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CASE REPORT
Cosmetic

Cerebral Hemorrhage after Cosmetic Facial Injection

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Summary: Cosmetic facial injection is a mini-invasive surgical procedure and is widely used at present. Meanwhile, it may also cause severe complications like cerebrovascular infarction occasionally. A 49-year-old female patient presented to a local hospital with dysphoria, left eye and head ache, unconsciousness, orthocolosis, right upper extremity hemiplegic paralysis, and emesia after receiving facial injection of hyaluronic acid into the glabella. Low molecular weight heparin and clopidogrel were used to treat the patient, but there was no relief of symptoms. The patient gradually developed coma and was transferred to our hospital. Brain computed tomography showed mixed density in left temporal lobe, multiple high density in left frontal temporal parietal lobe and sulcus, and subarachnoid hemorrhage and high density in brainstem. The patient was diagnosed with cerebral hematoma and hernia, who gradually developed respiratory and circulatory failure and eventually died. *(Plast Reconstr Surg Glob Open 2019;7:e2397; doi: 10.1097/GOX.0000000000002397; Published online 10 September 2019.)*

Cosmetic facial injection is widely used at present. Meanwhile, there are also some reports about cerebrovascular infarction as a rarely severe complication of cosmetic facial injection. We report a patient with cerebral hemorrhage after receiving facial injection of hyaluronic acid.

CASE REPORT

Under local anesthesia, a 49-year-old woman underwent facial injection of hyaluronic acid (1 ml) into the glabella. During the process of injection, the patient developed dysphoria, left eye and head ache, unconsciousness, and orthocolosis, which relieved after 10 s. There was also right upper extremity hemiplegic paralysis and emesia. Low molecular weight heparin was subcutaneously injected to the patient at a dose of 500 IU twice a day, after which the symptoms did not relieve. So the patient was transferred to a general hospital after 1 hour, where she received routine blood test, coagulation function, and nonenhanced computed tomography (CT) scan, which all showed negative results. Meanwhile, 0.4 ml of low molecular weight heparin (6150 u/0.6 ml), 150 mg of clopidogrel, and 125 ml of mannitol was used to treat the patient, after which she complained a relief of headache but no relief of hemiplegic paralysis and dysphoria. The patient developed coma later and was transferred to our hospital. Medical history showed a history of 8-year smoking, but no hypertension, diabetes mellitus, hyperlipidemia, heart disease, hypercoagulable state, arterial or venous occlusive disease, hematologic disease, surgery history, or trauma. Physical examination at our emergency department showed vital signs were normal, Glasgow coma scale was E3V1M5, dysphoria and aphasia. The left pupil was dilated of 5 mm and unresponsive to direct and indirect light stimulation. The right pupil was 2.5 mm and had slow reaction to direct and indirect light stimulation. There was fundus fuzzy in the left eye and gray and dropsical posterior pole of retina. The muscle force of right upper extremity was grade 1 and of right lower extremity was grade 3, mixed with hypermyotonia. Although the muscle force and muscle tension of left extremity was normal, the pathological reflex was not drawn out. When the patient transferred into our ER, brain CT showed that there was mixed density in left temporal lobe, indicating the amount of bleeding was around 15 ml. There was multiple high density in left frontal temporal parietal lobe and sulcus and subarachnoid hemorrhage and sulcus became shallow (Fig. 1).

After being admitted to our hospital, the patient was consulted by specialists of plastic surgery, ophthalmology, neurosurgery, and neology, who reached an agreement

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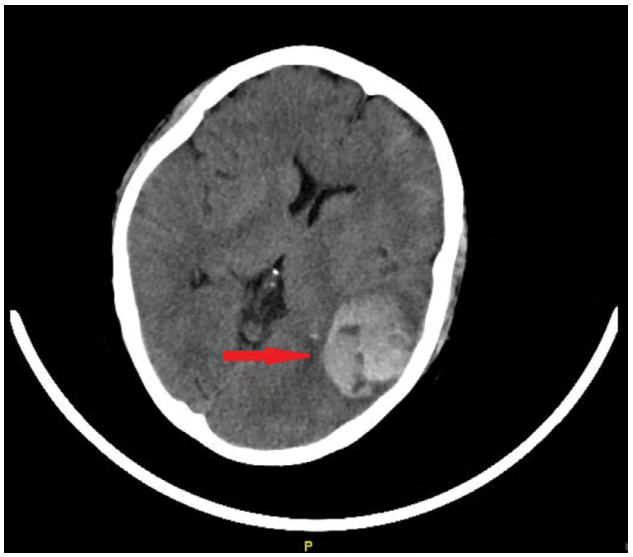

Fig. 1. There was mixed density of left temporal lobe, indicating the amount of bleeding was around 15 ml. There was multiple high density in left frontal temporal parietal lobe and sulcus and subarachnoid hemorrhage and sulcus became shallow.

that the cause of the hemorrhage was highly likely to be the treatment of anticoagulant and antiplatelet drugs after cerebrovascular infarction caused by the injection of hyaluronic acid. There was also a possibility that the patient had cerebrovascular malformation or cerebral venous thrombosis. Therefore, digital subtraction angiography was recommended for her, during which she immediately developed bucking, dyspnea, and severe hypoxemia. Brain CT was performed again and showed mixed density in left temporal lobe, multiple high density in left frontal temporal parietal lobe and sulcus, indicating the amount of bleeding was around 80 ml. There was subarachnoid hemorrhage and high density in brainstem (Fig. 2). Physical examination showed both pupils were dilated of around 4 mm and Glasgow coma scale was E1V1M2, which meant the enlargement of hematoma and the formation of cerebral hernia. The surgery was unable to be performed on the patient under such bad condition, who gradually developed respiratory and circulatory failure and eventually died 6 days after admission in hospital.

DISCUSSION

The Chinese have a strong aversion to glabellar wrinkles, which are regarded as a sign of bad luck, thus creating a strong desire to wipe them out. However, due to cultural background, many people refused to receive botulinum toxin injection in the early stages of glabellar wrinkles. After the stable and serious glabellar wrinkles appear, they can only choose hyaluronic acid injection instead, which turns out not as safe as they imagine.

Quite for the reason above, this patient chose to receive the injection of hyaluronic acid, but misfortune happened. The cause of the neurological symptoms on this patient was highly likely to be the cerebrovascular infarction by hyaluronic acid. After receiving the treatment of anticoagulant and antiplatelet drugs, the patient developed hemorrhage. There was also a possibility that the patient had cerebrovascular malformation or cerebral venous thrombosis. In terms of infarction, the arterial blood supply of the facial skin and soft tissues is derived from the branches of the external carotid artery and the internal carotid artery. There is plenty of communication between the external carotid artery and the internal carotid artery in the regions of eyes, nose, forehead, and other parts.[1] Therefore, some scientists like Hong et al[2] thought that, during the process of injection, filler could get into ophthalmic artery, the main branch of the internal carotid artery through the branches of the communicating small arteries under the great pressure, thus getting into the carotid artery, producing embolization and resulting in blindness or even cerebral infarction. However, there is a lack of relevant experimental study about correlation between injection pressure and embolism mechanism.

Although there was no autopsy, based on the symptoms and anatomy, we thought it should do with the damage of the supratrochlear artery. The ophthalmic artery system and the internal carotid artery system have extremely rich traffic branches on the face, such as supratrochlear artery, which is a bridge for communicating intracranial and extracranial blood flow. The supratrochlear artery runs between the orbicularis muscle and the frowning muscle, and then enters the superficial layer of the frontal muscle. During the injection of glabellar wrinkles, if the injection was too shallow, it might damage the supratrochlear artery. Hyaluronic acid then entered the intracranial vascu-

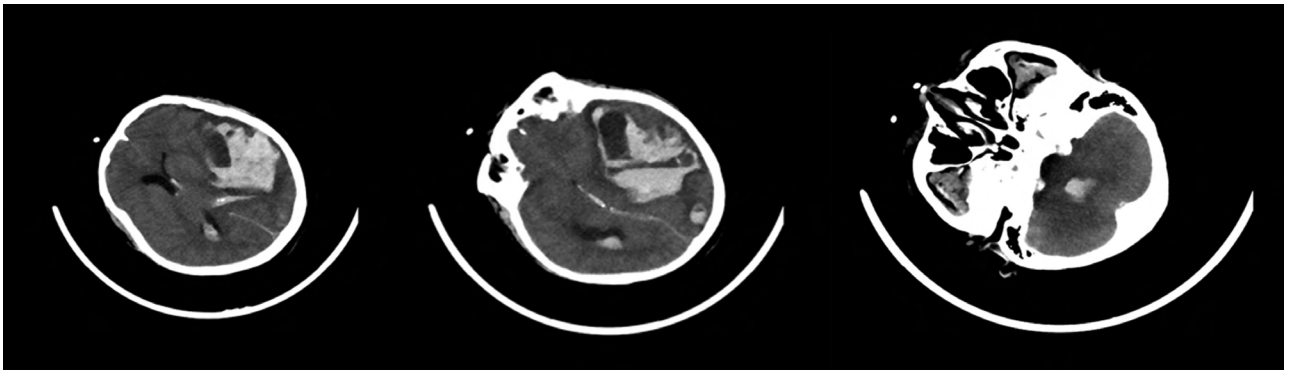

Fig. 2. There is mixed density in left temporal lobe, multiple high density in left frontal temporal parietal lobe and sulcus, indicating the amount of bleeding was around 80 ml. And there was subarachnoid hemorrhage and high density in brainstem.

lar system via the branch of supratrochlear artery, and was likely to block the anterior cerebral artery, which led to the cerebral infarction and the series of symptoms.

To avoid the complications, operators should be familiar with the basic knowledge of facial anatomy and the treatment of the related complications. Besides, medical history should be asked and the medical risk should be carefully valued and explained at length to patients.[2,3] Blunt needles are encouraged to be taken in the procedure to reduce the chance of filler entering small vessels.[2,4–6] Injection should be carried slowly with little pressure, and withdraw the needle with negative pressure.[2,7] We suggest the condition of patients' oxygen saturation, blood pressure, and neurological symptoms should be carefully observed during the procedure of injection. Once there are neurological symptoms, imaging examination should be carried as soon as possible. Magnetic resonance angiography, diffusion weighted imaging, and transcranial Doppler are more sensitive than brain CT scan, which will have positive results after 24 hours of cerebrovascular infarction.[4,8,9]

Whether anti-infarction therapy is effective or not on patients with cerebrovascular infarction of fillers still has not reached an agreement yet. Some cases received antiplatelet therapy[10] or thrombolysis therapy,[3] but there is no satisfactory result. Meanwhile, this will add more risk of cerebral hemorrhage. Neurotrophic therapy and hyperbaric oxygen therapy seem to have some positive results.[5] It is currently recommended that the first option taken in the event of embolization is to inject hyaluronidase immediately. Some basic medical studies carried out by Chiang et al[11] reported a combination of hyaluronidase and urokinase was used to treat the rats with ophthalmic artery embolism of hyaluronic acid, leading to positive results, which provided some physiological evidence for this operation.

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