



Case report

Resolution of pain with periocular injections in a patient with a 7-year history of chronic ocular pain



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ABSTRACT

Purpose: We report a case of a male patient with chronic ocular pain that resolved completely following peripheral nerve blocks.

Observations: A 66-year-old male presented with a seven-year history of severe left eye pain and photophobia. The pain began after retinal detachment repair with scleral buckle placement. Previous treatments included topical (autologous serum tears, corticosteroids, diclofenac, cyclosporine) and oral (gabapentin, diclofenac) therapies with no pain relief. The patient's pain was so severe that he requested enucleation. After discussion, the decision was made to perform periocular nerve blocks. Prior to the procedure, the patient reported an average pain intensity of 8 out of 10 and photophobia daily. Following left supraorbital, supratrochlear, infraorbital and infratrochlear injections with bupivacaine and methylprednisolone, pain intensity and photophobia improved to 1–2 out of 10. One week later, repeat infraorbital and infratrochlear nerve blocks were given, after which time the patient reported complete resolution of symptoms that lasted for 7 months. Repeat nerve blocks were administered with repeat resolution of pain. There were no complications associated with the procedures.

Conclusions and Importance: Chronic ocular pain can be a debilitating condition. Periorbital nerve blocks can provide pain relief and should be considered as a potential treatment option after medical management has failed.

1. Introduction

Chronic ocular pain can be seen in a number of clinical scenarios. Pain, as defined by the International Association for the Study of Pain, is an “unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.”¹ When applied to the eye, patients often describe their pain as “dryness”,² although other descriptors include “burning”, “tenderness”, and “aching”. These sensations can occur spontaneously or be evoked by stimuli such as light and wind.³ Pain in the eye can arise from nociceptive processes, such as chronic ocular surface disruption and inflammation, from neuropathic processes, or frequently from mixed mechanisms.^{4,5} As the cornea is the most densely innervated tissue in the body (7000 nerve terminals per millimeter square),⁶ it is no surprise that noxious stimulation can result in intense pain responses and that neuropathic pain can develop in response to nerve injury.^{2,4} In fact,

sensitization of corneal nerves has been demonstrated after exposure to inflammatory mediators,⁶ lacrimal gland transection, and hyperosmolarity.^{7,8}

One clinical scenario associated with chronic ocular pain is pain that occurs after eye surgery, with significant data centered around sensations of dryness after laser-assisted in situ keratomileusis (LASIK). After LASIK, approximately 20% of individuals develop persistent dry eye symptoms.⁹ Corneal nerve damage is thought to underlie these persistent symptoms, as corneal nerve density and sensitivity do not fully recover after surgery.^{10,11} For example, in vivo confocal microscopy has demonstrated corneal nerve alterations (e.g. decreased density, neuroma formation) years after LASIK.^{12,13} Hyperexcitability of peripheral and central nerves can explain chronic pain after eye surgery (including sensations of dryness and photophobia) that occur even in the absence of obvious noxious stimuli.^{14,15} Ocular pain has also been reported after other eye surgeries, including in 18% of individuals after

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retinal detachment repair with scleral buckle.¹⁶

While pain can significantly impact quality of life, it is not known how to best treat individuals with chronic ocular pain. Reported treatments have included topical therapies such as autologous serum tears,¹² oral therapies such as the $\alpha_2\delta$ ligands (e.g. gabapentin),^{2,4} and stimulation therapies.^{17,18} In this case report, we describe the therapeutic potential of sensory peripheral nerve blocks and discuss possible mechanisms for their effect on eye pain.

1.1. Case report

A 66 year-old male presented with a seven-year history of severe pain in his left eye with associated photophobia. He had radial keratotomy (RK) in both eyes many years prior. Seven years prior to presentation, he developed a total retinal detachment in the left eye that was treated with scleral buckle, pars plana vitrectomy, and gas placement. His cataract was also removed during the surgery and he was left aphakic. He then developed proliferative vitreoretinopathy and recurrent retinal detachment 3 months later, and underwent repeat pars plana vitrectomy, membrane peel, retinectomy, and silicone oil placement.

All ophthalmic procedures were done under monitored anesthesia with a retrobulbar block (bupivacaine, lidocaine). Post operatively, oil was found to fill approximately 80% of the anterior chamber (Fig. 1). Given the complex nature of the retinal detachment, the decision was made to observe the patient and keep the silicone oil in place. Best corrected visual acuity at that time was 20/80. Over time, progressive thickening was noted in the cornea with band keratopathy development. Furthermore, blood vessel growth was noted into the RK scars (Fig. 2). Vision slowly declined in the eye to a level of hand motion.

The patient developed constant left eye pain after his first retinal surgery. The pain involved the entire left eye but was most severe closest to the nasal region and extended to the periocular area with allodynia to light touch in the supraorbital and frontal regions. He described the pain as constant, sharp/stabbing and rated it as an 8 out of 10. The patient also had photophobia (extreme light sensitivity) and was dependent on dark sunglasses when he left the house. In fact, he preferred to stay at home and avoid light altogether, and over the years decreased his activities due to his photophobia.

Over the years, the patient tried several topical medications (artificial tears, nonsteroidal anti-inflammatory agents (NSAIDs), corticosteroids, cyclosporine, autologous serum tears) and oral pain medications (NSAIDs, opioids, gabapentin) with minimal to no relief. The pain was so severe that patient requested enucleation to eliminate his pain. Past medical, family, and social histories were noncontributory and he denied allergies and prescription medication use. He also denied headaches, dizziness, or constitutional symptoms.

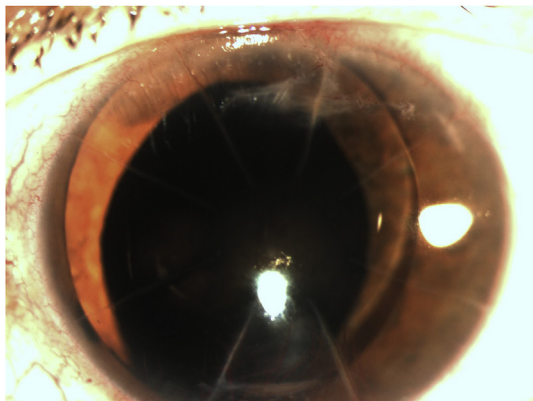


Fig. 1. Slit lamp photograph of left eye demonstrating radial keratotomy scars, an 80% oil fill in the anterior chamber, a surgical iris, and aphakia.

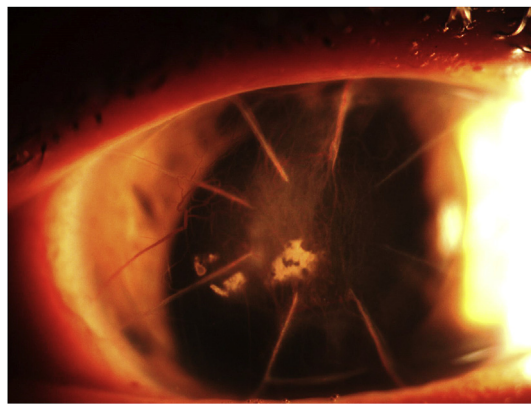


Fig. 2. Slit lamp photograph of left eye demonstrating neovascularization of the cornea into the radial keratotomy scars and central band keratopathy.

On physical examination, pertinent positive findings included dark sunglass use on entering the room. Tearing, ptosis, and conjunctival injection were noted in the left eye on removal of the sunglasses. The pain was minimally improved by topical anesthetic. The patient had mechanical (pressure) hyperalgesia over the left supraorbital ridge. Sensation to soft touch was intact. Pertinent negative findings included no significant intraocular inflammation on slit lamp examination, proper positioning and conjunctival coverage of the scleral buckle, and intraocular pressure within normal limits.

The patient's symptoms of hyperalgesia and allodynia in the absence of remarkable exam findings to explain his pain led to a presumed component of neuropathic ocular pain. Due to the refractory and severe nature of the pain, lack of response to medical therapies, and presumption of a neuropathic pain component to the patient's suffering, the decision was made to perform nerve blocks over several trigeminal nerve branches that contribute to the innervation of the periorbital region and the conjunctiva. Four ml bupivacaine 0.5% (Marcaine) and 1 ml methylprednisolone acetate (Depo-Medrol) 80 mg/ml were placed in a 5 cc syringe and attached to a 25 gauge needle. The supraorbital foramen was identified by palpation and 1 ml was injected over the supraorbital nerve, after the needle was inserted until contact with the ridge was established and then withdrawn 1 mm (Fig. 3). Another 0.5 ml was injected over the supratrochlear nerve at the point of maximum tenderness on the supraorbital ridge, approximately 1 cm medial to the supraorbital foramen. One ml of solution was injected into the infraorbital foramen below the inferior border of the infraorbital ridge. Through the same injection site, the needle was repositioned caudally and medially towards the bridge of the nose and another 0.5 ml was deposited over the infratrochlear nerve.

Following the procedure there was no noted bleeding, swelling, or paresthesia. The patient reported immediate and complete resolution of pain. He was able to remove his sunglasses and rated his pain and light sensitivity as a 0 out of 10. At one-week follow-up, the patient reported significant pain relief and rated the pain as intermittent with an intensity of 1–2 out of 10; he was unable to recall the exact time course of when his mild pain symptoms had recurred after the initial injection. Periocular sensation remained grossly unchanged from the pre-procedural exam. The left infratrochlear and infraorbital blocks were repeated by injecting 1 ml of a 2 ml bupivacaine/1 ml methylprednisolone mix at each site. The patient remained pain free until 7 months post-injection at which time he reported that the pain returned to 1–2 out of 10. He then underwent a 3rd series of injections targeting the supraorbital, supratrochlear, infraorbital and infratrochlear nerves. Four months after this latest series of injections, the patient has 0 out of 10 pain. He does still have mild photophobia symptoms, which are far less severe compared to pre-injection, and well-controlled with lightly tinted glasses.

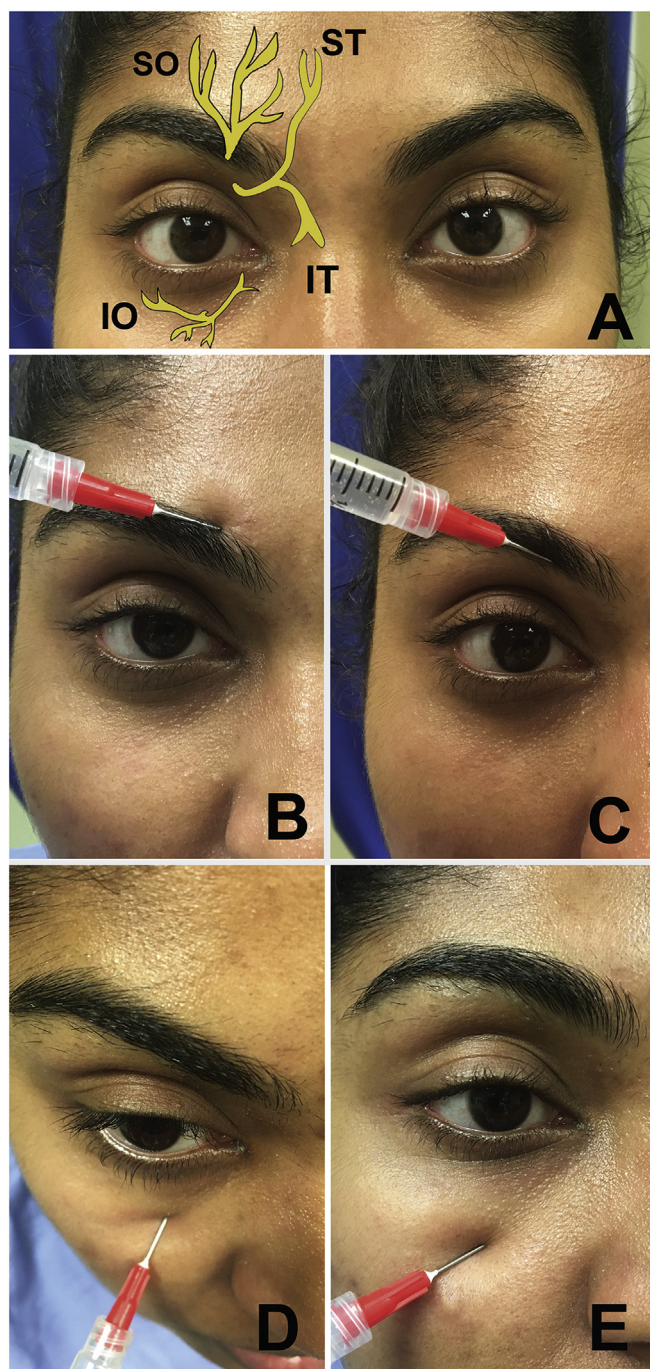


Fig. 3. Representative external photographs demonstrating location of nerve blocks: A) pictorial of nerve branches; B) supraorbital nerve block; C) supra-trochlear nerve block; D) infraorbital nerve block; E) infratrochlear nerve block through same injection site as infraorbital block achieved by repositioning needle.

2. Discussion

To summarize, we describe a patient with chronic ocular pain that began following ophthalmic surgery. The patient failed conservative measures and had symptoms consistent with neuropathic ocular pain; as such, the decision was made to inject the supraorbital, supratrochlear, infraorbital, and infratrochlear nerves. Following injection, the patient had complete resolution of pain and photophobia. To our knowledge, this is the first report of trigeminal nerve branch blocks as a treatment for chronic ocular pain. However, several studies have

reported the use of separate supraorbital, supratrochlear, infraorbital, and infratrochlear nerve blocks for the treatment of migraine headaches, supratrochlear neuralgia, infraorbital neuralgia, and lacrimal neuralgia.^{19–22} The majority of patients who underwent these peripheral nerve injections had several months of pain relief with no noted complications.

The immediate effect of the peripheral nerve injections is a long-acting reversible block of sodium channels by bupivacaine, which prevents depolarization and action potential generation.^{23,24} Bupivacaine has also been shown to inhibit *N*-methyl-D-aspartate (NMDA) receptor-mediated synaptic transmission in the dorsal horn of the spinal cord, and very likely in the spinal trigeminal nucleus, areas critically involved in central sensitization.^{25–28} The addition of methylprednisolone likely further enhances the therapeutic potential of blocks. Corticosteroids, by genomic actions, reduce peripheral inflammation by reducing levels of inflammatory mediators and inhibiting leukocyte infiltration, which in turn decreases ectopic neuronal discharge. Corticosteroids also suppress ectopic firing directly by acting on neuronal membranes,²⁹ including affecting membrane voltage gated calcium currents.^{30,31}

Several studies have highlighted that uninjured nerves adjacent to injured ones are important contributors to pain.^{32–34} Our findings of reduced ocular pain after periocular sensory nerve blocks support the idea that periocular nerves contribute to chronic ocular pain. We hypothesize that blockade of periorbital nerves, adjacent to injured corneal nerves, suppresses ectopic activity and decreases nociceptive signaling traffic to the spinal trigeminal nucleus, where ocular and periorbital pathways converge. We suspect that the long-lasting pain reduction over the course of weeks to months may be the result of modification of ongoing central sensitization within the spinal nucleus, in the absence of further evoked pain stimuli to the eye.

To conclude, we found that periocular sensory nerve blocks can be a successful strategy to reduce long standing ocular pain and photophobia after ophthalmic surgery. The benefit of this approach is that it is low risk, inexpensive, technically easy, and not dependent on complex equipment as landmarks for these nerves are easily identified. While our patient did not experience any complications from the injections, several theoretical complications exist, including changes in skin pigmentation, fat atrophy, and necrosis. The risk of more serious complications, such as brain toxicity, generalized toxicity, and sympathetic blockade can be limited by aspirating prior to injection to ensure that the needle has not cannulated a vessel. Specific to periocular blocks, this maneuver can also reduce the risk of an embolic event in the eye. Although a particulate steroid suspension was used for this patient based on the experience of the pain specialist performing the injections (CDS), using a non-particulate steroid suspension may further reduce the risk of such events. Although not an absolute contraindication, caution should be applied when considering nerve blocks in individuals at high risk of bleeding (e.g. anti-coagulation), with poor wound healing, and at risk for infection (including individuals with ocular pain in the setting of post-herpetic neuralgia).

While there are risks with any nerve block, local pain management does avoid side effects and long-term sequelae that are possible with oral medication. This case is one encouraging anecdote of a patient with presumed neuropathic ocular pain responding well to periocular nerve blocks. Further study is necessary to determine whether our findings are reproducible and sustainable. Patients with clinical evidence of neuropathic ocular pain who do not respond to more conservative measures may be appropriate candidates for this treatment.

Patient consent

Consent to publish the case report was not obtained. This report does not contain any personal information that could lead to the identification of the patient.

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Conflict of interest:

The following authors have no financial disclosures: (AC, RG, NZG, AG, RCL, CDS).

Authorship

All authors attest that they meet the current ICMJE criteria for Authorship

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None

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.ajoc.2019.02.001>.

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