

## Surgical management of infectious and noninfectious melting corneal ulcers in cats

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**Abstract:** A melting ulcer, characterized by the stromal dissolution of the cornea, is an important eye lesion that develops due to various reasons and threatens vision. Restoration of this lesion, which causes loss of corneal transparency, requires aggressive medical and surgical treatment. In medical treatment, stromal degeneration is controlled with agents that support topical antibacterial and stromal healing. Surgical treatment is applied using various techniques to remove structures damaged due to the progression of corneal melting and to promote corneal healing. In this study, lamellar keratectomy and the third eyelid flap techniques were combined with topical medical therapy in 20 cat cases with melting ulcers. The aim of this study was to alert to general practitioners that this condition is an important cause of progressive eye loss and necessitates urgent intervention and to show that this treatment approach is easy to implement and has successful results.

**Key words:** Cat, keratectomy, keratomalacia, third eyelid flap, stromal liquefaction

### 1. Introduction

Corneal ulcers are important eye diseases with a risk of blindness if not treated [1,2]. While most of these ulcers are healed with appropriate treatment, some progress into corneal melting ulcers [3,4]. A melting ulcer, also known as stromal liquefactive necrosis or keratomalacia, occurs acutely or with a rapid progression of an ulcer. It has been reported that the mechanism of the disease is closely linked with proteolytic enzymes, such as Matrix metalloproteinase (MMP) [5,6]. These enzymes are produced by certain bacteria (*Pseudomonas aeruginosa*, *Streptococcus* and *Staphylococcus* spp.), and inflammatory cells, such as leukocytes and corneal epithelial cells, are responsible for the removal of dead cells and debris from the ocular surface of the eye [6]. Normally, they are in balance with the proteolytic enzyme inhibitors of the cornea [6]. However, an increase in the number of proteolytic enzymes causes an imbalance between proteinase inhibitors, which leads to a rapid degeneration of the corneal stroma [6,7]. A melting ulcer can result in corneal perforation, even leading to the loss of eye, if adequate and timely treatment is not undertaken [8,9]. The treatment options are divided into two groups as medical and surgery according to the etiology, depth of the corneal stromal defect, and the presence of infection [8,9]. The aim of treatment is to ensure the integrity of the cornea, eliminate the infection, and prevent corneal degeneration.

In medical therapy, topical and/or systemic antimicrobials are used aggressively to prevent or eliminate infections. In order to reduce the progression of keratomalacia and facilitate epithelial healing, protease inhibitors, such as Ethylene Diamine Tetraacetic Acid (EDTA), autologous serum and/or tetracyclines are used [8,9]. Additionally, mydriatic-cycloplegic agents are administered to reduce secondary/reflex uveitis that is frequently seen in deep corneal ulcers. In patients not responding to medical treatment, several surgical methods are used, including keratectomy, conjunctival autografts, third eyelid flap, amniotic membrane graft, and biomaterial graft [10]. The removal of necrotic infected tissues with superficial keratectomy shortens the healing process and promotes vascularization. After keratectomy, the third eyelid membrane can be used as a flap to help protect the cornea and accelerate corneal healing [11,12].

The goal of this study was to determine the clinical results of lamellar keratectomy and the third eyelid flap method in the surgical treatment of corneal melting ulcer of varying severity in cats.

### 2. Materials and methods

#### 2.1. Animals

In this study, 20 cats, aged 3 months to 9 years of different sex and breeds, were treated for keratomalacia using keratectomy and third eyelid flap between 2014 and 2019 in

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the Surgery Department of İstanbul University-Cerrahpaşa Veterinary Faculty, Turkey. The history, clinical findings, medical and/or surgical interventions, and the treatment results of each animal were recorded. The clinical findings of these cases are summarized in Table 1.

Fluorescein stain test, the Schirmer tear test, and corneal culture analysis were performed for the ocular examination (Figure 1). The culture samples were collected directly from the corneal lesions and were immediately placed in a microbiological transport medium for processing without delay. The anterior segment was not visible, and the pupillary and menace reflexes were not evaluated in most cases due to corneal dullness. Furthermore, in most cases intraocular pressure could not be measured due to surface irregularity. Keratomalacia was diagnosed according to the presence of gray, loose appearance of the cornea, and rapid progression of the lesion despite the use of antibiotics and corneal healing accelerators (Figure 2). The etiology of the lesion was investigated according to the anamnesis, clinical examination findings, and culture results; however, it was not possible to determine the cause in some cases. In this study, animals with corneal lesions of different depth, size, and severity were evaluated, but the patients that presented with corneal perforation were not included. All cats were treated with the same surgical and medical treatment procedure. The necrotic and collagenolytic tissues in the cornea of all patients were removed by lamellar keratectomy using a corneal knife through gentle movements without any corneal puncture (Figure 3). A third eyelid flap was then applied to accelerate corneal healing. We decided to apply the corneal flap for 4 weeks. Third eyelid flap sutures were checked twice at a 2-week interval.

## 2.2. Preoperative

A topical broad-spectrum antibiotic, 0.3% ofloxacin (Exocin<sup>®</sup>, Abdi Ibrahim, Turkey), and artificial tear hyaluronate (Eyestil<sup>®</sup>, Teka Technique, Turkey) drops (one to two drops) every hour were administered prior to surgery. In all cases, the complete blood count (CBC) and serum biochemical analyses were performed before the surgery.

## 2.3. Anesthesia

The cats were premedicated with an intravenous injection of xylazine HCl (1 mg/kg, Basilazin<sup>®</sup>, Bavet, Turkey). General anesthesia was induced by the intravenous injection of ketamine HCl (5 mg/kg, Alamine<sup>®</sup>, Erse, Turkey). The animals were then intubated with a single-use endotracheal tube, and the continuation of general anesthesia was provided with 2–2.5% isoflurane (Forane<sup>®</sup>, AbbVie, Turkey). Analgesia was administered subcutaneously using meloxicam (0.1–0.2 mg/kg, Melox<sup>®</sup>, Nobel, Turkey) 1 hour prior to the surgery. In addition, intravenous antibiotic ceftriaxone (20–50 mg/kg, Novosef,

Zentiva, Turkey) was administered 15–20 min before the surgery.

## 2.4. Surgery

The operation was performed in the lateral recumbent position with the affected eye being on top, and the eyes were aseptically prepared. The ocular surface and conjunctival sacs were lavaged with 0.05% povidone-iodine solution. After ocular lavage, the eyes were flushed with sterile saline solution. The entire body of the animal except the operation area was closed with disposable sterile cloths. The eyes were fixed with two to three temporary sutures from the scleral conjunctiva using 3.0 monofilament non-absorbable suture material with an atraumatic needle. A surgical microscope was used in all operations. Nonviable, loose corneal tissue and ulcer margins were removed by lamellar keratectomy using 3.0 mm straight cornea blades and corneal scissors (Figure 3). The incision was made deep enough to allow the removal of the entire corneal defect without puncturing the eye (Figure 4). After keratectomy, the third eyelid flap was performed using 2/0 absorbable suture material (4–5 weeks effective) to accelerate corneal healing and protect the cornea (Figure 5).

## 2.5. Postoperative treatment

The treatment consisted of topical broad-spectrum antibiotics, 0.3% ofloxacin (Exocin<sup>®</sup>, Abdi Ibrahim, Turkey), and artificial tears, sodium hyaluronate (Eyestil<sup>®</sup>, Teka Technique, Turkey) every hour for the first 3 days, followed by 5 times per day for 2 weeks, 3 times per day for 2 weeks, and a topical administration of 1% cyclopentolate HCl (Sikloplejin<sup>®</sup>, Abdi Ibrahim, Turkey) twice a day for 1 week. Ceftriaxone (20 mg/kg, Novosef, Zentiva, Turkey) was intramuscularly administered once a day and meloxicam (Metacam, Boehringer, Italy) 0.2 mg/kg once a day for 1 week. An Elizabethan-collar was put on the cats during the healing process. The sutures were checked 2 weeks after the surgery. After 4–5 weeks, the sutures of the third eyelid flap were removed. On the 1st day after removing the sutures, the anterior segment was visible, and pupillary light and menace reflex were present. There was a variable degree of neovascularization and corneal fibrosis, granulation tissue but no corneal ulcer or malacic material (Figure 6). Fluorescein staining was negative. To minimize residual scar tissues and vessels of the cornea, tobramisin and dexamethasone eye drops (Tobradex, Novartis, Turkey) were used 4 times a day for 1 week, followed by drops were gradually reduced at 1 week intervals and artificial tear gel; karbomer (Thilo-Tears SE<sup>®</sup>, Alcon, Turkey) was used twice a day for 3–4 weeks, and the treatment was completed. In all cases, the cornea of the cats returned to their previous rigid structure over varying durations. In all cases, re-vision was obtained in the affected eyes (Figure 7). The eyes were examined and recurrence was assessed at postoperative 2nd week, 1st,

**Table.** The clinical findings of 20 cats with melting ulcer.

Case no.	Breed	Age	Sex	Anamnesis	Affected eye	Lesion Localization-Depth	Size of lesion (mm)	Duration of lesion	Associated findings	Bacteriology	Outcomes
1	Domestic	3 months	F	Infection	OS	Diffuse-medium	6-11	3 days	Corneal edema	Not performed	Good
2	Persian	2 years	F	Trauma	OD	Central-very deep	9-15	7 days	Corneal edema, descemetocele, severe hyperemia	No growth	Visual
3	Persian	3 years	M	Unknown	OD	Central-medium	5-6	11 days	Corneal vascularization Bullous keratopathy	No growth	Visual
4	Persian	7 years	M	Unknown	OS	Paracentral-medium	10-12	7-8 days	Corneal edema and vascularization, bullous keratopathy	Not performed	
5	Persian	6 years	F	Infection	OS	Diffuse-medium	14-15	4-5 days	Corneal edema, severe hyperemia and pain	<i>S.simulans</i>	Visual
6	Persian	5 years	M	Trauma	OD	Central- very deep	7-11	10 days	Corneal edema and vascularization, descemetocele	<i>S.wameri</i>	
7	Domestic	1 years	M	Ulcer	OD	Central-deep	10-11	5 days	Corneal edema and vascularization	Not performed	
8	Persian	2 years	F	Trauma	OD	Diffuse- very deep	12-14	7 days	Descemetocele, corneal edema and vascularization	No growth	
9	Domestic	4 months	M	Infection+steroid therapy	OS	Central- very deep	3-5	10 days	Corneal edema and vascularization, descemetocele, severe hyperemia	Not performed	
10	Persian	9 years	M	Corneal sequestrum	OS	Central-deep	4-6	20 days	Corneal edema and vascularization	No growth	
11	Domestic	4 months	M	Infection+Steroid therapy	OD	Central- very deep	6-9	7 days	Bullous keratopathy, corneal edema and vascularization	Not performed	
12	Persian	5 years	M	Corneal sequestrum	OD	Central-deep	8-8	30-35 days	Corneal edema and vascularization,	<i>S. wameri</i>	
13	Persian	3 months	M	Unkown	OS	Central-medium	9-11	10 days	Corneal edema, bullous keratopathy	No growth	
14	British Shorthair	6 months	M	Trauma	OD	Diffuse-medium	11-14	6 days	Corneal edema, bullous keratopathy	Not performed	
15	Persian	2 years	F	Infection + steroid using	OD	Diffuse very deep	12-14	3 weeks	Corneal edema, bullous keratopathy, vascularization	No growth	Visual
16	British Shorthair	4.5 years	M	Unknown	OD	Paracentral-deep	9-11	2 weeks	Corneal edema, vascularization	Not performed	Visual
17	Persian	1.5 years	F	Trauma	OD	Diffuse very deep	12-14	4 days	Corneal edema, vascularization, infection	No growth	Visual
18	Domestic	8 months	M	Unknown	OS	Diffuse-medium	12-13	3 days	Corneal edema, bullous keratopathy	No growth	Visual
19	Persian	5 years	M	Corneal sequestrum	OS	Central-medium	6-9	1 week	Corneal edema, vascularization, pain	<i>S. simulans</i>	Visual
20	British Shorthair	3.5 years	M	Unknown	OD	Diffuse-deep	13-15	4 days	Corneal edema, bullous keratopathy	Not performed	Visual

OD: Oculus Dexter OS : Oculus Sinister



**Figure 1.** Case 19; a central cornea malacia stained with fluorescein test in a persian cat.

2nd, and 3rd months, and within 1–3 year. No recurrence of lesion was observed in 2 to 3 years postoperatively.

### 3. Results

Between 2014 and 2019, 20 cats were diagnosed with keratomalacia and treated with lamellar keratectomy and the third eyelid flap technique by the same veterinarian. Table 1 presents the detailed information about the cats concerning breed, age, sex, history, ophthalmologic signs, etiology, and postoperative results. There were 5 domestic and 15 purebred cats, of which 15 were brachiocephalic. Of the 15 cats, 12 were Persian, and 3 were British shorthair. Six were female (1 intact, 5 neutered) and 14 are male (9 intact, 5 neutered). It was determined that four cats lived outside, thirteen cats lived indoors, and three cats spent time both indoors and outdoors. The mean age of the cats was 3.1 years, ranging from 3 months to 9 years. Melting ulcer was mostly seen in cats under 3 years of age. Disease frequencies in cats aged 0–2 years, 3–5 years, and 6–9 years were 55% (eleven cats), 25% (five cats), 20% (four cats),

respectively. Unilateral corneal malacia was diagnosed in all cases (n=20; 12 right eyes and 8 left eyes).

According to the anamnesis of the patient owners, the general complaints of the cats were ocular discomfort, such as pain, ocular discharge and redness, corneal opacity, and irregularity of the affected eye, as well as general inactivity. Ocular examination revealed blepharospasm, epiphora, conjunctival hyperemia, keratitis, varying degrees of collagenolytic stromal malacia in the ulcerated corneal region, edema and vascularization around the lesion in the eyes of the cats.

The depth and size of the corneal lesions ranged widely. The corneal lesion regions were classified as central (n=10), paracentral (n=3), and diffuse (n=7). In 50% of the cases, the lesion was located in the central cornea. In seven cats, the lesion was confined to the middle layers of the stroma while it was deeper in 12 cases and advanced to the descemet membrane in seven (Figure 8). The risk of corneal perforation was high in 12 cases, for which it was very difficult to perform corneal debridement of the malacic tissue by keratectomy. However, no corneal perforation occurred during keratectomy in any of the animals. The anterior segment could not be evaluated in eight cases due to liquefaction of varying degrees in the stroma and loss of transparency of the cornea. The menace response and pupillary reflex were present in eight cases; however, in the remaining 12 cases, such evaluation could not be made because of corneal changes. Three patients had concurrent ocular disorders, including superficial ulceration in the contralateral eye (n=1) and corneal necrosis in the affected eyes (n=3). The etiology of the lesions was determined as infection (n=5), trauma (n= 5), corneal sequestrum (n=3), corneal ulcer (n=1), and unclear causes (n= 6). Herpetic virus (n=1) was suspected based on the presence of dendritic ulcer findings in both corneas. In case 15, the cause of the lesion was thought to be due to herpes virus-induced infection, since bilateral ulcer formation after the topical cortisone was applied to the patient who came with



**Figure 2.** First clinical examination of cases 5, 16, and 17, respectively. Central deep stromal malacia, necrotic tissue, intense corneal edema, and neovascularization.



**Figure 3.** Keratectomy of nonvisible, loose malacic material and ulcer margins with corneal blade and scissors.

conjunctivitis, the rapid progress of the ulcer despite the treatment for discontinuation of the cortisone and healing of the ulcer (Figure 9).

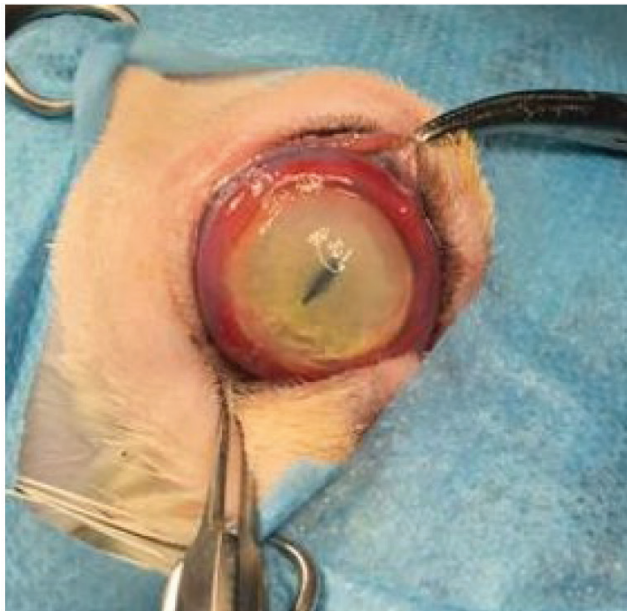
Samples for microbiological assessment were taken from 12 cats. In the remaining eight cases, samples were not obtained due to financial reasons. There was no bacterial growth in eight cases. *Staphylococcus spp.* (*S. wamari* and

*S. simulans*) were isolated from the samples obtained from four patients (Table 1).

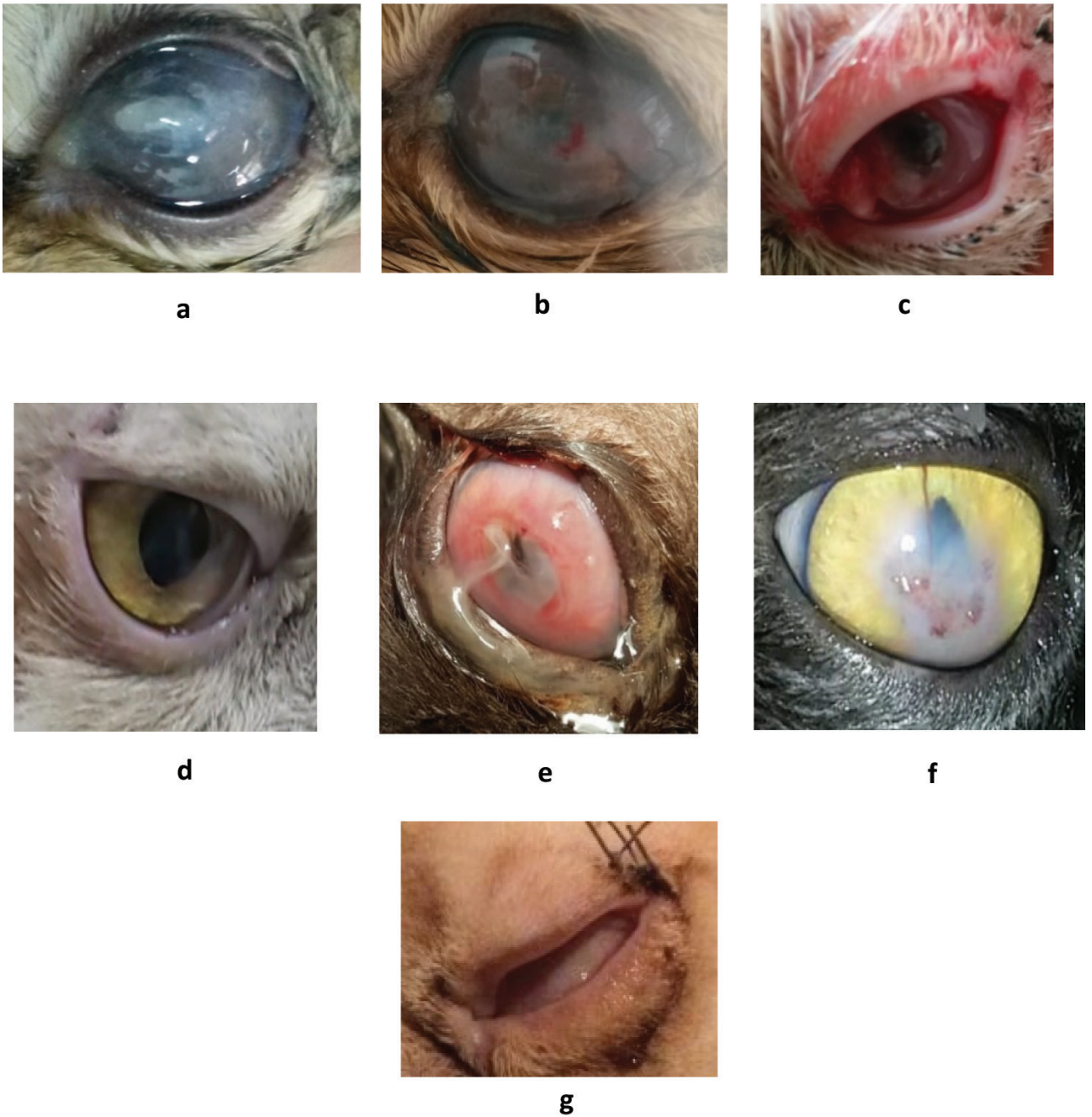
The results of culture analysis did not affect the treatment decision. Regardless of the etiology of the lesion, the same treatment method was applied in all cases. After the lesion was removed from the cornea by lamellar keratectomy, a third eyelid flap was applied for 1 month. In the postoperative period, neither pain nor any other discomfort symptoms were reported, but in almost all cases, scar and vascularization were present on the corneal surface for the first 1 to 2 weeks. These gradually attenuated and disappeared over time. Almost all corneas regained a transparent structure within 2 to 3 weeks and had good tonus by finger inspection. At postoperative months 1 to 1.5, vision was restored in all eyes. The corneal scar was mild in six (30%), thick and vascularized in eight (40%), and transparent in six cases (30%). Long-term topical use of artificial tear gel with eye drops containing cortisone and antibiotics was prescribed. It was checked periodically (every month) and was followed up in terms of corneal pigmentation and corneal necrosis.

#### 4. Discussion

Melting ulcer is a serious ocular lesion with risk of descemetocele, endophthalmitis, glaucoma, phthisis bulbi, and permanent loss of vision. This process involves a rapidly progressive stromal dissolution and loss of stiffness of the cornea secondary to proteolytic activity due to various infectious, noninfectious, or surgical/chemical agents [13,14]. Infectious causes that disrupt the anatomical barrier and physiological defense of the cornea are bacterial,



**Figure 4.** Appearance of the corneas after keratectomy of the necrotic tissue in cases 15 and 16.



**Figure 5.** a-g: Clinical presentation and results of the melting ulcer after treatment. Case 1) a: Before treatment of 3 months Domestic Long Hair. b: 4 weeks after treatment. Case 2) c: Before treatment of 2-year-old cat. d: 4 weeks after treatment. Case 6) e: before treatment of 5-year-old cat. f: 4 weeks after treatment. Case 5) g: Third eyelid flap used.

viral, and fungal agents while noninfectious causes include tear film deficiencies, low corneal sensation, trauma, exposure keratitis, and eyelid structural abnormalities, such as nasal fold trichiasis, macropalpebral fissure, and medial entropion. Eyelid anomalies are also known to be a predisposing factor, especially in brachycephalic breeds. The susceptibility of these breeds to melting ulcer is not

due to their direct genetic origin, but their anatomical structure. This brachycephalic facial structure plays an important role in the formation of melting ulcers in dogs and cats [8]. In previous studies, this lesion was mostly seen in dogs and cats, especially of Persian breed. Pot et al. [8] found that all cases with melting ulcers were Persian cats aged 6.9 to 14 years, but Famose [2] reported



**Figure 6.** a: The appearance of the cornea of the 15th case when the third eyelid sutures were removed 5 weeks after the surgery. b: Appearance of the cornea in case 16; corneal neovascularization and central granulation tissue was present sutures removed 5th weeks. c: Appearance of the cornea 4 weeks later in case 2. d: Appearance of the cornea in case 5. Granulation tissue was not seen.



**Figure 7.** Persistent superficial corneal neovascularization and fibrosis in cases 15 and 17 but vision was retained.



**Figure 8.** Case 8; large corneal malacia resulting in central descematocele.

that this lesion also developed in other cat breeds. In the current study, the most affected breed was Persian (60%) (brachicephalic breed predisposition), and the age range was 3 months to 9 years. The mean age of the disease was determined as 3.5 year in this breed. In addition, apart from the Persian breed, lesions of the same severity and depth were seen in a total of eight cats, five domestic and three British short hair. Except one of the domestic cats, all of them live outside and are aged 1 year or younger. The first year is considered to be the most active age for the animals, which increases the possibility of ocular trauma and secondary bacterial invasion but bacteriological test were not performed in mostly outdoor cats. We thought that these lesions might be caused by secondary infection after trauma due in outdoor cats. Moreover, the lesion spontaneously developed in the British shorthair cats.

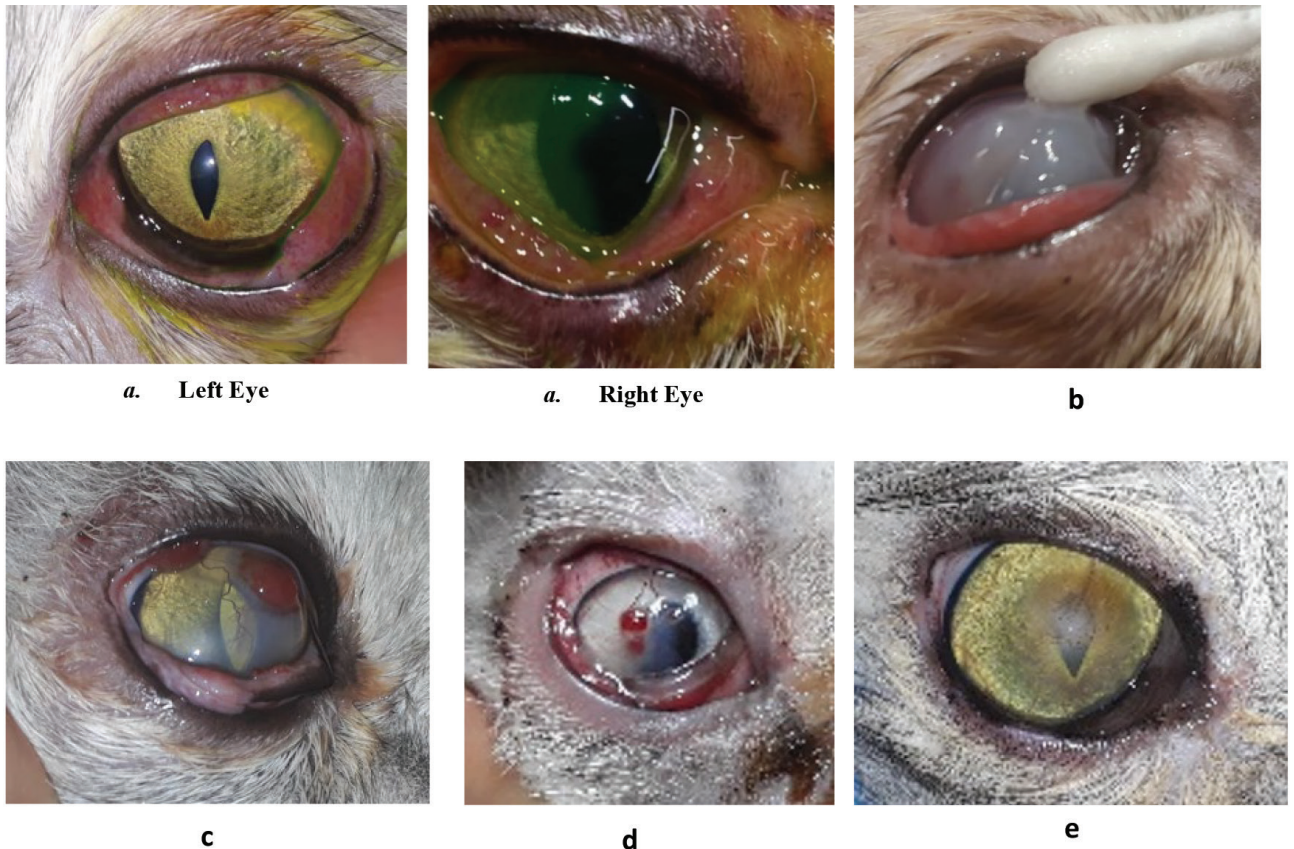
Melting ulcers generally occur in the center and/or close to the center of the cornea. They spread rapidly to the remaining surface and layers of the cornea. They are characterized by a gray, soft, mucoid, gelatinous appearance of the cornea [15,16]. Due to this appearance, these lesions recalled melting ulcer or keratomalacia [5,11,12]. In the clinical examination of the patients in our study, the melting regions of the corneas of all patients were gelatinous, mucoid, and soft, but the depth of the melting ulcer area and the intensity of the surrounding vascularized areas showed differences. However, all patients underwent the same medical and operative treatment. It was observed that the ulcer width and excess stromal loss had no significant effect on the duration and outcome of treatment. After the opening of the third eyelid flap, the clinical complaints of all patients disappeared, and the melting area of the cornea was completely healed.

There is no source related to sex predisposition and affected eye direction in cats diagnosed with this disease. Pot et al. [8] found that cat cases were predominantly male, and the affected eye was mostly the right side. Famose [2] also reported similar results on this subject. In our study, the right eye of the cases was mostly affected and the number of males were significantly higher than females. Three of the patients with trauma were nonsterile male and two were nonsterile female cats. This was considered to be the result of a fight after sexual activity-induced aggression. The infectious agents then penetrated the ocular surface and progressed rapidly, leading to the development of stromal melting ulcer.

Corneal melting ulcers are thought to be caused by vitamin deficiency in humans and microbial factors (bacteria, virus, fungi) in animals. Microbial factors are known to increase the dissolution process, albeit not always [9]. Primary or secondary bacterial agents, such as *Pseudomonas aeruginosa*, *Streptococcus*, and *Staphylococcus* have been found to be effective in melting corneal lesions. In addition, primary viral diseases (such as herpes virus) located in the upper respiratory tract may cause melting ulcers in cats [8]. Corneal melting ulcers have been investigated in dogs and cats, and reported that *Staphylococcus* spp. agent was mostly seen in cats [8]. Lin and Petersen-Jones found that *Staphylococcal* and *Pseudomonas* were the most commonly seen bacteria in the corneas of cats with ulcerative keratitis [17]. Famose [2] reported that six out of ten patients had no bacterial growth, three had *Pseudomonas*, and one had *Staphylococcus*. In the current report, the culture samples were analyzed in twelve of twenty patients, of which four were positive and eight were negative for bacterial growth. *Staphylococcus* spp. were isolated in accordance with previous studies [18]. However, in our case, the culture results did not affect the treatment decision, and the results were similar to those reported [8,17,18]. Good results were obtained after treatment in all cases with no recurrence being observed in the long term due to infectious agents.

Our treatment protocol included the application of aggressive medical drugs and various surgical procedures. The aim was to ensure the integrity of the cornea with minimal transparency changes [12]. Aggressive medical treatment with anti-collagenase and topical antimicrobials has been reported to halt the progression of stromal deterioration of the cornea, but some ophthalmologists argue that the clinical efficacy of medical treatments often has a weak or very slow effect to prevent ocular perforation [9]. The fight against bacterial microorganisms is extremely important regardless of whether the ulcer is deep or superficial. Broad-spectrum antibiotics should be applied to the ocular surface very often. In the medical treatment, topical fluoroquinolone, ciprofloxacin, and





**Figure 9.** Photographs shown clinical presentation and healing process of cases 15a–15e. a: First clinical presentation corneal ulcer with fluorescein test and conjunctival hiperemia in left and right eyes respectively. b: Deep corneal malacia in the right eye on 8 days and left eye was healed. c: Corneal neovascularization and granulation tissue 5 weeks after surgery. d: Appearance of ocular surface 6 weeks after surgery. e: Residual superficial corneal neovascularization 2 months after surgery.

aminoglycosides have been found to be highly effective against most agents [17]. In this study, topical ofloxacin was used in all patients. In corneal diseases, ofloxacin has been shown to be a highly effective and broad spectrum antibacterial agent with good corneal penetration. Although the eyes were closed, there were no complications related to the progression of infection in any of the cases, including those who were positive for culture.

The corneal stroma is largely composed of collagen tissue. Inhibition of the collagenase enzymes (MMPs), which tend to destroy this tissue continuously, is one of the main objectives of melting ulcer treatment [19]. Various medical agents are proposed as inhibitors of MMPs in the treatment of melting ulcer [1]. EDTA, acetylcysteine, ascorbate, tetracycline, cysteine, sodium citrate, penicillin, and fresh autogenous serum have been successfully used for anticollagenase activity [1,6,20]. These agents are used to reduce proteases produced by inflammation cells and to promote corneal healing.

Pain management has a critical importance during the treatment of corneal ulcers in all species but

there are differences of opinion regarding the use of topical antiinflammatory drugs. Some believe that antiinflammatory agents should be used to control pain and secondary/reflex uveitis, while others argue that these agents delay corneal epithelial healing [1,20]. In our study, nonsteroidal antiinflammatory drugs were not used due to the possibility of delaying corneal healing. Meloxicam was administered subcutaneously at the doses of 0.1–0.2 mg/kg dose of once daily for only 5 days in order to reduce pain after the operation. Even so, no reflex uveitis or other ocular complications were observed. Postoperative anterior chambers of all cases were normal in depth and transparent, and also the pupil diameters were normal.

In patients with progressive ulcers who do not respond to treatment, operative treatment is applied together with medical treatment. The aim of operative treatment is to remove the dead tissues and debris, accelerate healing, protect the cornea from external adverse effects, and fill the defect area with conjunctival or corneal tissue [19]. Many surgical techniques are used for this purpose, including keratectomy combined with the third eyelid

flap technique [15], corneo-conjunctival transposition [12], amniotic membrane transplantation, biomaterial grafts, conjunctival grafting, perforating keratoplasty, and collagen cross-linking [2,21,22]. The use of conjunctival pedicle flaps has been recommended by most surgeons in the treatment of melting ulcer in animals [8]. However, they cause permanent corneal opacities, which are usually located in the axial cornea and are too large, or require long-term medical treatment to achieve good corneal transparency, which may reduce vision [6]. Since corneal transparency prolongs the formation time, it was not preferred in our cases because the lesions were very large, even covering the entire surface of the cornea in seven cases. Other methods, such as amniotic membrane applied to the corneal surface were also not feasible considering that they take at least 2 months to be completely absorbed, and the required instruments are often very expensive. In this study, the melting tissue was removed by lamellar keratectomy, and then the third eyelid flap technique was used to stabilize corneal healing. There are no any articles about operative management of melting ulcer in cats using lamellar keratectomy and third eyelid flap in Turkey. The aim of keratectomy is to remove the infected and necrotic tissue, to eliminate anterior uveitis, to promote vascularization, to provide rapid healing, and to minimize scarring [13]. The third eyelid flap method is effective

in corneal reconstruction in most cases. It provides an excellent alternative to expensive and/or difficult corneal reconstruction, and offers physical support for weakened corneas. However, the use of this technique has been reported to be contraindicated in the treatment of progressive dissolution ulcers because of the inability to rapidly supply blood or fibrovascular tissue to the ulcers [14]. This method was applied by Ion et al. [15] in melting ulcers in cats and dogs, and contrary to the literature, they obtained positive results. In the current study, we applied this method after lamellar keratectomy regardless of the depth and width of the corneal lesions in 20 cat cases and we achieved excellent results in all cases. Vision was restored in all eyes, complete corneal transparency was achieved in approximately 1.5–2 months, which is shorter compared to other methods, and the duration of drug use was shortened. In the cases where the third eyelid flap suture was removed 1 month after the surgery, vascularization and fibrosis tissue in the cornea was thinner than after 5 weeks, when thick granulation tissue and veins were found in the corneal cavity. Therefore, we found that the treatment time was shorter and better after 4 weeks after the third eyelid flap sutures performed after keratectomy, no matter how wide and deep the melting ulcer was.

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