Visual Loss Secondary to Cosmetic Filler Injection

<table>
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<tr>
<th>Seriousness of complication</th>
<th>Frequency of complication</th>
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<td>Major complication</td>
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Title: Visual Loss Secondary to Cosmetic Filler Injection
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Date: February 2020
Version: 2.7
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Abstract

Visual loss is perhaps the most feared complication for any practitioner performing cosmetic injections and although extremely rare, if it does occur, the prognosis is very poor. This article produced by the Aesthetic Complications Expert Group provides practical support for injectors to try and minimise risk and management options for this devastating complication. Injectors need to be aware that there are no absolute safe areas on the face to inject and no completely safe products.

Keywords

Visual loss, blindness, hyaluronic acid, cosmetic, aesthetic, fillers, complication, hyaluronidase, ophthalmic, ocular massage, aesthetic complications expert group.

Definition

Any impairment or loss of vision (temporary or permanent) secondary to central retinal or retinal branch artery occlusion occurring as a direct consequence of percutaneous injection for aesthetic treatment1.

Introduction

Blindness after facial injection is extremely rare and was first reported by von Bahr over 50 years ago after scalp injection of a hydrocortisone suspension to treat alopecia2. The first cases after aesthetic filler treatments were reported in the 1980s (four cases) and rose to at least 16 reported cases between 2000-2010, presumably related to the increase in the number of treatments being performed1.

Depending on which artery is occluded, vision loss can be classified into six subtypes3,4,5:

1. Ophthalmic artery occlusion (OAO)
2. Generalised posterior ciliary artery occlusion with relative central retinal artery sparing (PCAO)
3. Central retinal artery occlusion (CRAO)
4. Branch retinal artery occlusion (BRAO)
5. Anterior ischaemic optic neuropathy (AION)
6. Posterior ischaemic optic neuropathy (PION)

There are also four subtypes of periorcular complications associated with blindness following cosmetic filler injection6:

Type I – Blindness without ophthalmoplegia (paralysis or weakness of ocular muscles) and ptosis
Type II – Blindness with ptosis but without ophthalmoplegia
Type III – Blindness with ophthalmoplegia but without ptosis
Type IV – Blindness with ophthalmoplegia and ptosis

Based on previously reported case studies, improvement of visual acuity in patients with vascular occlusion after filler injection is extremely rare. By contrast, periorcular symptoms such as ptosis and ophthalmoplegia often show dramatic recovery6.

Mechanism

Terminal branches of the ophthalmic artery, namely the supraorbital and supratrochlear, supply the medial forehead and anastomoses between these vessels and the terminal branches of the angular artery are well documented7. Similarly, anastomoses with the superficial temporal arteries and the orbit has also been demonstrated8. Injection of filler material into one of these vessels may lead to retrograde flow to beyond the point of the origin of the ophthalmic artery and when pressure from the plunger is released, systolic pressure drives the product forward and to
enter the ophthalmic artery or central retinal artery resulting in visual loss.

The requirements for blindness to occur include the retrograde and subsequent anterograde passage of material, injection pressure exceeding systolic pressure and sufficient amount of material within the lumen of the vessel. Findings indicate that the average entire volume of the supratrochlear artery from the glabella to the orbital apex is 0.085ml (range 0.04-0.12ml) and injection volume should not exceed this volume in critical injection points.

Incidence

Globally, at least 98 cases of visual loss after aesthetic facial injection have been reported prior to 2015. A review of the world literature by Belezany identified 98 cases of vision change. High risk areas were glabella (38.8%), nasal region (25.5%), nasolabial fold (13.3%), and forehead (12.2%). Autologous fat was responsible for most of the complications (47.9%) followed by hyaluronic acid (23.5%) and the outcome was worse in cases when autologous fat had been injected.

In 2012, the UK reported its first case, after injection to the temple with Poly-L-Lactic Acid, the first report with this product. In 2013, the first two cases of bilateral blindness were reported (calcium hydroxyapatite to the nose and hyaluronic acid to the glabella, which also led to cerebral infarction). The exact incidence of this devastating adverse event remains to be determined due to the heterogeneity of data.

Due to the seriousness of this complication, significant visual loss should be explained to the patient as a possible rare complication as part of the consent process and should be included on consent forms.
**Signs and symptoms**

- Visual loss (Complete or partial)
- Pain (Ocular, facial, headache or a combination)
- Paralysis or weakness of ocular muscles
- Nausea
- Ptosis
- Posterior displacement of the eye
- Strabismus (misalignment of the eyes when looking at an object)
- Corneal oedema
- Pupillary abnormality
- Iris atrophy
- Anterior chamber inflammation
- Phthisis bulbi (shrunken, non-functional eye)
- Livedo reticularis (a mottled, reticulated vascular pattern of the skin)

Visual loss following embolisation of dermal filler typically occurs within seconds of injection⁷ although visual loss has been reported 7 hours post-treatment in the case of a posterior ciliary artery occlusion¹⁰. Complete loss of vision is the normal presentation although there may be visual field defects. Visual loss is often accompanied by sudden onset of severe pain (ocular, facial, headache or any combination) although central retinal and retinal branch artery occlusions may present without ocular pain. Other symptoms include ophthalmoplegia (paralysis or weakness of ocular muscles), ptosis, enophthalmos (posterior displacement of the eye) and horizontal strabismus (abnormal alignment of the eyes). These symptoms accompany blindness due to disturbed flow to the superior and inferior branches which supply the extraocular muscles⁶.

Many cases with visual loss and periorcular symptoms also subsequently developed enophthalmos and surgery could be considered in patients demonstrating greater than 2mm descent within 6 weeks of the injury²⁵.

Other symptoms and signs include corneal oedema, anterior chamber inflammation, nausea, headache, pupillary abnormality, iris atrophy, phthisis bulbi and livedo reticularis⁷.

Cerebral infarction can accompany retinal artery occlusion and therefore signs and symptoms of this may also be present such as aphasia or even contralateral hemiparesis. Central nervous complications were seen in 23.5%¹² to 39%⁵ of cases where vision was affected.

An MRI scan should be performed in all patients who suffer visual loss or ocular pain because of filler injections⁴.

**Areas of caution**

Injections into the nose and glabella form most reported cases of blindness⁷ although moderate risk sites included the nasolabial folds, forehead, periorcular region, temple and cheek. Uncommon sites were the eyelid, lips and chin. Due to the complex vascularity of the face, any region of the face has the potential to cause this complication⁴.

**Minimising the risk**

The key preventative strategies are listed below⁶:

1. Know the location and depth of facial vessels and the common variations⁷. Injectors should understand the appropriate depth and plane of injection at different sites.
2. Inject slowly and with minimal pressure¹⁴,¹¹.
3. Inject in small increments¹,⁷ so that any filler injected into the artery can be flushed peripherally before the next injection. This prevents a column of filler traveling retrograde and subsequently anterograde. No more than 0.1 mL of filler should be injected at one time.
4. Move the needle tip while injecting⁴, so as not to deliver a large deposit in one location.
5. Aspirate before injection¹,⁴,⁷. This recommendation is controversial as it may...
not be possible to get flashback into a syringe through fine needles with thick gels. In addition, the small size and collapsibility of facial vessels limit the efficacy.

6. Use a small-diameter needle\(^1,7\). A smaller needle necessitates slower injection and is less likely to occlude a vessel. If a sharp needle is being used, then a perpendicular injection directly in contact with the bone is recommended. Injecting into a deeper plane may avoid vessels\(^7\).

7. Smaller syringes\(^4\) are preferred to larger ones as a large syringe may make it more challenging to control the volume and increases the probability of injecting a larger bolus. However, decanting filler material from its original syringe is not recommended.

8. Consider using a cannula (25 gauge or greater bore size), as they are less likely to pierce a blood vessel\(^1,7\). Some authors recommend use of the cannula in the medial cheek, tear trough, and nasolabial fold.

9. Use extreme caution when injecting a patient who has undergone trauma or a previous surgical procedure in the area\(^4\).

10. Ensure that you are adequately trained, using an appropriate product and are competent in the area in which you are treating as well as competent in the management of complications.

11. A technique to possibly prevent embolism of filler is digital compression of the inferior-medial orbital rim and the side of the nose\(^7\) whilst injecting.

Sometimes the ophthalmic artery does not arise normally from the internal carotid artery, but from the middle meningeal artery, which originates from the external carotid artery. Furthermore, the zygomatic-orbital artery raised from the superficial temporal artery has an anastomosis with branches of the ophthalmic artery and may be a retrograde arterial embolic route\(^4\). Facial anatomy can be diverse, and the facial artery originated from a single arterial trunk in 86% of specimens and branching patterns were only symmetrical in 53% of cases\(^16\). In conclusion, there is no absolute safe area of the face to inject\(^1\).

**Treatment of blindness after facial injection**

Once the retinal artery has been occluded there is a window of 60-90 minutes before blindness is irreversible\(^2\). It is essential to transfer the patient to the nearest specialist eye hospital via blue light ambulance as quickly as possible\(^4\). Transfer to a non-specialist emergency department may lead to inordinate delay and a worse outcome\(^7\). Ensure that you know where your closest specialist eye department is and contact the on-call team as soon as possible to inform them of the situation. Give medical staff as much information as possible about the product, area and volume of injection.

Although there is no generally agreed treatment regimen\(^17\), there are actions that may help. Prado\(^18\) suggests a 6-step therapy protocol with a ‘blindness safety kit’ that may be used in a clinical setting and then continued in hospital. The protocol was adapted from Lazzeri et al\(^1\).

**Treatment of Visual Loss after Facial Fillers**

Indications for treatment are sudden onset ocular pain and/or loss of vision. The goal is to quickly reduce the intraocular pressure to allow for the emboli to dislodge downstream and improve retinal perfusion\(^1\).

- Stop treatment immediately.
- Place patient in supine position\(^7\).
- Call 999 and prepare to transfer patient to hospital setting as soon as possible.

**Do not let any of the following measures delay referral to a specialist eye hospital.**

**Reduce Intraocular Pressure\(^7\)**

Administer Timolol\(^4,7\) 0.5% 1-2 drops in the affected eye only. This beta-adrenergic
antagonist will aim to reduce intraocular pressure by reducing aqueous humor production.
The patient should be encouraged to rebreathe in a paper bag. The aim is to increase CO₂ levels within the blood which will cause retinal arteries to vasodilate and could help dislodge blockage. An alternative to rebreathing through a paper bag is the inhalation of carbogen (95% oxygen, 5% carbon dioxide)⁴.

Oral acetazolamide⁴,⁷,¹⁴ may be considered although intravenous administration in hospital is likely to be of greater benefit.

Give the patient 300 mg of aspirin to prevent blood clotting¹⁴.

**Dislodge the embolus to a more peripheral position**

Massage the globe with repeated increasing pressure. Prolonged ocular massage attempts to dislodge emboli by rapidly changing intraocular pressure⁴ thereby changing the pressure and flow in the retinal arteries. Increasing the intraocular pressure also causes a reflexive dilation of the retinal arterioles and dropping it suddenly increases the volume of flow significantly.

Ocular massage is performed with the patient looking straight ahead with eyes closed. Gentle pressure is applied over the sclera with a finger, indenting the globe by a few millimetres and then releasing at a frequency of 2-3 times a second¹⁹. This should be continued until advised otherwise by staff at the eye hospital. Commonly, firm ocular massage is advised for several seconds and repeated only a few times. The alternative advice originates from 2 case studies where embolised retinal arteries were directly visualised during the massage process. This showed that even when the emboli were dislodged, more would occlude the vessel when massage stopped. Prolonged high frequency massage (up to 3 hours) had a better clearing effect¹⁹.

**Administer Hyaluronidase**

If hyaluronic acid filler has been used, administer hyaluronidase to the treatment area (Refer to the Aesthetic Complications Expert Group guideline: The Use of Hyaluronidase in Aesthetic Practice). Retrobulbar injection of hyaluronidase has been advocated by many plastic surgeons as emergency treatment, however an evaluation by Zhu et al³ failed to show any improvement in visual loss following 1500-3000 units of hyaluronidase injected into the retrobulbar space in 4 patients. Consensus from Ophthalmologists when consulted on the topic of retrobulbar hyaluronidase injections by aesthetic practitioners is that it is a technically difficult procedure even to a competent Ophthalmological Surgeon and the scope for causing more harm means the risks, including perforation of the orbit and haemorrhage, outweigh any benefit. However, Chestnut²⁰ reported in Dermatologic Surgery full restoration of vision after blindness was reported in a patient receiving hyaluronic acid fillers in the midface. Vision was restored following three retrobulbar hyaluronidase injections and aspirin. A total of 750 units were administered, 450 units as retrobulbar injections and 300 units to surround the supraorbital and infraorbital foramina.

A study has suggested that retrobulbar hyaluronidase injection is unlikely to alleviate vision loss caused by hyaluronic acid (HA) filler injections. The research was published in *Plastic and Reconstructive Surgery* journal and involved the study of six human cadaveric optic nerves in vitro. The study was designed to assess the ability of hyaluronidase to passively diffuse through the dura to degrade intraparenchymal optic nerve HA filler in parallel with various control conditions. The authors concluded that the results demonstrated the absence of transdural degradation of intraneural HA following direct immersion of the optic nerve in concentrated hyaluronidase. They stated that this suggested that HA occlusion of the central retinal artery is unlikely to be cleared by retrobulbar hyaluronidase injection by means of diffusion through the dural sheath.
The authors stated in the study, “Although extravascular hyaluronidase does appear to have utility in treating other ischemic complications, its benefits in reversing filler-induced blindness are tentative given these findings and previously demonstrated lack of efficacy in vivo. Further studies examining alternative routes of administration are needed to develop an effective protocol for treating this devastating complication.” In conclusion, hyaluronidase does not demonstrate the ability to cross the dural sheath of the optic nerve, suggesting that retrobulbar hyaluronidase injection is unlikely to alleviate hyaluronic acid gel–mediated central retinal artery occlusion and blindness. The goal was to see where hyaluronidase diffuses when it is injected with 4 following approaches; inferolateral orbit, supraorbital foramen, infraorbital foramen, and intravitreous were not able to demonstrate adequate hyaluronidase perivascular or intravascular infiltration in or around the retinal artery with any of the 4 approaches in this cadaver model. Various doses ranging from 150-1000 units have been documented in the literature. The volume suggested should not exceed 5ml per injection attempt due to the risk of compressing the contents of the retrobulbar space. Therefore, volume of hyaluronidase should be kept low but concentration high. It again should be stressed that the person available with the highest level of expertise should be handling this stage of treatment, and any treatment should not delay appropriate transfer. Treatment should be extremely timely because time to reverse the blindness is short.

Based on the current evidence regarding retrobulbar injections of hyaluronidase, the Aesthetic Complications Expert Group do not advocate this treatment for visual loss caused by hyaluronic acid filler. Injection of hyaluronidase into the supratrochlear or supraorbital arteries to reach the embolus seems a more sensible approach.

The use of hyaluronidase has been shown to be ineffective at recanalising the retinal artery occlusion or improving the visual outcome after 4 hours after onset of blindness.

**Specialist Treatment**

Once the patient has been transferred to the hospital setting, the aim is to further reduce intraocular pressure, remove/reverse central retinal ischaemia and increase blood flow to the retina.

- Injection of 500mg IV Acetazolamide. This should increase retinal blood flow and reduce intraocular pressure.
- Consider the use Enoxaparin subcutaneously or IV Heparin for anticoagulation. If the patient is having signs or symptoms of cerebral infarction, defer this step until a Neurologist has assessed the patient.
- Intravenous infusion of mannitol 20% (100ml over 30 minutes).
- Consider injection of hyaluronidase via the trans-orbital approach into the more prominent and tortuous post-septal ophthalmic artery.

**Other supportive therapies include**:  

- Anterior chamber paracentesis to immediately lower intraocular pressure.
- Steroid administration, intravenous dexamethasone.
- Judicious use of antibiotics for suspected infection.
- Hyperbaric oxygen may salvage vulnerable retinal damage. Practitioners should familiarise themselves with their nearest hyperbaric oxygen chamber.
- Intravenous prostaglandin E1 to increase blood flow to the retina and decrease activation of thrombocytes and neutrophils.

**Post-Event Administration**

- Ensure accurate and contemporaneous record keeping which include any discussions with secondary and tertiary care providers.
• Inform medical malpractice insurers at the earliest opportunity.
• Report the complication to the MHRA and medical affairs department of the manufacturer of the product used.

• Ongoing patient support and contact should be offered.
INITIAL MANAGEMENT AT PRIMARY LOCATION

PRINCIPAL SIGNS AND SYMPTOMS
Acute visual loss/impairment, pain, ocular muscle weakness

Stop treatment
Place patient in supine position
Call 999 and transfer to a Specialist Eye Unit

REDUCE INTRAOCULAR PRESSURE

Timolol 0.5% Eye Drops (1-2 drops into the affected eye)
Rebreathe into a paper bag

PREVENT THROMBUS FORMATION

Aspirin 300mg PO

DISLODGE EMBOLUS

Prolonged high frequency ocular massage

DISSOLVE HYALURONIC ACID

If hyaluronic acid is the causative agent:
Administer Hyaluronidase 450-1500 units in 1-2mls of saline,
injected into the supraorbital and supratrochlear foramina

Provide full information to the Specialist Eye Unit as patient transferred

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www.acegroup.online
The Aesthetic Complications Expert Group protocol for the management of visual loss secondary to cosmetic filler injection

**MANAGEMENT AT SPECIALIST EYE UNIT**

Review medical information and reassess patient, look for cerebral involvement

DO NOT ALLOW INVESTIGATIONS TO INTERFERE WITH STARTING TREATMENT

**REDUCE INTRAOCULAR PRESSURE**

**INCREASE RETINAL BLOOD FLOW**

- Acetazolamide 500mg IV
- Mannitol 20% IV infusion (100mls over 30 minutes)
- Consider anterior chamber paracentesis

**PREVENT FURTHER THROMBUS FORMATION**

- Enoxaparin SC or Heparin IV

**DISSOLVE HYALURONIC ACID**

- Trans-orbital approach allows more direct route to ophthalmic circulation
- Administer Hyaluronidase 450-1500 units in 1-2mls of saline

**JUDICIOUS USE OF ANTIBIOTICS TO PREVENT INFECTION**

**OTHER OPTIONS THAT HAVE BEEN REPORTED IN THE LITERATURE**

- Dexamethasone IV
- Prostaglandin E1 IV
- Hyperbaric oxygen
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Visual Loss Secondary to Cosmetic Filler Injection

The ACE Group have produced a series of evidence based and peer reviewed guidelines to help practitioners prevent and manage complications that can occur in aesthetic practice. These guidelines are not intended to replace clinical judgement and it is important the practitioner makes the correct diagnosis and works within their scope of competency. Some complications may require prescription medicines to help in their management and if the practitioner is not familiar with the medication, the patient should be appropriately referred. Informing the patient's General Practitioner is considered good medical practice and patient consent should be sought. It may be appropriate to involve the General Practitioner or other Specialist for shared care management when the treating practitioner is not able or lacks experience to manage the complication themselves. Practitioners have a duty of care and are accountable to their professional bodies and must act honestly, ethically and professionally.

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