INTRODUCTION

The cornea is an optically clear tissue permitting the transmission of light to the inner structures of the eye. Abnormalities of the corneal structure can result in ocular discomfort, reduced light refraction and blindness. Corneal endothelial dystrophy (CED) is a leading cause of corneal degeneration in dogs characterized by corneal edema, bullae formation and often ulcerative keratitis. This condition is characterized by a bilateral, slightly asymmetrical loss of ocular transparency noted in conjunction with decreased endothelial cellular density and aberrant morphology. When the endothelial cells become metabolically overwhelmed, fluid is permitted to enter the stroma posteriorly. Most dogs develop significantly debilitating recurrent corneal ulcerations as the bullae rupture superficially. These ulcerations are difficult to resolve due to the abnormal morphology, causing severe chronic ocular pain, blindness and often resulting in enucleation. The ulcers can initially be managed with topical antibiotics and often hypertonic saline agents; however, progression of the disease process occurs despite medical management. Breeds considered predisposed to CED include the Boston Terrier, Dachshund, German Shorthaired and Wirehaired Pointer and Chihuahua.1 Mean age of onset is 10 years and CED has a preponderance for females.2,3

Corneal endothelial dystrophy is similar to Fuchs’ endothelial corneal dystrophy (FEDC) and is considered a potential canine model for this disease process.4,5 Fuchs’ endothelial corneal dystrophy is characterized by progressive

ENDOTHELIAL KERATOPLASTY FOR CORNEAL ENDOTHELIAL DYSTROPHY IN A DOG

Micki D. Armour1 | Timothy E. Askew2 | Allen O. Eghrari3

1Armour Veterinary Ophthalmology, Washington, District of Columbia
2Hunt Valley PharmaLAB, Cockeysville, Maryland
3Johns Hopkins Wilmer Eye Institute, Baltimore, Maryland

Correspondence
Micki Armour, Armour Veterinary Ophthalmology, 4105 Brandywine St NW, Washington, DC 20016.
Email: Dr.armour@armoureyevet.com

Abstract

Objective: To assess the efficacy of an endothelial keratoplasty procedure at defined intervals to 1 year postoperatively for the treatment of corneal endothelial dystrophy (CED) in a canine patient.

Procedure: A dog diagnosed with CED with progressive corneal edema underwent an endothelial keratoplasty. The patient was examined pre- and postoperatively with slit lamp biomicroscopy and ultrasonic pachymetry.

Results: Mean central corneal thickness (CCT) measured with pachymetry was >1400 μm preoperatively and decreased postoperatively to 725 μm. The transplanted donor tissue became transparent 2 weeks postoperatively and incorporated with the recipient cornea. The graft remained transparent throughout the duration of the postoperative period evaluated in this study (2 weeks postoperatively to 1 year). The canine patient was comfortable pre- and postoperatively.

Conclusions: Endothelial keratoplasty is a potential therapeutic option for canine cases with progressive corneal thickening due to CED. As this is a single case study, further investigation into the use of endothelial keratoplasty to treat CED is warranted. Moreover, canine patients with CED might serve as a surgical model for human patients with Fuchs’ Endothelial Corneal Dystrophy.

Keywords

canine, corneal endothelial dystrophy, Fuchs’ endothelial corneal dystrophy
corneal edema often initiates with corneal haziness but vision is still intact. However, through the formation of guttae and chronic dysfunction of the endothelial cells, FECD can lead to significant vision loss. Patients with FECD are often treated successfully surgically with a corneal transplant. Endothelial keratoplasties (Eks), in particular Descemet’s Stripping Endothelial Keratoplasty (DSEK) and Descemet’s Membrane Endothelial Keratoplasty (DMEK) are pioneering surgical techniques to address FECD in human ophthalmology. In human patients with FECD, the use of fewer cells transplanted and localized to the region that requires repair (DSEK or DMEK) vs a penetrating keratoplasty (PK) has been the focus of the human ophthalmology field for a faster surgical procedure and recovery, lower antigenic load, lower risk of postoperative astigmatism and discomfort, a smaller incision with lower risk of traumatic injury postoperatively and maintenance of corneal sensation postoperatively.6-11

To date, treatment options for CED is limited but includes thermokeratoplasty3 and superficial keractectomy and conjunctival advancement hood flap (SKCAHF)12 each of these options results in significant keratitis, limited corneal clarity postoperatively and does not address the primary disease process: endothelial cell loss. Progressive corneal edema develops over months to years and is monitored using pachymetry or optical coherence tomography.1-3,6,12,13 Topical and systemic anti-inflammatory therapy and topical sodium chloride are frequently used to delay advancement of clinical signs, although clinical trials to determine effectiveness are required.14 The purpose of this clinical study is to evaluate the effects of an endothelial keratoplasty (EK) as a novel approach for the treatment of CED in a dog.

2 | MATERIALS AND METHODS

A single patient, 10-year-old male neutered Boston Terrier with chronic CED was selected for inclusion into this case report. The client completed a clinical trial owner consent-release form prior to enrollment, and completion of the study was within the guidelines directed by the ARVO Statement for the Use of Animals in Ophthalmic and Vision Research. The Eye Care for Animals Research Committee and Medical Quality Board, composed of board-certified veterinary ophthalmologists charged with maintaining ethical and humane treatment of veterinary patients, reviewed and approved the study. Prior to enrollment, the patient underwent a complete physical exam and was considered well enough to undergo general anesthesia. A complete blood count and serum biochemical profile was performed prior to general anesthesia and was unremarkable and within normal limits for surgery. A complete ophthalmic examination including slit-lamp biomicroscopy (SL-17; Kowa American Corporation), binocular indirect ophthalmoscopy (Keeler Instruments Inc.) using a 28 diopter and 20 diopter indirect lens (Volk Optical Inc.), schirmer tear-test 1 (STT1; Intervet Inc.), rebound tonometry (TonoVet, Jorvet, Loveland, CO) and fluorescein stain (Altafluor benox; Altaire pharmaceuticals, Inc.). The right cornea was classified according to a grading scheme as described by Thomasy et al2 as Stage 3 demonstrating moderate edema in the central, temporal paraxial and perilimbal cornea with axial anterior stromal bulla (Figure 1). Ultrasound pachymetry (Sonogage Corneogage Plus) was utilized to evaluate the corneal thickness of the right and left cornea.

The patient was pre-medicated with buprenorphine (0.01 mg/kg; Par Pharmaceuticals), maropitant (1 mg/kg; Zoetis Services LLC) and famotidine (1 mg/kg; West-ward Pharmaceutical Group) intravenously 2 hours prior to induction. Glycopyrrolate (0.01 mg/kg; Somerset Therapeutics, LLC) was injected intravenously 5 minutes prior to induction. The patient was induced with 0.2 mg/kg midazolam (Akorn, Inc.) followed by 4 mg/kg PropoFlo28 (Abbott Laboratories) and placed in dorsal recumbency under an operating microscope. The patient was maintained on 5 mL/
kg/h intravenous fluid supportive therapy for the duration of the procedure. Cefazolin (22 mg/kg; WG Critical Care, Inc.) and dexamethasone (0.15 mg/kg; VetOne) was injected intravenously shortly after induction. The patient was maintained on isoflurane inhalant anesthesia for the duration of the surgery.

2.1 Presurgical preparation of donor tissue

In cases of corneal transplantation, identification and procurement of viable, healthy donor tissue is paramount to the success of the procedure. Much like the field of human transplantation, tissue availability is dependent on a well-informed and passionate community of pet owners who will consent to the recovery of their pet's tissue for transplantation and/or research. For this case, the team developed a brochure to educate pet owners by answering such questions as “Why should I donate my pet’s eyes?” and “How do research and education benefit from eye donation?” Furthermore, our authors worked with community veterinarians to establish appropriate and effective consenting language and recovery protocols. As a result of these efforts, multiple donor corneas were recovered, ensuring that the patient was able to receive healthy tissue.

As in human eye tissue donation, we focused on establishing medically-sound donor eligibility and tissue suitability criteria prior to initiating a recovery program. The surgical team's donor eligibility standards for this case were a known cause of death, and exclusion criteria included viral encephalitis, active bacterial or viral meningitis, active bacterial or fungal endocarditis, rabies, and neoplasia. We excluded eyes with active ocular or intraocular inflammation, glaucoma, or prior ocular surgery. Upon meeting the pre-recovery tissue suitability criteria, pairs of corneas were recovered by one of the authors (MDA). Final tissue suitability criteria were the following: (a) Cell count greater than 2500 cells/mm², (b) time from euthanasia to preservation ("E to P") less than 4 hours, and (c) time from euthanasia to transplant ("E to T") less than 4 days.

A donor cornea of a 1-year-old male neutered staffordsire bull terrier mix was identified. A complete blood count and serum biochemistry profile performed on the donor patient was within normal limits. The donor selected was up to date on vaccines including rabies. The donor had a normal physical examination. A complete ophthalmic examination, including slit lamp biomicroscopy and indirect ophthalmoscopy, was performed on the donor canine patient within 1 minute of humane euthanasia. No malignant ocular tumors were identified. No active intraocular or periorbital inflammation was identified. The patient did not have a history of previous ocular diseases or ophthalmic surgeries. Congenital and acquired ophthalmic diseases that would potentially preclude a successful outcome could not be identified. The donor canine did not have a history of previous or active encephalitis, meningitis, or endocarditis. The donor had a history of aggression towards other pets in the household and the owner and was unresponsive to extensive therapy with a behavior specialist. The owner elected humane euthanasia and consented in writing to the donation of their pets’ eyes to benefit research in field of veterinary medicine and the vision of another dog. This patient had a known cause of death (humane euthanasia).

Five minutes following humane euthanasia, the corneas were aseptically prepared with 1:50 povidone iodine solution. Sterile drapes were placed around the patients’ right globe. A lateral canthotomy was performed and a barraquer eyelid speculum was placed. A peritomy was performed to remove the conjunctiva from the underlying sclera. A 360-degree circular partial depth incision was created with a number 64 beaver blade 2 mm posterior to the limbus. This incision was followed with westcott tenotomy scissors to puncture into the globe and to free the cornea and 2-mm scleral rim from the underlying globe. The iris was separated from the cornea. The donor tissue was placed in Optisol GS (Bausch & Lomb) corneal storage media and kept refrigerated at 2-8°C for 2 days until tissue preparation began the day of surgery.

Two certified eye bank technicians (CEBT, Eye Bank Association of America) trained in contraindications to corneal transplantation performed a physical inspection of the donor tissue 2 days following procurement of the tissue. The CEBTs evaluated the tissue for trauma, fibrosis, scarring, and any infiltrates that would decrease cell viability or in any way negatively impact transplantation of the tissue. The thickness of the cornea was evaluated with pachymetry, and determined to be 580 μm. A specular microscope (Hai Labs) imaged and analyzed the endothelial cells for pleomorphism, polymegathism and to determine the cell density. The corneal endothelial cell viability was considered excellent with 3307 cells/mm². (Figure 2)

The cornea was removed from the specular microscope and was mounted on a nitrogen-powered microkeratome (Moria). The team utilized existing microkeratome equipment from Moria to perform a lamellar transectional dissection of the anterior segment of the cornea. The maximum diameter attainable from the equipment was 10 mm. The targeted thickness of the postprocessed graft was less than 100 μm. Final graft thickness was 81 μm (and therefore considered an Ultra-thin DSEK). This very thin section of corneal tissue is created with two separate passes of the microkeratome; the first pass removes the anterior ⅗ of the cornea, and with guidance from a pachymeter, the second pass cut is selected to result in an ideal corneal thickness. The tissue was deemed suitable for transplantation by the authors, was brought into the surgery suite, and was gently placed endothelium side-up on a Jaeger lid plate, then manually trimmed with right and left Castroviejo corneal scissors to approximately 6 mm in
diameter to provide ease of insertion. Marking of the cornea with a surgical pen was not performed as this could result in endothelial cell loss, but careful evaluation of the orientation of the graft was maintained by the surgeon.

2.2 Surgical approach for the recipient

The patient was placed in dorsal recumbency under an operating microscope (Zeiss) with head stabilization. Periorcular adnexa underwent a sterile preparation and was treated with 1:50 povidone iodine. A sterile drape (3M Health Care) and fenestrated drape was placed over the patient's right eye, and an oval opening in the sterile drape was created. A barraquer eyelid speculum was positioned on the upper and lower eyelids. The donor tissue was prepared for transplant. While floating in the optisol solution, drops of Trypan blue 0.06% (DORC International, the Netherlands) were placed on the donor tissue.

An irrigation/aspiration port was created at the 1 o'clock position of the perilimbal cornea with a 1.8-mm keratome on the recipient's right globe. An incision was created at the 3 o'clock position with a 1.8-mm keratome. A dorsal perilimbal three-stepped keratotomy was performed at the 10 o'clock position, first with a 2.8-mm keratome and then expanded with a 4.5-mm keratome. An infusion cannula was placed into the anterior chamber through a side port incision. The donor tissue was gently placed endothelium-down on a bed of viscoelastic (Ixium 1.8%, LCA Pharmaceuticals), on a spatula brought adjacent to the globe. Using 25-G serrated forceps introduced through a side port incision, across the anterior chamber and exiting the wound, the tissue was grasped and pulled into the anterior chamber. With light tapping to open the graft, addition of balanced salt solution and air bubbling, the graft was positioned centrally and slightly ventrally, over the area of greatest edema (Video S1). An air bubble was expanded to fill the anterior chamber behind the graft and permitted to sit for 10 minutes while the patient was under general anesthesia. A dorsal and a ventral 10-0 Ethilon Nylon simple interrupted suture (Ethicon) was placed at the edge of the graft, and the knots of these sutures were buried into the corneal stroma. The 4.5-mm perilimbal dorsal incision was closed with two simple interrupted 10-0 Ethilon Nylon sutures. A subconjunctival injection of 0.2 mL Kenalog (10 mg/mL; Bristol-Meyers Squibb Co.) and 20 mg gentamicin (100 mg/mL, VetOne) was performed. The recipient had a routine recovery and extubation. Postoperatively, rebound tonometry was performed of the right eye and was estimated at 11 mm Hg.

The recipient remained at the emergency hospital overnight to maintain a level of sedation. While resting, he was positioned in dorsal recumbency and with his chin resting close to his sternum, as much as he would permit this position, to facilitate the air bubble positioning directly below the cornea. Postoperative medical protocol included topical Dorzolamide 2% (Hi-Tech Pharmacal Co) q8hours, Durezol (0.05% difluprednate ophthalmic solution, Alcon Laboratories, Fort Worth, TX) q1hour, Moxifloxacin (Sandoz, Inc.) q6hours, Optixcare (CLC Medica) q6hours, as well as a tapering dose of oral prednisone (0.25 g/kg West-Ward Pharmaceutical, Eatontown, NJ) per os (PO), Tramadol (2 mg/kg; Amneal Pharmaceuticals, Eatontown, NJ) PO q8 hours and Clavamox (13 mg/kg; Zoetis Services LLC) PO q12 hours. A protective Elizabethan collar was in place at all times. (Figure 1) Modified cyclosporine (25 mg, (5 mg/kg/d) IVAX Pharmaceuticals) was added to the medical regimen orally q24 hours 1 week postoperatively.

At the 2 week postoperative appointment, the Nylon sutures were ligated with a 30 g needle. At evaluation 3 weeks postoperatively, significant corneal edema was present at the superior edge of the graft, and it was unclear if the graft was still in contact at the recipient bed dorsally. The patient was admitted and administered preoperative medications identical to the day of his initial surgery. The graft was evaluated under the operating microscope and was determined to have a 1 mm 360-degree rim of detachment with corneal clarity ventrally but haziness dorsally. Air was placed in the anterior chamber via a 27-G needle and 1-cc syringe to create a 65% “fill” of the anterior chamber. Two sutures were placed at the 10 o'clock and at the 2 o'clock position dorsally with the knots buried into the stroma. The patient recovered uneventfully from the procedure. The patients’ oral prednisone was increased back to q12 hours. Oral antibiotics were extended for another two weeks q12 hours. Moxifloxacin and Durezol were continued topically at q6 hours.
3 | RESULTS

Prior to surgery, ultrasound pachymetry of the left cornea was considered less affected at 600 μm and optically free of corneal edema. Ultrasound pachymetry of the right eye was greater than 1400 μm prior to surgery. Mild nuclear sclerosis was identified in both eyes. Mild pupillary zone iris atrophy was identified in both eyes. Slit lamp biomicroscopy was otherwise considered within normal limits. Indirect ophthalmoscopy was considered within normal limits in both eyes. Diagnostic testing including Schirmer tear test (15 mm wetting/min in both eyes), tear film breakup time (>20 seconds in both eyes), rebound tonometry (15 mm Hg in both eyes) and fluorescein stain (negative for uptake in both eyes) were all considered within normal limits prior to surgery. Postoperative intraocular pressure 2 hours following the procedure was estimated at 16 mm Hg, and intraocular pressure remained normotensive postoperatively (between 10 and 18 mm Hg). The patient presented for a recheck examination daily for the first week, and then was evaluated once weekly for the duration of 1 year.

At 1 week following the procedure, there was no sign of corneal malacia or ocular irritation. The transplanted graft was still hazy and edematous, but no overt intraocular inflammation was identified. (Figure 1) Rebound tonometry of the right eye was estimated at 8 mm Hg. The dorzolamide, Optixcare, and the Tramadol were discontinued. The moxifloxacin and Durezol were maintained at q6 hours. The prednisone was tapered to once daily administration. Modified cyclosporine was initiated in the absence of overt infection or corneal malacia.

At the 2 weeks postoperative recheck, the cornea was clear and appeared thinner in the transplanted window; the moxifloxacin was discontinued and the Durezol was maintained at q6 hour therapy. (Figure 1) The sutures were replaced 3 weeks postoperatively. One day following the suture replacement procedure, the graft appeared much clearer dorsally and the sutures were intact. That week a line of fibrosing keratitis at the graft periphery became evident, especially ventrally, but the axial corneal clarity continued to improve. Postoperative IOP was estimated at 13 mm Hg with rebound tonometry. At the 1 month recheck examination, the graft was optically clear and comfortable. The moxifloxacin was discontinued. The prednisone was tapered to 0.25 mg/kg every third day. The Durezol was maintained at q8 hours topically for the right eye.

Two months following the patient’s initial surgery, the cornea surrounding the graft appeared slightly more edematous. (Figure 1) An injection of kenalog was performed subconjunctivally in the right eye, the Durezol was increased to q6 hours and the moxifloxacin was restarted at q6 hours topically. The surrounding corneal edema appeared to subside following the medical alterations and kenalog injection, and the topical and systemic medications were slowly tapered once more.

Three months following the patient’s initial surgery, an acute onset of hyperemia was encountered; the patient had pinpoint fluorescein stain corneal uptake lateral and paraxial to the transplanted graft. Subepithelial microbullae were identified in the cornea surrounding the graft, consistent with progression of his CED. Moxifloxacin was restarted. Once the ulceration healed, another injection of subconjunctival kenalog was performed. Again 4 months postoperatively, the patient exhibited mild periorcular hyperemia; although no ulceration was noted on his examination that day, there was evidence of a recently resolved epithelialized peripheral bullae. He was maintained on moxifloxacin topically for 1 week duration, but then this was discontinued due to the restoration of his ocular comfort.

The patient was maintained on topical Durezol q12 hours, topical sodium chloride 0.5% q24 hours and modified cyclosporine 25 mg PO q24 hours for months 4-10 postoperatively. He had one episode of blepharospasm in month 8; however, this resolved before his ophthalmology appointment. At 9 months postoperatively, the graft remained clear. Pachymetry was conducted in both the operative eye (725 μm centrally, >1400 μm peripherally) and nonoperated eye (828 μm centrally), indicating that the improvement in pachymetry was maintained. At 12 months postoperatively, the graft remained clear ventrally, although there was a 2-mm dorsal encroachment of corneal edema, leading to a smaller window of clear cornea (4 mm vertical; 6 mm horizontal). The patient was comfortable and tolerating the topical therapy well. (Figure 1)

4 | DISCUSSION

Corneal endothelial dystrophy (CED) is an inherited progressive condition leading to corneal bullae formation, blindness, corneal ulceration, and secondary infection.

With rapid progression to persistent superficial corneal ulceration, often complicated with secondary infection and chronic ocular irritation, endophthalmitis and enucleation are unfortunately too often selected for canine patients with CED. Several forms of prophylactic surgical intervention have been published to intervene early and prevent disease progression, including thermokeratoplasty, superficial keratectomy and conjunctival advancement hood flap. Given that they do not address the primary cause of lost corneal endothelial function, these procedures are limited in their ability to restore ocular comfort and clear central vision long-term in our canine patients.

Corneal endothelial dystrophy has been considered an animal model for FECD, which also demonstrates progressive central corneal edema and visual deficits. Whereas Fuchs’ patients are affected first in the central cornea, CED canine
patients demonstrate temporal edema that spreads centrally and then finally nasally. In canine patients with CED, endothelial cell count reduction directly correlates with advancement of the corneal edema. Therefore, it would be suggested that repopulation with viable endothelial cells would result in reversal of clinical signs.

The corneal tissue in canines is distinctly different from a human; the tissue is less rigid and friable. This could be secondary to a combination of the absence of a Bowman’s layer as well as different types and locations of stromal collagen, and fewer collagen intertwinings than in humans.21,22 The process of cutting tissue, while comparable to humans, must account for the larger corneal size and change in corneal consistency. The thickness of the corneal tissue transplanted into our patient was less than 100 μm thick, and therefore considered an ultra-thin DSEK.

The inflammatory response in canine eyes is robust compared to humans, which carries potentially higher risk for rejection. In this case, we opted for ultra-thin DSEK grafts to minimize the antigenic load and facilitate a faster speed of visual recovery. While minimizing graft thickness through DMEK or PDEK is feasible in humans, the tight scroll of canine descemet membrane tissue, combined with a deep anterior chamber and reduced canine corneal rigidity25,26 prevents tapping and opening a graft in the eye. Moreover, DMEK in humans frequently requires rebubbling that can be conducted in clinic with topical anesthesia, whereas rebubbling the canine eye may require returning to the operating room with risks of general anesthesia.

Potential complications following canine corneal transplantation include graft detachment, graft rejection and secondary infection. These are comparable to complications of human DSEK including pupillary block, chronic glaucoma, dislocation of the transplanted graft, and graft failure.28 However, several distinctions exist that require modification of the human DSEK technique to avoid complications. In contrast to the peripheral iridotomy and postoperative supine positioning commonly performed in humans, dogs have a highly vascularized and thick iris, preventing iridotomy; they are also unable to position postoperatively. Therefore, we sutured the graft and started the patient prophylactically on anti-glaucoma therapy. Despite being a breed predisposed to glaucoma, we did not encounter pupillary block. In humans, the operative eye is kept away from dirt and contaminants to avoid infection given frequent steroid use in the initial postoperative period; however, given the proximity of the canine eye to the ground, we opted to use antibiotics perioperatively to decrease the pathogenic load. In order to minimize postoperative complications, a combination of careful care, modified minimization of movement in the primary 24 hours period, and diligent use of anti-rejection medications is imperative. Postoperative immunomodulation is a key component to avoid graft rejection and stimulate adherence of the new tissue to the adjacent injured cornea.

Unlike DSEK or DMEK procedure in classic FECD in which guttae require removal, no guttae have been reported in Descemet’s membrane of the canine eye. In this case study, Descemet’s membrane was left in place and the donor tissue was directly apposed. Although corneal transparency was achieved, perhaps removal of the Descemet membrane could result in improved apposition of the donor and recipient cornea. Guttae-like excrescences in the canine may exist, and evaluation of the abnormal Descement’s membrane in CED cases at the time of surgery should be investigated.

Relative to DMEK, a limitation of DSEK is that the raised edge of the graft can prevent migration of cells. Cell migration across a flat surface allows corneal clearance in humans from DMEK, descemet stripping only, or descemet membrane endothelial transfer from a misplaced graft. However, after one year, we did not notice significant clearance of the cornea surrounding the graft. Rather, a distinct area of clarity remains in the region of transplantation with distinct margins.

Another limitation of this study is that the authors reported clarity subjectively. Corneal clarity is a very important aspect of this surgery. Photographs do not necessarily demonstrate corneal clarity accurately due to artifact inherent to photography, and although the surgery provided for an obvious improvement in clarity at the transplant site, the use of a corneal clarity score would have been desirable in addition to the staging of the CED. Only one validated corneal clarity score (ie with a known, high inter- and intrauser variability) is available in the veterinary literature at present.29 Further studies into EK procedures should make use of validated corneal clarity scoring systems before and after surgery, in order to allow for the objective comparison of results between cases as well as between studies, whenever possible.

As a new field, canine transplantation will require collaboration across institutions to determine the optimal balance between donor and tissue rule-out criteria and the availability of transplantable tissue. Donor eligibility standards and cornea suitability standards must be developed to provide the highest quality tissue possible to maximize long-term surgical outcomes. This case report highlights EK as a potential therapeutic surgical option for cases of CED in our canine patients. Additionally, EK in CED patients might serve as a canine model for FECD patients, allowing collaboration and maximization between human and veterinary ophthalmology fields. Successful restoration of vision and ocular comfort depends on optimization of the technique and the perioperative therapeutic protocol.

ACKNOWLEDGEMENTS

The authors would like to thank Ms. Cindy Stroup and Lucky for pioneering the field of EK in dogs; Ms. Rebecca Britner, Katerina Kesten, Shanelle Anderson and Pathway/Eye Care for Animals for their technical support of this project. This study was supported by the Eye Care for Animals Research Committee and medial Quality Board.
CONFLICT OF INTEREST

There is no conflict of interest to disclose.

ORCID

Micki D. Armour https://orcid.org/0000-0002-4705-5880
Allen O. Eghrari https://orcid.org/0000-0003-2798-038X

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

How to cite this article: Armour MD, Askew TE, Eghrari AO. Endothelial keratoplasty for corneal endothelial dystrophy in a dog. *Vet Ophthalmol*. 2019;00:1–7. https://doi.org/10.1111/vop.12670