Yellow Dye Laser Treatment of Vascularized Corneal Stromal Scars in Pediatric Patients

Patients with a variety of corneal diseases may develop stromal scars that interfere with vision. Vascularization of these scars may lead to inflammation, extravasation of lipid, and progressive opacification. Laser treatment of the neovascular vessels may induce regression of these vessels, with resultant increased corneal clarity and improved vision. We describe the treatment and outcome of 3 pediatric patients with vascularized corneal stromal scars treated with yellow dye laser. Using a PubMed search, we were unable to find any previous reports of this treatment in pediatric patients.

Informed consent was obtained from the patients and parents before performing the treatment. The affected eye was pretreated with pilocarpine hydrochloride, 2%. The laser was used with the patients awake using topical proparacaine hydrochloride. An iridotomy contact lens using high (∗H1100325) slitlamp magnification was used. A laser (Lumenis, Santa Clara, California) was used with a wavelength of 568 nm. The spot size was 50 µm. The power ranged from 200 to 300 mW, with a duration of 0.1 second. The laser was begun at the distal (corneal) end of the vessels and continued backward toward the limbus. The power was increased until occlusion of the vessels was visualized. The number of spots per session ranged from 30 to 501. Postoperatively, patients were treated with topical prednisolone acetate, 1%, drops 4 times a day and cyclopentolate hydrochloride, 1%, drops twice a day for 3 days.

Report of Cases. Case 1. A 6-year-old boy was referred 1 month after corneal laceration repair and lens extraction following a penetrating pencil injury. Visual acuity with aphakic correction was 20/125 OS. The patient had a vascularized inferior corneal scar with traumatic corectopia (Figure 1). The patient underwent secondary intraocular lens implantation. One month postoperatively, the visual acuity was 20/80, with −0.25 + 2.25 × 140°, and the corneal vascularization and scarring had progressed. The patient underwent 3 treatments with a yellow dye laser. Fourteen months after treatment, his visual acuity was 20/40, with −3.75 + 2.0 × 155° (Figure 2).

Case 2. A 16-year-old boy was struck in the left eye with a paintball, sustaining a corneoscleral laceration with iris prolapse that was surgically repaired. Postoperative ultrasonography revealed a retinal detachment, for which he underwent pars plana lensectomy and vitrectomy. His visual acuity 3 months following surgery was 20/200 with aphakic correction. He had a vascularized corneal scar. Three months following his third laser treatment, the central vascularization had resolved and his visual acuity was 20/20 with an aphakic contact lens.

Patient 3. A 15-year-old girl with hemifacial microsomia was observed since the age of 3 years. She had left-sided amblyopia, anisometropia, exotropia, ptosis with lagophthalmos, exposure keratopathy, and retinal and iris colobomas.

Figure 1. Patient 1 had vascularized inferior corneal scar.

Figure 2. Patient 1 had a decreased central vascularity and opacity 1 month following laser treatment.
Previous treatment included spectacles, patching, and penalization for amblyopia, strabismus surgery, and ocular lubrication. Her Snellen visual acuity maximized at 20/60 OS at the age of 6 years. She developed a dendritic corneal lesion at the age of 11 years, which was presumed to be herpetic. She was treated with acyclovir and topical corticosteroids. The active corneal lesion stabilized, but the patient continued to have a heavily vascularized corneal scar (Figure 3) and her visual acuity decreased to 20/200 because of the opacity and irregular astigmatism. She underwent 4 treatments with a yellow dye laser, with improvement in visual acuity to 20/70, with −1.25 + 1.5 × 90°, and markedly decreased corneal vascularization (Figure 4).

Comment. Patients with corneal diseases may develop neovascularization of the cornea because of the growth of vessels across the limbus into the corneal stroma. This may result in opacification of the cornea, which may be exacerbated by leakage of lipid from the abnormal blood vessels. Laser occlusion of the feeder vessels, initially with argon laser, has been used as a treatment for this condition. The goals of treatment are to induce regression of neovascular vessels, resorb lipid, and improve visual function. Based on the absorption peak of hemoglobin in the yellow range, the yellow dye laser was demonstrated in animal studies to decrease neovascularization in animal models. Baer and Foster used the yellow dye laser to treat 23 patients with corneal neovascularization, with improvement in all patients except those with extensive neovascularization. Using a PubMed search, we were unable to find any previous reports of this treatment in pediatric patients.

The causes for the corneal neovascularization in our patients were traumatic corneal lacerations in patients 1 and 2 and a combination of chronic exposure keratopathy and presumed herpetic disease in patient 3. All patients demonstrated improvement in visual acuity. Possible complications of treatment include intrastromal hemorrhage, iris atrophy, and corneal thinning, none of which occurred in our patients. The laser is not adaptable for use under general anesthesia, so patients must be able to cooperate with applications at the slitlamp, including the use of a contact lens. The procedure is not painful but does require successive treatment sessions. Despite this, even our 6-year-old patient tolerated the treatment without difficulty.

The advantages of successful treatment of corneal neovascularization include improvement of visual acuity and appearance of the corneal scar. In addition, treatment may interrupt progressive opacification and lipid deposition and decrease the need for penetrating keratoplasty. In our 3 pediatric patients, the yellow dye laser safely and effectively decreased corneal neovascularization and improved visual function.

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Figure 3. Patient 3 had a heavily vascularized corneal scar.

Figure 4. Patient 3 had decreased vascularity and opacity 3 weeks following laser treatment.
NMO Antibody–Positive Recurrent Optic Neuritis Without Clear Evidence of Transverse Myelitis

When a laboratory test becomes available for a disorder previously diagnosed solely on clinical grounds, the spectrum of that disorder is apt to be expanded. With the discovery of a blood test (the NMO antibody) that has an estimated sensitivity of 76% and a specificity of 94%, Devic disease is proving to be such an example. Devic recurrent neuromyelitis optica was considered a demyelinating disease in which the cardinal features were a bilateral optic neuropathy and a cervical myelopathy without other clinical or magnetic resonance imaging (MRI) evidence of involvement elsewhere in the nervous system and with a poor prognosis for recovery. With the advent of the NMO-antibody test, the picture of the syndrome is expanding. We report on the cases of 3 women who suffered from recurrent optic neuritis (ON) in association with the NMO antibody but who never developed clear evidence of a transverse myelitis in approximately a decade of follow-up.

Report of Cases. Case 1. A 43-year-old woman was seen in October 1994 with a severe bifrontal headache and left eye pain with eye movement. Over the next week visual acuity declined to 20/50 OS. Dyschromatopsia, a superior altitudinal visual field defect, and an afferent pupillary defect were all present in the left eye. Her fundi, general neurological examination results, and findings from contrast MRI of the brain were unremarkable. Her vision did not improve and optic atrophy developed. She was stable until late 2001 when the visual acuity OS decreased painlessly to the level of counting fingers. Brain MRI findings were again normal. She was observed without any improvement. In the spring of 2002, she had similar symptoms in the right eye attributed to ON and was not treated; details of her examination findings at that time were not available to us. We first evaluated her condition in July 2002 by which time her best-corrected visual acuity had recovered to 20/20 OD with normal color and full visual fields. Her visual acuity OS was 1/200 and her remaining visual field was a small inferonasal island. Both optic discs were pale. Contrast MRI of the brain and cervical spine showed no lesion and she was not treated. Results of a repeated contrast MRI of the brain and cervicothoracic cord in April 2003 were normal. She was well until June 2005 when she developed “blotchy” vision in her right eye that was accompanied by discomfort with eye movement. Visual acuities were 20/400 OD and hand motion perception OS. There was an inferior altitudinal visual field defect in the right eye. An MRI of the brain and orbits showed only 1 small T2-weighted hyperintense lesion in the subcortical white matter of each parietal lobe. She was treated with intravenous (IV) methylprednisolone sodium succinate, 1g/d, for 5 days, and 2 months later visual acuity OD improved to 20/20 and the inferior altitudinal visual field defect improved. The NMO-antibody test that time was positive. She also complained of intermittent, fleeting paresthesias that could occur anywhere in her body, including the face and head. Findings from a neurological examination were normal except for a left Hoffman sign. Results from a contrast MRI of the total spine were normal (Figure 1).

Case 2. In July 1997, a 39-year-old woman had 2 episodes of blurry vision in both eyes that appeared like “wavy water” after taking a hot shower. Four months later, vision in her left eye declined over 5 days, accompanied by pain on eye movement and occasional photopsias. Best-corrected visual acuities were 20/10 OD and 8/200 OS and she could only correctly identify the control Ishihara plate with her left eye. There was a large central scotoma and a relative afferent papillary defect in the left eye. The right fundus was unremarkable and the nasal portion of the left optic disc was swollen without hemorrhages or exudates. Findings from neurological examination were normal. An MRI of the orbits showed increased inversion recovery signal and enhancement of the left optic nerve that extended through the optic canal but spared the optic chiasm. The brain parenchyma was unremarkable. Based on a history of an abdominal rash several months earlier and an antinuclear antibody titer positive at 1:5120, she was treated with IV methylprednisolone for presumed lupus optic neuropathy. However, there was no other clinical evidence of systemic lupus and findings for double-stranded DNA were negative. One month later, the acuity OS decreased to hand motion perception, but it improved to 20/40 by 8 months after her first visit. She was then unchanged until 7 years later when she experienced pain with eye movements in the left eye that was followed by a progressive loss of visual acuity to no light perception (NLP) over 2 days. Acuity OD was 20/20, but a temporal field defect was found. An MRI showed enhancement of the left optic nerve to the chiasm but no white matter lesions in the brain parenchyma (Figure 2). The visual acuity OS improved to 8/200 over 8 months, but the acuity OD dropped to 20/25 prompting another course of IV methylprednisolone. In February 2005, she saw a yellow band across the center of her left eye accompanied by pain in each eye. She im-