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Mooren's Ulcer Following Salmonella Gastroenteritis: A Case Report

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Introduction

Peripheral ulcerative keratitis (PUK) is a potentially sight-threatening condition. The peripheral cornea has a predilection to be ulcerated in association with a variety of inflammatory disorders because of its anatomic position, especially the presence of blood vessels and lymphatic channels in the adjacent conjunctiva. PUK can be the result of local and systemic infectious disorders. Noninfectious causes of PUK include trauma, and neurologic, neoplastic, and autoimmune disorders such as rheumatoid arthritis, Granulomatosis with Polyangiitis (formerly called Wegener's), relapsing polychondritis, systemic lupus erythematosus, and polyarteritis nodosa. PUK may be the presenting manifestation of life-threatening systemic vasculitis and therefore is an indication for immunosuppressive therapy, if such a disorder is present or discovered. Duke-Elder described marginal corneal ulcers in patients with systemic infection including bacillary dysentery.

In this study, we report a patient who developed bilateral Mooren's ulcer type of PUK following Salmonella gastroenteritis.

Case Report

A 78-year-old white man was referred to our clinic for light sensitivity, pain, and redness in both eyes. He had been treated with topical ciprofloxacin for one month, with minimal improvement. The patient's best-corrected visual acuity was counting fingers at 2 feet and 20/100, in the right and left eye respectively. Slit-lamp examination revealed bilateral peripheral ulcerative keratitis: 360° involvement extending 3 mm into the cornea in the right eye and 270° involvement extending 2 mm into the cornea in the left eye with no scleral involvement. The lesions in both eyes extended to approximately 80% of the corneal depth and lacked overlying epithelium. Peripheral corneal neovascularization extending into the

ulcer beds was noted into both eyes. The rest of the ocular examination was normal. The past ocular and medical history was unremarkable, except for a severe, culture-positive *Salmonella* diarrheal illness six weeks prior to presentation. Due to extensive progression of peripheral ulcerative keratitis in the right eye, conjunctival resection, keratectomy, and cyanoacrylate application with soft contact lens placement were performed.

A complete laboratory work-up for keratitis was performed. This included a complete blood count, urinalysis, rheumatoid factor, antinuclear antibody, antineutrophil cytoplasmic antibody, hepatitis B and hepatitis C (HCV) serology, circulating immune complexes measured by C1q and Raji cell assays were negative. Erythrocyte sedimentation rate, C-reactive protein, and interleukin 2 receptor levels were mildly elevated. HLA typing was negative for HLA-B27.

Histopathology of the resected conjunctiva revealed perivascular inflammatory cell infiltration and microangiopathy. Based on the history, clinical findings, and laboratory results, the patient was diagnosed with Mooren's ulcer and treated postoperatively with topical 1% prednisolone acetate hourly while awake, ofloxacin four times daily, and cyclopentolate 1% three times daily, with gradual tapering and discontinuation. The patient's ocular inflammation gradually subsided, and the PUK slowly resolved in both eyes, with vision improving to 20/100 OD (primarily secondary to cataract) and 20/25 OS.

Discussion

As mentioned earlier, the unique anatomic position and physiological characteristics of the peripheral cornea predispose it to a variety of inflammatory disorders. Compared to the center of the cornea, the peripheral cornea possesses more antigen-presenting cells, mast cells, eosinophils, as well as immunoglobulins and complement components. Additionally, its proximity to conjunctival and episcleral vessels, as well as the dense packing of collagen bundles and lamellae, may help explain the accumulation of immunologic cells and immunoreactants in the peripheral cornea. PUK represents the end result of proteolytic enzymes released by inflammatory cells and their immunoreactants in a variety of inflammatory disorders with diverse etiologies. In the patient we report here, salmonella gastroenteritis was the only significant associated condition.

Duke-Elder described a type of marginal corneal ulceration without accompanying conjunctival inflammation occurring in association with systemic infections such as influenza, brucellosis, and bacillary dysentery. Our patient had culture-confirmed salmonella dysentery two weeks prior to the onset of ocular symptoms, and the PUK was accompanied by severe conjunctival inflammation.

Mooren type corneal ulceration (progressive PUK without associated scleritis, with an undermined advancing edge) was reported in association with HCV infection. Similar to our patient, In another study, two patients with Mooren-type corneal ulcers associated with chronic HCV infection were reported. The ulcers markedly improved with systemic interferon alpha 2b treatment for the HCV infection. The improvement of the ulcers paralleled the normalization of the liver enzyme levels; however, the corneal ulcers in these cases were unlikely to be a result of direct infection of the corneas by HCV since HCV was not detected in the conjunctival specimen of one of this patients. Similarly in our case, the PUK was not the result of direct salmonella infection since scrapings and cultures were negative for salmonella and other infectious agents.

One possible pathogenic mechanism could be the deposition of immune complexes in the limbal vessels or in the peripheral cornea, with resultant release of proteolytic enzymes. We found no elevated circulating immune complexes by C1Q of Raji cell assays in our patient. Another possible mechanism is an autoimmune phenomenon because of molecular mimicry between the infectious agents and self-antigens. For example, molecular mimicry between HLA-B27 and subspecies of Salmonella, Shigella, Klebsiella, and Yersinia was demonstrated. Specifically, a cationic outer membrane protein of salmonella typhimurium shares homology with five amino acids of HLA-B27 in a nonlinear fashion. This molecular mimicry could explain the fact that individuals with HLA-B27 account for 40-80% of those with post-salmonella reactive arthritis.

Despite the fact that our patient was HLA-B27 negative, it is possible that the subspecies of salmonella causing our patient's gastroenteritis shared common epitopes with corneal stromal antigens, triggering an immune response after the salmonella infection, creating antibodies or antigen-antibody complexes that targeted corneal cells.

Treatment of PUK and Mooren's ulcer follows a "stepladder" approach combining medical and surgical interventions to halt inflammation and prevent perforation. Initial treatment involves intensive topical steroids, cycloplegics, and antibiotics. Progressive cases require conjunctival resection, systemic immunosuppression (e.g., conventional immunomodulatory therapy, biologic response modifiers, and cyclophosphamide), and tectonic surgery for corneal repairs.

You can find more detailed information on PUK and Mooren's ulcer in the references.

References:

1) Hassanpour K, H ElSheikh R, Arabi A, R Frank C, M Elhusseiny A, K Eleiwa T, Arami S, R Djalilian A, Kheirkhah A. Peripheral Ulcerative Keratitis: A Review. J Ophthalmic Vis Res. 2022;17(2):252-275.

2) Maleki, A.; Valerio, T.; Massoudi, Y.; Ruggeri, M.L.; Foster, C.S.; Anesi, S.D. Updates on Systemic Immunomodulation in Peripheral Ulcerative Keratitis. *J. Clin. Transl. Ophthalmol.* 2024, *2*, 131-139.
3) Alhassan MB, Rabiu M, Agbabiaka IO. Interventions for Mooren's ulcer. Cochrane Database Syst Rev. 2014;2014(1):CD006131.
4) Ou S, Zhang Y, Feng Y, Zheng X, Lin Y, Zhang L, Zhao S, Su Y, Cai H, Lin L, Gu H, Wu H, Wu Y. Mooren's ulcer: a multifactorial autoimmune peripheral ulcerative keratitis and current treatment protocols. Front Med (Lausanne). 2025;12:1630585.

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