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Comprehensive Management of Mooren's Ulcer with Conjunctival Resection and Double-Layer Amniotic Membrane Transplantation: A Case Report

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Abstract

Mooren's ulcer is a rare, progressive peripheral corneal inflammatory disease that may lead to vision loss due to corneal destruction. The disease is thought to be associated with an autoimmune mechanism without clear evidence of infectious causes or systemic disease involvement. Its management remains challenging, particularly in progressive cases, with a combination of conjunctival resection and Amniotic Membrane Transplantation (AMT) serving as one of the therapeutic options to control inflammation and preserve corneal integrity. A 23-year-old woman presented with severe pain in her left eye for approximately 3 weeks, accompanied by redness, epiphora, photophobia, foreign body sensation, and decreased visual acuity. The patient had previously been diagnosed with corneal ulcer and had received initial treatment without clinical improvement. Ophthalmological examination revealed a left eye visual acuity of 1/300, intraocular pressure of 8 mmHg, and a peripheral corneal ulcer with stromal excavation extending from the 9 to 15 o'clock position, accompanied by superior conjunctivalization. There was no history of ocular trauma, contact lens use, previous ocular infection, or systemic autoimmune disease. The patient was diagnosed with Mooren's ulcer with a differential diagnosis of Peripheral Ulcerative Keratitis (PUK) and underwent conjunctival resection combined with Double-Layer Amniotic Membrane Transplantation (AMT). Postoperative evaluation showed that the amniotic membrane was well-positioned with adequate apposition, and the patient received further treatment consisting of antibiotics, corticosteroids, topical immunomodulators, and ocular lubricants.

Introduction

Mooren's ulcer is a rare, progressive inflammatory disease of the peripheral cornea that carries the potential for permanent vision loss. The condition is characterized by distinctive peripheral corneal ulceration, featuring inflammatory infiltration and destruction of the corneal stroma, in the absence of scleral involvement or an identifiable infectious cause. Although the exact etiology remains incompletely understood, evidence suggests that Mooren's ulcer is linked to autoimmune mechanisms specifically, an abnormal immune response against corneal antigens in genetically predisposed individuals (Höllhumer, 2022; Świerczyńska et al., 2023; Mohan et al., 2023; Venkateswaran et al., 2022).

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Clinically, Mooren's ulcer may present with severe eye pain, photophobia, epiphora, a foreign-body sensation, conjunctival hyperemia, and reduced visual acuity. Lesions typically originate in the peripheral cornea and may progress circumferentially toward the center (Höllhumer, 2022). Without adequate treatment, the disease's progression can lead to severe corneal thinning ("corneal melt"), corneal perforation, and loss of visual function. A major diagnostic challenge lies in distinguishing it from other conditions with similar presentations particularly peripheral ulcerative keratitis (PUK) associated with systemic autoimmune diseases, infections, or trauma (Sabhapandit et al., 2022; Baquet-Walscheid et al., 2024; Guerrero-Acosta et al., 2026; Pelegrín et al., 2026; Świerczyńska et al., 2023).

Management of Mooren's ulcer requires a stepwise approach tailored to disease severity. Conservative therapies such as topical corticosteroids, immunosuppressive agents, and systemic immunomodulators may be employed in early-stage cases or those with mild inflammatory activity. However, in cases that are progressive, refractory to medical therapy, or involve significant corneal tissue destruction, surgical intervention is necessary to control the inflammatory process and preserve the integrity of the eyeball (Ou et al., 2025; Dang et al., 2022; Fischer et al., 2025; Rafizadeh et al., 2025). Conjunctival resection is a surgical procedure employed in the management of Mooren's ulcer, aimed at removing conjunctival tissue suspected of being the source of inflammatory mediators and autoantibodies that drive disease progression. Additionally, Amniotic Membrane Transplantation (AMT) serves as a reconstructive therapeutic option due to its anti-inflammatory and anti-angiogenic properties, as well as its ability to support ocular surface epithelialization. The double-layer AMT technique offers the added benefits of filling deeper stromal defects and providing structural support to severely thinned corneas (Widowati et al., 2023; Shen et al., 2023; Ibukun et al., 2026). Although various therapeutic modalities have been reported, scientific evidence regarding the optimal management strategy for Mooren's ulcer remains limited due to the disease's low incidence and the fact that most existing literature consists of case reports or case series. Therefore, a case report detailing the combination of conjunctival resection and double-layer Amniotic Membrane Transplantation is significant for illustrating a multidisciplinary approach to managing progressive Mooren's ulcer and for evaluating clinical outcomes following the surgical intervention (Widowati et al., 2023; Huang et al., 2026; Yu et al., 2025; Bin Helayel et al., 2026).

Case Presentation

Patient Identity

A 23-year-old female presented to Makassar Eye Hospital with a chief complaint of severe pain in the left eye, which had been present for approximately three weeks prior to admission. The patient is a housewife residing in Pekkabata. She was referred from Lasinrang Regional General Hospital (Pinrang) with an initial diagnosis of corneal ulcer for further evaluation and management.

History of Present Illness

A 23-year-old female presented with severe pain in the left eye, experienced for approximately three weeks prior to admission. The pain was constant, stabbing in nature, and radiated to the area surrounding the eye. These symptoms were accompanied by redness, itching, swelling of the left eyelid, and increased tearing (epiphora). Additionally, the patient reported photophobia, a foreign body sensation in the eye, decreased visual acuity in the left eye, and difficulty opening the eyelid due to the pain. There was no history of prior ocular trauma, contact lens use, chemical exposure, or eye-rubbing habits. Prior to referral, the patient had been examined at Lasinrang Regional General Hospital and diagnosed with a corneal ulcer. She was treated with Cendo Lyteers eye drops, but her condition did not improve. She also applied ice compresses to the left eye to alleviate the pain, but the symptoms persisted.

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The patient denied any history of ocular trauma, soft contact lens use, wearing glasses, or excessive exposure to dust, wind, or chemicals. There was no history of recurrent eye infections. The patient had no history of systemic diseases such as hypertension, diabetes mellitus, autoimmune disorders, or tuberculosis. A history of sexually transmitted diseases, including syphilis, was also denied. There was no history of prior ocular viral infections, such as Herpes Simplex Virus keratitis. No history of drug or food allergies was found.

Clinical Examination

Upon initial examination, the patient appeared moderately ill but fully conscious (compos mentis). Vital signs showed a blood pressure of 128/84 mmHg, a pulse rate of 59 beats per minute, a respiratory rate of 18 breaths per minute, a body temperature of 36°C, and an oxygen saturation of 99% on room air. Ophthalmological examination revealed

Table 1. Clinical Examination Results

Structure	Right Eye (OD)	Left Eye (OS)
Eyelid	Normal	Edema, hyperemia
Eyelashes	No discharge found; no trichiasis	No discharge found; no trichiasis present.
Lacrimal apparatus	Normal	Increased tear production (epiphora)
Conjunctiva	Normal	Ciliary flush around the limbus
Cornea	Clear	Cloudy; a peripheral corneal ulcer with an overhanging edge is visible, located in the temporal to superior region spanning approximately the 9 to 3 o'clock positions with the lesion extending from the temporal toward the superior aspect, accompanied by superior conjunctivalization.
Anterior Chamber	Deep, clear	Deep, relatively clear, hypopyon (-)
Iris	Brown, difficult to assess	Difficult to assess
Pupil	Round, isochoric	Drawn superiorly
Light Reflex	(+/+)	Cannot be assessed
Lens	Clear	Cannot be assessed
Posterior Segment	No abnormalities	Examination not performed



Figure 1. Preoperative clinical appearance of a peripheral corneal ulcer in the patient's left eye.

Anterior Segment Examination

REVISION NO. 1: Examination of the left eye revealed edema and perilimbal injection (ciliary flush) surrounding the lesion, accompanied by increased lacrimation. A peripheral corneal ulcer with an overhanging edge was observed, extending from the temporal to the superior region (involving the area between the 9 o'clock and 3 o'clock positions) and progressing from temporal to superior, with associated superior conjunctivalization. The anterior chamber

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appeared deep and relatively clear, with no hypopyon present. The iris was difficult to evaluate, while the pupil appeared drawn superiorly. Posterior segment examination could not be performed due to limited visualization of the anterior segment.

Ancillary Tests

Initial laboratory testing was conducted on March 30, 2026, at Lasinrang Regional General Hospital (RSUD). Routine blood test results showed:

Hemoglobin: 14,8 g/dL

Leukosit: 15,4 $\times 10^3/\mu L$

Trombosit: 215.000/µL

Leukocyte differential count analysis revealed a predominance of neutrophils (72.2%) and elevated eosinophils (18%).

Ocular ultrasonography (B-scan) was performed on March 31, 2026. The results showed the eyeball to be intact with a regular posterior wall contour. The retina-choroid-sclera complex appeared attached to the posterior wall of the eyeball. No evidence of retinal detachment, vitreous hemorrhage, intraocular mass, or suprachoroidal effusion was found. Impression:

Retina attached

No retinal detachment observed

No vitreous hemorrhage observed

Posterior segment within normal limits

The erythrocyte sedimentation rate (ESR) was 20 mm/hour within the normal range indicating the absence of significant systemic inflammation.

The ancillary tests performed were tailored to the patient's clinical condition, the availability of testing facilities, and cost considerations. Autoimmune serological tests such as antinuclear antibody (ANA), antineutrophil cytoplasmic antibody (ANCA), and rheumatoid factor (RF) and tests for specific infections (e.g., VDRL/TPHA) were not conducted; this was due to their unavailability at the primary care facility and the lack of strong clinical indications derived from the patient's history or physical examination suggesting systemic autoimmune disease or specific infections. The diagnosis of Mooren's ulcer in this case was established primarily based on characteristic clinical features and the exclusion of infectious causes and systemic diseases through a comprehensive clinical evaluation.

Diagnosis

Based on the patient's history, clinical examination, and ancillary tests, the diagnosis established was Mooren's ulcer of the left eye, with a differential diagnosis of Peripheral Ulcerative Keratitis (PUK). The diagnosis was based on lesion characteristics specifically a progressive peripheral corneal ulcer, severe pain, and conjunctival involvement surrounding the lesion along with the absence of infectious factors or underlying systemic disease.

Management

The patient received initial management consisting of supportive care, medication, and anti-inflammatory and antimicrobial therapies. Supportive therapy involved the administration of intravenous Ringer's Lactate solution at a rate of 20 drops per minute. Systemic antibiotic therapy consisted of intravenous ceftriaxone (1 gram every 12 hours), while anti-inflammatory therapy involved intravenous dexamethasone (every 8 hours) to control the inflammatory process driving the ulcer's progression. To alleviate pain, the patient was administered intravenous ketorolac (1 ampoule every 12 hours).

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Topical therapy for the left eye included moxifloxacin eye drops (every 2 hours), Giflox eye drops (every 2 hours), and 1% prednisolone acetate eye drops (6 times daily) for local antimicrobial and anti-inflammatory effects. Additionally, the patient was prescribed cyclosporine eye drops twice daily as an immunomodulatory agent and Cendo Lyteers as an ocular lubricant to maintain ocular surface moisture.

Despite pharmacological treatment, the patient exhibited progression of the peripheral corneal ulcer, carrying a risk of further corneal thinning and complications such as corneal perforation. Consequently, a surgical procedure was performed on the left eye, consisting of conjunctival resection combined with a double-layer Amniotic Membrane Transplantation (AMT) technique

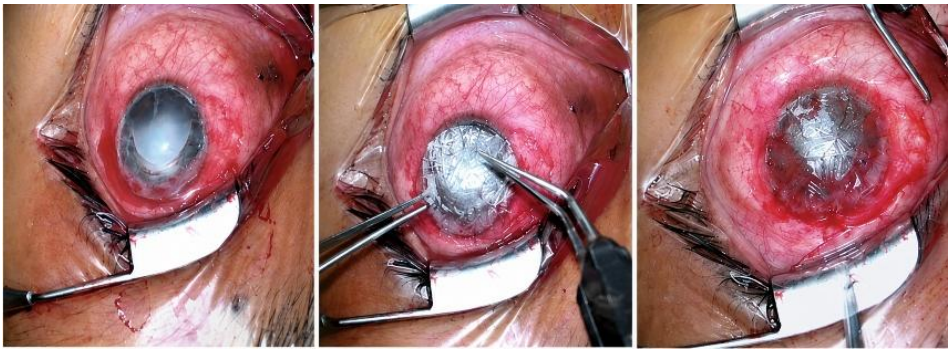


Figure 2. Intraoperative documentation of conjunctival resection and double-layer Amniotic Membrane Transplantation (AMT) on the patient's left eye.

Conjunctival resection was performed to eliminate conjunctival tissue acting as a source of inflammatory mediators and local immune responses that contribute to disease progression. Subsequently, a double-layer Amniotic Membrane Transplantation technique was employed to cover the corneal defect, provide structural support to the thinned corneal area, accelerate epithelialization, and exert anti-inflammatory and anti-scarring effects on the ocular surface. The combination of these procedures was intended to preserve the integrity of the eyeball and prevent severe complications such as corneal perforation and loss of vision.

Follow-up and Outcome

Postoperative evaluation was conducted following the conjunctival resection and double-layer Amniotic Membrane Transplantation (AMT) on the left eye. Examination revealed a visual acuity of 1/300 in the left eye (VOS) and an intraocular pressure (TOS) of 7 mmHg. Anterior segment examination showed blepharospasm, ciliary flush in the conjunctiva surrounding the limbus, the presence of a bandage contact lens (BCL), and an amniotic membrane that was well-positioned with 10 sutures, ensuring adequate apposition.



Figure 3. Clinical appearance of the patient's left eye in the postoperative period following conjunctival resection and double-layer amniotic membrane transplantation (AMT), showing the BCL and amniotic membrane with adequate apposition.

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Postoperative therapy consisted of oral ciprofloxacin (500 mg twice daily), methylprednisolone (16 mg; 2.5 tablets once daily), lansoprazole (30 mg once daily), and vitamin C (500 mg once daily). Topical therapy was continued with 0.5% moxifloxacin eye drops every 2 hours, cyclosporine eye drops twice daily, and carboxymethylcellulose sodium as a lubricant every 4 hours for the left eye.

At the postoperative follow-up, the patient showed clinical improvement, evidenced by reduced pain, decreased conjunctival hyperemia, and a stable amniotic membrane. No signs of corneal perforation or ulcer progression were observed during the monitoring period. Topical therapy was continued with gradual adjustments based on the patient's clinical response until the next follow-up visit.

Table 2. Clinical progression of patients with Mooren's ulcer before and after surgery

Time	Clinical Condition	Inspection	Governance	Outcome
±3 weeks before hospital admission	Left eye redness, severe pain, gritty sensation, discharge, and a sharp decline in vision.	Treated at Lasinrang Regional General Hospital (RSUD) with a diagnosis of corneal ulcer.	Cendo Lyteers®, kompres dingin, rawat inap ±6 hari	No improvement; referred to Makassar Eye Hospital.
Day of admission to the Eye Hospital	Severe pain, redness, epiphora, photophobia, and reduced visual acuity; findings include a progressive peripheral corneal ulcer with an overhanging edge extending from the temporal region superiorly, accompanied by superior conjunctivalization and ciliary flush around the limbus.	VOS 1/300, IOP 8 mmHg; working diagnosis: Mooren's ulcer, differential diagnosis: Peripheral Ulcerative Keratitis (PUK).	Pre-operative preparation and internal medicine consultation for surgical clearance.	The patient is deemed fit for surgery.
Day of surgery	A surgical procedure was performed.	Conjunctival resection + double-layer amniotic membrane transplantation (AMT) with anterior chamber reformation	Systemic and topical antibiotics, corticosteroids, cyclosporine, lubricants	The operation went well.

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Post-operative Day 0	Conjunctival hyperemia	BCL in place, AMT secured with 10 sutures showing good apposition, anterior chamber formed	Intravenous antibiotics were switched to oral ciprofloxacin; topical therapy was continued.	Postoperative condition is stable.
Post-operative Day 1	Blepharospasm persists; hyperemia has decreased.	BCL in place, AMT secured with 10 sutures showing good apposition	IV dexamethasone is planned to be replaced with oral methylprednisolone, prednisolone drops are temporarily stopped	No early postoperative complications were observed.
Post-operative Day 2 (Discharge)	The pain has decreased, but the redness in the eye persists.	VOS 1/300, TOS ±7 mmHg; BCL and AMT remain securely in place; amniotic membrane apposition is good.	The patient was discharged with antibiotics, oral steroids, lubricants, cyclosporine, and vitamin C.	A follow-up visit is scheduled for one week later.
Follow-up Day 8	No severe complaints, minimal pain, significant reduction in redness, evaluation of healing.	Right eye visual acuity (VOD) 20/40, left eye visual acuity (VOS) 1/300; mild conjunctival hyperemia; cornea remains hazy; good apposition of the BCL and AMT.	Topical therapy and oral steroids are continued.	Good healing, inflammation controlled.
Follow-up Day 21	My left eye still feels uncomfortable	Visual acuity (OD) 20/50, (OS) 1/60; mild conjunctival hyperemia, superior corneal haze, pupillary synechiae, lens opacity.	Moxifloxacin, Cyclosporine, Prednisolone tetes, Methylprednisolone oral	Planned suture removal (suture removal)

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Definition and Epidemiology of Mooren's Ulcer

Mooren's ulcer (MU) is a rare, chronic, progressive, and destructive inflammatory disease of the peripheral cornea. It is characterized by peripheral corneal ulceration occurring without scleral involvement and is not directly associated with infection or an underlying systemic disease. This condition is classified as a form of idiopathic peripheral ulcerative keratitis (PUK) and can lead to severe corneal damage and loss of vision if not properly treated. Unlike PUK, which is typically associated with systemic autoimmune diseases such as rheumatoid arthritis or vasculitis, Mooren's ulcer is primarily confined to ocular tissues and is thought to involve an abnormal immune response to corneal antigens (Alberta, 2022)



Figure 4. Illustration of the clinical characteristics of Mooren's ulcer on slit-lamp examination, showing a progressive peripheral corneal ulcer with an overhanging edge. Adapted from: Vitaplana, F (2026)

Mooren's ulcer is a very rare disease with an incidence rate that remains imprecisely known. It is reported to occur more frequently in specific regions such as sub-Saharan Africa, South Asia, China, and various tropical areas compared to countries in the Northern Hemisphere. This geographical disparity suggests the potential influence of genetic factors, environmental factors, or exposure to specific antigens on the disease's development. Studies also indicate variations in clinical characteristics across populations, including age of onset, disease aggressiveness, and the tendency for unilateral versus bilateral involvement (Priscilia et al., 2026).

Epidemiologically, Mooren's ulcer is more common in adults and shows a higher prevalence in men than in women. However, it can still occur in young women, as seen in this case involving a 23-year-old female patient with unilateral involvement of the left eye. Clinical manifestations typically include severe ocular pain, photophobia, epiphora, a foreign-body sensation, conjunctival injection, and a sharp decline in visual acuity resulting from progressive peripheral corneal involvement. Initial lesions generally present as inflammatory infiltrates in the peripheral cornea that evolve into crescent-shaped ulcers characterized by a distinctive overhanging edge (Atima et al., 2024).

Despite its low incidence, Mooren's ulcer carries significant clinical implications; the process of corneal destruction can be rapid, leading to complications such as stromal thinning, corneal melt, perforation, and even blindness. Consequently, early diagnosis and aggressive therapy are essential to halt disease progression and preserve the integrity of the eyeball (Lucchino et al., 2024).

Pathogenesis and Immunological Mechanisms

The pathogenesis of Mooren's ulcer remains incompletely understood; however, various lines of evidence suggest an autoimmune basis mediated by both cellular and humoral immune responses. The prevailing hypothesis posits that initial corneal injury leads to the release of previously sequestered corneal antigens, subsequently triggering immune system activation in genetically predisposed individuals. This uncontrolled immune response results in chronic inflammation within the limbus and peripheral cornea (Guerrero-Acosta et al., 2026).

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At the cellular level, the inflammatory process involves the infiltration of various immune cells primarily T lymphocytes, neutrophils, macrophages, and plasma cells—into the ulcerated area. T-lymphocyte activation leads to the release of pro-inflammatory cytokines such as interleukin (IL)-1, IL-6, IL-8, and tumor necrosis factor-alpha (TNF-α), which sustain the local inflammatory process. Furthermore, the activation of these immune cells enhances the production of proteolytic enzymes, such as matrix metalloproteinases (MMPs), which play a key role in the degradation of corneal stromal collagen (Guerrero-Acosta et al., 2026).

Elevated MMP activity specifically MMP-2 and MMP-9 causes damage to the corneal extracellular matrix, driving progressive stromal thinning. An imbalance between tissue degradation and regeneration leads to "corneal melting," a hallmark feature of Mooren's ulcer. Beyond local mechanisms, certain external factors are suspected triggers, including ocular trauma, prior surgery, parasitic infections, and specific viral infections such as hepatitis C; however, a definitive causal link for these factors has not yet been fully established (Guerrero-Acosta et al., 2026).

In addition to the cellular immune response, humoral autoimmune mechanisms also play a role through the formation of antibodies against corneal components. These antibodies can trigger complement activation and amplify the inflammatory process through neutrophil recruitment and the release of tissue-destructive mediators. The combination of adaptive immune activation, chronic inflammation, and increased collagenolytic enzyme activity leads to progressive damage to the peripheral cornea (Guerrero-Acosta et al., 2026).

In this case, no precipitating factors such as trauma, contact lens use, prior eye infection, or systemic autoimmune disease were identified. This clinical picture supports the likelihood of a local autoimmune mechanism characteristic of Mooren's ulcer: peripheral corneal inflammation occurring in the absence of an underlying systemic disease.

Diagnosis of Mooren's Ulcer

The diagnosis of Mooren's ulcer is a clinical diagnosis established based on the characteristics of the corneal lesion, the disease course, and the exclusion of other causes of peripheral corneal ulceration. To date, there is no specific laboratory test to confirm the diagnosis of Mooren's ulcer; therefore, the diagnostic approach relies primarily on clinical evaluation and ancillary testing to rule out other conditions (Najdawi et al., 2025; Howaldt et al., 2025; Kochaji, 2026). The classic clinical presentation of Mooren's ulcer involves a peripheral corneal ulcer originating at the limbus, exhibiting a circumferential or crescentic shape, and accompanied by inflammatory infiltration, severe pain, photophobia, conjunctival hyperemia, and progression toward the central cornea. A key characteristic is the presence of an overhanging ulcer edge resulting from the destruction of the corneal stroma proceeding from the periphery toward the center. Unlike infectious ulcers, Mooren's ulcers typically do not present with predominant purulent infiltrates or signs of active infection (Höllhumer, 2022). Evaluation of suspected Mooren's ulcer begins with a meticulous slit-lamp examination to assess the location, depth, and morphology (including edge characteristics) of the lesion, limbal involvement, the depth and extent of corneal thinning, and the presence or absence of scleritis or episcleritis. Anterior segment OCT (AS-OCT) can be used to objectively determine corneal thickness and the depth of the stromal defect, while also facilitating the monitoring of healing. Fundus examination is performed to assess the posterior segment, whereas B-scan ultrasonography may be used if media opacities obscure the view of the fundus. Corneal scrapings for staining and culture should be performed if an infectious etiology is suspected. These examinations assist in differentiating Mooren's ulcer from infectious keratitis and other causes of peripheral corneal ulceration (Ou et al., 2025). Furthermore, a systemic history and examination are required to rule out autoimmune diseases and infections that could cause peripheral ulcerative keratitis (PUK). Laboratory tests that may be considered include complete blood count, ESR/CRP, renal

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and hepatic function tests, urinalysis, rheumatoid factor (RF), anti-cyclic citrullinated peptide (anti-CCP), antinuclear antibody (ANA), and antineutrophil cytoplasmic antibody (ANCA). Depending on clinical indications, serological testing for syphilis, hepatitis B and C, and HIV, as well as tests for tuberculosis (such as QuantiFERON), may be performed. These tests are not intended to confirm a diagnosis of Mooren's ulcer; rather, their primary purpose is to rule out systemic diseases, infections, and other conditions that could present with a clinical picture resembling peripheral ulcerative keratitis (PUK). (Ou, et al., 2025), (Lucchino, et al., 2024).

Table 3. Diagnostic examinations for Mooren's ulcer (MU)

Type of Examination	Specific Tests/Procedures	Potential Objectives and Findings
Eye examination	Slit-lamp biomicroscopy	Identifies peripheral corneal ulceration, epithelial defects, and the characteristic overhanging edge of Mooren's ulcer. Helps rule out infectious keratitis or traumatic ulcers.
	AS-OCT (anterior segment optical coherence tomography)	Visualizes corneal structures in detail, including stromal thinning and the depth of ulceration.
	Fundus examination	Supports diagnosis and monitoring of healing. Helps rule out deeper infectious or structural pathologies.
Image building	Chest X-ray	Assesses the possibility of systemic diseases such as tuberculosis (TB) or sarcoidosis.
	B-scan ultrasound	Used when media opacity obstructs fundus examination, particularly in severe uveitis.
	Sinus and KUB X-rays	Performed to rule out other causes of systemic inflammation.
Laboratory examination	Complete blood count (CBC)	Assesses systemic inflammation, anemia, and infection. Helps rule out systemic causes of ulceration.
	ESR and CRP	Marker of systemic inflammation; may be elevated in some patients with MU. Helps distinguish MU from systemic autoimmune diseases.
	Hepatitis B and C serology	Investigates a possible viral link to MU. Some cases show positive Hepatitis C results.
	HIV serology	Assesses for immunosuppression as a potential contributing factor.
	Urinalysis and stool examination	Assesses for systemic infection or parasitic infestation.
	Liver and kidney function tests	Evaluates systemic health status and rules out contributing metabolic factors.
	Serum ACE and QuantiFERON test	Assesses for possible sarcoidosis and tuberculosis.
	Corneal scraping and culture	Ruling out infectious keratitis. All cases undergoing this examination showed negative microbiological results.

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	Corneal or conjunctival biopsy	Histological confirmation of MU characteristics. Helps rule out neoplastic or infectious causes.
	Autoimmune markers	Ruling out autoimmune diseases that can mimic or contribute to PUK. Results were negative in all MU cases, supporting an idiopathic etiology.

Table 4. Autoimmune Serology Panel to Rule Out Diseases in the Differential Diagnosis of Mooren's Ulcer

Autoimmune marker	Diseases Ruled Out
ANA (Antinuclear antibodies)	Systemic sclerosis, Sjögren's syndrome, systemic lupus erythematosus (SLE)
ANCA (Anti-neutrophil cytoplasmic antibodies)	Granulomatosis with polyangiitis, microscopic polyangiitis, inflammatory bowel disease (IBD)-associated vasculitis
RF (Rheumatoid factor)	Rheumatoid arthritis
Anti-CCP (Anti-cyclic citrullinated peptide)	Rheumatoid arthritis
Anti-dsDNA (Double-stranded DNA antibody)	Systemic lupus erythematosus (SLE)
SSA/Ro (Anti-Ro antibodies)	Systemic lupus erythematosus (SLE), Sjögren's syndrome
SSB/La (Anti-La antibodies)	Sjögren's syndrome
Cardiolipin antibodies	Antiphospholipid syndrome, systemic lupus erythematosus (SLE)
Complement (C3, C4)	SLE, immune complex-mediated disease, infection-associated immune activation
MPO (Myeloperoxidase antibody)	Microscopic polyangiitis
PR3 (Proteinase 3 antibody)	Granulomatosis with polyangiitis
Lupus anticoagulant	Antiphospholipid syndrome
RNP (Anti-ribonucleoprotein antibody)	Mixed connective tissue disease

Note: MU = Mooren's ulcer; SLE = systemic lupus erythematosus; IBD = inflammatory bowel disease..

In this case, the diagnosis of Mooren's ulcer was established based on the presence of a progressive peripheral corneal ulcer in the left eye, complaints of severe pain and reduced visual acuity, and the absence of any history of trauma, contact lens use, prior infection, or systemic autoimmune disease. These clinical findings are consistent with the characteristics of Mooren's ulcer, with peripheral ulcerative keratitis (PUK) serving as the primary differential diagnosis. B-scan ultrasonography, which showed the posterior segment to be within normal limits, also helped rule out intraocular complications such as retinal detachment or vitreous hemorrhage (Baquet-Walscheid et al., 2024).

Although immunological tests—such as ANA, ANCA, and rheumatoid factor—are part of the evaluation for peripheral ulcerative keratitis to rule out systemic autoimmune disease, they were not performed in this case due to facility limitations and the absence of clinical manifestations suggestive of autoimmune disease. Therefore, the diagnosis of Mooren's ulcer was established based on characteristic clinical features and the exclusion of infectious causes and systemic diseases through patient history, physical examination, and available ancillary tests. A similar approach has been reported in other Mooren's ulcer cases where immunological testing was unavailable.

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Principles of Mooren's Ulcer Management

Managing Mooren's ulcer remains challenging due to the disease's variable clinical course and the lack of a universally standardized treatment protocol. The primary goals of therapy are to control the inflammatory process, halt the progression of corneal destruction, preserve the anatomical integrity of the eyeball, and optimize visual function. Treatment generally follows a stepwise approach (stepladder approach) based on disease severity, the extent of corneal involvement, response to initial therapy, and the risk of complications such as corneal melting and perforation (Priscilia et al., 2026).

In mild to moderate cases, conservative therapy using anti-inflammatory and immunosuppressive agents is the initial choice. Topical corticosteroids serve as first-line therapy due to their ability to suppress inflammatory cell activation and the production of pro-inflammatory cytokines, which contribute to corneal tissue damage. Additionally, immunomodulatory agents such as topical cyclosporine A can be used to inhibit T-lymphocyte activation and reduce local autoimmune responses. Artificial tears and bandage contact lenses may also be employed to enhance patient comfort and support corneal epithelialization (Sabhapandit et al., 2022).

In more aggressive disease—particularly in bilateral or progressive cases, or those unresponsive to topical therapy—systemic immunosuppressive therapy is required. Oral corticosteroids, methotrexate, azathioprine, cyclophosphamide, and certain biological agents have been reported for use in refractory cases. However, the use of systemic therapy necessitates consideration of side effects and must be tailored to the patient's clinical condition (Alberta, 2022). Surgical intervention is considered if the ulcer progresses despite maximal medical therapy, or in the presence of significant corneal thinning, corneal necrosis, or a risk of perforation. Surgical modalities include conjunctival resection, keratectomy, amniotic membrane transplantation, lamellar keratoplasty, and penetrating keratoplasty for cases involving severe corneal destruction. In this instance, conjunctival resection combined with double-layer amniotic membrane transplantation (AMT) was selected due to the presence of a progressive peripheral ulcer carrying a risk of further damage to corneal structures (Fu & Jones, 2026).

The Role of Conjunctival Resection in Mooren's Ulcer

Conjunctival resection is a surgical procedure that has long been employed in the management of Mooren's ulcer, particularly in cases suspected of involving a local autoimmune mechanism. The rationale for this procedure stems from the hypothesis that inflamed perilimbal conjunctival tissue serves as a source of various immune mediators, antibodies, and proteolytic enzymes that drive corneal stromal destruction. Consequently, resecting the conjunctiva adjacent to the ulcer aims to eliminate or reduce the tissue responsible for this immune response and the associated destructive mediators; this is expected to alleviate local inflammation and halt the progression of the ulcer (Guney et al., 2021; Sabhapandit et al., 2022).

Pathophysiologically, immunologically activated conjunctiva can produce various inflammatory mediators such as pro-inflammatory cytokines and increase the activity of collagenolytic enzymes, including matrix metalloproteinases (MMPs). These enzymes cause the degradation of the extracellular matrix and corneal collagen, leading to progressive corneal thinning. Therefore, resecting the affected conjunctival tissue aims to reduce antigenic exposure and inhibit the infiltration of immune cells into the damaged corneal area (Guney et al., 2021).

Several reports indicate that conjunctival resection can yield favorable results in controlling the progression of Mooren's ulcer. Brown et al. reported that excising a 3–4 mm wide ring of limbal conjunctiva adjacent to the ulcer in 10 eyes with progressive Mooren's ulcer resulted in healing in 8 eyes within three weeks. However, recurrence remains a possibility, and

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subsequent studies have shown that conjunctival resection does not always prevent recurrence or definitively halt the disease; thus, the procedure's effectiveness remains a subject of debate (Brown, 1975). The limitations in the evidence are primarily due to the small number of cases and the variability in patient characteristics. Therefore, conjunctival resection is often combined with other therapies, particularly in cases with extensive corneal damage (Guney et al., 2021)

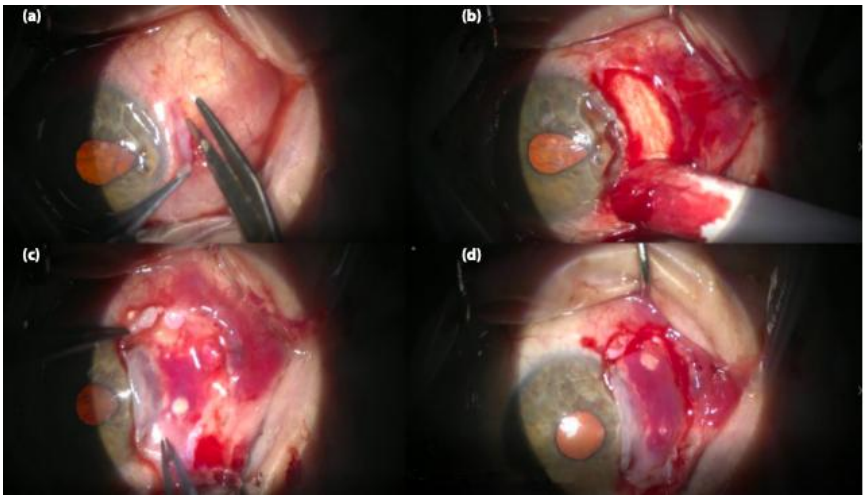


Figure 5. Schematic illustration of the conjunctival resection and double-layer amniotic membrane transplantation procedure (adapted from Guney et al., 2021)

In this patient, conjunctival resection was performed to control the source of local inflammation, aiming to reduce the source of immune mediators and proteolytic enzymes suspected of driving progressive corneal destruction. This procedure was combined with AMT to provide structural support to the corneal defect and accelerate ocular surface regeneration (Guney et al., 2021).

The Role of Double-Layer Amniotic Membrane Transplantation (AMT)

Amniotic Membrane Transplantation (AMT) is an ocular surface reconstruction technique widely used for various corneal conditions, including non-infectious corneal ulcers, persistent epithelial defects, corneal melting, and conditions at risk of perforation. The amniotic membrane possesses various biological characteristics that support tissue healing, such as anti-inflammatory, anti-fibrotic, and anti-angiogenic effects, as well as the ability to enhance corneal epithelial cell migration and differentiation (Walkden, 2020).

The anti-inflammatory effect of the amniotic membrane stems from its ability to suppress the expression of pro-inflammatory cytokines, such as interleukins and tumor necrosis factor-alpha (TNF-α). Additionally, the amniotic membrane contains various growth factors that aid in epithelial regeneration, accelerate corneal defect closure, and reduce scar tissue formation. These characteristics make AMT a suitable therapeutic choice for corneal diseases involving active inflammation, such as Mooren's ulcer (Baranwal et al., 2021).

The double-layer AMT technique offers advantages over single-layer amniotic membrane placement, particularly in cases involving deeper stromal tissue loss. In this technique, the first membrane layer acts as a filler or graft to fill the stromal defect and provide structural support, while the second layer serves as a biological dressing that covers the corneal surface and supports the epithelialization process. This approach provides superior mechanical stability to the thinned cornea, thereby reducing the risk of progression to perforation (Ou et al., 2025). In Mooren's ulcer, the use of AMT serves not only to cover the defect but also to modulate the corneal inflammatory environment. By reducing local inflammation and providing a biological matrix for epithelial regeneration, AMT can help maintain corneal integrity and serve as an

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alternative prior to more invasive corneal reconstruction procedures, such as keratoplasty (Walkden, 2020)..

In this case, Amniotic Membrane Transplantation (AMT) was performed as a reconstructive procedure to support the healing of a cornea damaged and thinned by ulceration. The amniotic membrane possesses a basement membrane that acts as a biological scaffold for corneal epithelial cell adhesion, migration, and proliferation, thereby facilitating re-epithelialization. Beyond this structural role, the amniotic membrane exerts anti-inflammatory, anti-fibrotic, and anti-angiogenic effects; these help create an ocular surface environment conducive to healing while limiting inflammation and scar tissue formation. Thus, for this patient, AMT served not merely to cover the corneal defect but also to provide biological support for corneal surface regeneration (Ahmed et al., 2025).

For deep corneal defects involving stromal tissue loss, the double-layer AMT technique provides both structural support and surface protection. The first layer is placed at the base of the defect as an inlay or filler to compensate for tissue loss and support the thinned cornea. The second layer is placed over the defect as an overlay or biological dressing to protect the damaged area and provide a substrate that supports epithelial migration and regeneration. Consequently, the two layers serve complementary functions: the first primarily supports the deeper stromal defect, while the second helps protect the surface and facilitates epithelialization. This approach is particularly relevant for patients with ulcers causing significant corneal thinning and deep tissue loss (Ahmed et al., 2025).

In this patient, the combination of conjunctival resection and double-layer AMT served complementary purposes. Conjunctival resection aimed to reduce the local inflammatory component suspected of driving the progression of Mooren's ulcer, whereas the double-layer AMT aimed to fill and support the thinned corneal defect, protect the corneal surface, and provide a biological environment conducive to re-epithelialization and healing. Thus, the primary objective of this combined approach is to control ulcer progression, preserve corneal integrity, and support the healing process, rather than solely to directly improve visual acuity. While the literature supports the biological and reconstructive functions of AMT, specific evidence regarding the combination of conjunctival resection and AMT for Mooren's ulcer remains limited and stems primarily from case reports or case series. Therefore, the outcomes observed in this patient must be interpreted with caution and cannot be generalized to all patients with Mooren's ulcer. (Ahmed et al., 2025)

Visual Acuity Progression and Postoperative Healing Process

The improvement in visual acuity from 1/300 in the early postoperative period to 1/60 at the 21-day follow-up likely reflects the gradual recovery of the ocular surface and cornea. In the early postoperative phase, factors such as inflammation, perilimbal ciliary flush, blepharospasm, and corneal surface opacity and irregularity can limit visual function. In this case, visual acuity remained at 1/300 until the 8-day follow-up while the cornea still appeared cloudy and subsequently improved to 1/60 by day 21 as the conjunctival ciliary flush subsided, despite persistent superior corneal opacity. A pattern of delayed visual improvement in the early postoperative period was also reported by Al Motowa and Al Zobidi (2015) in patients with Mooren’s ulcer undergoing Amniotic Membrane Transplantation (AMT); in their study, visual acuity on the first postoperative day remained unchanged from the preoperative state but improved to 20/160 (with pinhole) at the four-week postoperative evaluation (Motowa & Zobidi, 2015). In Mooren’s ulcer, the perilimbal conjunctiva is thought to play a role in the process of corneal destruction, as it contains antibodies and enzymes that can contribute to corneal damage. Therefore, conjunctival resection may be considered to limit immune infiltration and assist in controlling inflammation and the ulcer healing process (Ou et al., 2025). Furthermore, Amniotic Membrane Transplantation (AMT) is known to facilitate

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epithelial cell adhesion and migration, induce epithelial differentiation, and suppress interleukin secretion, thereby helping to reduce corneal inflammation (Ou et al., 2025). REVISION NO. 5: Thus, in this case, the improvement in visual acuity is likely the multifactorial result of inflammation control following conjunctival resection and the corneal surface healing process supported by AMT. The patient's visual acuity improved from 1/300 to 1/60 by day 21. This change is likely associated with the resolution of inflammation, the restoration of corneal integrity and surface quality, and the epithelialization process facilitated by AMT. Amniotic membrane possesses anti-inflammatory properties and supports the migration, adhesion, and differentiation of corneal epithelial cells; consequently, corneal surface repair can contribute to improved visual function (Ahmed et al., 2025). Improvements in visual acuity following AMT have also been reported in various cases of corneal ulcers, with a meta-analysis showing a vision improvement rate of 53% (Liu et al., 2019). Multicenter studies have also reported that 92% of patients with non-healing corneal ulcers experienced stable or improved visual acuity after AMT (Ramos et al., 2021). However, a direct causal link between the procedure and the improvement in visual acuity cannot be definitively established, as the patient still exhibited corneal opacity, pupillary synechiae, and lens opacity on day 21 factors that could potentially limit visual acuity.

Limitations

This case report has several limitations. More comprehensive immunological and serological tests such as antinuclear antibody (ANA), antineutrophil cytoplasmic antibody (ANCA), rheumatoid factor (RF), anti-cyclic citrullinated peptide (anti-CCP), and serological screening for various infectious diseases could not be performed due to limited testing facilities at the hospital where the patient received care. Such tests are crucial, particularly for ruling out systemic autoimmune diseases and infections that can cause peripheral ulcerative keratitis (PUK) and present with clinical features resembling Mooren's ulcer. Given that the diagnosis of Mooren's ulcer is primarily one of exclusion, the unavailability of certain tests means the possibility of subclinical systemic disease cannot be entirely ruled out. Nevertheless, the diagnosis in this patient is supported by the clinical presentation of a progressive peripheral corneal ulcer featuring characteristic overhanging edges and perilimbal involvement as well as the disease course and the absence of any history of systemic disease or infectious factors based on the available medical history and examinations. Furthermore, the observation period in this report was relatively short extending only to postoperative day 21 and thus does not reflect long-term outcomes, including the potential for Mooren's ulcer recurrence. As this is a single case report, the results of the combination therapy involving conjunctival resection and double-layer amniotic membrane transplantation (AMT) cannot be generalized to all patients with Mooren's ulcer. These limitations should be taken into account when interpreting the clinical outcomes observed in this patient.

The authors hope that future evaluations of patients with Mooren's ulcer will incorporate more comprehensive immunological and serological testing to strengthen the diagnosis of exclusion and identify potential underlying systemic diseases. Long-term follow-up is also necessary to assess corneal stability, changes in visual acuity, and the risk of recurrence following conjunctival resection and amniotic membrane transplantation (AMT). Given that Mooren's ulcer is a rare disease and evidence regarding the efficacy of medical and surgical interventions remains limited, further research involving larger patient cohorts, multicenter studies, and comparative analyses is required to evaluate various therapeutic modalities and establish more standardized management strategies. Future research could also explore immunological biomarkers and novel diagnostic methods that might help distinguish Mooren's ulcer from other causes of peripheral ulcerative keratitis (PUK)..

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Conclusion

Mooren's ulcer is a rare peripheral corneal inflammatory disease characterized by a high potential for progression and the risk of permanent vision loss. Diagnosis is established based on clinical findings specifically a progressive peripheral corneal ulcer and severe ocular inflammation in the absence of identifiable infectious causes or underlying systemic diseases. Management requires a tailored approach based on disease severity, ranging from medical therapy to control inflammation to surgical intervention for progressive cases or those unresponsive to conservative treatment. In this case, a combination of conjunctival resection and double-layer amniotic membrane transplantation (AMT) was selected to control the source of local inflammation while preserving corneal structural integrity. Conjunctival resection serves to reduce tissue acting as a source of inflammatory mediators, while the double-layer AMT technique provides mechanical support for the corneal defect and facilitates epithelial regeneration through anti-inflammatory and reparative effects. This combined approach offers a viable therapeutic alternative for progressive Mooren's ulcer cases at risk of severe corneal damage, although long-term evaluation remains necessary to assess disease stability and visual outcomes.

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