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NON-SURGICAL MANAGEMENT IN BILATERAL TOTAL LIMBAL STEM CELL DEFICIENCY IN ATOPIC DERMATITIS: A SUCCESSFUL OUTCOME

Ophthalomology

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ABSTRACT

Purpose: To report a case of successful non-surgical management of atopic keratoconjunctivitis (AKC) with bilateral total limbal stem cell deficiency (LSCD). **Methods:** This is a retrospective case report describing successful conservative management of a sight-threatening bilateral total limbal stem cell deficiency in atopic keratoconjunctivitis. **Results:** A 53-year-old man, a known case of atopy, presented with complaints of pain, watering, severe itching, photophobia, lid swelling and non-ambulatory vision in both eyes of 5 years duration. Diagnosed as a case of atopic dermatitis, atopic keratoconjunctivitis with bilateral total limbal stem cell deficiency. Patient managed conservatively with topical and systemic steroids and steroid-sparing agent with remarkable improvement in symptoms, visual acuity, and quality of life. **Conclusions:** Total LSCD is a sight-threatening complication of atopic keratoconjunctivitis often requiring surgical interventions. But, rigorous follow-up, outstanding patient compliance and inter-departmental collaboration are crucial to the successful conservative management of total limbal stem cell deficiency in atopic keratoconjunctivitis.

KEYWORDS

Atopic dermatitis; Atopic keratoconjunctivitis; Cornea; Limbal stem cell deficiency

INTRODUCTION

Atopic keratoconjunctivitis (AKC) is a bilateral, non-infectious, chronic inflammatory eye disease with an incidence of <1% to 8% among adults, most often occurring between the late teens and fifth decade of life.[1] About 87% and 95% of these cases are associated with bronchial asthma and atopic dermatitis, respectively, with a definitive history of atopy among vast majority of cases.[2] The symptoms of itching, tearing, burning of eyes and severe eczema of eyelids and periorbital skin, wax and wane, with variable progression.[1,3] Patients with poor compliance to medications or aggressive disease often develop limbal stem cell deficiency (LSCD) with consequent corneal neovascularization in about 60% of cases, resulting in conjunctivalization of the cornea, corneal opacity and visual loss.[4] Although early cases present with mild symptoms that can be adequately managed with conservative management, advanced cases with 360^{o} limbal stem cell deficiency present with significant risk of progressive corneal damage, visual impairment and poor quality of life.[5] These advanced, recalcitrant cases may require deep anterior lamellar keratoplasty or high-risk penetrating keratoplasty with ocular surface limbal stem cell transplantation for visual rehabilitation. These procedures for ocular surface reconstruction are challenging requiring detailed preoperative evaluation and management, intricate surgical techniques, and comprehensive postoperative medical management for successful outcome.[4,6] There are only anecdotal reports of visual improvement in severe cases of AKC with total LSCD using non-surgical approaches such as oral or topical Cyclosporin A and Topical Tacrolimus.[6-8] Advanced cases of total LSCD associated with atopic keratoconjunctivitis have very poor visual outcome with conservative management.[9] Herein, we report a rare case of bilateral 360-degree limbal stem cell deficiency, managed non-surgically with a successful outcome.

Case Report

A 53-year-old man, a known case of atopic dermatitis and bronchial asthma for 12 years and 15 years respectively, presented with complaints of pain, watering, severe itching, photophobia, lid swelling and diminution of vision in both eyes of 5 years duration. Initially, the symptoms would wax and wane with symptom-free periods, but symptoms increased in frequency and severity over past 2 years, with no symptom-free period, indicating progression of the disease. Visual acuity dropped drastically over 2 weeks prior to presentation. Histopathologically-proven atopic dermatitis involved the entire body surfaces including the periorbital areas.

On ocular examination, patient had best corrected visual acuity (BCVA) of 3/60 in right eye (RE) and hand movements close to face with accurate projection of rays in left eye (LE). Right eye examination revealed gross lid oedema, circumcorneal congestion, features suggestive of 360 LSCD (conjunctival epithelial migration onto cornea, vascularization and inflammation), and Grade III shield ulcer. (**Figure 1**). Similarly, LE examination showed gross lid oedema, features suggestive of partial LSCD 360^{o} LSCD and Grade III shield ulcer (**Figure 2**).

For atopic dermatitis, patient was receiving treatment in form of oral Prednisolone 25 mg daily during active stage followed by slow tapering dosage along with oral Azathioprine 50 mg two times a day for maintenance. Oral antacids and calcium were given as well. Ocular features were managed with topical steroids (Eyedrop Predinsolone acetate 1% 12 times a day with a weekly tapering dosage), cycloplegic (Eyedrop 2% Homatropine 2 times a day) and preservative-free lubricant (Eyedrop Genteal 12 times a day).

Debridement was done for Shield ulcers in both eyes. Initially, patient was scheduled for a weekly follow-up which was gradually extended to a 3-weekly follow-up subsequently. With an elaborate treatment plan and strict follow-up, the patient's symptoms gradually improved with a non-surgical approach, using systemic steroids, steroid-sparing agents along with topical medications. The patient did not develop any local or systemic complications, either from the disease or the aggressive treatment measures.

At 5 months from the first visit, the patient improved both subjectively and objectively. BCVA improved to 6/18 in RE and 1/60 in LE respectively. RE was quiet with multiple, irregular, superficial scars of varying sizes without any features of LSCD and with complete resolution of Shield ulcer (**Figure 3**). Similarly, LE showed complete resolution of LSCD and Shield ulcer, although a superficial corneal scar persisted involving the pupillary area (**Figure 4**). Subsequently, deep anterior lamellar keratoplasty (DALK) is planned in the left eye. Patient's quality of life improved significantly with improvement in visual acuity and resolution of symptoms.

Patient had non-ambulatory vision at first visit and was assisted by a friend, with steady improvement to ambulatory vision over 5 months.

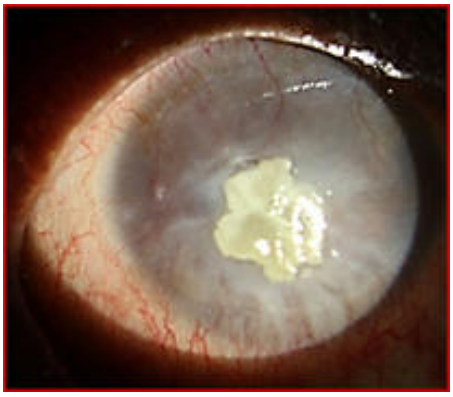

Figure 1: 360°LSCD with Stage III Shield Ulcer in RE

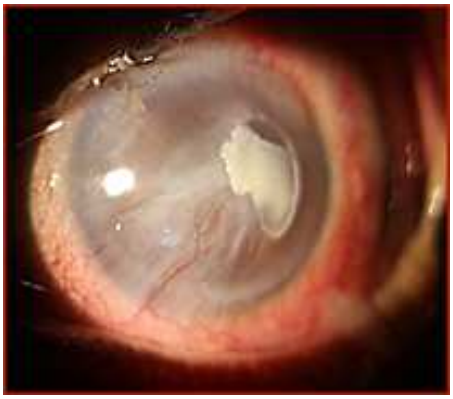

Figure 2: 360°LSCD with Stage III Shield Ulcer in LE

Figure 3: Resolution by non-surgical management in RE

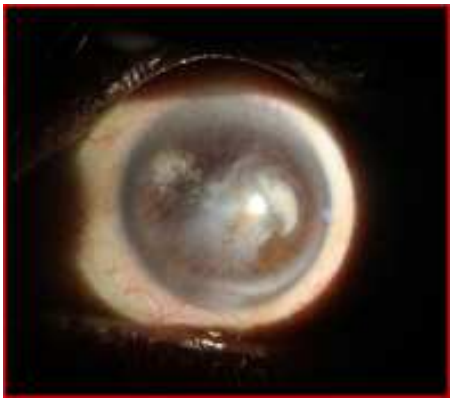

Figure 4: Resolution by non-surgical management in LE

DISCUSSION

AKC is bilateral, chronic inflammatory eye disease among patients with atopic dermatitis caused by both IgE-mediated type-I and type-IV delayed hypersensitivity reactions characterized by progressive cicatrizing conjunctivitis with corneal neovascularization and opacification secondary to chronic epithelial disease and limbal stem cell dysfunction.[6] Limbal stem cell deficiency (LSCD), either diffuse (total) or sectoral (partial), is a common and severe complication affecting cases of atopic keratoconjunctivitis.[3] Recognizing LSCD is an important step in the surgical management of corneal opacity because of the high rates of graft failure in eyes with LSCD.[9] LSCD eventually leads to visual impairment, even blindness in advanced cases. The management of mild to moderate atopic keratoconjunctivitis hinges on topical and systemic antiallergics, and on occasion, topical and systemic steroids. However, the management of severe cases involves frequent use of systemic steroids, and steroid-sparing agents in addition to topical measures.[6] Currently, optimal topical therapy includes the use of topical calcineurin inhibitors like ciclosporin and tacrolimus. Systemic therapy is widely used in specialized centres but without sufficient literature to support its clinical use.[2] Advanced cases with 360° LSCD and complications like shield ulcer, corneal perforation, and total corneal opacification, require definitive surgical management such as DALK, ocular surface limbal stem cell transplantation, therapeutic penetrating keratoplasty and keratoprosthesis.[4]

The waxing and waning pattern of the disease, its chronic clinical course, strict therapeutic and follow-up regimens, and poor patient compliance make the management of atopic keratoconjunctivitis challenging for both the doctor and the patient.[10] Accompanying dermatological symptoms complicate its management, with the patient having to follow up with two or more specialists. While partial LSCD may be tackled by mechanical debridement of conjunctiva-like corneal epithelium with or without amniotic membrane transplantation, topical medications and local measures, a robust team effort with relentless patient co-operation to a comprehensive, non-surgical treatment plan is key to a favourable outcome in cases with total LSCD.

Eyes affected by AKC that have developed visual impairment from LSCD often require surgical intervention for visual rehabilitation.[9] Total limbal stem cell deficiency requires corneal surface reconstruction, achieved by limbal auto- or allotransplantation, often combined with or followed by keratoplasty. This eradicates altered corneal epithelium and pannus, replacing it with healthy corneal epithelium from the donor limbus.[11] Although a novel technique, simple limbal epithelial transplantation (SLET) has shown promising results in patients with ocular burns, evidence for its clinical application in the treatment of total LSCD in conjunction with AKC is not known.[12] Penetrating keratoplasty in cases of AKC is associated with high incidence of graft failure (50%) even though there was significant improvement in visual acuity in some studies.(4) However, the studies were limited in their scope and hence, PK is reserved for cases with significant corneal scarring, vascularization or perforation.[13] Favourable outcomes following non-surgical management of advanced atopic keratoconjunctivitis with 360 LSCD are anecdotal.[1,6,8] In this rare case, it is noticed that meticulous follow-up with conservative therapeutic measures, inter-disciplinary effort and good patient compliance resulted in an acceptable outcome, without the side-effects associated with a more invasive therapeutic management.

CONCLUSION

Atopic keratoconjunctivitis is a potentially blinding ocular condition with disabling symptomatology and complications that requires a comprehensive treatment plan, cohesive collaboration between specialists and relentless patient counselling. We present a case of 360° LSCD in a case of atopic keratoconjunctivitis, who was successfully managed conservatively with an acceptable visual outcome. Despite total LSCD, the patient was managed conservatively with systemic and topical steroids, steroid-sparing agents and local measures. Rigorous follow-up protocols and outstanding patient compliance were crucial in the management of this case. Collaborative effort between the ophthalmologist and dermatologist is key to finding the most appropriate treatment regimen to manage both dermatological and ocular manifestations of atopic keratoconjunctivitis, especially in the management of advanced cases of AKC. Newer surgical options like SLET for the management of LSCD is promising, but needs studies validating its use in LSCD due to AKC.

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