OPEN

PRS GLOBAL OPEN

CASE REPORT
Cosmetic

Partially Reversible Episode of Blindness after Intravascular Hyaluronic Acid Filler Injection

Patricia Barrera, MD*
Gillian Murray, MPharm†
Alejandra Pantano, MD‡

Summary: Blindness from hyaluronic acid filler injections is an uncommon but devastating complication. We present a case of blindness related to a "skin booster." It is often assumed that skin boosters have no risk of visual problems, and this case underlines the need to understand products' rheology and chemistry. Given the lack of agreed consensus in bedside and secondary care management, it is important to report and describe all cases of vision loss caused by fillers, including assessment and management, to allow understanding of what may give rise to better outcomes.
(Plast Reconstr Surg Glob Open 2024; 12:e6316; doi: 10.1097/GOX.0000000000006316; Published online 20 November 2024.)

Many "skin boosters" are cross-linked with a high G prime, leading to ischemic issues if injected intravascularly, and non-cross-linked products can cause blindness if they reach the ophthalmic territory.[1] Cross-linked products are harder to dissolve than non-cross-linked ones. Contrary to common beliefs, non-cross-linked fillers can cause vascular adverse events, and most skin boosters are cross-linked. Although non-cross-linked products disperse within 24 hours, they can still pose a danger in end-arterial circulation like the retina or brain. Cross-linked hyaluronic acid (HA) persists longer, and its intravascular behavior depends on its rheological properties. Both types pose risks in ophthalmic circulation, underscoring the importance of understanding skin booster crosslinking. We aim to present a case of partially resolved blindness following a skin booster treatment, detailing its management and resolution, and to provide insights into the importance of understanding the rheology and chemistry of the products used in these procedures.

CASE REPORT

A 54-year-old woman presented seeking treatment for static wrinkles in the forehead following previous botulinum toxin injections. Restylane Vital Skin Booster (Q-Med AB, Sweden) was administered using a 23G × 38 mm cannula in the subcutaneous plane, targeting a vertical static wrinkle. After confirming negative aspiration with an unprimed cannula, 0.1 mL of filler was retrogradely injected on the left side. The same technique was replicated on the right side using a primed cannula. During the injection, immediate and sustained blanching of the skin occurred over the right supraorbital, supratrochlear, and dorsal nasal segments of the ophthalmic angiosome, leading to the cessation of the injection. (**See figure, Supplemental Digital Content 1**, which displays immediate blanching of the supraorbital, supratrochlear, and dorsonasal segments of the ophthalmic angiosome after HA filler placement on the superficial branch of the supraorbital artery. **http://links.lww.com/PRSGO/D641**.) Subsequently, 1500 IU of hyaluronidase was subdermally administered followed by vigorous massage. The patient experienced severe pain in the right temporal fossa, ocular discomfort, and nausea, prompting immediate cessation of the procedure. At this point, 2 additional 1500 IU vials of hyaluronidase were reconstituted and injected around the supraorbital foramen. The patient was transferred to the emergency department and discharged after 8 hours with a diagnosis of psychosomatic symptoms. However, the patient later reported blurred vision and delayed capillary refill time (10 seconds) in the supratrochlear artery territory. Despite recommendations for further hyaluronidase treatment, the patient declined.

The following day, the patient was admitted to the hospital. Ophthalmological examination revealed right palpebral ptosis, ipsilateral headache, severely impaired vision ("bulk vision"), conjunctival hyperemia, chemosis, ophthalmoparesis, intact pupillary reflex, and a pallid retina. A magnetic resonance imaging angiogram of the brain and orbit was conducted. Treatment included intravenous

*From the *Aesthetic Medicine Department, PB Medicina Estética, Entre Ríos, Argentina; †Pharmaceutical Science Department, Division of Life Sciences and Medicine, Kings College London, London, United Kingdom; and ‡Private Consult, Aesthetic Medicine Department, Mendoza City, Argentina.*

Received for publication April 19, 2024; accepted September 23, 2024.

DOI: 10.1097/GOX.0000000000006316

Disclosure statements are at the end of this article, following the correspondence information.

Related Digital Media are available in the full-text version of the article on www.PRSGlobalOpen.com.

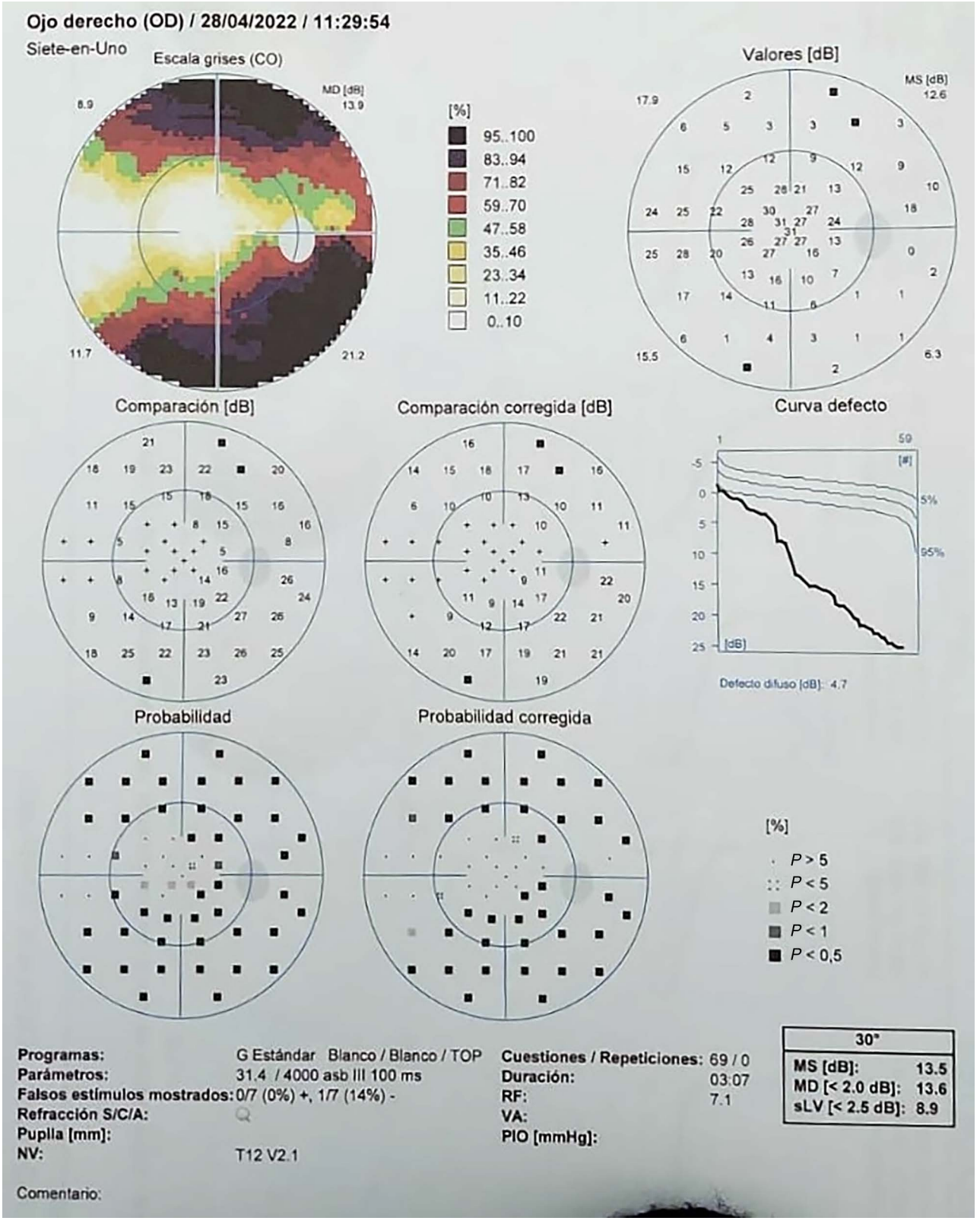

Fig. 1. Ophthalmic report on day 9 showing superior and inferior scotoma.

ketorolac (30 mg every 8 hours), pantoprazole (40 mg every 8 hours), subcutaneous enoxaparin (60 mg every 12 hours), and methylprednisolone (1000 mg every 24 hours).

On the second day of hospitalization, a retinal specialist diagnosed the patient with grade IV central retinal artery occlusion (CRAO). Examination showed

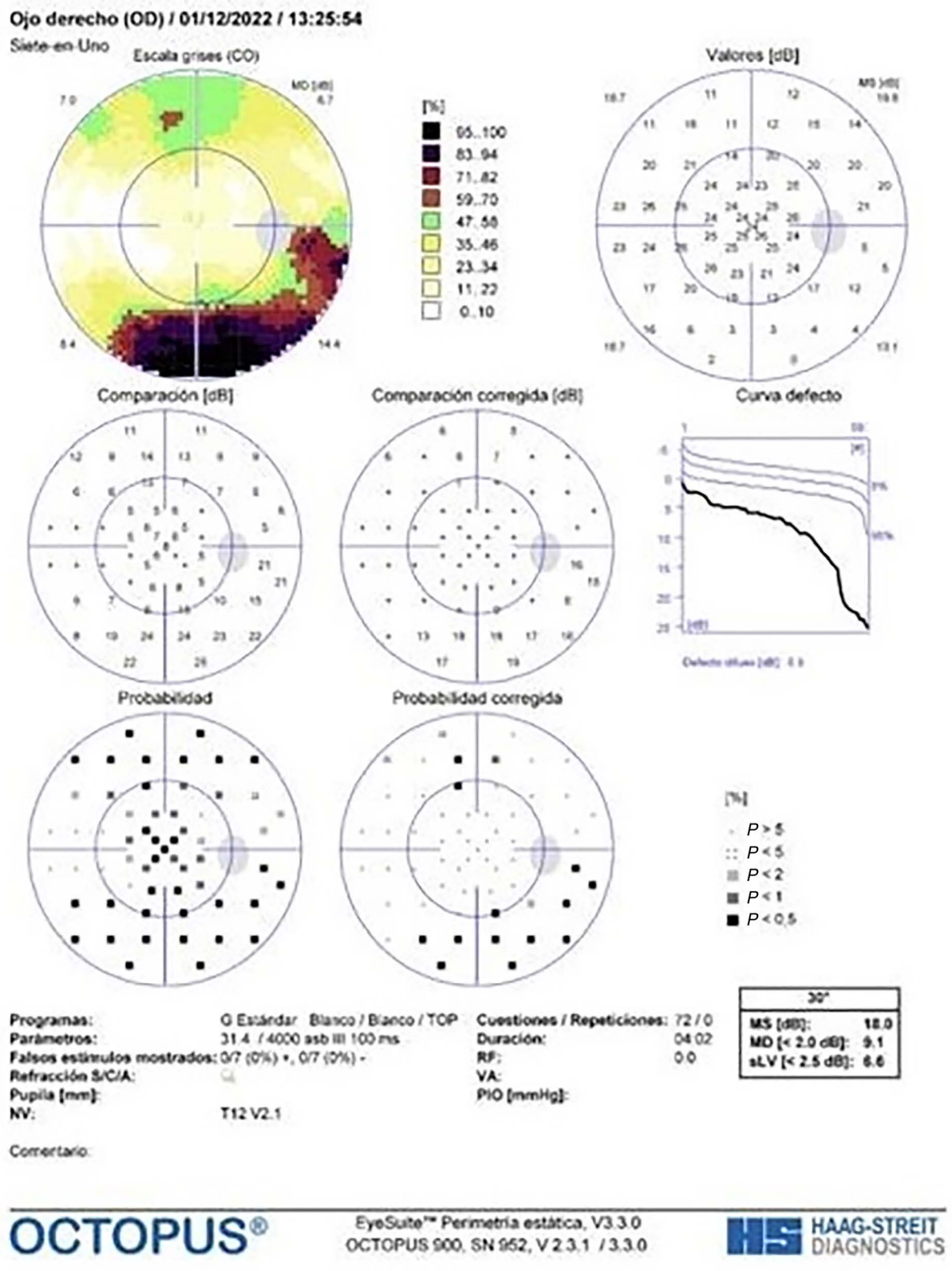

Fig. 2. Final visual defect observed 8 months later, characterized by inferior and temporal scotoma.

significant corneal edema, a central epithelial corneal ulcer, nasal conjunctival injection, a normal anterior chamber and inotraocular pressure hyporeactive pupil, and ophthalmoparesis, which improved with pain relief. Magnetic resonance imaging angiogram confirmed grade IV CRAO. The patient was discharged with eye lubricants,

antibiotic drops, and acenocoumarol 2 mg/d for 6 months, with a reassessment scheduled in 24 hours. Further evaluations with optical coherence tomography and computerized visual field testing were advised.

By day 4, the patient could count fingers and showed necrosis on the skin supplied by the supratrochlear artery. (**See figure, Supplemental Digital Content 2**, which displays day 4 necrosis observed on the skin supplied by the supratrochlear artery. **http://links.lww.com/PRSGO/D642**.) By day 9, optical coherence tomography and computerized visual field testing indicated the right optic nerve was better. The patient had superior and inferior scotomas with a visual acuity of 20/60 (Fig. 1). By day 14, visual acuity improved to 20/25. However, 20 days posttreatment, the patient developed frontal alopecia, managed with topical minoxidil. After 8 months, the wound and alopecia resolved, but the inferior and temporal scotomas persisted (Fig. 2). (**See figure, Supplemental Digital Content 3**, which displays an image of the patient showing her evolution 8 months after the episode. **http://links.lww.com/PRSGO/D643**.)

DISCUSSION

Understanding the chemical composition and rheological properties of skin boosting products is crucial due to their close association with injection-related risks. The misdiagnosis in the emergency department highlights a lack of awareness regarding this issue. In our case report, initial vision loss was attributed to type IV dissemination following cannulation of the right superficial branch of the supraorbital artery.[2]

The optimal timeframe for reversing CRAO is generally within 15 minutes, though intermittent reperfusion may extend this window.[3] Notably, 15%–30% of cilioretinal arteries independently supply the macula, potentially preserving central vision despite CRAO-induced infarction.[3]

Various treatment modalities for CRAO have been proposed. Although super selective intra-arterial thrombolysis was once thought limited by a narrow therapeutic window, recent studies suggest interventions up to 39 hours postonset may restore vision, though availability remains limited.[4] Endovascular urokinase and hyaluronidase require high doses and face hesitancy due to infusion risks.[5]

Retrobulbar administration remains debated due to hyaluronidase's slow diffusion rate and the short exposure time of the artery before it enters the optic nerve dura.[6] Ophthalmologists might consider peribulbar injection according to The Royal Australian and New Zealand College of Ophthalmologists guidelines.[7] Sharudin et al[8] reported vision restoration with subcutaneous hyaluronidase targeting the ischemic area, suggesting an alternative mechanism.

Two cases of blindness reversal following hyaluronidase injection into the supraorbital/supratrochlear foramen have been documented, yet specifics on intravascular injection were not clarified by the authors.[9,10] Given risks such as stroke and hemorrhage with vessel cannulation, consensus favors hyaluronidase administration at the supraorbital/supratrochlear foramen.[7] We suggest a concentrated saline solution to avoid lidocaine-induced blurred vision.

Additional measures such as oral acetazolamide, aspirin, timolol drops, ocular massage, and rebreathing in a paper bag may aid initial management. However, numerous questions persist, including vessel behavior postinjury, HA filler interactions, and hyaluronidase diffusion mechanisms. Concerns about HA embolization and its potential exacerbation remain unresolved within this case report's scope.

CONCLUSIONS

Our findings stress the importance of documenting all cases of blindness to guide appropriate treatment strategies. They also highlight a lack of awareness in hospitals about the potential risks of these procedures, underscoring the need for healthcare providers to fully understand anatomy and product properties before performing injections.

Patricia Barrera, MD
PB Medicina Estética
Laprida 192, Paraná
Entre Ríos, Argentina
E-mail: drapatriciabarrera@gmail.com

DISCLOSURES

Both authors are board members of the Complication in Medical Aesthetics Collaborative (CMAC), a not-for-profit organization supporting clinicians worldwide. Dr. Barrera is the Chair of CMAC IA (iberoamerica). Murray is in Founding Board CMAC.

REFERENCES

1. Soares DJ, McCarthy AD. Commentary on "Histopathologic analysis of hyaluronic acid composite solution following intravascular injection: variability and safety." *J Cosmet Dermatol.* 2024;23:1925–1928.
2. Soares DJ. Bridging a century-old problem: the pathophysiology and molecular mechanisms of HA filler-induced vascular occlusion (FIVO)-implications for therapeutic interventions. *Molecules.* 2022;27:5398.
3. Tobalem S, Schutz JS, Chronopoulos A. Central retinal artery occlusion—rethinking retinal survival time. *BMC Ophthalmol.* 2018;18:101.
4. Nguyen HH, Tran HTT, Duong QH, et al. Significant vision recovery from filler-induced complete blindness with combined intra-arterial injection of hyaluronidase and thrombolytic agents. *Aesthetic Plast Surg.* 2022;46:907–911.
5. Xu X, Zhou G, Fu Q, et al. Efficacy of intra-arterial thrombolytic therapy for vision loss resulting from hyaluronic acid filler embolization. *J Cosmet Dermatol.* 2021;20:3205–3212.
6. Navarro-Hernandez E, Pérez-López M. Effectiveness of retrobulbar hyaluronidase in the treatment of visual loss caused by periocular hyaluronic acid injection. A systematic review. *Arch Soc Esp Oftalmol (Engl Ed).* 2022;97:521–538.
7. The Royal Australian and New Zealand College of Ophthalmologists. RANZCO Guideline for Filler Blindness. 2024. Available at https://ranzco.edu/wp-content/uploads/2024/06/RANZCO-Filler-Blindness-Guidelines_2024.pdf. Accessed July 12, 2024.
8. Sharudin SN, Ismail MF, Mohamad NF, et al. Complete recovery of filler-induced visual loss following subcutaneous hyaluronidase injection. *Neuroophthalmology.* 2018;43:102–106.
9. Goodman GJ, Clague MD. A rethink on hyaluronidase injection, intraarterial injection, and blindness: is there another option for treatment of retinal artery embolism caused by intraarterial injection of hyaluronic acid? *Dermatol Surg.* 2016;42:547–549.
10. Thanasarnaksorn W, Cotofana S, Rudolph C, et al. Severe vision loss caused by cosmetic filler augmentation: case series with review of cause and therapy. *J Cosmet Dermatol.* 2018;17:712–718.