Necrotising Scleritis Post Trans-Scleral Diode Cyclophotocoagulation-A Novel Approach

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Abstract:

Introduction: Surgically induced necrotizing sclerokeratitis (SINS) is a destructive form of scleritis.

Presentation of Case: We report two cases of necrotising scleritis after inferior 180 degrees trans-scleral cyclophotocoagulation (TSCPC). Both patients presented after one week with symptoms of pain and redness. We tried a novel therapy of surgical patch graft rather than systemic steroid therapy.

Discussion: High dose pulse systemic steroid with gradual tapering is the initial treatment for which majority cases respond. Some cases do require systemic immunosuppression by drugs other than steroids. We tried a novel therapy of surgical patch graft rather than systemic steroid therapy.

Conclusion: Scleral patch graft is a novel treatment in the management of post-TSCPC SINS.

Introduction

Surgically induced necrotizing sclerokeratitis (SINS) is a destructive form of scleritis reported following cataract surgery, trabeculectomy, squint surgery, and retinal detachment surgery and pterygium excision [1-3]. It is recognized by a focus of intense scleral inflammation associated with severe ocular pain that progresses to scleral thinning and even globe perforation due to collagen destruction. It is often associated with systemic autoimmune conditions. Trans-scleral diode cyclophotocoagulation destroys ciliary processes and reduces aqueous production and has been advocated in treatment of refractory glaucomas since 1930 [4]. Postoperatively it can cause intense inflammation, pain, redness a hypotony and visual loss [4]. We report two cases of SINS following TSCPC and different management strategy.

Presentation of Case

A 68 years old diabetic on treatment for 20 years presented with history of dimness of vision, pain and redness in left eye for 1 week. Patient had undergone cataract surgery (clear corneal phacoemulsification) in both eyes ten months back following which he developed proliferative diabetic retinopathy (PDR) changes in both eyes. Intravitreal Avastin (Bevacizumab) injection and panretinal photocoagulation (PRP) was performed to stabilise his PDR. On examination his BCVA was 6/9 (RE) and 3/60 (LE). Anterior segment examination his BCVA was 6/9 (RE) and 3/60 (LE). On anterior segment examination his BCVA was 6/9 (RE) and 3/60 (LE). Gonioscopy showed 3600 peripheral anterior synechiae and vision worsened to counting fingers ½ m in left eye. Patient continued to experience pain in his left eye and hence cycloablative procedure like trans-scleral cyclophotocoagulation was planned after an informed and a written consent was taken explaining the guarded visual prognosis. The semiconductor solid state diode laser system for trans-scleral diode cyclophotocoagulation with an 810 nm wavelength exhibits less scleral transmission but considerably greater absorption by melanin. The laser energy was transmitted by a 600-micron- diameter quartz fibre with a spherical polished tip called the “G-Probe”. Spherical footplate matches the scleral curvature and has 0.7 protruding fibre optic tip centrally which is 1.2 mm away from the curved edge of footplate. TSCPC was performed under peribulbar anaesthesia. Duration was set at 2000 ms (2 seconds), and the initial power setting was 1750 mW. The power was increased in 250 mW increments to a maximum of 2500 mW until an audible “pop” (caused by tissue explosion of the ciliary process, the iris root anteriorly or the retina posteriorly) was heard, then the power was backed off 250 mW and treatment was completed at this power level. TSCPC was given under peribulbar block for the first sitting for 180 degrees with 26 shots and power settings of 2250 mw and 2000 ms duration. Patient was continued with antiglaucoma medications, topical steroids and cycloplegics. In the first postoperative week patient developed ciliary congestion associated with sclera thinning at the nasal limbus from 9 O’clock to 7 O’clock with iris incarceration. Patient was evaluated in detail to rule out any systemic collagen vascular diseases like rheumatoid arthritis (RA), Wegener’s granulomatosis, systemic lupus erythematosus etc. All the investigations turned out to be normal ruling out collagen vascular diseases. G-probe used was also inspected for any damage or break, which was negative. Also, the same probe was used for another few patients for cyclophotocoagulation and they lowering of intra-ocular pressure (IOP) and improving corneal clarity. Neovascularization regressed with PRP but IOP was 52 mmHg in left eye. Gonioscopy showed 3600 peripheral anterior synechiae and vision worsened to counting fingers ½ m in left eye. Patient continued to experience pain in his left eye and hence cycloablative procedure like trans-scleral cyclophotocoagulation of inferior 180 degrees was performed after an informed and a written consent was taken explaining the guarded visual prognosis. The semiconductor solid state diode laser system for trans-scleral diode cyclophotocoagulation with an 810 nm wavelength exhibits less scleral transmission but considerably greater absorption by melanin. The laser energy was transmitted by a 600-micron- diameter quartz fibre with a spherical polished tip called the “G-Probe”. Spherical footplate matches the scleral curvature and has 0.7 protruding fibre optic tip centrally which is 1.2 mm away from the curved edge of footplate. TSCPC was performed under peribulbar anaesthesia. Duration was set at 2000 ms (2 seconds), and the initial power setting was 1750 mW. The power was increased in 250 mW increments to a maximum of 2500 mW until an audible “pop” (caused by tissue explosion of the ciliary process, the iris root anteriorly or the retina posteriorly) was heard, then the power was backed off 250 mW and treatment was completed at this power level. TSCPC was given under peribulbar block for the first sitting for 180 degrees with 26 shots and power settings of 2250 mw and 2000 ms duration. Patient was continued with antiglaucoma medications, topical steroids and cycloplegics. In the first postoperative week patient developed ciliary congestion associated with sclera thinning at the nasal limbus from 9 O’clock to 7 O’clock with iris incarceration. Patient was evaluated in detail to rule out any systemic collagen vascular diseases like rheumatoid arthritis (RA), Wegener’s granulomatosis, systemic lupus erythematosus etc. All the investigations turned out to be normal ruling out collagen vascular diseases. G-probe used was also inspected for any damage or break, which was negative. Also, the same probe was used for another few patients for cyclophotocoagulation and they...
did not develop any scleral melting postoperatively. Surgical treatment of free hand fashioned scleral patch graft with amniotic membrane graft (AMG) over the sclera graft was planned over the scleral melt area and patient was treated with postoperative topical steroids, cycloplegics and antiglaucoma medications. Systemic steroids were withheld as patient was diabetic and patient did not give consent for pulse steroid therapy. Patient responded to scleral patch graft and topical steroids, cycloplegics and antiglaucoma medications. IOP was 16 mmHg in left eye and inflammation had reduced by two weeks. No recurrence of sclera thinning was noticed in graft site as also in the inferior 180° in the follow of period of 16 months. Antiglaucoma medications were also reduced from three medications to single medication and IOP was well controlled. Visual acuity improved to 5/60 during the follow up period.

Case 2

A 65 years old diabetic on treatment for 15 years presented with history of dimness of vision since few weeks and severe pain and redness in left eye for 1 month. Patient was diagnosed with PDR in the right eye and neovascular glaucoma in the left eye. On examination his BCVA was 6/9 (RE) and perception of light (PL+ve) (LE). Anterior segment in right eye showed quiet anterior chamber but left eye showed hazy edematous decompensating cornea with neovascularization on iris, ectropion uveae and mild hyphema. Other details were not visible. IOP in right eye was 18 mmHg and left eye was 58 mmHg. Neovascular glaucoma in left eye was treated with antiglaucoma medications, topical steroids and cycloplegics. Pan retinal photocoagulation could not be performed due to media haze. So injection Avastin (Bevacizumab) and anterior retinal cryopexy was performed. But patient did not get relief from pain even after waiting for 3 weeks. So we decided to plan inferior 180 degree diode cyclophotocoagulation after taking an informed and written consent from the patient. Prognosis was clearly explained to the patient and also the palliative nature of whole treatment. TSCPC was performed under peribulbar anaesthesia block for the first sitting for 180 degrees with 24 shots and power settings of 2500 mw and 2000 ms duration. Laser was delivered at subthreshold level after hearing the delayed pop sound. Patient was continued with antiglaucoma medications, topical steroids and cycloplegics. In the first postoperative week patient developed ciliary congestion associated with sclera thinning at the nasal limbus from 3 O’clock to 7 O’clock with uveal show (Figure 1).

Patient was evaluated in detail to rule out any systemic collagen vascular diseases which turned out to be negative. G-probe used was also inspected for any damage or break, which was negative but still that G-probe was changed for future cases. Scleral melt was large extending four clock hours, we decided to plan for a scleral patch graft. A free hand fashioned scleral patch graft (Figure 2) was performed over the scleral melt area along with an AMG and patient was treated with postoperative topical steroids, cycloplegics and antiglaucoma medications. Patient responded to scleral patch graft and topical steroids, cycloplegics and antiglaucoma medications. IOP was 10 mmHg in left eye and inflammation had reduced by two weeks. No recurrence of sclera thinning was noticed in graft site as also in the inferior 180° with a follow up period of 16 months. Patient was off all antiglaucoma medications subsequently. Visual acuity improved to 4/60 during the follow up period.

Discussion

SINS is reported from first postoperative day to 40 years after ocular surgery and after noncontact procedures like YAG capsulotomy to surgeries like cataract, pterygium, trabeculectomy, squint surgery, scleral buckling and vitrectomy [3]. It also has been reported after multiple surgeries where primary surgery is the inciting factor. Mechanism is thought to be hypersensitivity reaction to antigens revealed or altered after tissue injury. High dose pulse systemic steroid with gradual tapering is the initial treatment for which majority cases respond. Some cases do require systemic immunosuppression by drugs other than steroids. Refractory cases require surgical grafting in addition to systemic immunosuppression [3]. SINS is associated with many systemic conditions like collagen vascular diseases, thyroid disorders, diabetes, hyperuricemia etc. We tried a novel treatment option in our case that is surgical correction without systemic steroids. Also our patients were diabetic and hypertensive with proliferative diabetic retinopathy with one having undergone multiple procedures like PRP, intravitreal injections and cataract surgery. They also had precarious diabetic control and were not willing for systemic steroid therapy. Our patients responded well with donor scleral patch graft and topical anti-inflammatory therapy and scleral melt has not recurred in the graft area with a follow up of 16 months. Similarity in our case and reports by Shen et al. [5] and Sudha et al. [6] was occurrence of SINS in areas where TSCPC was administered. Sangwan et al. [7] described one case of SINS in aphakic glaucoma following TSCPC managed with sclera patch graft but the eye had to be eviscerated.
**Conclusion**

Scleral patch graft is a novel treatment in the management of post-TSCPC SINS.

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