Complete recovery of filler-induced visual loss following subcutaneous hyaluronidase injection

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ABSTRACT

The rise in popularity of hyaluronic acid (HA) dermal filler injection has caused an exceptional increase in the number of cases of reported irreversible blindness. Here, we reported a case of ischemic optic neuropathy and ophthalmoplegia following subcutaneous HA filler injection with complete visual recovery. A 31-year-old Chinese woman presented with sudden onset of right monocular visual impairment associated with diplopia. Patient had received a hyaluronic acid-containing filler injection for nasal dorsum augmentation twelve hours prior to presentation. Visual acuity of the right eye was counting finger. A right relative afferent pupillary defect was demonstrated with ophthalmoplegia. Humphrey visual field test disclosed a right inferior altitudinal field defect with impairment of colour vision. Computed tomography of the orbit revealed mild enlargement of the right medial and inferior recti muscles. Our patient showed a tremendous improvement of vision after a subcutaneous hyaluronidase injection with complete visual recovery within 2 weeks.

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Introduction

Hyaluronic acid (HA) is a biodegradable and biocompatible dermal filler which is a widely used technique for modern facial cosmetic enhancement.¹ Their rise in popularity recently has caused a substantial increase in the number of cases of irreversible blindness reported.² Here, we reported a case of posterior ischemic optic neuropathy (PION) with ophthalmoplegia following dermal HA filler injection, which had a complete visual recovery.

Case report

A 31-year-old Chinese woman presented with a sudden onset of right monocular visual impairment associated with diplopia. The patient had received a HA-containing filler injection into the glabella and dorsum of her nose for nasal dorsum augmentation 12 h prior to presentation. She experienced a severe right periorbital pain and headache immediately after the injection followed by a sudden and profound ipsilateral visual loss in the inferior half of the visual field.

On examination, there was multiple reticulated and erythematous skin discoloration over the site of injection (Figure 1). Her best corrected visual acuity of the right eye was counting finger while that of the left eye was 6/9. A right relative afferent pupillary defect (RAPD) was present. There was exotropia of the affected eye on primary gaze. However, there was no ptosis or proptosis noted. The extraocular movement of the right eye showed limitation of adduction, depression and elevation on the left gaze (Figure 2). The right anterior segment and fundus examination revealed no abnormalities. The fellow eye and the rest of the systemic examination were normal.

Humphrey perimetry disclosed a right inferior altitudinal field defect (Figure 3). Standard colour vision test with Ishihara plate was documented as 1/21. Routine blood tests, thyroid function and connective tissue screening were normal. Computed tomography of the orbit revealed mild enlargement of the right medial and inferior recti muscles with increased intensity, heterogeneous appearance of the superior rectus muscle (Figure 4). Both optic nerves were normal. Fundus fluorescein angiography (FFA) was not

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Figure 1. Multiple reticulated skin lesions and erythematous discoloration over the glabella, dorsum and tip of the nose.

available at the time of presentation. A diagnosis of right posterior ischemic optic neuropathy (PION) with ophthalmoplegia secondary to hyaluronic filler injection was made based on clinical ground.

She was treated with a total of 60 IU/mL of hyaluronidase enzyme which was given subcutaneously over the glabellar and dorsum of the nose twelve hours after the symptoms started. Fortunately, our patient showed a tremendous improvement of vision 2 weeks later with right eye visual acuity of 6/9 following subcutaneous hyaluronidase injection alone. The necrotic skin healed with minimal scarring after an intensive wound care. Ophthalmoplegia and visual field defect had resolved 3 months post treatment. Her best corrected visual acuity of both eyes was 6/6 with the absence of RAPD.

Figure 3. Humphrey 24-2 visual field of the right eye showed inferior altitudinal visual field defect.

Discussion

In recent years, the injectable soft tissue fillers have become an increasingly popular option because of its versatility and promising results. This case heightened the awareness of the uncommon but potentially disastrous consequences of this minimally invasive procedure. The most serious complications are vascular in nature which has been reported to include blindness, cerebral ischemic events and even death.²

In a review of world literature on blindness from fillers by Belezney et al., the authors found that HA was the second most common type of filler associated with ocular complications after autologous fat injection.¹ Based on the selective ophthalmic angiographic findings as noted by Kim et al., the authors concluded that patients who received HA were more likely to get a more distal and localised occlusion which explained the milder symptoms and better visual prognosis than those who received autologous fat.³

Our patient experienced a sudden painful partial visual loss with normal fundus findings at presentation. The presence of relative afferent pupillary defect and a demonstrable inferior altitudinal field loss were consistent with ipsilateral

Figure 2. Nine cardinal positions of gaze showed limitation of right extraocular movements on adduction, depression and elevation.

Figure 4. Axial (A) and coronal view (B) of CT orbit showing enlargement of right medial and inferior rectus.

ischemic optic neuropathy post HA filler injection. The diagnosis of PION was made and can be distinguished clinically from anterior ischemic optic neuropathy (AION) by a normal-appearing optic nerve head. The posterior segment of the optic nerve is usually supplied by a pial capillary plexus and is derived from collateral branches of the ophthalmic artery.⁴ Hence, the potential retrograde embolization of the HA particles into the limited collateral circulation of these peripheral branches will cause sudden disruption of the vascular supply to the affected area.⁵

In contrast, Chen et al. reported a case of acute branch retinal artery occlusion (BRAO) following HA filler injection where the segmental retinal whitening was seen on examination due to significant swelling of the inner retinal layers from the ischemic event.⁶ FFA would provide a confirmation of BRAO, showing a delayed filling of the involved retinal artery.⁶

Our case also demonstrated ipsilateral ophthalmoplegia with enlarged recti. Few cases were reported to have visual loss and ophthalmoplegia secondary to oculomotor nerve palsy following nasal dorsum injection of calcium hydroxyapatite and HA-based fillers.^{6–8} In contrast to our present case, none of these cited case reports have shown to have radiological evidence of extraocular muscle enlargement. The radiological findings in our present case were more in keeping with restrictive ophthalmoplegia rather than neurological cause. To the best of our knowledge, filler-induced enlarged recti has not been previously reported.

We postulated that the inflammation and tissue oedema of the affected muscles could be due to direct infiltration of the filler materials into the surrounding

tissues and muscles or due to the volume-expansion properties of HA itself.¹ Another explanation includes the possibility of hypersensitivity reaction to the HA particles that has extended through the septum into the anterior orbit where the muscles were presented.⁹ Furthermore, the enlargement of the extraocular muscles involved mainly the right medial and inferior recti probably due to their close proximity to the injection site and also due to the gravitational force which caused a denser filler substance to be distributed in downward direction.

Following hyaluronic acid embolism, there is a chance that it can be dissolved by hyaluronidase administration within the window period of 60–90 min.¹⁰ Hyaluronidase is a soluble protein responsible for the enzymatic degradation of glycosaminoglycans including HA. The half-life of hyaluronidase is about 30 min in the subcutaneous tissues of rodents.¹¹ Adverse reactions to hyaluronidase are rare.^{11,12} In a fortunate case like ours, although presented twelve hours post filler injection, she showed a favourable sequelae in terms of visual recovery with subcutaneous hyaluronidase injection alone.

Proposed mechanism includes our patient probably have received a smaller size of HA particles, which has the tendency to obstruct the more distal part of the ophthalmic artery branches. The patient also would have received a smaller amount and less concentrated of HA filler thus a possibility of a transient embolism could not be ruled out. It would be highly suggested that the emboli could have dislodged spontaneously over time or an adequate dosage of perivascular hyaluronidase was able to cross the vessel wall and was carried downstream by the arterial pressure into the embolised region.

Many studies have shown that subcutaneous hyaluronidase is effective in rescuing tissue necrosis following intravascular HA injection.^{11,12} However, there was very little evidence concerning this method in cases of accidental ocular circulation embolization. Our case further supports the ex vivo study which showed hyaluronidase enzyme was able to degrade HA filler material trans-arterially.¹¹ Our present case has shown that the rescue with subcutaneous administration of hyaluronidase might well be one of the effective option to dissolve intraorbital and intravascular hyaluronan without direct canalization of the affected artery.⁷

To date, there was only one report of a successful subcutaneous hyaluronidase injection in reversing HA-induced vision loss by Goodman GJ and Claque MD.¹³ The authors experienced a reversible visual loss by injecting 300 units of hyaluronidase into the superomedial orbit. However, there was no details regarding their patient's visual loss whether it was a result of vascular occlusion or vasovagal response associated transient visual loss.

Other methods of delivering hyaluronidase enzyme have been advocated including retrobulbar and direct intra-arterial infusion in rescuing vision loss. However, recent case series reported that both methods have not been found to be effective in restoring visual outcome in at least 4 h after receiving HA-based filler.^{3,12,14} The long term visual outcome of intravascular filler injection is depending on the site of embolization and visual acuity at presentation. The more distal the obstruction site and good visual acuity at presentation will have a better long-term outcome.³

Conclusion

We reported a rare case of a complete recovery of inadvertent visual loss associated with right PION with restrictive ophthalmoplegia following cosmetic facial filler injection. It is very crucial for the practitioner to be alert and aware of the devastating complication of intravascular injection from this cosmetic procedure. Subcutaneous hyaluronidase injection may be a potential treatment strategy in managing vision loss in HA-filler-associated subtle ophthalmic artery occlusion and preserving good visual outcome.

Hence, a good understanding and using of applied anatomy of facial vasculature is vital to minimize this catastrophic event.

Conflict of interest

The authors have no proprietary or commercial interest in any materials discussed in this case.

Patient Consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

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