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Treatment Outcomes of Cystoid Macular Edema in Patients with Boston Type I Keratoprosthesis

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INTRODUCTION

Cystoid macular edema (CME) is a poorly studied complication of type 1 Boston Keratoprosthesis (Kpro). The incidence of CME following Kpro implantation in recently published Kpro series ranges from 0.74% to 33.3%[1-7]. A small number of series indicate specific treatments for CME and even fewer report on outcome of treatment[1-7]. None of these series reported on the criteria for diagnosis, specific treatment regimens, the effect on visual acuity or optical coherence tomorgraphy (OCT) measurement, or the presence of treatment-related complications. In this study we report our experience in treating post-Kpro CME using several established treatment modalities including topical steroids and non-steroidals (NSAIDs), peri-ocular and intravitreal steroid injections, and intravitreal bevacizumab.

METHODS

The medical records of all patients who underwent implantation of Boston type I Kpro at the Illinois Eye and Ear Infirmary between February 2007 and November 2012 were retrospectively reviewed. Approval for the study was granted by the institutional review board of the University of Illinois at Chicago.

In all, 105 type 1 keratoprosthesis procedures in 91 eyes of 85 patients were retrospectively reviewed. Eyes with CME as confirmed on optical coherence tomography (OCT) in the postoperative period were included. The decision to obtain an OCT was made based on clinical suspicion of the trained examiner (vitreoretinal or corneal specialist). CME was defined as central subfield thickness (CST) greater than 315 µm or the presence of cystoid spaces or subretinal fluid on spectral domain-OCT[8]. All OCT images were obtained on either the Stratus OCT (Carl Zeiss Meditech Ophthalmic Systems Inc., Dublin, CA, USA) or Spectralis OCT (Heidelberg Engineering, Heidelberg, Germany). When available, the calculated value of CST by the Spectralis OCT was recorded. In cases of poor Spectralis OCT signal strength or those with only Stratus OCT data available, retinal thickness measurements were obtained manually, measured from the posterior surface of the retinal pigment epithelium layer to the internal limiting membrane.

Outcome measures included visual acuity (VA), OCT macular thickness, type, frequency, and duration of CME treatment. Other information recorded included reason for Kpro implantation (underlying diagnosis), comorbid ocular disease, concomitant ocular procedures, and complications including elevation of intraocular pressure, endophthalmitis, Kpro explantation, or repeat Kpro procedures.

Patients were categorized by treatment group and by final anatomic outcomes for analysis. Treatment groups included Topical (topical prednisolone acetate or NSAIDS – usually ketorolac), Intravitreal dexamethasone implant (Ozurdex), and Triamcinolone/bevacizumab (either intravitreal or periocular triamcinolone, intravitreal bevacizumab, or combination of both). Patients were divided into four different anatomic outcome groups based on the difference between the final and initial OCT thickness: Resolved (R), Improved (I), Stable (S), and Worsened (W). CME was designated as "resolved" based on resolution of cystoid spaces on OCT. Improved CME was defined as persistent cystoid spaces with greater than 50 µm improvement of final OCT macular thickness compared to initial measurement. Stable CME was defined as a final OCT macular thickness within 50 µm of the initial measurement. Worsened CME was a final OCT macular thickness increase of more than 50 µm.

RESULTS

INDICATIONS FOR KERATOPROSTHESIS

	Preoperative Diagnosis	Number of Eyes (%)
No Prior Corneal Transplant	Chemical burn	3 (15.8)
	Corneal scar	1 (5.3)
	Corneal ulcer	1 (5.3)
	Aniridia/Limbal stem cell deficiency	1 (5.3)
	Total	6 (31.6)
Failed Corneal Transplant	Infectious keratitis	3 (15.8)
	Chronic uveitis	3 (15.8)
	Corneal dystrophy	2 (10.5)
	Unknown	2 (10.5)
	Thermal burn	1 (5.3)
	Keratoconus	1 (5.3)
	Bullous keratopathy	1 (5.3)
	Total	13 (68.4)

TABLE 1
Indications for keratoprosthesis in 19 eyes (21%) which developed postoperative CME were consistent with other large published series[1-7].

TREATMENT ALGORITHM

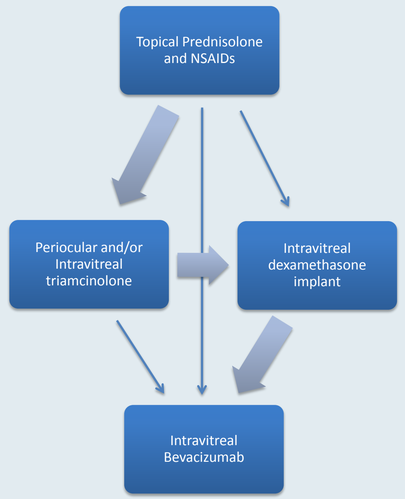

FIGURE 1
Treatment algorithm devised for patients with keratoprosthesis and cystoid macular edema. Generally, treatment is initiated with topical medications (prednisolone acetate and NSAIDs). If not successful, then either periocular and/or intravitreal triamcinolone or intravitreal dexamethasone implant is used. History of steroid response or concern for elevated intraocular pressure may lead to earlier use of intravitreal bevacizumab instead of steroids. Large arrows show a dominant algorithm with smaller arrows as alternates.

TREATMENT OUTCOMES

Treatment Group	Number of Patients (%)	Time from Kpro to CME diagnosis (months)	OCT thickness at diagnosis of CME (µm)	Visual acuity at diagnosis of CME (LogMAR)	Duration of CME (months)	Total follow up time (months)	OCT thickness at final follow up (µm)	Visual Acuity at final follow up (LogMAR)	Final CME Anatomic Outcomes
Topical	9 (47%)	4.1	394	20/200 (1.0)	5.6	20.0	331 p = 0.10	20/300 (1.18), p= 0.11	3 R, 3I, 2S, 1W
Dexamethasone implant	6 (32%)	1.6	703	20/200 (1.0)	6.8	28.4	419 p <0.05	20/400 (1.3), p= 0.41	1R, 4I, 1S
Triamcinolone/bevacizumab	4 (21%)	6.1	585	20/280 (1.15)	1.8	24.0	501 p = 0.25	20/200 (1.0), p=0.30	1I, 2S, 1W

TABLE 2
Patients were divided into three treatment groups for analysis. The topical group received only topical 1% prednisolone aceatate and/or NSAIDS. Patients in the intravitreal dexamethasone implant group each received at least one Ozurdex injection. The triamcinolone/bevacizumab group received either posterior sub-tenon's and intravitreal triamcinolone, intravitreal bevacizumab, or a combination of both medications. All numerical values are calculated median value unless otherwise specified. Final CME outcomes were divided into four groups: Resolved (R), Improved (I), Stable (S), and Worsened (W).

DISCUSSION

- Nineteen of 91 eyes (20.9%) were diagnosed with postoperative CME.
- Four eyes experienced an intraocular pressure spike requiring treatment while undergoing therapy for CME. Two of these eyes were from the "Topical" group and two were from "triamcinolone/bevacizumab" group. Two were managed medically, one underwent diode cyclophotocoagulation and one eye with pre-existing glaucoma underwent repeat glaucoma drainage implantation.
- Limitations due to retrospective design include variations in the number of patients in each treatment group, the duration of time from keratoprosthesis implantation to diagnosis of CME, and the initial OCT thickness.
- The intravitreal dexamethasone implant group was the only treatment subgroup with improvement in OCT thickness reaching statistical significance. This may be influenced by the markedly higher starting OCT thickness, making more room for improvement possible.
- No group had a statistical difference in final visual acuity compared to baseline. Likely limiting factors for visual acuity improvement included small sample size, eye pathology (glaucoma, aniridia, chronic uveitis, etc.), and severity and chronicity of CME.
- 12 of 19 eyes (63%) resolved or improved their CME with treatment; 5 of these 12 also improved visual acuity.

CONCLUSION

Treatment of CME following type 1 Boston keratoprosthesis can be challenging. Corticosteroids remain a mainstay of treatment and anatomic improvement may occur in the absence of visual acuity improvement. Twelve of 19 eyes (63%) exhibited resolution or improvement of CME, including 5 of 6 eyes treated with intravitreal dexamethasone implant; five of these 12 eyes had improved visual acuity. Intravitreal dexamethasone implant may be beneficial in treating chronic Kpro CME. The chronic nature of the CME in these eyes may influence visual recovery.

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