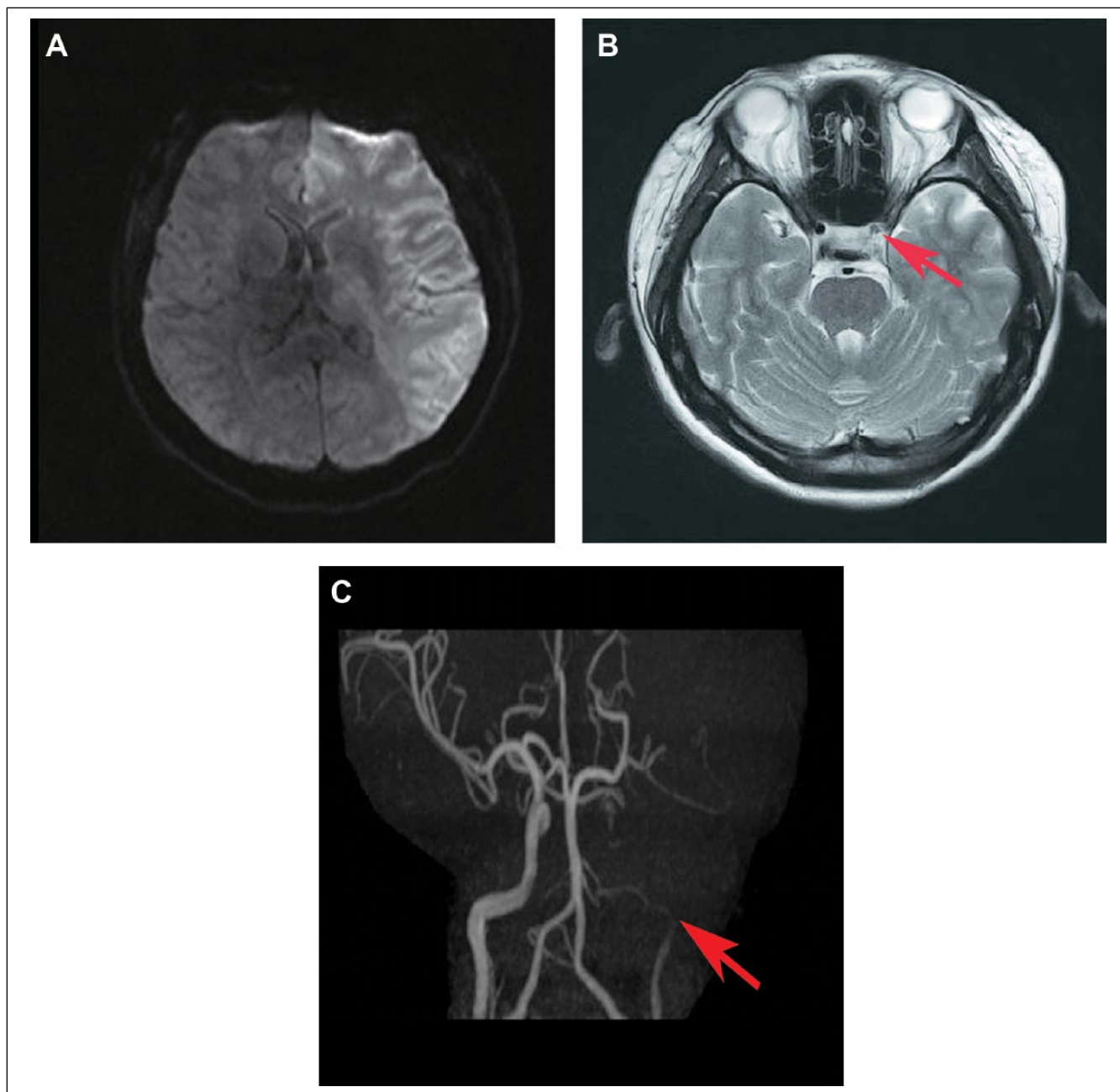
The patient received intravenous mannitol for treatment of cerebral edema. One month after discharge from the intensive care unit, she was able to raise her right leg against minimal resistance; however, her right arm remained paretic. Two months later, there was improvement in the aphasia, but the vision in her left eye was not recovered. The soft-tissue lesion of the left temple was repaired using intermediate split-thickness skin grafts.

## DISCUSSION

Autologous fat injection is a procedure aimed at eliminating deep defects in the skin surface by subcutaneous injection of the patient's own fatty tissue.<sup>3</sup> Fat embolism is a rare but serious complication of this procedure.<sup>4-17</sup> Preconditions, local increase in pressure, and tissue with rich vasculature are proposed causes of intravasation of fat resulting in fat embolism.<sup>18</sup> Excessive force and velocity of injection may cause an increase in local pressure. Thus, facial fat injection should be performed slowly, with low pressure, and after ensuring the lack of blood reflow in the delivering syringe.<sup>19</sup>

In the present case, the diagnosis of fat embolus was established on the basis of negative Hounsfield unit attenuation in the MCA.<sup>12</sup> If fatty particles were intravasated by entering facial veins, they would have had to travel through the pulmonary circulation. However, the patient had no signs of pulmonary fat embolism. The lesions of fat embolism in this case were confined to the region of the left internal and external carotid artery. Moreover, the blood supply to the face is mainly from branches of the external carotid (facial and superficial temporal artery) and internal carotid (ophthalmic artery). Thus, we believe that fragments of fatty tissue were injected into left superficial temporal artery. Under the pressure of injection, the fat was likely forced retrograde into the left internal carotid artery, followed by movement of the emboli distally into left ophthalmic artery, left anterior artery, and middle cerebral artery.

We believe that the force applied during injection of facial fat is a major contributor to fat embolism. Thus, the surgeon should ensure that only minimal force is exerted when fat is injected into facial tissue. Moreover, aspiration should be performed before the fat is introduced to ensure avoidance of veins and arteries. If fat embolism develops perioperatively, early recognition and supportive therapy are essential for optimizing clinical outcomes. It may be



**Figure 1.** (A) Diffusion-weighted magnetic resonance imaging showed high signal intensities in the left cerebral hemisphere. (B) T2-weighted imaging showed high signal intensity in the left internal carotid artery (arrow). (C) Magnetic resonance angiography showed occlusion of the left intracranial internal carotid artery, left anterior artery, and middle cerebral artery (arrow).

possible to achieve vascular recanalization via mechanical thrombectomy devices.<sup>14</sup>

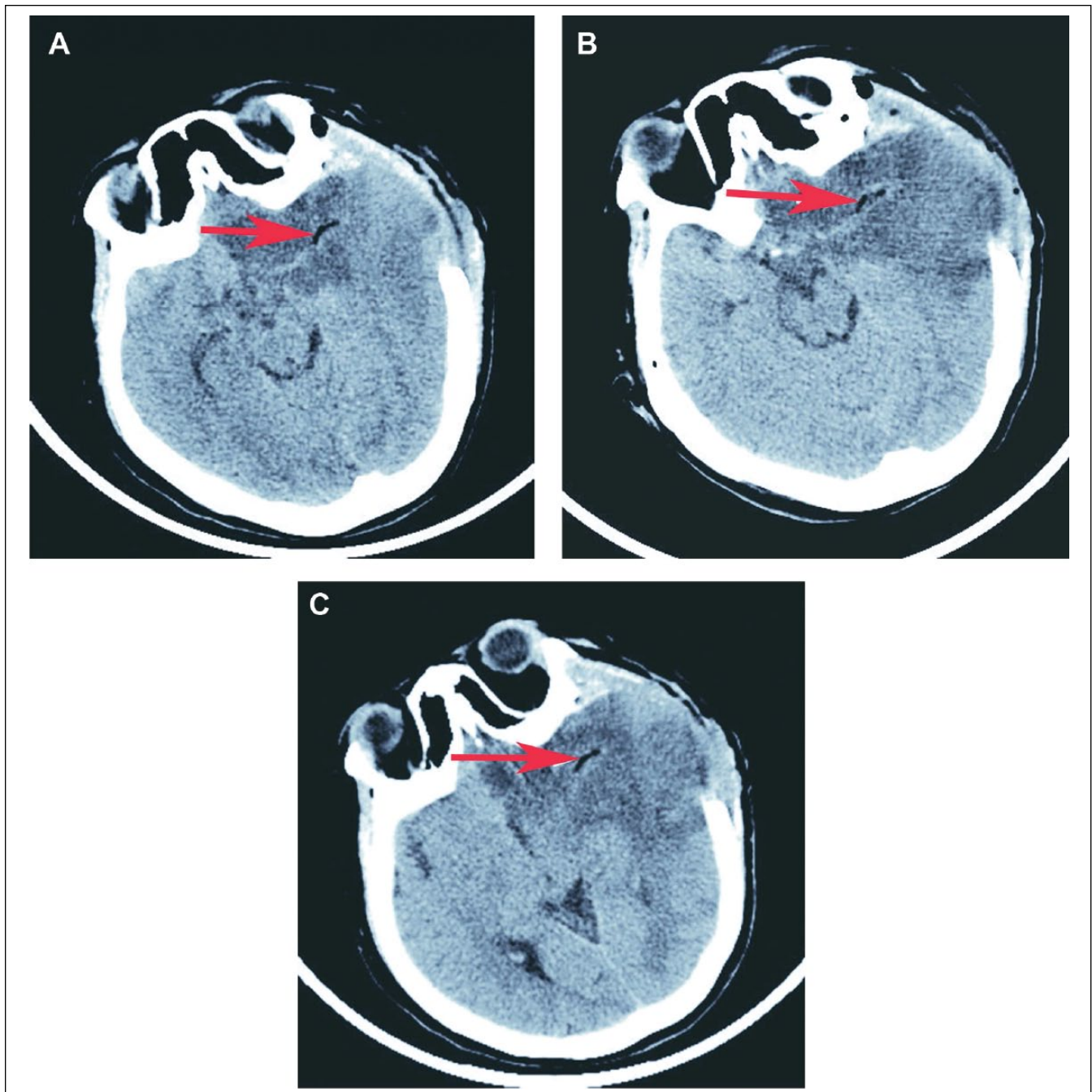
## CONCLUSIONS

This unusual case serves as a caution to plastic surgeons to exert only minimal force when injecting autologous fat

into facial tissues. Accidental injection of fat into the arterial system can result in fat embolism in the internal and/or external carotid artery.

## Disclosures

The authors declared no potential conflicts of interest with respect to the research, authorship, and publication of this article.



**Figure 2.** Posttreatment computed tomography scan images obtained on (A) day 3, (B) day 4, and (C) day 6 demonstrated a left middle cerebral artery infarct. The filling defect (arrow) indicated a fat globule in the trunk of the proximal left middle cerebral artery.

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