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Case report

Severe ocular complications following facial calcium hydroxylapatite injections: Two case reports

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ABSTRACT

Dermal soft-tissue augmentation using a filler is a technique widely used for facial cosmetic enhancement. However, potential complications following facial cosmetic injections have heightened the possibility of iatrogenic visual loss. We report two cases of severe ocular complications after nasal cosmetic enhancement. Both cases had poor visual outcomes in spite of emergency management. The second patient is a rare case with bilateral anterior ischemic optic neuropathy after dermal soft-tissue augmentation. The visual outcome was correlated with the location and the extent of the arterial embolization. Unfortunately, there is still no standard treatment protocol for vision-threatening complications. Clinicians should always keep in mind that embolic arterial occlusion may occur after augmentation.

1. Introduction

Dermal soft-tissue augmentation using a filler is a widely used technique for modern facial cosmetic enhancement due to its convenience, high predictability, and good results. However, a serious complication of arterial embolization can occur due to accidental injection of the filler material into small vessels leading to tissue necrosis. In recent years, complications after facial cosmetic injections have heightened the awareness of the possibility of iatrogenic visual loss. Most previous case reports have shown fair visual recovery and spontaneous resolution of the embolization episodes.

Herein, we present two cases whereby severe ocular complications occurred after nasal cosmetic enhancement. One patient suffered from unilateral irreversible visual loss leading to eyeball atrophy. The other patient encountered bilateral ischemic optic neuropathy and diffuse chorioretinal ischemia in one eye. These cases serve as a reminder that adverse events after cosmetic enhancement can cause permanent ocular complications and can occur bilaterally.

2. Case reports

2.1. Case 1

The first case was a 47-year-old female hepatitis B virus carrier with normal liver functions, who denied having a previous history of other systemic diseases. She received a facial calcium hydroxylapatite injection in the glabellar area for augmentation rhinoplasty. She complained of a headache immediately while the injection was being given. Sudden left eye blindness and general weakness developed 10 minutes after the injection. Severe headache and left ocular pain occurred 1 hour later, and she was sent to our hospital.

In the emergency room, there was no light perception in her left eye, with retinal cherry-red spots, moderate ptosis, and limited eye movements. The tentative diagnosis was central retinal artery occlusion and ophthalmoplegia of the left eye. Although the intraocular pressure of her left eye was within normal limits, we performed anterior chamber paracentesis and administered timolol and acetazolamide in order to push the emboli to the distal side. A neurological examination revealed mild right central facial palsy and right limb weakness. Brain magnetic resonance imaging showed multiple small infarctions with intracranial hemorrhages in the left cerebral hemisphere (Fig. 1A). Carotid duplex showed an

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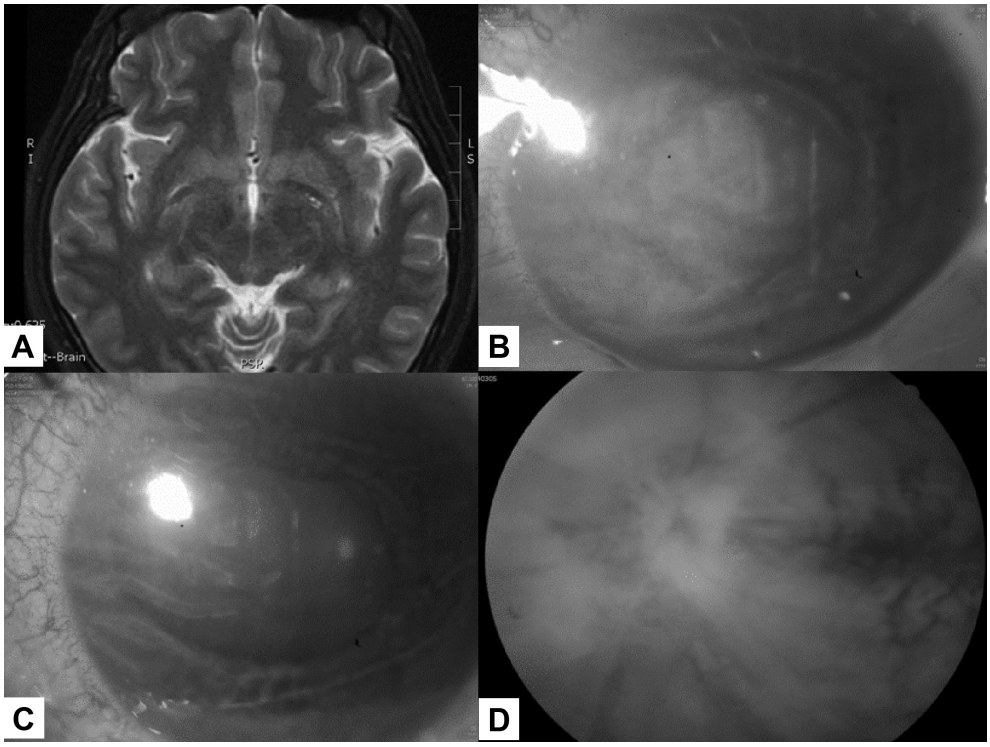

Fig. 1. Case 1: (A) Brain magnetic resonance imaging (T2 FS) showing multiple small infarctions and multiple intracranial hemorrhages in the left cerebral hemisphere. (B) The cornea of the left eye 1 day after the injection became edematous. (C) The corneal edema of the left eye is markedly improved 3 weeks after the injection. (D) Fundus photography of the left eye 3 weeks after the injection showing total exudative retinal detachment with retinal atrophy.

adequate flow amount without obstruction or atherosclerosis. During admission, she received hydration under the impression of multiple artery embolizations; meanwhile, oral antiplatelet agents were also given. Systemic work-ups for diabetes, rheumatologic disorders, and hypercoagulation status were done, but all findings were negative.

Orbital infarction syndrome developed 1 day after the calcium hydroxylapatite injection (Fig. 1B). The cornea became edematous, and a mild anterior chamber reaction was noted. Her left eye became hypotonous. The fundus was blurred due to an edematous cornea; however, the retina was generally pale. We then added topical betamethasone. Multiple reticulated, erythematous-to-violaceous ulcerative skin lesions were noted over the glabella, perinasal, and periorbital areas. The ocular pain and ophthalmoplegia gradually improved (Fig. 1C), but the retina eventually became totally detached (Fig. 1D). No light perception in her left eye was noted during follow-up.

2.2. Case 2

In the second case, a 33-year-old female with no history of systemic diseases received a dermal calcium hydroxylapatite injection for nasal bridge augmentation. Sudden eye pain and visual impairment occurred in the left eye during the injection. Bilateral lower visual field defects were noted several hours later, and she was sent to our hospital.

The initial visual acuity was 20/60 in the left eye and 20/20 in the right eye. Fundoscopy of the right eye was relatively normal (Fig. 2A). However, the fundus of the left eye showed both diffuse drusen-like lesions in the retina and diffuse crystal-like lesions in the level of the choroid. Retinal whitening was seen around the disc with a radial peripapillary capillary distribution (Fig. 2B). A visual field examination revealed altitudinal visual field defects in both eyes and generalized depression over the left eye (Fig. 3). No extraocular muscle limitation was noted. The tentative diagnosis was bilateral anterior ischemic optic neuropathy and chorioretinal vascular occlusion over the left eye. She received hyperbaric oxygen therapy, systemic low-dose steroids, antiaggregants, and topical and oral antiglaucomatous agents.

Five days later, a follow-up fundoscopy of the left eye showed disc swelling, extensive small white embolic particulates in the choroidal vessels, and peripapillary retinal whitening with a flame-shaped intraretinal hemorrhage (Fig. 2C). The intraocular pressure was only 5 mmHg over the left eye, with a visual acuity of 20/20 in the right eye, and 20/50 in the left eye.

Because the ocular condition of the patient did not show obvious improvement, systemic treatments, including systemic steroid and anticoagulants, were gradually tapered. The topical steroid treatment was maintained, but no apparent visual improvement was noted. The antiglaucomatous agents were also discontinued due to persistent intraocular pressure below the normal range (around 5–10 mmHg) after 2 months of treatment.

Two months later, the visual acuity had decreased to 20/200 in the left eye and 20/20 in the right eye. The retinal hemorrhage over the left eye resolved; however, the crystal-like linear deposition in the choroidal layer persisted, and the vessels became attenuated. Bilateral optic discs were pale (Fig. 2D). A visual evoked potential examination revealed a low amplitude waveform with prolonged latency over the left eye and a relatively normal waveform over the right eye. A follow-up visual field test still showed altitudinal visual field defects in both eyes and generalized depression in the visual field of the left eye.

3. Discussion

Calcium hydroxylapatite is composed of microspheres of a synthetic bone suspended in a methylcellulose gel matrix. It is thicker than hyaluronic acids and has longer lasting effects for facial correction.[1] It is widely used to treat severe or moderate wrinkles of

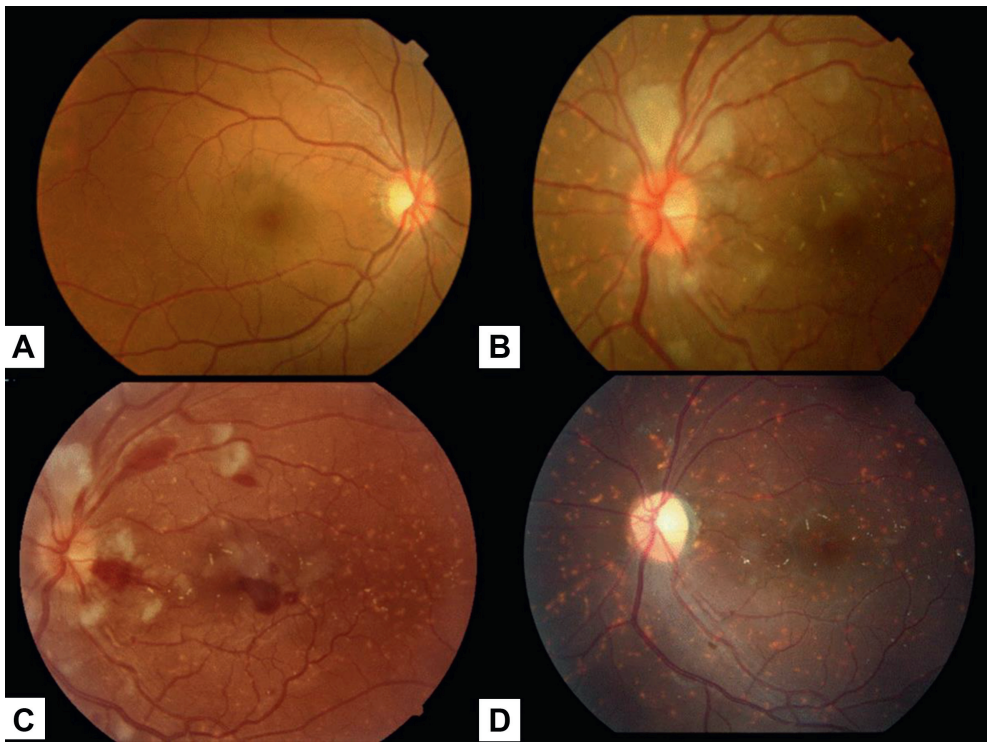

Fig. 2. Case 2: (A) Fundus photography of the right eye showing a relatively normal retinal presentation. (B) Ocular fundoscopy over the left eye revealed diffuse drusen-like lesions in the retina and diffuse crystal-like lesions in the level of the choroid. Retinal whitening around the disc is seen in a radial peripapillary capillary distribution. (C) Follow-up fundoscopy of the left eye 5 days later shows disc swelling, extensive small white embolic particulates in the choroidal vessels, and retinal whitening with a flame-shaped intraretinal hemorrhage. (D) The retinal hemorrhage over the left eye resolved; however, the crystal-like linear deposition in the choroidal layer persisted, and the vessels became attenuated. The optic discs were pale.

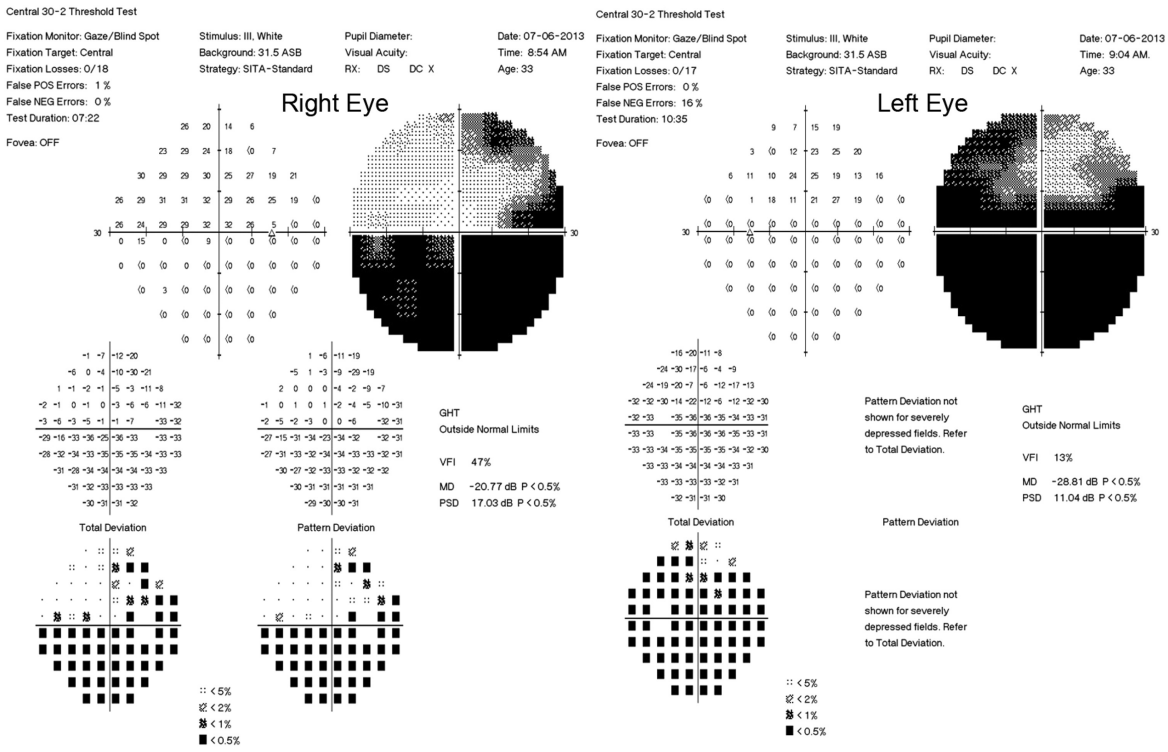

Fig. 3. Case 2: Visual field examination 2 days after the injection reveals altitudinal visual field defects in both eyes, and generalized depression over the left eye.

the forehead, and facial aesthetic enhancement. However, the bony particles can act as potential emboli, which can cause small vessel occlusions if incidental intravascular injection occurs. In Case 1 of the present study, the embolization resulted in multiple small infarctions with intracranial hemorrhages in the left cerebrum, ophthalmoplegia, central retinal artery occlusion, orbital ischemic syndrome, discoloration, and necrosis of the skin after injection of the filler into the glabellar area. Previous reports have hypothesized that the injection pressure might retroactively push filler materials into arteries and create multiple distal site occlusions.[2–4] However, in Case 2, the filler with calcium hydroxylapatite not only resulted in acute vision damage, but also progressive choroidal and retinal vessel occlusion caused gradual visual loss. The most common dosage of dermal fillers for nasal bridge augmentation is about 1.0–1.5 mL among Asians. According to surgeon statements, both of the patients in this study complained of eye pain and blurred vision when $<$ 1.0 mL filler has been injected, and therefore complications are not likely to be dose related.

Although embolic arterial occlusion after cosmetic procedures is rare, the influence on vision is devastating. Risk factors include the site of injection, techniques used during the injection, and the skill of the practitioner.[5] We inspected previous case reports and none addressed the relationship between systemic diseases and complications. The glabellar area is one of the most common sites associated with severe ocular complications after injection. Many filler materials have been reported to cause blindness after cosmetic injections, including fat tissue, paraffin, silicone oil, bovine collagen, hyaluronic acid, polymethylmethacrylate, and calcium hydroxylapatite.[6] The mechanism of orbital ischemic syndrome may be due to direct entry of the needle tip into the branches of the lateral nasal and dorsal nasal arteries. The filler materials may then enter the ophthalmic artery and occlude the central retinal, short, and long posterior ciliary arteries. Another possibility is that the filler materials are injected into Kiesselbach's area, an area of anastomosis between the internal and external carotid arteries, and then enter the central retinal artery through the anterior ethmoidal artery. Blockage of the long posterior ciliary artery can cause ciliary body ischemia leading to ocular hypotony, which has been reported to be associated with decreased aqueous humor production.[7] Ophthalmoplegia occurs due to the involvement of the muscular branch of the ophthalmic artery. In addition, the materials may flow in a retrograde manner from the ophthalmic artery into the intracranial middle cerebral artery through the internal carotid artery, resulting in cerebral infarction and distal embolism with secondary hemorrhagic transformation.

Various protocols have been reported with regards to the management of skin lesions, including immediate discontinuation of the injection and gently massaging the affected area, applying warm water on gauze, nitroglycerine paste, injecting hyaluronidase (for hyaluronic acid gel), and low molecular weight heparin.[5] However, no protocols currently exist for ocular complications. In previous reports, several different individualized treatments were applied, including medical and surgical approaches such as administration of systemic steroids, carbonic anhydrase inhibitor, vasodilators, antiplatelet agents, performing ocular massage, and anterior chamber paracentesis.[6] However, the visual prognosis was still reported to be very poor despite the management, and $<$ 10% of the patients (3 of 32 cases) achieved visual recovery.[6] If ocular ischemic syndrome develops, topical steroids can be given to suppress anterior segment inflammation, along with cycloplegics to stabilize the blood-aqueous barrier and prevent spontaneous hyphema. In cases of chronic ischemia, preventing neovascularization is very important. Methods of prevention include aspiration prior to the injection, injecting superficially and medially, limiting the bolus size and syringe size, and avoiding sharp needles.

In conclusion, facial calcium hydroxylapatite injection is a widely performed dermal soft-tissue augmentation procedure, however it may cause central retinal artery occlusion, ocular and orbital ischemic syndrome, and chronic retinal and choroidal vessel occlusion. Although it is a rare adverse event, clinicians should be aware of the potential risk of embolization when injecting dermal fillers in the glabellar area.

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