Case Report

Surgically Induces Scleral Melting Improved by Topical Erythropoietin: A Case Report

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Abstract

Surgically induced scleral necrosis is a rare complication after many types of ocular surgery, including cataract surgery, pterygium surgery, glaucoma surgery, and vitrectomies. This report discusses a 35-year-old woman with Type I diabetes mellitus with multiple episodes of ocular surgeries, including glaucoma device implantation. The subject presented to our clinic with ocular pain and vision loss in the right eye following reoperation for glaucoma device implantation. The subject was managed successfully with topical erythropoietin in her episode of scleral melting. In this case, successful epithelialization and vascularization of the conjunctival defect were observed with the use of topical erythropoietin, resulting in putting off another reoperation. This indicates the usefulness of topical erythropoietin in successfully managing surgically-induced scleral melting cases.
Keywords: Scleral Melting; Erythropoietin; Glaucoma.

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Introduction

Surgically induced scleral necrosis is a rare complication of ophthalmologic procedures. This complication occurs due to vascular ischemia accompanied by scleritis. It is associated with cataract surgeries, vitreoretinal surgeries, pterygium excision, and glaucoma procedures. Its association with predisposing factors is largely unproven [1].

The use of erythropoietin in various ophthalmic diseases is gaining ground. Successful use of topical erythropoietin in scleral necrosis was reported in the rabbit models and then in humans by Feizi et al [2].

This article discusses a 35-year-old female patient who successfully managed surgically-induced scleral melting followed by Ahmed glaucoma valve implantation treated with topical erythropoietin.

Case Report

The subject referred to Imam Hussein Hospital's Glaucoma Clinic complaining about pain in the right eye that began three months ago. The subject had a history of Type I diabetes mellitus, a surgical history of cataract surgery and vitrectomy in the right eye, intravitreal anti-VEGF injections in both eyes, panretinal photocoagulation and Ahmad glaucoma valve implantation in both eyes, and Yag laser capsulotomy in the right eye.

Her best-corrected visual acuity was counting fingers from 3 meters in the right eye and 5/10 in her left eye.

In her anterior segment examination, conjunctival injection and melting and underlying scleritis had led to tube exposure in her right eye. The left eye's anterior segment examination was unremarkable.

Her tonometry applanation with the Goldman tonometer was 30 mmHg in her right eye and 17 mmHg in her left eye with the use of latanoprost, timolol, dorzolamide, and brimonidine in both eyes at the highest possible dosage.

In her fundus examination, the right eye showed a pale optic disc with a 0.45 cup/disc ratio and signs of regressed proliferative diabetic retinopathy. She had a pink disc with a 0.7 cup/disc ratio and signs of regressed proliferative diabetic retinopathy in the left eye. Both eyes had laser scars due to laser photocoagulation in the past.

Due to assessment of glaucoma device tube exposure, reimplantation of the new Ahmad glaucoma valve in the superior nasal area with simultaneous repair of the first AGV with scleral patch graft was planned.

One month after her surgery, her second tube was exposed to about 0.5 mm on the nasal side with adjacent conjunctival melting and vascularizing of the adjacent sclera in an area of about 3*4 mm with uveal exposure at two sites in an area of 0.5*0.5 mm, with a 2 mm distance from the limbus. There was no leakage from these sites.

Due to probable diagnosis of surgically induced scleral necrosis leading to exposure of the second AGV tube, lubrications plus antibiotic and erythropoietin drops 3000 IU in milliliter 4 times a day had been made by diluting 1.5 mL of commercially available recombinant human erythropoietin solution for intravenous use (PDpoetin, 10,000 IU/0.5 mL; Pooyesh Darou Biopharmaceutical Co., Tehran, Iran) with 8.5 mL of isotonic normal saline to arrive at a final concentration of 3000 IU/mL.

Four days after starting topical erythropoietin therapy, vascularization was seen on the tube's site, tube exposure was resolved, and vascularization progressed to the avascular sclera (Figure 1).

Two weeks after topical erythropoietin therapy,

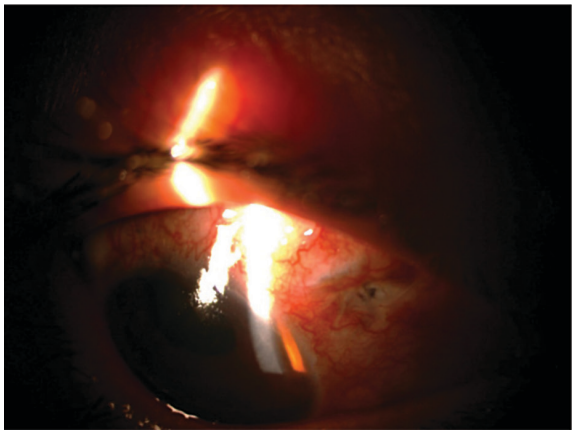

Figure 1: shows vascularization on the tube's site, and vascularization progressed to the avascular sclera

one site of uveal exposure was covered entirely with the conjunctiva. Also, a thin vascularized conjunctiva layer covered the second site of uveal exposure. (Figure 2)

Due to the healing of the defects, erythropoietin drops were discontinued, but lubrication was continued.

Discussion

Surgically induced scleral necrosis is rare after various ophthalmic surgeries, with the underlying mechanism including inflammation and vascular ischemia. Its underlying factors are currently unknown, but this complication is thought to be related to excessive use of cauterization, excessive manipulation of tissue with forceps, and use of beta irradiation and mitomycin. It has been associated with underlying autoimmune disease. It has been reported with contradicting numbers and mostly with cases of surgically-induced scleral necrosis after cataract surgery. Also, the association of surgically induced scleral necrosis with infections is currently unproven, but cases of superimposed infections are reported in the literature. Due to the relative rarity of this complication, it is usually a diagnosis of exclusion [1, 3, 4].

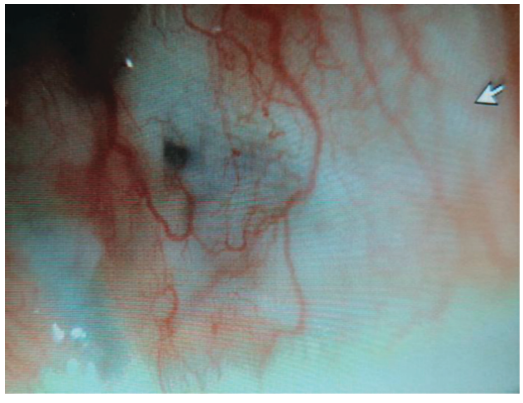

Figure 2: One site of uveal exposure was covered entirely with the conjunctiva. The second site of uveal exposure was covered by a thin vascularized conjunctiva layer

The treatment of this complication includes medical and/or surgical therapy. Medical therapy includes topical antibiotics, lubrication and topical and systemic corticosteroids, and hyperbaric oxygen, while surgical therapy consists of conjunctival flap tenoplasty, amniotic membrane transplant, or scleral patch graft. Erythropoietin is a glycoprotein hormone responsible for the differentiation of hematopoietic cells and essential for maintaining homeostasis. Feizi et al. experimented on rabbits and reported that topical erythropoietin used every six hours could offset many scleral necrosis mechanisms by promoting angiogenesis, inhibiting apoptosis, and downregulating proinflammatory cytokines [2, 5].

Our result agrees with the findings of Feizi et al. regarding both human and animal subjects. Our patient responded well to topical erythropoietin, and her scleral necrosis resolved without further intervention.

Conclusions

Surgically induced scleral necrosis is a rare but devastating occurrence after ophthalmic surgeries that can be successfully treated with

topical erythromycin.

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Footnotes and Financial Disclosures

Conflict of interest:

The authors have no conflict of interest with the subject matter of the present manuscript.