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Ocular complications in patients with Atopic Dermatitis – Review Article

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ABSTRACT

Atopic dermatitis is a chronic inflammatory disease that affects skin but is frequently associated with a variety of ophthalmological manifestations that can have a significant impact on patients quality of life. This review aims to gather current evidence on the pathophysiology, epidemiology, and clinical spectrum of ocular complications in AD, including eyelid dermatitis, blepharitis, conjunctivitis, keratoconus, cataract, retinal detachment, and dupilumab-associated ocular surface disease (DAOSD). Almost half of AD patients will experience ocular complications with main risk factors being disease duration and involvement of the periocular area. Diagnosis should primarily be based on thorough clinical evaluations and ophthalmological assessment. Management requires use of both topical and systemic drugs, patient education and in the most severe cases surgical intervention. Multidisciplinary cooperation most importantly between dermatologists and ophthalmologists is key for early diagnosis, effective treatment and prevention of complications associated with vision loss. Regular ocular screening is recommended for all patients with AD, especially those who are being treated with dupilumab.

KEYWORDS: atopic dermatitis, conjunctivitis, blepharitis, cataract, glaucoma, keratoconus, keratitis, retinal detachment

INTRODUCTION

Atopic dermatitis (AD) is a chronic inflammatory disease that affects skin but is frequently associated with a variety of ophthalmological manifestations that can have a significant impact on patients quality of life[1]. AD is primarily considered to be a dermatological condition, but it can frequently involve ocular structures - manifestations range from mild symptoms like itching to pathologies such as cataracts and keratoconus which can threaten the vision[2].

Pathophysiology of concomitant skin and ocular symptoms in AD is based on shared **IL4/IL13** signaling pathways that disrupt both cutaneous and ocular barrier. Mentioned cytokines reduce **filaggrin** (filament-associated protein that binds to keratin fibers in epithelial cells) expression in keratinocytes and conjunctival goblet cells which leads to impaired tear film stability and Staphylococcus aureus colonization. This pathology creates a cycle of inflammations that exacerbates ocular surface damage[3].

A new emerging challenge is **dupilumab-associated ocular surface disease (DAOSD)**, that can occur even in 32-50% of the patients receiving this kind of treatment[4,5]. Most often DAOSD manifests itself as chronic inflammation, conjunctival cicatrization and madarosis - all requiring long-term use of topical immunosuppressants. Pathogenesis is dependent on inhibition of IL-13 that causes impaired mucin production by goblet cells[4]. DAOSD is a perfect example of delicate balance between systemic AD control and preserving the homeostasis of ocular surface.

This review aims to synthesize current evidence on AD-related ophthalmological manifestations, emphasize the importance of interdisciplinary management strategies and most importantly guide to early diagnosis of vision-threatening complications in this high-risk population.

PATOPHYSIOLOGY

Common Immunological Mechanisms Between AD and Ocular Manifestations

The core of AD pathophysiology is the activation of type 2 helper T-cells (Th2) mediated by IL-4, IL-13 and IL-5. Involvement of these cytokines explains why ocular inflammation is often seen in AD patients[6].

IL-4 and IL-13 cause the recruitment of eosinophils and other white cells to the ocular surface which amplifies the process of inflammation and results in tissue remodeling[6].

IL-4 and IL-13 share a receptor called IL-4Rα which plays a pivotal role in conjunctival inflammation, as demonstrated in both human studies and animal models[6].

Cytokines connected to Th2 immune response induce vascular adhesion molecules (e.g. V-CAM1) and chemokines (e.g. CCL11, CCL24) which helps to recruit inflammatory cells to the conjunctiva[7].

Role of Barrier Dysfunction in Both Skin and Ocular Surface

Barrier dysfunction is a key feature of both AD and ocular complications.

The skin barrier in AD patients is compromised due to reduced expression of structural proteins like filaggrin, which increases susceptibility to allergens and pathogens. Ocular surface barrier is disrupted in a similar fashion - this leads to increased exposure to many different environmental allergens and irritants[7].

Production of tight junction proteins (e.g. ZO-1, E-cadherin) which are responsible for epithelial integrity is downregulated in AD-patients affected by atopic keratoconjunctivitis[7].

Conjunctival epithelium, when treated with fluorescein drops, demonstrates increased permeability in AD patients. This compromised barrier facilitates allergen penetration, triggering immune responses via pathogen recognition receptors and strengthening the process of chronic inflammation[7].

Inflammatory Pathways Involved (IL-4, IL-13 Signaling)

As mentioned before IL-4 and IL-13 are two main cytokines responsible for Th2 mediated inflammation in both AD specific skin lesions and inside the ocular surface.

These cytokines:

1. Lower the expression of structural proteins that are necessary for proper barrier function (e.g. filaggrin)[3].
2. Lower antimicrobial peptide production, which increases susceptibility to infections[3].
3. Increase collagen deposition and tissue remodeling by activating fibroblasts - this contributes to thickened conjunctival tissue[3].

Animal models show that blocking IL-4Rα can decrease conjunctival inflammation by reducing Th2 cytokines production and eosinophil infiltration[8]. However, therapies that target these pathways may also disrupt homeostasis of the ocular surface, as seen in dupilumab-associated ocular surface disease (DAOSD)[9].

Impact on Conjunctival Goblet Cells

Goblet cells are epithelial cells that specialize in secreting mucins that maintain tear film stability. AD patients suffer from significant reduction of goblet cell density compared to healthy controls[9]. This results in a vicious cycle of tear film instability, increased allergen exposure, and chronic inflammation that worsens ocular symptoms over time.

EPIDEMIOLOGY OF OCULAR MANIFESTATIONS IN ATOPIC DERMATITIS

Ocular manifestations are both frequent and significant complication of atopic dermatitis (AD) that affects patients in all age groups. Some studies indicate that almost half of AD patients experience ocular symptoms – prevalence rates range from 25% to 50% depending on the population that was studied and the severity of the disease[10,11]. These complications range from mild such as allergic conjunctivitis to severe vision threatening ones including keratoconus and cataracts[12].

Frequency of ocular involvement increases with the duration of AD and is very often associated with facial skin lesions. Periocular dermatitis is a strong risk factor for ocular complications because it facilitates allergen exposure and colonization of the bacteria on the ocular surface[13]. Even though both children and adults can be affected, ocular complications are more common and severe in adults as a consequence of disease duration and progressive exposure to inflammatory mediators[11].

Allergic conjunctivitis and periocular dermatitis are the most common condition in children. These conditions are often mild but over time they may progress to more severe ones, such as atopic keratoconjunctivitis or keratoconus[11]. In adults, the spectrum broadens to include cataracts, glaucoma, and other intraocular complications[14].

The use of corticosteroids is also linked to ocular complications. Long-term application of topical corticosteroids around the eyes contributes to formation of cataract and ocular hypertension. Using systemic corticosteroids may exacerbate these conditions, especially in patients who have been ill afflicted for a long time[15].

Ocular manifestations may occur not only with severe AD, but also in patients with mild or moderate disease. This proves how important it is to provide regular ophthalmological examinations for patients with AD in order to detect and early treat these complications[12].

CLINICAL MANIFESTATIONS

EYELID INVOLVEMENT

Eyelid Dermatitis and Eczema

Eyelid dermatitis manifests itself in edema, pruritus, erythema and skin peeling around the eyes. In AD it is mostly caused by the thin epidermis, weak skin barrier and heightened sensitivity to allergens.

One of the most common causes of eyelid dermatitis in ADS patients is allergic contact dermatitis (ACD). It may be triggered by irritants and allergens such as metals, fragrances or preservatives in some products. Patch testing is used to identify specific allergens[16].

Lichenification (thickening of the skin) may be caused by chronic scratching which also exacerbates inflammation[11].

Blepharitis

Blepharitis characterized by chronic inflammation of the eyelid margins, occurs in two types: posterior and anterior.

Anterior blepharitis concerns the base of the eyelashes. Most common symptoms are redness, irritation and skin peeling. It is often associated with colonization of *Staphylococcus aureus* or seborrheic dermatitis[17].

Posterior blepharitis involves meibomian gland dysfunction (MGD), which results in dry eye symptoms due to altered tear film composition. In AD patients MGD is exacerbated by chronic inflammation[17].

Potential Complications of Chronic Eyelid Inflammation:

Prolonged eyelid inflammation can lead to eyelash abnormalities such as loss of eyelashes (madarosis), misdirected growth (trichiasis), or discoloration[10].

Another consequence of chronic inflammation is scarring that may lead to ectropion (outward turning) or entropion (inward turning) of the eyelids[10].

Chronic inflammation also leads to dysfunction of meibomian glands which directly contribute to dry eye symptoms and increased irritation of ocular surface[17].

CONJUNCTIVAL MANIFESTATIONS

Allergic Conjunctivitis

Allergic conjunctivitis is the single most common ocular manifestation in AD patients - some studies indicate prevalence of up to 31.7%. This dysfunction is an IgE-mediated hypersensitivity reaction triggered by different allergens such as pollen, dust mites and animal fur. Main symptoms are bilateral conjunctival hyperemia, pruritus, tearing and mild mucous discharge[18].

Atopic Keratoconjunctivitis (AKC)

Atopic keratoconjunctivitis (AKC) is a form of chronic and severe ocular allergy associated with AD. Most often it affects young adults, but it tends to persist throughout life. AKC manifests itself as intense bilateral itching, burning, tearing and mucoid discharge. Examination of the eye might show findings in form of hypertrophy of tarsal conjunctiva, conjunctival scarring and Horner-Trantas dots (chalky white limbal deposits). Involvement of cornea is common and may result in punctate epithelial erosions, corneal ulcers, neovascularization and scarring[19].

Vernal Keratoconjunctivitis (VKC)

Vernal Keratoconjunctivitis (VKC) is a rare form of allergic conjunctivitis. Symptoms mimic other types of conjunctivitis including intense itching, photophobia, tearing and thick mucoid discharge. Key clinical findings include giant papillae on the superior tarsal conjunctiva (cobblestone appearance), limbal Horner-Trantas dots and in the most severe cases shield ulcers in the cornea. When left untreated, VKC leads to corneal scarring and vision impairment[20].

Papillary Reactions and Other Findings

One of the hallmark symptoms in both AKC and VKC is papillary hypertrophy of the conjunctiva. In AKC the hypertrophy is diffuse and involves both the superior and inferior tarsal conjunctiva in contrast to VKC where we find giant papillae that tend to be localized on the superior tarsal conjunctiva[20,21].

CORNEAL COMPLICATIONS

Keratitis

Keratitis (inflammation of the cornea) is another common complication in AD patients, with some studies indicating a prevalence of up to 9.7% in adult population. Patient with keratitis will mainly report pain, photophobia, tearing and gritty sensation in the eyes. In severe cases it may lead to ulceration or scarring of the cornea – risk factors include prolonged disease duration, periocular eczema and frequent use of contact lenses. Early diagnosis and topical anti-inflammatory treatment are critical in preventing permanent vision loss[14].

Keratoconus

Keratoconus is a progressive thinning and protrusion of the cornea leading to a cone-like shape. This directly impacts the vision by causing irregular astigmatism. It is one of the most well-documented corneal complications in AD – characteristic pruritus leads to chronic eye rubbing

which is the most important contributing factor to formation of keratoconus. Prevalence is ranging from 0.5% to even 39%, depending on the populations studied and severity of AD[22].

OTHER OCULAR MANIFESTATIONS

Cataract Formation

AD patients are at risk of developing anterior subcapsular cataracts, which are more specific to AD than posterior subcapsular cataracts which are usually steroid induced. Chronic inflammation induces oxidative lens damage by reduced superoxide dismutase activity in AD patients. Even though steroid therapy exacerbates the risk of cataract development, as evidenced it can develop independently. During disease flares, AD patients may experience rapid-onset cataracts which in severe cases can lead to early surgical intervention[23].

Glaucomatous Changes

Notably, severe AD exacerbates the risk of glaucoma progression, necessitating surgical intervention[24]. Intraocular inflammation may damage trabecular meshwork which results in elevated intraocular pressure (IOP). This emphasizes how important it is to monitor intraocular pressure in AD patients, event without steroid use.

Retinal Detachment

AD patients experience a higher incidence of bilateral rhegmatogenous retinal detachment. The pathogenesis involves eye rubbing and vitreoretinal traction caused by inflammation[25].

Herpetic Eye Disease

AD increases a risk of ocular infections caused by herpes simplex virus (HSV). This includes eczema herpeticum with keratoconjunctivitis. Due to immune dysfunction, viral clearance is damaged which may lead to stromal scarring and prolonged epithelial healing[26].

DUPILUMAB-ASSOCIATED OCULAR SURFACE DISEASE (DAOSD)

Dupilumab is a monoclonal antibody targeting the IL-4 receptor alpha (IL-4Rα) used to treat moderate-to-severe atopic dermatitis. The most common adverse effect of dupilumab is **Dupilumab-associated ocular surface disease**. DAOSD includes a spectrum of inflammatory ocular conditions for instance blepharitis, conjunctivitis, dry eye disease and keratitis[4].

Incidence Rates

The incidence of DAOSD can vary depending on the studied population. In adults clinical report rates of 8.6-22.1% however real-world data indicate higher appearance, ranging from 27% to 62%[4,27,28].

In the pediatric population incidence is higher in adolescents compared to younger children and can range from 5.6% to 34.6%[27,28].

Clinical Presentations and Symptoms

Commonly reported symptoms of DAOSD include pruritus, tearing, redness, photophobia, dryness and foreign body sensation. DAOSD may also manifest with varied signs, such as conjunctival injection, meibomian gland dysfunction, superficial punctate keratitis and papillary reactions. Severe cases may involve conjunctival cicatrization or limbal nodules.

Time Course of Development

Median onset of DAOSD is approximately **13 weeks** in pediatric patients and **4 months** in adults[27,28]. Symptoms may emerge as early as **3–7 weeks**, with some cases presenting up to **16 months** post-initiation[27,28].

Severity Spectrum

The severity of DAOSD ranges from mild to severe[29]. Mild cases are often self-limiting and can be treated with lubricants or artificial tears. More severe cases require anti-inflammatory therapies such as topical corticosteroids or calcineurin inhibitors. The most severe cases involve vision-threatening complications like corneal scarring, which requires discontinuation of dupilumab therapy.

Proposed Pathophysiological Mechanisms for DAOSD

The actual mechanisms of DAOSD remain not fully understood but are studied multiple factors related to IL-4/IL-13 inhibition and ocular surface homeostasis which include following:

1. Dupilumab inhibits IL-13 signaling, which is critical for conjunctival goblet cell proliferation and mucin production. Goblet cell hypoplasia leads to tear film instability and increased susceptibility to inflammation[4].
2. Dupilumab suppresses Th2 cytokines which results in a compensatory shift toward Th1/Th17 immune responses. Elevated levels of IFN-γ and IL-17 lead to ocular surface inflammation and epithelial damage[4].
3. During dupilumab therapy there are changes in the ocular surface that may promote overgrowth of organisms like *Demodex* exacerbating inflammation[4].

DIAGNOSIS AND ASSESSMENT

Condition	Tool	Key Parameters
AD Severity	EASI	Erythema, edema, excoriation, lichenification (0–72 scale)
Ocular Surface Disease	DEQ-5	Dryness, discomfort, visual fatigue (0–22 scale)
Keratitis	NEI Corneal Staining	Punctate epithelial erosions (0–15 scale)
AKC Severity	Oxford Scheme	Conjunctival hyperemia, papillae, corneal involvement

Diagnosing ocular complications in atopic dermatitis (AD) requires a multidisciplinary approach. Key steps include patient history and physical examination. It is important to measure AD severity using tools like Eczema Area and Severity Index (EASI) or SCORing Atopic Dermatitis (SCORAD)[30]. Identifying a risk factors like facial/periocular dermatitis, chronic eye rubbing, prolonged corticosteroid use is of great importance[15]. Assess symptoms, especially itching, redness, tearing, photophobia, or vision changes[11]. Considering the physical examination, special attention should be paid to:

- Eyelids: Check for dermatitis, blepharitis, madarosis, or structural changes (ectropion/entropion)[31].
- Conjunctiva: Look for hyperemia, papillae (giant papillae in AKC), Horner-Trantas dots, or symblepharon[31].
- Cornea: Screen for keratitis, ulcers, vascularization, or keratoconus[31].
- "Red flag" signs warranting urgent ophthalmology referral include corneal opacification, limbal nodules, or suspected herpes simplex keratitis[31].

Table 1. Grading Systems for Severity Assessment

Table 2. Differential Diagnosis Considerations

TREATMENT OF OCULAR MANIFESTATIONS

Treatment can be divided into topical therapies, systemic therapies and surgical interventions. Key focus of topical therapies is to control inflammation and stabilize ocular surface. Preservative-free artificial tears (e.g., hyaluronic acid 0.15–0.3%) improve dryness and reduce mechanical trauma from rubbing. Severe dry eye may require autologous serum eye drops[5]. Topical corticosteroids (e.g., fluorometholone 0.1%): Used short-term (<2 weeks) for acute flares of keratoconjunctivitis or blepharitis. Chronic use risks glaucoma and cataracts[31,32].Calcineurin inhibitors: Tacrolimus 0.03% ointment applied to the periocular area reduces eyelid dermatitis recurrence[5,31]. Cyclosporine 0.05% drops mitigate chronic conjunctival inflammation[32].Antihistamine/mast cell stabilizers: Olopatadine 0.1% or ketotifen 0.025% control allergic conjunctivitis symptoms[31,32].

Condition	Distinguishing Features
Vernal keratoconjunctivitis (VKC)	Seasonal exacerbations, giant tarsal papillae, onset <18 years
Allergic conjunctivitis	No corneal involvement, minimal lid changes, IgE-mediated
Ocular rosacea	Telangiectasia, absence of AD history, facial erythema
Herpetic eye disease	Dendritic ulcers on fluorescein staining, unilateral onset
DAOSD	Onset post-dupilumab initiation, goblet cell loss on confocal microscopy

Systemic therapies are reserved for refractory or bilateral disease. Dupilumab improves AD severity but requires concurrent topical therapy for DAOSD[5,31].Immunosuppressants like oral cyclosporine (3–5 mg/kg/day) reduces Th2-driven ocular inflammation[31,32]

Surgical Interventions are indicated for structural complications[31,33] including keratoconus, cataracts, retinal detachment and corneal scarring.

MANAGEMENT OF DAOSD

Treatment algorithms vary depending on severity of a disease[4,34,35]. In mild cases (symptoms ≤2) main treatment includes preservative-free artificial tears (4–6×/day) and eyelid hygiene.

Topical corticosteroids (e.g., fluorometholone 0.1% 2–4×/day for ≤2 weeks) or calcineurin inhibitors (cyclosporine 0.05% 2×/day) are first-line therapy in moderate cases.

Severe/refractory cases (cicatrization, limbal nodules) require immunomodulators (tacrolimus ointment 0.03% periocular) or systemic therapies (oral doxycycline 100 mg/day for MGD). Ophthalmology referral is mandatory for severe presentations.

Even though long-term or maintenance therapy may be needed, most patients experience symptomatic improvement or complete resolution with these interventions. Fortunately, discontinuation of dupilumab is required only rarely in severe or refractory cases. Effective management of DAOSD usually allows for the continuation of dupilumab therapy[4].

MULTIDISCIPLINARY COLLABORATION

Effective management of ocular manifestations in atopic dermatitis (AD) demands a multidisciplinary approach involving both dermatologists and ophthalmologists.

Dermatologists should routinely ask all AD patients about ocular symptoms such as redness, pain, photophobia, tearing, and vision changes, and perform a basic examination for signs like conjunctival redness, eyelid eczema, or blepharitis. Mild cases (e.g., mild allergic conjunctivitis or blepharitis) can be managed by dermatologists however, it should be emphasized that referral to an ophthalmologist is recommended for moderate-to-severe or persistent symptoms, worsening discomfort, changes in vision, or suspicion of complications such as keratitis, keratoconus, or herpetic eye disease.

Multidisciplinary collaboration provides early diagnosis, timely intervention, and optimal outcomes. Empowering patients with information about ocular risks and clear referral pathways is a key component of compliance.

CONCLUSIONS

Ophthalmological manifestations in Atopic Dermatitis are common and clinically significant, affecting nearly half of patients regardless of age or AD severity[10]. These complications range from mild conditions such as eyelid dermatitis and allergic conjunctivitis to severe, vision-threatening diseases including keratoconus, cataract, retinal detachment, and dupilumab-associated ocular surface disease (DAOSD).

Due to the risk of developing serious ocular complications, even in patients with mild skin involvement, early and regular ophthalmological evaluation is crucial[10]. Children and adults with AD-especially those with facial or periocular involvement, long disease duration, or a history of eye rubbing-should undergo routine eye examinations to enable timely detection and intervention, minimizing the risk of irreversible visual impairment[10]. The emergence of DAOSD as a frequent adverse effect of biologic therapy underscores the importance of multidisciplinary collaboration[5]. The partnership between dermatologists and ophthalmologists is essential for optimal care, ensuring early diagnosis and appropriate treatment.

Clinically, management focuses on a tailored approach that includes topical and systemic anti-inflammatory therapies, patient education, and, when necessary, surgical intervention for a severe cases[10].

Final recommendations for practice include:

1. Regular ophthalmological screening for all AD patients, regardless of disease severity[10].
2. Early referral to ophthalmologist if any ocular symptoms or signs are noticed, particularly in patients on dupilumab[5,12].
3. Patient education, especially on avoiding eye rubbing and recognizing ocular symptoms[10].
4. Multidisciplinary collaboration to provide comprehensive care[5,12].

Continued research and awareness are needed to further clarify risk factors and optimize prevention and management strategies for ocular complications in AD.

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Author's contribution

Conceptualization: S.Kosek; methodology: J.Klonowska; check: W.Wasiniewska; formal analysis: S.Kosek; investigation: M.I.Sroka; resources: T.Kandefer; data curation: J.Klonowska; writing - rough preparation: M.Barański; writing - review and editing:

R.J.Walkowski; visualization: M.I.Sroka; supervision: W. Wasiniewska; project administration: T.Kandefer; receiving funding- no specific funding.

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In preparing this work, the authors used Perplexity for the purpose of checking language accuracy. After using this tool, the authors have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

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