Clinical and Experimental Vision and Eye Research (2021), 4, 46–49

CASE REPORT

Unilateral dry eye and hypoesthesia caused by trigeminal nerve compression: Importance of imaging

Asaf Achiron[1,2,3], Daniel Rappoport[2,3], Oriel Spierer[1], Arie L Marcovich[2,3]

[1]Department of Ophthalmology, Wolfson Medical Center and Sackler School of Medicine, Tel Aviv University, Tel Aviv, Israel, [2]Department of Ophthalmology, Kaplan Medical Center, Rehovot, Israel, [3]The Hebrew University Medical School, Jerusalem, Israel

Key words:

Punctate epithelial erosions, hypoesthesia, magnetic resonance imaging, trigeminal nerve compression

Address for correspondence:
Asaf Achiron, Department of Ophthalmology, Wolfson Medical Center, 62 Ha'Lochamim St., Holon, Israel. Phone: +972-3-5028706
E-mail: achironasaf@gmail.com

Received: 06-03-2021;
Accepted: 25-09-2021
doi: 10.15713/ins.clever.68

Abstract

Reduced corneal sensitivity due to trigeminal nerve injury results in the appearance of epithelial erosions. Here we would like to present 2 cases with asymmetric dry eye findings and decreased corneal sensation that subsequently were found to have a trigeminal nerve injury. We would like to emphasize the importance of corneal and facial sensation and brain imaging in cases with asymmetrical ocular surface changes.

Introduction

The cornea is one of the most densely innervated tissues in the body. Those dense corneal nerves and the neuropeptides produced by their fibers are essential for maintaining the health of the cornea.[1]

Corneal nerves and epithelial cells mutually support each other by the release of trophic factors promoting epithelial cell proliferation, migration and differentiation, as well as nerve development and survival, all are necessary for balanced ocular surface homeostasis and wound healing.[2]

Neurotrophic keratitis, characterized by a reduction in corneal sensitivity, spontaneous epithelium breakdown, and impairment of corneal healing, is a result of trigeminal damage. The most common etiologies include herpetic keratitis, diabetes, and ophthalmic and neurosurgical procedures.[3]

We report on two patients who presented with decreased corneal sensation and asymmetric ocular surface disease due to primary neurological abnormalities. The report supports the role of brain imaging in the management of cases with asymmetrical ocular surface changes.

Case 1

A 26-year-old female was referred to a cornea consultation at the Kaplan Medical Center because of recurrent corneal ulcers in her right eye. She was myopic and used daily wear soft contact lenses. Her past medical history was unremarkable. Visual acuity was 6/12 (20/40) in her right eye and 6/9 (20/30) in her left. The anterior segment examination of the right eye revealed a severely dry eye, with marked punctate epithelial corneal erosions [Figure 1A]. There was hypoesthesia in the right side of her face, including the cornea. The left eye examination was normal, and there were no other abnormal findings. Pupil reactions and ocular movements were normal. She then reported that, for several years, she had not felt pain in the right side of her face. She first noticed this lack of sensation after cosmetic treatment. After this significant asymmetric finding, she underwent a brain magnetic resonance imaging (MRI), which revealed a hypoplastic right trigeminal nerve due to pressure from a right pontine artery in the right pre-pontine cistern [Figure 1B]. She was treated with lubricating drops and punctal plugs and remained stable.

Case 2

A 7-year-old boy was referred to the Pediatric Corneal Service at the E. Wolfson Medical center due to unstable myopia, irregular astigmatism, and difficulty in finding the correct refraction with suspected keratoconus in his right eye. The child was known to have signs of a right 3rd nerve palsy with asymmetric pupils and restricted eye movement, since the age of two. He was not under regular follow-up since then.

On examination, uncorrected visual acuity was 20/100 in the right and 20/30 in the left eye. Refraction was not reliable in the right eye and −0.5 + 1.00 × 65 in the left eye. The slit-lamp exam revealed right central superficial keratitis with reduced sensation. Corneal topography showed irregular astigmatism in the right eye and with the rule astigmatism in the left eye, both without keratoconus pattern [Figure 2B and 2C].

In addition, 20° of left face turn was noticed with limited right adduction, elevation, and depression [Figure 2A]. A right exotropia of 18 prism diopters for distance and 25 prism diopters for near was documented. A 1 mm right eyelid ptosis was also noticed with a 2 mm of anisocoria with right mid dilated pupil non-responding to light. A diagnosis of right 3rd cranial nerve palsy with pupil involvement and dry eye was made. The patient underwent MRI which demonstrated right anterior-lateral cavernous sinus thickening [Figure 2D]. No Definite diagnosis was made but it was assumed the lateral cavernous sinus thickening most probably caused the right 3rd and 5th nerve damage. As the third nerve palsy seems to be irreversible, the neurosurgeon decided to only follow-up with the patient. The patient was treated with lubricating drops and ointment and remained stable.

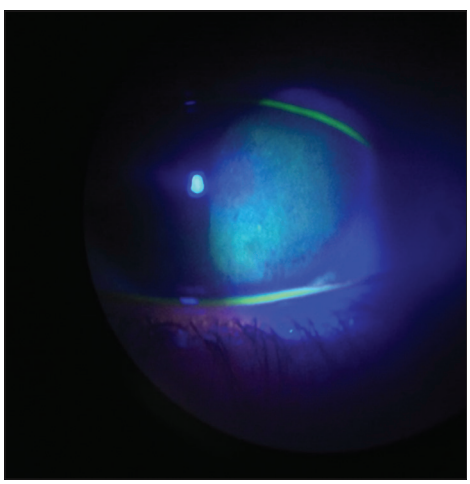

Figure 1A: Right cornea of the patient in case 1. Staining with fluorescein shows multiple punctate corneal erosions

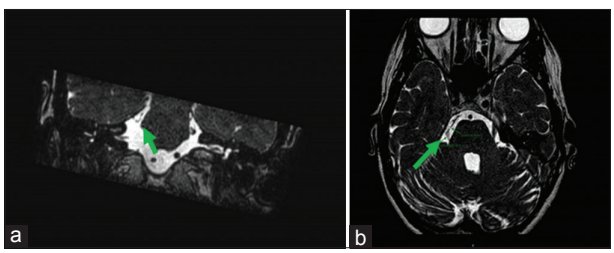

Figure 1B: (a) Coronal magnetic resonance imaging T2W VISTA showing hypoplastic trigeminal nerve, touched by a pontine artery in the pre-pontine cistern (green arrows); (b) Axial view

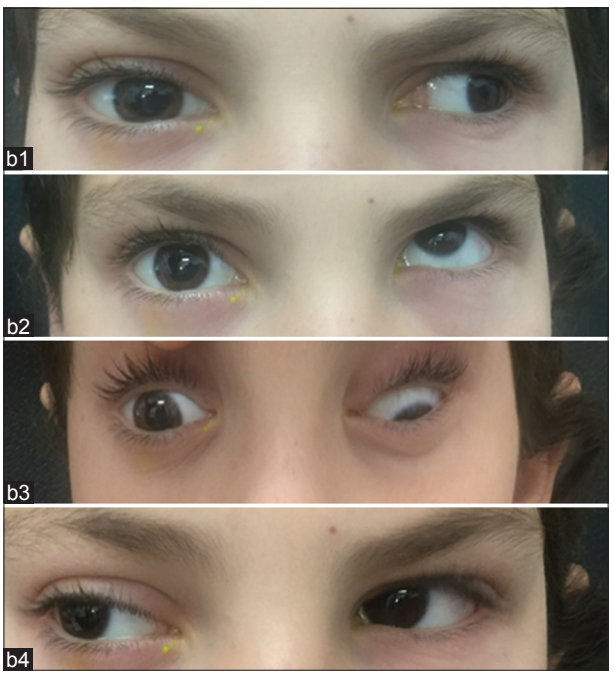

Figure 2A: A severe limitation of the right eye on adduction, elevation, and depression. Abduction is preserved. Anisocoria can also be observed. (b.1): Limited right adduction, (b.2) Limited right elevation, (b.3) Limited right depression, (b.4) Normal right abduction

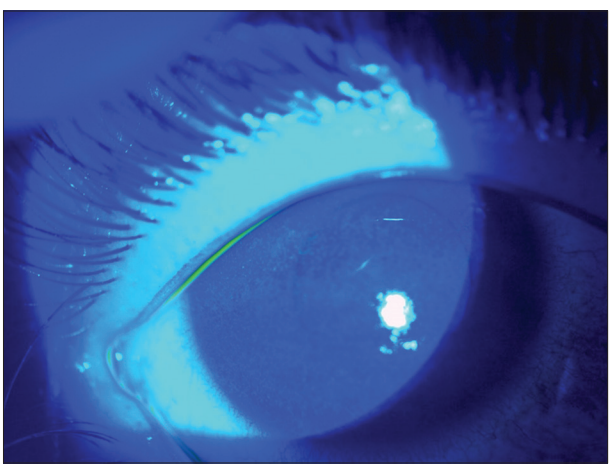

Figure 2B: Right cornea of the patient in case 2. Staining with fluorescein shows multiple punctate corneal erosions

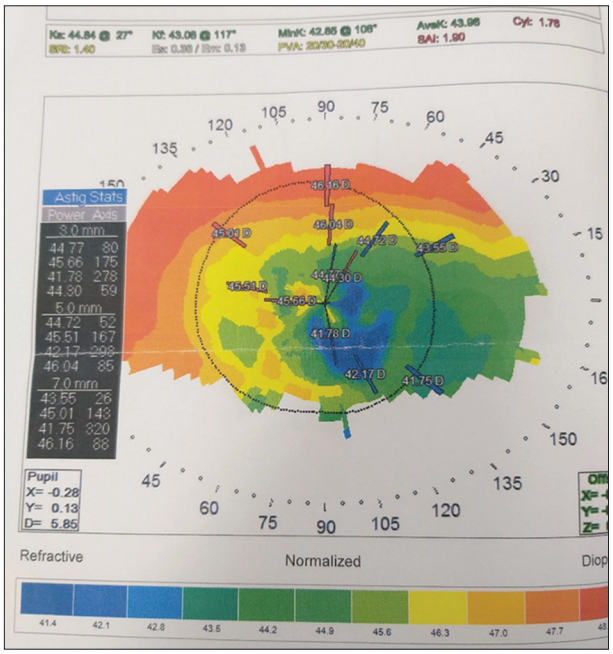

Figure 2C: Corneal topography of the right eye shows irregular astigmatism without a clear keratoconus pattern

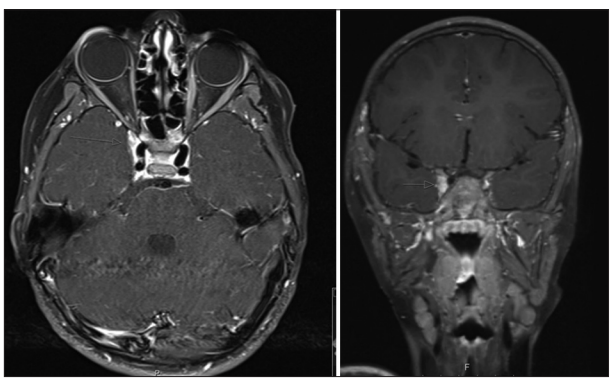

Figure 2D: Magnetic resonance imaging T1 with contrast and fat suppression protocol. Axial and corneal cuts demonstrate thickening of the right cavernous sinus, originating in its anterior-lateral aspect and advancing toward the orbital apex (gray arrow)

Discussion

The cornea is densely innervated with sensory fibers from the ophthalmic branch of the trigeminal nerve, which respond to mechanical, thermal, and chemical stimulation of the cornea. The ocular surface epithelium, tear gland, and sensory and autonomic nerve fibers release cytokines, neuropeptides that modulate the ocular surface environment.[1]

Corneal sensory nerve damage leads to marked changes in the levels of neuromodulators, causing impairment to epithelial cell vitality and imbalance in the metabolic activity of the epithelial cells. This can affect the mitosis of epithelial cells, leading, consequently, to an epithelial breakdown and loss of the microvilli that are responsible for maintaining the mucin layer of the tear film.[4] There is also an associated reduction in lacrimation reflex when the sensory nerves are not properly stimulated, which can worsen the damage by drying the eyes. In addition, bidirectional communication between nerves and the immune system forms a negative feedback loop that keeps both systems in check. Minor insults to the ocular surface are rapidly healed within a continuous trophic environment, maintained by corneal innervation and the tear film.[3]

Table 1: Summary of conditions associated with damage to the trigeminal nerve

Peripheral	Systemic diseases
Herpetic keratitis	Diabetes
Thermal and chemical burns	Multiple sclerosis
Long-term use of contact lenses	Riley-Day syndrome
Corneal surgery	Goldenhar-Gorlin syndrome
Ablative procedures for trigeminal neuralgia	Mobius syndrome
Surgical procedures for reduction of jaw fracture	Familial corneal hypesthesia
Space-occupying intracranial masses (e.g., schwannoma, meningioma and aneurysms)	Congenital insensitivity to pain with anhidrosis
Cranial neurosurgery	

The impairment of corneal trigeminal innervation leaves the cornea in a vulnerable state, in which it may become susceptible to infections and minor traumas that lead to the development of recurrent or persistent epithelial defects.[2] Injury to any part of the trigeminal cranial nerve, from the nucleus to the corneal nerve endings, may cause ocular surface disease [Table 1 and Figure 3].

Reduced corneal sensitivity due to trigeminal nerve injury results in the appearance of epithelial erosions. Therapeutic measures may include daily lubricating eye drops, night creams, and autologous serum. Additional procedures such as punctal plug insertion, amniotic membrane implantation, and surgical tarsorrhaphy may become necessary if the disease worsens.[5]

Conclusion

As presented in our two cases, we would like to emphasize the importance of corneal and facial sensation examination. In asymmetric findings, 5th nerve involvement must be ruled out and therefore brain imaging is required to evaluate any trigeminal nerve pathology.

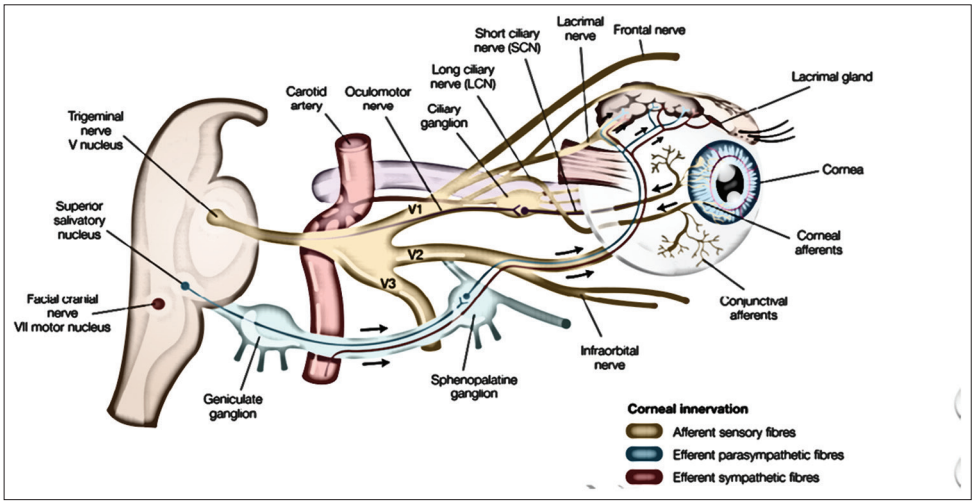

Figure 3: Diagram illustrating the afferent sensory pathway from the cornea and conjunctiva to the trigeminal ganglion, and the efferent sympathetic and parasympathetic nerve pathways. Adapted and modified from Dua *et al.* 2018.[5]

References

1. Cho KJ, Mok JW, Choi MY, Kim JY, Joo CK. Changes in corneal sensation and ocular surface in patients with asymmetrical Keratoconus. Cornea 2013;32:205-10.
2. Versura P, Giannaccare G, Pellegrini M, Sebastiani S, Campos EC. Neurotrophic keratitis: current challenges and future prospects. Eye Brain 2018;10:37-45.
3. Dua HS, Said DG, Messmer EM, Rolando M, Benitez-Del-Castillo JM, Hossain PN, *et al.* Neurotrophic keratopathy. Prog Retin Eye Res 2018;66:107-31.
4. Sacchetti M, Lambiase A. Diagnosis and management of neurotrophic keratitis. Clin Ophthalmol 2014;8:571-9.
5. Cidad P, Boto A, Del Hierro A, Capote M, Noval S, Garcia A, *et al.* Unilateral punctate keratitis secondary to Wallenberg syndrome. Korean J Ophthalmol 2014;28:278-83.

How to cite this article: Achiron A, Rappoport D, Spierer O, Marcovich AL. Unilateral dry eye and hypoesthesia caused by trigeminal nerve compression: Importance of imaging. Cli Exp Vis Eye Res J 2021;4(1):46-49.