Delayed-onset cerebral infarction after cosmetic facial injection using hyaluronic acid

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A 25-year-old woman without any known systemic disease reported experiencing a sudden onset of right eye blindness after hyaluronic acid injection into the nose, and a delayed course of left upper limb weakness developed 9 hours later. Initially, the patient was brought to our emergency department for right eye blindness. The fundus revealed right central retinal artery occlusion (Figures 1A and 1B). Nine hours later, acute onset of left upper limb weakness with muscle power Grade 4 in the left elbow, wrist, and hand and Grade 5 in the left shoulder and other three extremities was noted. Brain magnetic resonance imaging (MRI) revealed many small acute infarcts, mainly in the right middle cerebral artery territory (Figure 1C). The survey of young stroke was within the normal limit, except transesophageal echocardiography showed an atrial septal defect (ASD) with a size of 2.7 cm. Finally, the visual loss and left upper limb weakness remained after discharge.

Cosmetic facial injection using hyaluronic acid as a filler is often performed because of its convenience, ease of implementation, and safety profile. However, several possible complications, including local erythema, infection, skin necrosis, blindness, panophthalmplegia, pulmonary embolism, and cerebral infarction may occur. In previous reports, the retrograde embolic mechanism in the artery route was investigated to determine if an injection performed in the face, orbit, and nasal area can result in fundus artery occlusion or brain infarction through rich vascular anastomosis. However, explaining the delayed course by this mechanism is difficult because when the retrograde emboli are forced from the ophthalmic artery into the internal carotid artery, the emboli may be pumped into the cerebral circulation immediately.

From the perspective of the venous return system, a delayed course of foreign body embolism could occur. Rare cases of nonthrombotic pulmonary embolism (NTPE) have been reported, with a delayed course of symptoms after injection of hyaluronic acid. In the current case, if foreign body emboli were injected into the venous system of the face, the emboli may have returned to the heart and possibly caused NTPE syndrome. However, the symptoms of pulmonary embolism did not develop in this patient; instead, cerebral infarction occurred. We posit that the emboli of the hyaluronic acid might have entered the vessel not only through the artery by the retrograde mechanism, which caused central retinal artery occlusion, but also...
through the antegrade venous system return to the right atrium, and might have then passed through ASD, which caused paradoxical embolism and ultimately resulted in cerebral infarction.

In the current case, the new onset of weakness developed 9 hours after visual loss and was distributed only in the left elbow, wrist, and hand. The special distribution of monoparesis can be attributed to Penfield and Boldrey's cortical homunculus, that is, lesion of the primary motor cortex, which could be correlated with the distribution, presented in the brain MRI (Figure 1D) and confirmed the delayed course of cerebral infarction. In conclusion, we highlight the importance of further evaluation and careful history taking of the cardiovascular condition of patients in preprocedure surveys.

Figure 1  (A) Fundus examination of the right eye demonstrates a cherry-red spot (red arrow), which indicates central retinal artery occlusion. (B) Fundus examination of the left eye revealed normal retina and optic disc appearance. (C) Brain magnetic resonance imaging revealed many small acute infarcts distributed over primary motor cortex (orange cross line). (D) Primary motor cortex with infarction site (red line).

References