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CASE REPORT

Bandage Contact Lens Associated Infection after Amnion Membrane Transplantation in Peripheral Ulcerative Keratitis Case with Spondyloarthritis

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Abstract

Introduction: Bacterial keratitis causes around 90% of all cases of microbial keratitis. The global rise in contact lens usage has contributed to an increased risk of microbial keratitis. Peripheral ulcerative keratitis (PUK) is essential to diagnose as it can be the first presenting feature of a sight-threatening and associated with rheumatic autoimmune disease. **Case Presentation:** The case presents a 35-year-old woman with redness, discharge, and tenderness in her right eye (RE) since the day before, along with light sensitivity and tearing. Three months prior, she underwent multilayer amniotic membrane transplantation (AMT) surgery for corneal thinning due to peripheral ulcerative keratitis (PUK) and wore a contact lens postoperatively as a bandage. She had a history of conjunctival resection related to the PUK a year ago. She received oral cyclosporin and methylprednisolone for spondyloarthritis. Initially, her visual acuity of the RE was limited to hand movement. Diagnosis included RE keratoconjunctivitis related to contact lens and PUK post-AMT surgery; treatment comprised intravenous and topical antibiotics, artificial tears, cycloplegics, analgesics, and oral ascorbic acid. After four days of treatment, clinical signs were improved, with visual acuity progressing from hand movement to counting fingers at one meter. **Conclusions:** Careful management is essential for PUK patients after AMT surgery, especially those using contact lenses as bandages due to the potential risk of infection. Early PUK identification is crucial, as it may indicate sight-threatening issues and underlying systemic diseases. Meticulous examination and multidisciplinary management are required to ensure patient safety.

Keywords: contact lens; microbial keratitis; peripheral ulcerative keratitis

Introduction

Nearly 90% of microbial keratitis (MK) cases are caused by bacterial infections, presenting with light sensitivity, eyelid swelling, discomfort, redness, and diminished vision.[1] Accurate identification of the infection and its virulence factors, coupled with the appropriate antibiotics, can prevent prolonged and harsh treatments and the development of antibiotic resistance, leading to better patient outcomes and fewer surgeries.[2]

Over recent decades, the global increase in contact lens usage has become a significant risk factor for MK.[3] Corneal inflammation from MK is a severe and potentially life-threatening complication. Contact lens wearers face an annual incidence rate of 2 to 20 cases per 10,000 users.[4],[5] Factors increasing the risk of MK include professional occupations, infrequent contact replacement, showering with contacts, and sleeping with them. Treatment delays can lead to corneal scarring, perforation, endophthalmitis, and potential vision loss. MK can be caused by bacterial, fungal, protozoal, and viral agents.[6],[7],[8]

Bandage contact lenses (BCLs) play an important role in therapeutic ophthalmology by protecting and promoting healing in various corneal conditions. They manage corneal abrasions, recurrent corneal erosion, post-surgical healing, and chronic conditions like bullous keratopathy. BCLs act as a barrier, relieving pain, shielding healing epithelial cells from trauma, and improving topical

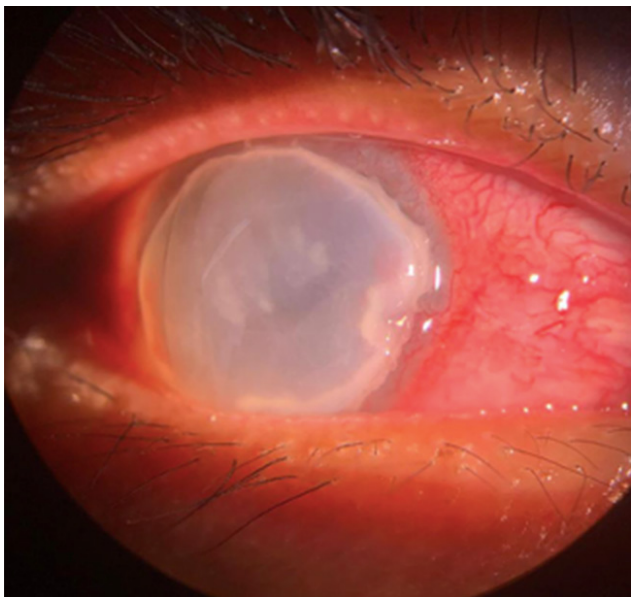

Figure 1. The initial clinical presentation. Conjunctival and pericorneal injections were present in the right eye (RE), with mucopurulent discharge appearing. The cornea was hazy, with infiltrates and thinning surrounding the peripheral edge.

medication delivery. However, BCLs pose a risk of MK, a severe corneal infection that can threaten vision if not promptly treated. Studies[9] indicate a notable incidence of MK with BCL use, especially with extended wear or in patients with compromised corneas. In a study of patients undergoing corneal collagen cross-linking (CXL), 2.85% of those using BCLs developed MK, emphasizing the need for careful monitoring for infection signs, particularly in compromised corneal conditions. It highlights the importance of diligent follow-up and monitoring for signs of infection, especially in compromised corneal conditions.

Peripheral ulcerative keratitis (PUK) affects the juxtalimbal cornea, causing stromal lysis and epithelial defects. It results from a complex interaction of environmental factors, peripheral cornea morphology and physiology, and host autoimmunity.[10] PUK can be caused by systemic or local, infectious or non-infectious factors, with up to 53% of cases linked to systemic lupus erythematosus (SLE), granulomatosis with polyangiitis (GPA), and rheumatoid arthritis (RA).[10] Collagen vascular diseases and vasculitides can also cause PUK. Progressive stromal lysis can lead to corneal perforation, posing significant morbidity and mortality risks for patients with underlying autoimmune diseases. PUK in scleritis indicates a poor prognosis.[10]

PUK without systemic association, known as Mooren's ulcer (MU), accounts for 31.5% of PUK cases.[11] Initially described by Bowman in 1849 and McKenzie in 1854 as "rodent ulcer" of the cornea, Mooren's ulcer is diagnosed by exclusion when scleritis is absent.[12] It starts in the cornea periphery and progresses centrally and circumferentially, with a characteristic overhanging edge. Identifying PUK is crucial as it may signal a potentially fatal systemic illness. Ensuring safe patient outcomes requires meticulous clinical investigation and multidisciplinary management.[13]

This study aims to provide a thorough understanding of the clinical applications, benefits, and potential complications of BCLs use in therapeutic ophthalmology to guide safer and more effective patient management practices.

Case presentation

This case report presents a 35-year-old woman who came to the outpatient clinic with complaints of redness and purulent discharge in the right eye (RE) since a day ago. Her complaints came along with tenderness, watery eyes, and light sensitivity. Two days earlier, she felt her RE became blurry. The patient wore a contact lens as a bandage after undergoing multilayer amniotic membrane transplantation. The bandage contact lens was changed two weeks ago.

The vision has worsened since the patient had the complaints. There was no history of any foreign body in her eye, and she never had any scratches from plants or other solid things. Two months ago, she underwent a multilayer amniotic membrane transplantation surgery due to corneal thinning caused by PUK. She also had a history of conjunctival resection a year ago, which was related to her PUK. The patient had a history of spondyloarthritis and was treated with 50 mg of oral cyclosporin once a day and 4 mg of oral methylprednisolone every eight hours (Figure 1).

Visual acuity of the RE was hand movement, while the left eye (LE) was 6/6. Intraocular pressure was normal in palpation for both eyes. Anterior segment evaluation of the RE revealed lid oedema, blepharospasm, diffuse conjunctival injection, peri corneal injection, and mucopurulent discharge on the RE. The corneal examination revealed corneal haziness, thinning, and infiltrates 3600 around the peripheral edge. Details of the anterior chamber, iris, pupil, and posterior segment were challenging to evaluate due to corneal haziness. The anterior and posterior segments of the LE were within normal limits.

We assessed the patient for RE keratoconjunctivitis-related contact lens and PUK post amniotic membrane transplantation. Upon previously scheduled follow-ups, the patient showed significant signs of impairment and was instructed to keep the application of the bandage contact lens. The BCL's hygiene factor, which was relatively hard to maintain, made the suspicion about contact lens-induced infection. Therefore, the usage of contact lenses was stopped, and the patient was hospitalized with consideration of intravenous antibiotic treatment. The initial medication given was intravenous antibiotic, topical antibiotic, non-preservative artificial tears, cycloplegic, analgesics, and ascorbic acid orally. Ceftriaxone injection every 12 hours, moxifloxacin eyedrop every five minutes in the first 30 minutes

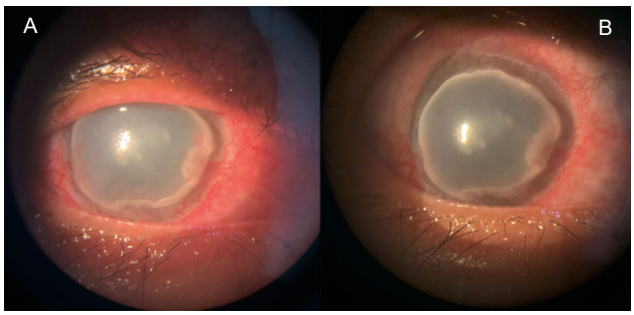

Figure 2. Follow up after treatment; (A) First day, the mucopurulent secret was reduced, however, there were no significant improvements in the corneal evaluation; and (B) Fourth day, clarity of the right cornea improved, followed by better visual acuity. The peripheral infiltrate seemed less active.

continue to every hour, atropine eyedrop every 12 hours, artificial tears eyedrop every two hours for both eyes, doxycycline capsule 100 mg every 12 hours orally, and vitamin C tablet 500 mg every 12 hours orally were prescribed. The ceftriaxone injection intravenously was given because the initial topical antibiotic medication showed no significant impairment. The patient also had an autoimmune condition, making the immunological state of the patient an utmost concern to prevent further infection. The first diagnostic procedure was a corneal scraping and culture sensitivity test with no bacterial or fungal results. Consultation with the internal medicine department was made, and the patient was advised to take cyclosporin treatment 50 mg once a day and to temporarily discontinue oral methylprednisolone while an acute infection is still going on (Figure 2).

On the first follow-up day, the patient complained of less pain than the day before, and the discharge rate was reduced. Visual acuity remains the same as before. There was no significant improvement on the corneal haziness in the first follow-up day. On the fourth follow-up day, the patient showed improvement in signs and symptoms. The patient complained of no significant tenderness. The visual acuity improved from hand movement to one meter counting finger. Eyelid oedema and spasm reduced so the patient could open the eyelid more quickly, and the discharge was disappeared. The corneal clarity improved, and the infiltrates became less active. The patient was informed to be followed up at the ophthalmology outpatient clinic after discharge.

Discussion and conclusions

To prevent corneal morbidity and vision loss, contact lens-induced keratitis must be addressed swiftly and effectively, as it constitutes a medical emergency. The most common risk factor for infectious keratitis in individuals with previously healthy eyes is the use of contact lenses.[14] The annual incidence of bacterial MK varies by lens type, with approximately two cases per 10,000 for rigid lenses, 2.2 to 4.1 cases per 10,000 for daily-wear soft lenses, and 13.3 to 20.9 cases per 10,000 for extended-wear soft lenses. Therapeutic contact lenses present an even higher risk, at 52 cases per 10,000 annually.[15] Factors such as corneal trauma, exposure to contaminated water, homemade saline solutions for lens disinfection, poor lens care, non-compliance with cleaning solutions, overnight lens wear, and extended wear schedules contribute to contact lens-related infectious keratitis. Soft contact lenses worn overnight significantly increase the risk, particularly with prolonged non-stop wear. Other associated risks include smoking, HIV, and lower socioeconomic status.[16],[17],[18]

The pathophysiology of contact lens-induced keratitis involves alterations in the tear film dynamics and ocular surface caused by contact lenses and their care systems. These changes can lead to corneal infection by introducing foreign contaminants to the corneal surface, disrupting natural tear flow essential for corneal immunity, causing microtrauma to the corneal epithelium, altering ocular surface immunity, and inducing corneal hypoxia. Poor hygiene practices increase the risk of infectious and non-infectious corneal inflammatory events.[19],[20]

Patients typically present with symptoms such as increasing discomfort, photophobia, mucopurulent discharge, limbal or conjunctival hyperemia, and impaired vision. Contact lens-induced keratitis can manifest as either ulcerative or infiltrative keratitis. Infiltrates associated with contact lens use might not always be clinically significant. Holden et al.[21] categorize contact lens-related events into serious, clinically significant, and clinically non-significant. Symptoms of MK in contact lens wearers include mucopurulent discharge, tears, photophobia, lid edema, abrupt onset of moderate to severe pain, and intense redness of the limbus and bulbar conjunctiva.[9],[14],[22],[23],[24]

Symptoms of contact lens-related keratitis include moderate to severe eye redness, tearing, photophobia, and mild to moderate pain, typically noticeable after waking up. The condition prompts an inflammatory response in the conjunctiva and cornea immediately after eye closure, leading to diffuse and focal infiltrates in the corneal mid-periphery to the periphery. Infiltrative keratitis features mild to moderate irritation, redness, and occasional discharge, with anterior stromal infiltration in the corneal mid-to-periphery, potentially with or without epithelial involvement. Calcofluor-white staining is particularly useful for detecting fungal and acanthamoeba elements, while fluorescein-conjugated lectins and indirect immunofluorescent staining methods can identify Acanthamoeba species, though these require a fluorescent microscope. Non-nutrient agar with an E. coli overlay enhances acanthamoeba recovery, indicated by snail-tract clearing from trophozoites.[25],[26]

The primary treatment goal is to preserve vision and corneal clarity, as bacterial infections can cause irreversible corneal scarring rapidly. Broad-spectrum

topical antibiotics are the first-line treatment, with fluoroquinolone monotherapy achieving results comparable to combination therapy. Initially, antibiotics should be applied every 30 to 60 minutes, reducing frequency based on clinical response. In severe cases, loading doses every five minutes for 30 minutes can quickly achieve therapeutic concentrations.[27],[28]

Topical combination therapy, including fortified antibiotics for increased corneal stroma concentrations, is recommended, particularly when methicillin-resistant Staphylococcus aureus (MRSA) is suspected or with vision-threatening ulcers. Most infectious keratitis cases are culture-negative after 48–72 hours of effective treatment. Once the offending microbe is identified or clinical response improves, appropriate monotherapy may be considered to reduce toxicity.[14] Systemic antibiotics are indicated for suspected scleral and/or intraocular infection extension, with fluoroquinolones offering excellent ocular penetration. The use of corticosteroids in bacterial keratitis is debated due to their dual effects on inflammation and host response inhibition. Evidence suggests that corticosteroids can worsen prognosis if administered before antibiotics, though they may improve outcomes if used 48 hours after starting antibiotics for bacterial keratitis.[12],[29] Patients should adhere to antibiotic regimens and return for regular follow-ups when using corticosteroids. Moderate dosages of corticosteroid drops can be given, with frequency adjusted based on clinical response. Collagen cross-linking is an emerging adjunctive therapy for bacterial keratitis, though its precise role is still evolving. Penetrating keratoplasty (PK) may be necessary if the condition worsens or fails to improve with antibiotic therapy. The infected area should be identified and contained before surgery, and postoperative treatment includes strong topical corticosteroids, cycloplegics, and appropriate antibiotics.[30]

Regular cleaning of contact lens cases, heat disinfection, and replacement are essential to prevent infection. Daily disposable contact lenses are recommended due to their lower infection risk.

PUK affects the juxtalimbal cornea, causing stromal lysis and epithelial defects. It involves a complex interplay between the environment, peripheral corneal morphology, and host autoimmunity. Systemic diseases like SLE, GPA, and RA account for up to 53% of PUK cases. PUK in scleritis indicates a poor prognosis, with progressive stromal lysis potentially leading to corneal perforation and significant morbidity.[31] Ophthalmologic manifestations in rheumatic patients are expected, with inflammatory non-infectious disorders more prevalent than non-inflammatory ones. Multidisciplinary rheumatology-ophthalmology clinics can facilitate early diagnosis and management of ocular manifestations, which can precede or follow rheumatic disease onset. Autoimmune PUK, often associated with RA, typically affects the peripheral cornea unilaterally. Early detection and treatment with topical and systemic therapies are crucial to manage inflammation and prevent severe complications.[7]

In cases where there is a suspicion of scleral and/or intraocular extension of infection, systemic antibiotics—particularly the fluoroquinolones, which have significant ocular penetration and extensive topical antibiotics are indicated. There is ongoing debate on the use of corticosteroid therapy for bacterial keratitis. The combination of the bacteria's direct impacts and the ferocious human inflammatory response, mainly caused by polymorphonuclear leukocytes and proteolytic enzymes even after corneal sterilization, leads to tissue loss. Although corticosteroids are good at changing this response, they also prevent the host from reacting to an infection. Research indicates that prognosis is worsened by corticosteroid medication given before proper antibiotic therapy.[30],[32] However, as seen by a randomized clinical trial where topical corticosteroids were administered 48 hours after topical antibiotics were started for bacterial keratitis, the literature is inconclusive regarding using steroids concurrently with antibiotic therapy or after it is commenced.[33] The final visual outcome and complication rate showed no change at three months; however, patients with the lowest initial vision who got corticosteroids showed a trend toward better outcomes at the one-year follow-up. The study[34] found that Nocardia keratitis, an uncommon condition in the United States, responded poorly to corticosteroid treatment. There is still a significant risk associated with corticosteroid use in patients with bacterial or other forms of infectious keratitis not appropriately treated. The recommended criteria for instituting corticosteroid therapy for bacterial keratitis, including corticosteroids, should not be used without appropriate antibiotic therapy. The patient must be able to return for frequent follow-up examinations.[35]

It is not advisable to use corticosteroids without the proper antibacterial therapy. The patient needs to be able to show that they are taking their antibiotics as prescribed and be able to return for regular check-ups. No other suspected or discovered related virulent or challenging-to-addicted organisms exist.[34] Moderate dosages of corticosteroid drops, such as 1% prednisolone acetate or phosphate every six hours, can be given, and the patient should be observed 24 and 48 hours after medication starts. Based on clinical response, the frequency of administration may be changed if the patient exhibits no side effects. Collagen cross-linking is increasingly used as an adjunctive therapy for bacterial keratitis, with anecdotal success; as this technology becomes more available in the United States, its precise role and application are evolving.[36]

If the condition worsens despite treatment, if there is a descemetocele development or perforation, or if

the keratitis is not improving with antibiotic therapy, PK may be necessary. Before surgery, the affected area should be identified, and every effort should be taken to contain the infection. Because seclusion of the pupil may result from inflammatory pupillary membranes, peripheral iridectomies are recommended. It is advised to use interrupted sutures. Following surgery, the patient must be treated with strong topical corticosteroids, cycloplegics, and the proper antibiotics.[19] Regularly cleaning the inside surfaces of the contact lens case with a cotton ball or Q-tip saturated with contact lens cleanser is necessary to break up infection-resistant biofilms. The contact lens case should be periodically replaced and heat disinfected with hot water exposure. After air drying, the case should be replaced. Since daily disposable contact lenses are linked to a lower risk of infection, we advise using them.[14],[35]

The juxtalimbal cornea is affected by PUK, which often manifests as stromal lysis and epithelial defect. The environment, the peripheral cornea's morphology and physiology, and host autoimmunity interact intricately due to this inflammatory disease. The root cause may be systemic or local, infectious or non-infectious. Up to 53% of PUK cases can be attributed to SLE, GPA, and RA. PUK can also result from collagen vascular disease and vasculitides.[10] PUK in scleritis is a poor prognostic factor. Progressive stromal lysis can cause corneal perforation, which is an emergency and, in patients with an underlying autoimmune disease, indicates significant morbidity and mortality.[13]

As many as 26.9% of all consecutive rheumatic patients were diagnosed with an ophthalmologic manifestation or disease at the ophthalmologic clinic. This percentage is in line with the available literature on patients with specific rheumatic diagnoses like RA and spondylarthritis. This high number of ophthalmologic diagnoses in rheumatic patients may argue for a multidisciplinary rheumatology–ophthalmology clinic.[37]

While most ophthalmologic diagnoses were more frequently non-inflammatory without malignancy than inflammatory non-infectious disorders, inflammatory non-infectious diagnoses were more common than non-inflammatory diagnoses without malignancy. Undoubtedly, non-inflammatory ophthalmologic diagnoses that do not indicate cancer may be undervalued because they are also treated in healthcare facilities outside of university hospitals. Pure ophthalmologic counseling and self-management suggestions can help these patients become more knowledgeable.[38] The decision to treat RA with immunosuppressive medication may depend on the presence of inflammatory non-infectious ophthalmologic diagnosis. Regarding particular diagnoses, PUK was common (16.5%) in spondyloarthritis. This confirms the possible role of PUK in axial spondyloarthritis, as already mentioned in the existing literature. Still, this fact emphasizes the need for rheumatologists to consider ocular manifestations.[39]

Rheumatic disease and ophthalmologic symptoms do not always show simultaneously in clinical practice. An ophthalmologic manifestation might appear concurrently with, after, or prior to the commencement of a rheumatic illness. Thus, one of the most critical difficulties is considering ocular signs as soon as possible and seeing an ophthalmologist before visual damage or even blindness happens. An ophthalmologic visit may help identify an inflammatory ocular disease early if suspected, even in patients with non-inflammatory rheumatic diseases. Conversely, inflammatory non-infectious rheumatic diseases coexist in 73.5% of individuals with inflammatory non-infectious eye diseases.[40] Therefore, both disciplines may see a single patient to manage different manifestations of the same disease, two separate disease entities, or even to manage ocular side-effects of medications for rheumatic diseases. Nevertheless, it may be helpful for both specialists to have their patients rapidly assessed by the cooperating specialist. This is made possible by the commitment of both the rheumatologists and the ophthalmologists, and not only provides an advantage for earlier diagnosis.[41]

Patients with rheumatic and systemic immune-mediated disorders may develop autoimmune peripheral keratitis. Although it can occasionally occur in other illnesses, PUK is most frequently associated with RA. Although it is not a routine diagnostic practice, a biopsy of the conjunctival tissue next to marginal corneal illness usually reveals indications of immune-mediated vaso-occlusive disease. In the context of systemic collagen-vascular illness, central corneal melting could be caused by a separate mechanism linked to a T-lymphocyte infiltration.[42] A history of connective tissue disease is often (but not invariably) present, although, in some patients, the ocular finding of peripheral corneal infiltration or frank stromal melting may be the first sign of the underlying systemic illness. Autoimmune PUK generally correlates with exacerbations of systemic disease activity. Follow-up of patients with autoimmune PUK reveals that if they are treated inadequately, severe disease-related morbidity may occur in a high number of these patients. The term keratolysis refers to the significant (and often rapid) stromal melting in some cases of immune-mediated PUK associated with systemic autoimmunity.[36],[43]

Autoimmune PUK typically affects one sector of the peripheral cornea and is unilateral, though it can occasionally be widespread and bilateral. There are variable degrees of vaso-occlusion of the surrounding limbal vascular networks in conjunction with the first lesions, which occur in a zone within 2 mm of the limbus.[44],[45] The underlying stroma thins and the epithelium is usually absent in the affected area; however, if the disease is discovered early, the stroma may still be almost normal in thickness, and the epithelium may be patchy. Ulceration may or may not be associated with a significant cellular infiltrate in

the corneal stroma, and the adjacent conjunctiva can be minimally or severely inflamed. The sclera can be involved in patients with systemic immune-mediated diseases (e.g., necrotizing scleritis in patients with RA), so a careful, complete examination must be performed.[46]

Therapy aims to reduce melting by using local supporting measures. This is accomplished via actions meant to enhance wetness, encourage epithelialization, and reduce systemic and local immune-mediated inflammation. Increased lubrication of the ocular surface is crucial because lubrication may help reduce the impact of inflammatory cytokines in the tear film and because keratoconjunctivitis sicca (KCS) is a common sign of secondary Sjogren syndrome in RA patients. If the epithelium can be coaxed to recover using lubricants, patches, or BCLs, melting will halt or slow down noticeably.[34] Several topical collagenase inhibitors (e.g., sodium citrate 10%, acetylcysteine solution 20%, medroxyprogesterone 1%) and systemic collagenase inhibitors such as tetracyclines (e.g., doxycycline) are of potential value. Topical cyclosporine is effective in patients with central melting, likely due to a T-cell-mediated process rather than occlusive vasculitis.[47]

Acute ocular inflammation is usually treated with topical corticosteroids and cycloplegia, like idiopathic PUK. However, peribulbar injections or systemic corticosteroid therapy may be necessary in more severe cases and eyes with vitreous involvement. Difluprednate 0.05%, prednisolone acetate 1%, and dexamethasone 0.1% are examples of topical treatments for PUK. Compared to dexamethasone, difluprednate, and prednisolone acetate reach greater and longer-lasting aqueous concentrations. Prednisolone acetate 1% eight times per day was not inferior to difluprednate 0.05% four times daily.[50] Compared to prednisolone, difluprednate clears the anterior chamber reaction more quickly and in a higher percentage of patients.[48]

Numerous research studies[39] have looked into the function of antibiotics in treating spondyloarthritis, taking into account the importance of gut flora. As a mainstay of care for other spondyloarthritis, Tumor necrosis factor inhibitors (TNFIs) have gained prominence. In patients who are receiving Non-steroidal anti-inflammatory drugs (NSAID) treatment, however, still have substantial disease activity, they are necessary.[38] There are no particular recommendations for selecting TNFIs for spondyloarthritis; thus, the use of TNFIs for these conditions depends on their indications and contraindications in each case. Etanercept, adalimumab, certolizumab pegol, and infliximab are commonly used to treat spondyloarthritis. A summary of the different TNFIs, their dosing, and routes of administration is provided.[37]

Topical corticosteroids can have various effects, as they can block the operation of collagenase. Generally speaking, a doctor treating inflammation must balance the potential hazards of poor healing with the advantages of treating inflammation. Recession or excision of the surrounding limbal conjunctiva frequently results in ulcer healing, most likely because the operation removes a source of collagenolytic enzymes and inflammatory cells.[49] Local treatments alone are frequently insufficient to achieve definitive control; systemic treatment, such as immunosuppressive therapy with oral prednisone, cytotoxic medicines like cyclophosphamide, or immuno-modulatory drugs like methotrexate or cyclosporine, must be started or escalated. Biologic agents such as infliximab have reportedly been used with some success in more severe cases. Patients with severe, rapid melting may require intravenous therapy with high-dose cyclophosphamide, with or without corticosteroid therapy.[50]

Because lamellar and penetrating grafts are equally prone to melting, threatened perforations should be treated with temporizing techniques like cyanoacrylate glue and bandage contact lens insertion until systemic therapy has been started. Numerous tectonic grafts may be needed to maintain the globe while the systemic therapy is being modified. Keratoplasty can restore vision once the underlying disease process has been managed.[51] Conjunctival flaps are probably best avoided in immune-mediated disease, even though they can be beneficial in managing stromal melting in difficult-to-manage MK. Bringing the conjunctival vasculature even closer to the area of corneal disease could accelerate melting. It is essential to partner with a rheumatologist in caring for patients with immune-mediated disease, as their risk of morbidity and death is significant.[52]

There is a higher risk of PUK recurrence, and graft melts with surgery, so this should be delayed until adequate control of inflammation is achieved.[53] Graft survival is less than 50% at six months, so multiple grafts are required in many patients.[54] Surgical emergency care is contingent upon the indication. A tectonic indication would be a perforation or descemetocele; an optical indication would be for vision rehabilitation; and a therapeutic indication would be ulcers expanding circumferentially and causing corneal melt. The surgical approach selected depends on the magnitude of the corneal defect. In regions where the cornea is thinning, options include conjunctival excision, recession, flaps, and a multilayered amniotic membrane transplant.[55]

The patient's previous treatment for her PUK involved a multilayer amnion membrane transplantation (AMT). Corneal thinning, especially in the peripheral region, led to the decision to perform AMT. The amniotic membrane (AM) is the placenta's innermost layer, consisting of an avascular stromal matrix, a thick basement membrane, and a single layer of metabolically active epithelium. It is an effective intervention for treating corneal ulcers and perforations because of its many uses as an onlay, graft, patch, or combination. Its non-immunogenic nature, combined with its multiple qualities such as delaying

apoptosis exhibiting antibacterial, antifibrotic, anti-inflammatory, and antiangiogenic traits, contributes to its therapeutic efficiency. AM enhances epithelialization by boosting the migration and differentiation of epithelial cells, fortifying the adherence of basal epithelial cells, and modifying the proliferation of normal corneal, conjunctival, and limbal fibroblasts. Its efficacy in treating corneal ulcers and perforations is further increased by its use as a single or multilayered graft. AMT has become a common treatment for ocular surface illnesses because of the availability of AM donor tissues, its low risk of graft rejection, and improvements in storage techniques. The appeal of AM as a therapy option for corneal diseases stems from its accessibility and ease of surgery compared to donor cornea.[25],[56] Conjunctival resection removes the tissue supplying inflammatory mediators to the cornea. Lamellar patch grafts reduce graft rejection risk compared with a full-thickness patch or tectonic grafts.[57],[58] Corneal glue (cyanoacrylate) with a bandage contact lens can be deployed if the perforation is less than 3 mm in diameter.[59]

Careful management of PUK with AMT and subsequent use of BCLs in patients with autoimmune diseases are required, as well as the balance between therapeutic benefits and the risk of infection. This case illustrates the critical need for early recognition and aggressive management of infections to prevent severe ocular morbidity. Multidisciplinary collaboration and patient education are pivotal in achieving favorable outcomes in complex cases.

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