



## ABSTRACT

There are multiple treatment strategies proposed for the management of vision loss related to the injection of soft tissue fillers. Currently, there is no internationally accepted consensus on the immediate management of soft tissue filler induced vision loss (STFIVL). A recent systematic review of the literature concluded that there is not enough evidence to support retrobulbar hyaluronidase, and alternative treatments require exploration. The available literature demonstrates the inconsistent and unproven success of retrobulbar and peribulbar hyaluronidase in reversal of soft filler induced vision loss. Various therapeutics have been used to aid the reversal of vision loss but with mixed outcomes. The current evidence base does not support the use of retrobulbar and peribulbar hyaluronidase. The use of retrobulbar hyaluronidase for reversing soft tissue filler induced vision loss is controversial. Its efficacy remains unproven and there is mixed evidence within the literature. The current evidence suggests that there may be an increased risk of introducing severe adverse events associated with retrobulbar hyaluronidase and may even exacerbate the problem for those clinicians who are not ophthalmology trained. Therefore, we recommend two alternative treatment pathways for ophthalmology and non-ophthalmology trained practitioners. The suggested goal of this publication is to understand the pathophysiology of STFIVL, recognize signs and symptoms, and to propose algorithms to manage vision loss for both non-ophthalmology and ophthalmology trained clinicians. Clinicians must act swiftly and arrange immediate transfer to an emergency department or ophthalmology specialist setting to give the patient the best chance of vision restoration. The focus of any intervention for non-ophthalmology trained clinicians should be based around the immediate use of non-invasive techniques.

**KEYWORDS:** Vision loss, blindness, hyaluronic acid, filler, complications, soft tissue, ophthalmology, central retinal artery, ophthalmic artery, hyaluronidase, risk, algorithm.

# Consensus Opinion for The Management of Soft Tissue Filler Induced Vision Loss

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Vision loss after facial injection is an extremely rare event, estimated at 0.001 percent<sup>1</sup> but we must assume the incidence is higher due to under-reporting. Since the first 98 cases documented in 2015, there has been an increase of 49 percent based on the most recent global review in 2019.<sup>2</sup> Furthermore, the incidence has evolved from mainly autologous fat injections to a more recent rise in hyaluronic acid filler related cases.<sup>3</sup> Since the incidence of this potentially catastrophic complication is on the rise with the use of hyaluronic acid dermal fillers, the responsible clinician must be competent to mitigate known risk factors, recognize the symptoms, and instigate safe, evidence-based management strategies.

## PATHOPHYSIOLOGY

There are three factors required for blindness to occur: injection pressure exceeding systolic pressure, sufficient amount of material within the lumen of the vessel, and retrograde and subsequent anterograde passage of material into the ophthalmic circulation.

**1) Injection pressure exceeding systolic pressure.** The average injection pressure required to embolize the ophthalmic artery is 166.7 mmHg. Monitoring of the injection pressure showed that it is easy for the injector to exert

a pressure well above 200mmHg within 2 to 3 seconds.<sup>4</sup>

**2) Sufficient amount of material within the lumen of the vessel.** Findings indicate that only a small amount of filler is required to occlude branches of the ophthalmic artery. 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 0.085mL in critical injection points.<sup>1,5</sup>

**3) Retrograde and subsequent anterograde passage of material into the ophthalmic circulation.** The pressure during the injection is likely to be dispersed to multiple vessels rather than transmitted to one artery.<sup>4</sup> Terminal cutaneous 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 documented. Similarly, anastomoses with the superficial temporal arteries and the orbit have also been demonstrated. Injection of filler material into one of these vessels can lead to retrograde flow to beyond the point of the origin of the ophthalmic artery. When pressure from the plunger is released, systolic pressure drives the product forward to enter the ophthalmic

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artery and specifically, the central retinal artery, resulting in visual loss.<sup>1,5</sup>

**RISK ZONES**

There are areas of the face with a higher incidence of vision loss. Table 1 shows the rates of blindness relative to the anatomical zone injected. The nasal region now has the highest associated incidence of vision loss, overtaking the glabella as the highest risk area for soft tissue filler injections.<sup>2</sup> The risks have been zoned according to treatment indications, by a 2020 consensus, as illustrated in Figure 1.

**TYPES OF BLINDNESS**

Several types of blindness/vision loss have been described in the literature.<sup>1,6,7</sup> The degree of subsequent visual disturbance relates to the type of soft tissue filler, the volumes injected, cohesivity, particle size, and the branches of the ophthalmic artery affected.<sup>1</sup>

There are various classifications based on the artery occluded, the presentation of symptoms, and the nature of the onset.

**Occluded artery (Figure 2).** Based on which artery is occluded, vision loss/blindness can be classified into six subtypes.<sup>3,8</sup>

1. *Ophthalmic artery occlusion (OAO).* Total obstruction of the ophthalmic artery would tend towards visual loss, ptosis, and ophthalmoplegia
2. *Generalized posterior ciliary artery occlusion with relative central retinal artery sparing (PCAO)*
3. *Central retinal artery occlusion (CRAO).* Isolated central retinal artery occlusion would tend towards visual loss alone
4. *Branch retinal artery occlusion (BRAO).* Branch retinal artery occlusion may cause a partial visual loss
5. *Anterior ischaemic optic neuropathy (AION)*
6. *Posterior ischaemic optic neuropathy (PION)*

Combinations of these patterns can occur.<sup>1</sup>

Each type of occlusion carries a distinct prognosis, which is worse in the diffuse occlusion group.<sup>7</sup>

**Classification of blindness/vision loss based on presentation of symptoms.** Table 2 shows four presentation subtypes of periocular complications associated with blindness following cosmetic filler injection.<sup>6</sup> Figure 3 shows an example of Type IV blindness.

**Classification of blindness/vision loss based on time to onset.** In a publication by

Taylor et al,<sup>9</sup> blindness induced by filler fell into three distinct clinical patterns and is summarized in Table 3.

**PROGNOSIS**

Degree of vision loss predicts the location of the embolus, which might be the most important prognostic factor.<sup>3</sup> Eighty percent of fat emboli present as complete vision loss, whereas 50 percent of HA emboli present as partial vision loss, accounting for its better prognosis. Presentation with complete vision loss/blindness (i.e., no light perception [NLP]) is most often associated with ophthalmic artery occlusion (OAO) or central retinal artery occlusion (CRAO), and most do not recover.<sup>3</sup>

Presentation with partial vision loss (i.e., blurry vision to diminished light perception [DLP]) is less commonly associated with OAO/CRAO, and more often involves more distal branches of both the posterior ciliary arteries or the central retinal artery, likely due to smaller emboli. Partial vision loss after HA filler has a better prognosis than complete vision loss.<sup>3</sup>

Branch retinal artery occlusion (BRAO) has the most favourable prognosis. In the literature it shows that all fully and partially recovered vision loss patients received some form of treatment.<sup>3</sup> Table 4 summarizes the affected artery and degree of vision loss associated.

**SIGNS AND SYMPTOMS**

The signature features of periocular embolism are instant-onset and simultaneous blindness and ocular pain, which contrasts with ischemia of the facial skin that manifests as blanching followed by delayed pain.<sup>10</sup>

In an updated review of the world literature of 48 new cases of vision loss, 26 cases (54.2%)

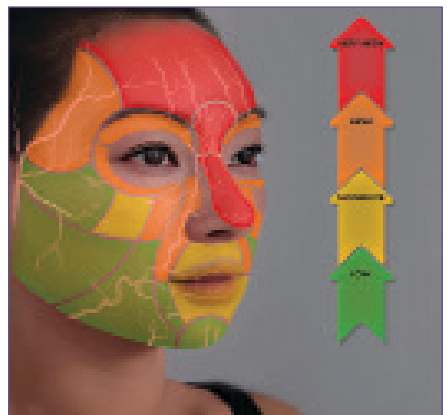
**TABLE 1.** Rates of blindness relative to the anatomical zone injected

ANATOMICAL AREA	INCIDENCE 2015	INCIDENCE 2019
Nose	25.5%	56.3%
Glabella	38.3%	27.1%
Forehead	12.2 %	18.8%
Nasolabial fold	13.3%	14.6%

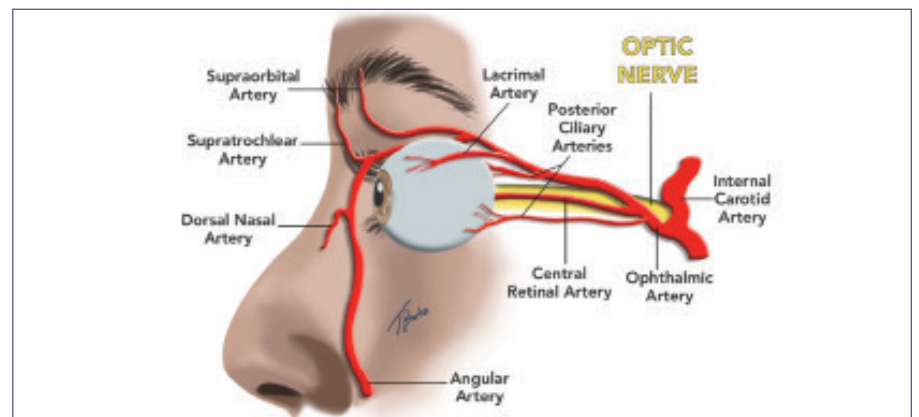
**TABLE 2.** Four presentation subtypes of periocular complications associated with blindness following cosmetic filler injection

DESCRIPTION OF BLINDNESS	PRESENTATION
Type I	Blindness without ophthalmoplegia or ptosis
Type II	Blindness with ptosis but without ophthalmoplegia
Type III	Blindness with ophthalmoplegia but without ptosis
Type IV	Blindness with ophthalmoplegia and ptosis

Adapted from Yujin et al<sup>6</sup>



**FIGURE 1.** Regions of associated risk of blindness; adapted from Goodman et al<sup>1</sup>

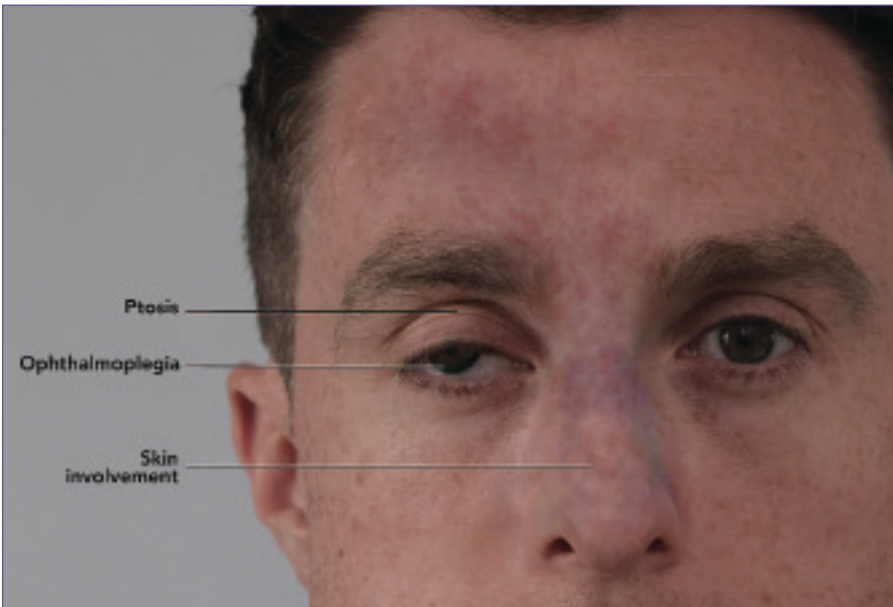


**FIGURE 2.** Ophthalmic artery and associated branches

**TABLE 3.** Clinical patterns of filler-induced blindness

DESCRIPTION OF BLINDNESS	TIME TO ONSET AFTER INJECTION	MECHANISM/THEORY
Type I (Acute Onset)	Immediate to minutes	Embolus of main trunk ophthalmic artery with associated choke spasm
Type II (Delayed Onset)	1–24 hours	Progressive migration of embolus further along ophthalmic circulation
Type III (Late Onset)	Days to weeks	Arteriovenous shunt of emboli via connections in glabella and orbit

Adapted from Taylor et al<sup>9</sup>



**FIGURE 3.** Example of symptoms associated with type IV blindness

**TABLE 4.** Degree of vision loss associated with the affected artery

ARTERY OCCLUDED	DEGREE OF VISION LOSS
Ophthalmic Artery	Complete blindness/ NLP
Central Retinal Artery	Complete blindness/NLP
Branch Retinal Artery	Partial Blindness/DLP
Posterior Ciliary Artery	Blurred vision/DLP

Adapted from Jones et al<sup>3</sup>

**TABLE 5.** Incidence of signs and symptoms of soft tissue filler induced vision loss

SIGN/SYMPTOM	INCIDENCE
Complete Blindness	54.2%
Unilateral Blindness	45.8%
Ocular Pain	56.3%
Skin Changes	43.8%
Ophthalmoplegia	54.2%
Ptosis	52.1%
Nausea/Vomiting	16.7%
CNS symptoms	18.8%

Adapted from Belezney et al<sup>2</sup>

were found to have complete vision loss, whereas the remaining cases had complete unilateral vision loss.<sup>2</sup> In 27 cases (56.3%) pain was reported as one of the initial symptoms, described as periorbital, ocular, periocular, orbital, eye pain, or headache. In 21 cases (43.8%) associated skin changes, commonly described as erythematous to violaceous mottling or skin necrosis, were reported. Ophthalmoplegia (i.e., decreased extraocular muscle movement) was reported in 26 cases (54.2%) and ptosis was seen in 25 cases (52.1%). Most commonly, the ophthalmoplegia and ptosis recovered completely. Nausea and/or vomiting were described as a presenting symptom in eight cases (16.7%). Among the 48 cases, there were nine cases (18.8%) of central nervous system (CNS) complications, including stroke-like features, such as unilateral weakness or evidence of brain infarction on imaging.<sup>2</sup> Table 5 summarizes the incidence of signs and symptoms of STFIVL.

### TREATMENT OPTIONS IN THE IMMEDIATE MANAGEMENT OF STFIVL

There are numerous therapeutic options for the immediate management of STFIVL. The key for clinicians is not to delay any intervention which might reverse vision loss. One big challenge in evaluating potential therapies for STFIVL involves the uncertainty regarding retinal tolerance time or the duration of retinal ischemia prior to irreversible damage. The evidence is uncertain when it comes to how long the retina can survive hypoxia.

Research in primates demonstrated retinal survival if perfusion is restored within 90 to 240 minutes following occlusion.<sup>11</sup> However, evidence now suggests that human retinal ischemic survival time might be considerably shorter.<sup>12–14</sup> A shorter retinal tolerance time undermines the result of some studies which have purported visual benefit of CRAO treatments given up to 24 to 48 hours after occlusion occurs.<sup>12</sup> The actual range is likely to be 15 to 90 minutes.<sup>13</sup> In a publication by Tobalem et al<sup>14</sup> in 2018, it was suggested that retinal infarction is most likely to occur after only 12 to 15 minutes of complete CRAO. This may help to explain why delayed therapeutic interventions for CRAO are often ineffective. Therefore, interventional time management is critical for any chance of vision restoration.

Management strategies reported in the literature usually include polypharmacy, which makes it difficult to identify key effective interventions.<sup>2</sup> Some important interventions require specialist knowledge and skills and access to advanced diagnostic imaging, therefore it is in the patient's best interests that the treating clinician is able to administer first aid and transfer to specialist care without delay.

Treatment options and management strategies should be divided in two distinct categories: interventional options for non-ophthalmology trained clinicians, and interventional options for ophthalmology trained clinicians.

Most of these therapeutic treatment options are derived and extrapolated from ophthalmic literature for the management of central retinal artery occlusion.<sup>2,15,16</sup>

Table 6 shows the pharmacological and non-pharmacological options described within the literature. These options have now been adapted in aesthetic medicine to aid retinal

**TABLE 6.** Summary of treatments for soft tissue filler induced vision loss

CURRENT TREATMENT STRATEGIES	MODE OF ACTION	MECHANISM OF ACTION
Non-pharmacological		
Ocular massage	Dislodge	Repeated compression of the eyeball to dislodge blockage: a sudden drop in intraocular pressure (IOP) with release increases the retinal perfusion
Anterior chamber paracentesis	Reduce intraocular pressure	Extracting 0.1-0.2ml aqueous fluid via needle to reduce IOP and to allow an increase in perfusion pressure
Hyperbaric oxygen	Increase perfusion	Increase oxygen tension and oxygen delivery to ischaemic retinal tissue—involves intermittent inhalation of 100% oxygen under a pressure greater than 1 atm
CO2 rebreathing	Increase perfusion	Rebreathing into a bag produces both hypercapnia which is known to increase retinal blood flow and hypoxia which causes vasodilation. In addition, both hypercapnia and hypoxia can increase cardiac out-put and raise systemic arterial blood pressure, which in turn, increases ocular perfusion pressure.
Pharmacological		
Timolol drops	Reduction in intraocular pressure	Suppress aqueous humour formation, reduce IOP and increases perfusion
IV Mannitol, Acetazolamide	Reduce intraocular pressure	Medication used in glaucoma to reduce intraocular pressure
Aspirin	Prevents clot formation	Prevents platelet aggregation, allowing body to breakdown embolus element of blockage
Topical and systemic steroids	Reduce intraocular pressure	Reduction in vascular endothelial oedema
Sublingual isosorbide mononitrate	Increase perfusion	Causes a mild decrease in intraocular pressure along with corresponding dilation of retinal vasculature and increased perfusion in the retinal artery

perfusion, reduce intraocular pressure, reduce retinal edema, aid the lysis of hyaluronic acid embolus, and restore vision loss in STFIVL.

**Interventional options for non-ophthalmology trained clinicians (Appendix 1).**

1. Stop treatment immediately.
2. The best chance of vision restoration is in a hospital/specialist setting.<sup>1</sup> Specialist treatment at an appropriate facility should be instituted as soon as possible to optimize all possible treatment pathways.<sup>17</sup> Therefore, the treating clinician should arrange immediate transfer of the patient by calling an ambulance or driving the patient directly to hospital. The authors strongly recommend that the treating clinician accompanies the patient. Accompanying the patient may assist with liaison and prompt hand over in the specialist setting, while also providing support to the patient during this difficult time.
3. While awaiting transfer, test and document visual acuity.<sup>1</sup> Using simple methods (i.e., counting fingers, hand motions and light perception via a hand torch or mobile phone light) can provide useful information to the treating ophthalmologist on hand over.<sup>18</sup> Recording vision from worst to best may also prove valuable to the treating specialist. The order of worst to best regarding vision is as follows: No light perception, light

- perception, hand motion, counting fingers.<sup>18</sup>
4. Give the patient 300mg aspirin. There is no direct evidence that aspirin prevents platelet aggregation in the event of a hyaluronic acid-related occlusion; however, it is reasonable, based on the extrapolation of the evidence from acute coronary syndrome, to prescribe 300mg as an immediate dose with 75mg daily until the occlusion is dissolved and vision has been restored. Safety must be considered in the context of the individual patient, and if the patient is allergic to aspirin, clopidogrel at a dose of 300mg immediately, then 75mg daily may be used.<sup>19</sup>
5. Reduce intraocular pressure by administering 0.5% timolol drops.<sup>18</sup> One drop is administered to the affected eye while awaiting transfer to the hospital. The authors recommend that timolol should form part of a vision loss kit that is readily available at the clinician's place of work.
6. Begin ocular massage. Ocular massage is known to cause retinal arterial dilatation and large fluctuations in IOP,<sup>20</sup> promoting the dislodgment of the causative embolus. The embolus then either disintegrates or migrates into a peripheral portion of the retinal vasculature, allowing for retinal reperfusion.<sup>12</sup> Place the patient in a supine position with the head raised 15 to 45 degrees (Semi Fowler Position), perform digital ocular massage by applying direct pressure through the closed eyelid of the affected eye for five seconds and then releasing and waiting for 10 seconds before applying pressure for another five seconds. The procedure should be repeated in a pulsatile fashion for five minutes, after which vision should be checked. A total of three sets of this pressure/release can be performed if necessary, so that the patient has a total of 15 minutes of pressure/release.<sup>18</sup>
7. Instruct the patient to re-breathe into a paper bag for approximately 10 minutes every half an hour.<sup>18</sup> This assumes that an increase in carbon dioxide promotes dilation of arterioles.<sup>21</sup>
8. If the skin is ischemic in the area of injection, consider prompt administration of hyaluronidase. The use of hyaluronidase in dissolving cross-linked hyaluronic acid is highly effective and has been shown to prevent tissue necrosis. Rapid deployment might rescue threatened tissue. The authors recommend using 1,500 units per 1mL with lidocaine 1% or 2% without adrenaline, employing a high-dose pulsed model of dosing.<sup>19</sup>
9. If patient has no medical contraindications, the use of sublingual glycerol trinitrate is a quick and effective method for increased vasodilatation.<sup>1</sup>

**Interventional options for the ophthalmology trained clinician. For**

the ophthalmology trained clinician, the primary focus is also based around reduction of intraocular pressure and increasing retinal perfusion. Other supportive therapies which have a more invasive nature can be considered; these are suitable for those with an ophthalmology background. HA filler emboli degradation can also be attempted using retrobulbar/peribulbar hyaluronidase. In the eventuality of a patient presenting with immediate visual loss, the course of action of the practitioner should be as follows:

1. Stop treatment immediately.
2. The best chance of vision restoration is in a hospital/specialist setting.<sup>1</sup> Specialist treatment at an appropriate facility should be instituted as soon as possible to optimize all possible treatment pathways.<sup>17</sup> Therefore, the treating clinician should arrange immediate transfer of the patient by calling an ambulance or driving the patient directly to hospital. The authors strongly recommend that the treating clinician accompanies the patient. Accompanying the patient may assist with liaison and prompt hand over in the specialist setting while also providing support to the patient during this difficult time.
3. While awaiting transfer, test and document visual acuity.<sup>1</sup> These more detailed tests should include relative afferent pupillary defect (RAPD) which is an objective measurement of relative optic nerve damage. RAPD is tested by the “swinging light test,” assessing for pupillary constriction on exposure to light.<sup>22</sup> Extraocular muscle movement assessment for ophthalmoplegia and ptosis assessment, including measurement of marginal reflex distance, must also be recorded.<sup>22</sup>
4. If available, preferably intravenous rather than oral acetazolamide (Diamox) 500mg bolus plus topical ocular antihypertensives (e.g., timolol) to reduce intraocular pressure should be administered.<sup>23</sup>
5. Perform anterior chamber paracentesis. This technique has been demonstrated to effectively reduce intraocular pressure with subsequent increase in retinal perfusion rate up to 20 percent. It should be noted that this technique entails the intrinsic risk of intraocular complications.<sup>24</sup>

6. Start ocular massage. Ocular massage is known to cause retinal arterial dilatation and large fluctuations in IOP.<sup>20</sup> Use a Goldman lens or transpalpebral two-finger approach.<sup>24</sup>
7. Instruct the patient to re-breathe into a paper bag for approximately 10 minutes every half an hour.<sup>18</sup> This assumes that an increase in carbon dioxide promotes dilation of arterioles.<sup>21</sup>
8. If the skin is ischemic in the area of injection, consider prompt administration of hyaluronidase. The use of hyaluronidase in dissolving cross-linked hyaluronic acid is highly effective and has been shown to prevent tissue necrosis. Rapid deployment might rescue threatened tissue. The authors recommend using 1,500 units per 1mL with lidocaine 1% or 2% without adrenaline, employing a high-dose pulsed model of dosing.<sup>19</sup>
9. Consider retrobulbar/peribulbar hyaluronidase 1,500IU in 4mL saline with 25G retrobulbar needle.<sup>23,25</sup> A systematic review of the literature reported that hyaluronidase was used in 86 percent of vision loss cases; however, only 11.1 percent of cases were successful.<sup>26</sup> There are case reports of success using retrobulbar/peribulbar hyaluronidase,<sup>27–30</sup> so there may be value in the ophthalmology trained clinician attempting this procedure. An initial volume of 7mL is recommended to avoid orbital compartment syndrome.<sup>18</sup>
10. If the patient has no medical contraindications, the use of sublingual glycerol trinitrate is a quick and effective method for increased vasodilatation.<sup>1</sup>

## CONCLUSION

Vision loss due to soft tissue filler injection is a rare, but catastrophic event. When vision loss occurs, early recognition and prompt treatment are critical. There is currently no internationally accepted gold standard for the treatment of vision loss, and even though there have been consensus recommendations, no specific guidelines are available that have been universally successful in reversing this complication.<sup>31</sup> The best chance for vision restoration is a hospital or specialist setting.<sup>1</sup>

However, it should be noted that a recent survey of British oculoplastic consultants

demonstrated a limited awareness of visual vascular complications with dermal fillers and their emergency management. The majority (88%) did not have local management guidelines in place, nor were they aware of guidance to manage the complication (75%).<sup>32</sup> A similar situation has been observed in the United States, where a survey of 45 institutions found that only 20 percent had a formal policy, guideline, or white paper to standardize the approach to CRAO treatment.<sup>18</sup>

The current evidence suggests that there might be an increased probability of introducing severe risks associated with retrobulbar hyaluronidase and may even exacerbate the problem for those clinicians who are not ophthalmology trained. Expert opinion and the reviewed case reports has suggested that many non-ophthalmology trained clinicians would not be adequately prepared or equipped to accurately diagnose and document a CRAO and address these complications should they arise. Therefore, the authors suggest two alternative treatment pathways for the ophthalmology and non-ophthalmology trained practitioner.

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Complications in Medical Aesthetics Collaborative (CMAC) is an organization set up to provide support and education to medical aesthetic clinicians. Members are part of a collaboration, and through working with the membership, CMAC aims to capture data to help improve patient safety. For more details, please see <https://www.cmac.world/>.

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## APPENDIX 1. Vision Loss Algorithm for Non-ophthalmology Trained Clinicians

### Signs and symptoms suggestive of visual loss with soft tissue fillers<sup>\*\*</sup>:

- Bilateral or unilateral vision loss
- Ocular pain
- Ophthalmoplegia (decreased extraocular muscle movement)
- Double/Blurred vision
- Ptosis
- Headache
- Nausea/vomiting
- Stroke-like symptoms
- Skin blanching/reticulation \*

\* may or may not precede other symptoms

\*\*Please note the visual disturbance may occur as a single symptom or a combination of symptoms

### Stop injecting and arrange immediate transfer to eye hospital or emergency department, inform patient nature of emergency.

- *Rationale: Time critical event due to retinal intolerance to hypoxia, best chance of successful outcome*

- Test eye movements and note any ptosis
- Test visual acuity: count fingers, hand motions and light perception
- Administer 300mg aspirin, 1 drop 0.5% timolol
- Place patient in supine position (Semi Fowler position)
- Perform ocular massage: Manually press the globe firmly for cycles of 5 to 15 seconds followed by rapid release, repeat for a total length of 5 minutes, rest (a few minutes), then repeat. This may be continued while patient is being transferred to hospital.
- Inform patient to rebreathe into paper bag
- *Rationale: Record of baseline visual loss, reduce thrombus risk, reduce intraocular pressure, increase retinal perfusion*

### While awaiting ambulance prepare a pack of the following:

- 4 x 1500 IU hyaluronidase
- CMAC ophthalmology protocol
- Reception to contact family
- Consider documentation of case with photographs
- *Rationale: Ophthalmology department might not hold hyaluronidase, might be unaware of association between soft tissue fillers and vision loss; inform family of emergency*

### Upon arrival of ambulance:

- If you can, stay with patient (recommended)
- Hand over, offer support and help liaise with hospital
- *Rationale: Very distressing and traumatic time for patient, support and reassurance are helpful at this critical point. Liaison with hospital will help expedite correct treatment strategy*

### Upon return to clinic review visual loss record form:

- Record all treatment interventions, time of onset, type, amount of filler, site injected, cannula or needle, signs/symptoms visual acuity treatment administered post vision loss, time of ambulance arrival, and any immediate hospital interventions.
- Contact indemnity provider
- Keep in regular contact with patient
- *Rationale: Good, accurate recordkeeping can help with any medicolegal issues which might ensue*

APPENDIX 2. Interventional Options for Non-Ophthalmology Trained Clinicians

## SOFT TISSUE FILLER INDUCED VISION LOSS

### INTERVENTIONAL OPTIONS for non-ophthalmology trained

- 1



**Stop treatment**
- 2



**Arrange transfer to specialist**
- 3

Light perception 

Hand motion 

Counting fingers 

**Assess visual acuity**
- 4

Digital pressure for 5 seconds, release for 10 seconds. Repeat for 5 minutes. Complete up to 3 cycles. 

15-45°  **Ocular massage & rebreathing**

Semi Fowler Position

Paper bag rebreathing for 10 minutes every half an hour. 
- 5

 300mg stat, then 75mg daily **Aspirin**

**Timolol** 0.5% Timolol, one drop 

 Sublingual GTN to increase vasodilation **Glycerol trinitrate**
- 6

 1500 units per 1ml with lidocaine, 1 or 2%, without adrenaline

**Ischaemic skin hyaluronidase**



VISION LOSS EMERGENCY KIT



**APPENDIX 3. Visual Loss Recording Form**

Patients Name:

DOB:

Treating Clinician:

Date:

**Treatment Details**

*Anatomical site:*

- Glabella
- Forehead
- Nose
- Tear trough
- Midface
- Temple
- Lips
- Chin
- Jaw
- Other

*Type of Filler used:*

- Hyaluronic Acid (BRAND)
- Radiesse
- Ellanse
- Sculptra
- Other

*Depth of Injection:*

- Intradermal
- Subcutaneous
- Supraperiosteal

Amount of Filler:

Needle or Cannula:

Gauge of Needle/Cannula:

Length of Needle/Cannula:

**Signs, Symptoms and Interventions**

Right eye, left eye or both

Ocular pain Y/N

Ophthalmoplegia (decreased extraocular muscle movement) Y/N

Double/Blurred vision Y/N

Ptosis Y/N

Skin blanching/reticulation Y/N

Headache Y/N

Nausea/vomiting Y/N

Stroke like symptoms Y/N

Other

Time of first symptom:

Time called Ambulance:

**Visual Acuity Test**

Can see hand movements Y/N

Can count fingers Y/N

Can see light Y/N

Photographs/video taken: Y/N

**Treatment Interventions**

Aspirin 300mg Y/N

- Time:

0.5% Timolol Drops Y/N

- Time:

Ocular Massage Y/N

- Time:

Rebreathing CO2 in paper bag Y/N

- Time:

Other Interventions

- Time:

Time of arrival of Ambulance:

**APPENDIX 4. Vision Loss Algorithm, Ophthalmology Department**

Hand over Visual Loss Recording Form, which gives a history of complaint and treatments administered prior to hospital take over. This expedites specialist intervention in this time-critical event.

**Interventions to consider immediately**

- High dose pulsed injection of Hyaluronidase. 1500iu diluted in 1ml lidocaine without adrenaline. Infiltrate supratrochlear, supraorbital notch, retrobulbar and any visible areas of skin ischaemia. Inject 1ml of 1500iu hyaluronidase every 15 minutes – dissolve filler emboli
- Retrobulbar/peribulbar hyaluronidase 1500IU in 4ml saline with 25G retrobulbar needle
- Limbal paracentesis with removal of 0.1-0.2ml of aqueous from anterior chamber – reduce intraocular pressure
- IV acetazolamide (Diamox) plus topical ocular hypertensives – reduce intraocular pressure
- IV steroid – reduce retinal oedema
- Hyperbaric oxygen
- Sublingual GTN - vasodilation

**Ophthalmic investigations<sup>25</sup>**

- Consider neuroimaging with angiography
- Fundus fluorescein angiography
- OCT and photograph facial skin and eye movements

APPENDIX 5. Interventional Options for Non-Ophthalmology Trained Clinicians

SOFT TISSUE FILLER INDUCED

# VISION LOSS

INTERVENTIONAL OPTIONS for ophthalmology trained

- 

**Stop treatment**
- If possible, accompany the patient for support and to ensure a prompt handover



**Arrange transport**
- 

**Assess visual acuity**
- 

**Ocular antihypertensives**
- 

**Anterior chamber paracentesis**
- Ocular pressure for **5 seconds**, release for **10 seconds**

Repeat for **5 minutes** & recheck vision.

Complete up to **3 cycles**




**Ocular massage**
- Paper bag rebreathing for **10 minutes** every half an hour



**Bag rebreathing**
- 

**1500 units per 1ml with lidocaine 1 or 2%, without adrenaline**

**Ischaemic skin hyaluronidase**
- Retro/Peribulbar hyaluronidase **15000U** in 4ml with 25G retrobulbar needle



**Retrobulbar hyaluronidase**
- Sublingual GTN to increase vasodilation



**Glycerol trinitrate**