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 0.7% to 33.3%1-7. A small number of series indicate specific treatments for CME and even fewer report on outcome of treatment1-7. None of these series reported on the criteria for diagnosis, specific treatment regimens, the effect on visual acuity or optical coherence tomography (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-steroids (NSAIDs), peri-ocular and intravitreal steroid injections, and intravitreal bevacizumab.

METHODS

The medical records of all patients who underwent implantation of Boston type 1 Kpro at the Illini 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, 205 type 1 keratoprosthesis procedures in 215 eyes of 210 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 based on clinical suspicion of the trained examiner (intracameral or coaxial specular). CME was defined as central subfield thickness (CST) greater than 315 µm or the presence of cystoid spaces or subretinal fluid on the spectral domain-OCT. All OCT images were obtained with either the Spectral OCT (Carl Zeiss Meditec Ophthalmic Systems Inc., Dublin, CA, USA) or Spectralis OCT (Heidelberg Engineering, Heidelberg, Germany). When available, the calculated value of CST by the Spectral OCT was recorded. In cases of poor Spectral OCT signal strength or with only dubious OCT data available, clinical 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 (LogMAR), OCT macular thickness, frequency, and duration of CME treatment. Other information recorded included frequency and duration of CME treatment. Treatment algorithm devised for patients with keratoprosthesis and cystoid macular edema. Generally, treatment is initiated with topical medications (prednisolone acetate and topical nonsteroidal anti-inflammatory agents). Intravitreal injection is reserved for patients with acute uncontrolled intraocular pressure or severe visual loss. Intravitreal dexamethasone implant may be beneficial in treating chronic Kpro CME.

RESULTS

INDICATIONS FOR KERATOPROSTHESIS

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

Randee C. Miller, MD, Kaitlyn M. Wallace, MD, Joshua H. Hou, MD, Clement C. Chow, MD, Jose de la Cruz, MD, Maria S. Cortina, MD, Felix Y. Chau, MD

UIC Department of Ophthalmology and Visual Sciences, Illinois Eye and Ear Infirmary, Chicago, IL, United States

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) 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-steroids (NSAIDs), peri-ocular and intravitreal steroid injections, and intravitreal bevacizumab.

The incidence of CME following Kpro implantation in recently published Kpro series ranges from 0.7% to 33.3%1-7. A small number of series indicate specific treatments for CME and even fewer report on outcome of treatment1-7. None of these series reported on the criteria for diagnosis, specific treatment regimens, the effect on visual acuity or optical coherence tomography (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-steroids (NSAIDs), peri-ocular and intravitreal steroid injections, and intravitreal bevacizumab.

The incidence of CME following Kpro implantation in recently published Kpro series ranges from 0.7% to 33.3%1-7. A small number of series indicate specific treatments for CME and even fewer report on outcome of treatment1-7. None of these series reported on the criteria for diagnosis, specific treatment regimens, the effect on visual acuity or optical coherence tomography (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-steroids (NSAIDs), peri-ocular and intravitreal steroid injections, and intravitreal bevacizumab.

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 0.7% to 33.3%1-7. A small number of series indicate specific treatments for CME and even fewer report on outcome of treatment1-7. None of these series reported on the criteria for diagnosis, specific treatment regimens, the effect on visual acuity or optical coherence tomography (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-steroids (NSAIDs), peri-ocular and intravitreal steroid injections, and intravitreal bevacizumab.

METHODS

The medical records of all patients who underwent implantation of Boston type 1 Kpro at the Illini 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, 205 type 1 keratoprosthesis procedures in 215 eyes of 210 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 based on clinical suspicion of the trained examiner (intracameral or coaxial specular). CME was defined as central subfield thickness (CST) greater than 315 µm or the presence of cystoid spaces or subretinal fluid on the spectral domain-OCT. All OCT images were obtained with either the Spectral OCT (Carl Zeiss Meditec Ophthalmic Systems Inc., Dublin, CA, USA) or Spectralis OCT (Heidelberg Engineering, Heidelberg, Germany). When available, the calculated value of CST by the Spectral OCT was recorded. In cases of poor Spectral OCT signal strength or with only dubious OCT data available, clinical 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 (LogMAR), OCT macular thickness, frequency, and duration of CME treatment. Other information recorded included frequency and duration of CME treatment. Treatment algorithm devised for patients with keratoprosthesis and cystoid macular edema. Generally, treatment is initiated with topical medications (prednisolone acetate and topical nonsteroidal anti-inflammatory agents). Intravitreal injection is reserved for patients with acute uncontrolled intraocular pressure or severe visual loss. Intravitreal dexamethasone implant may be beneficial in treating chronic Kpro CME.

RESULTS

INDICATIONS FOR KERATOPROSTHESIS


REFERENCES


SUPPORT

Research to Prevent Blindness