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# Ocular Leishmaniasis Presenting as Chronic Ulcerative Blepharoconjunctivitis: A Case Report

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#### Abstract

Leishmaniasis is caused by unicellular eukaryotic obligate intracellular protozoa of the genus Leishmania that is endemic in over 98 countries in the world-most of which are developing countries including Ethiopia. It is transmitted by phlebotomine sandflies. The eye may be affected in cutaneous, mucocutaneous and Post Kala-Azar Dermal Leishmaniasis. We report a case of ocular leishmaniasis with eyelid and conjunctival involvement that had simulated ulcerative blepharoconjunctivitis not responding to conventional antibiotics. The patient was diagnosed by microscopy of a sample obtained via direct smear from the lesions. He was treated with systemic sodium stibogluconate (20 mg/kg/day) for 45 days and was clinically cured with this treatment.

**Keywords:** Ocular leishmaniasis; Chronic Ulcerative Blepharoconjunctivitis; Leishmaniasis

# **Case Report**

A 29 year-old male patient presented to our center with bilateral eyelid swelling associated with redness of the eyes, itching sensation, scant whitish discharge and mild photophobia. He was seen at our center one month prior for the same complaint and was treated for chronic ulcerative blepharoconjunctivitis with topical and systemic antibiotics but did not improve. He lives in a village in northwestern Ethiopia, in close proximity to the Ethio-Sudan border, where visceral leishmaniasis (VL) is endemic. Review of his medical history revealed that he was treated for VL in the nearby hospital with sodium stibogluconate five times in the past.



Figure 1: Appearance of the eyelids and conjunctiva at presentation.

The first episode occurred six years ago and his recent infection one year previously had responded well to treatment. He has no

concomitant HIV infection. He claimed to have no ocular involvement when he was treated for VL. In his latest presentation, his visual acuity was 6/6 in both eyes. His eyelids were erythematous, edematous, indurated and ulcerated with decreased density of lashes. He had bilateral conjunctival inflammation with chemosis (Figure 1). His cornea was clear and quiet anterior chambers in both eyes. Systemic examinations revealed mild hepatosplenomegaly but no lesions on the skin. Given that he is from a VL endemic area and his prior episodes of VL led to the suspicion of ocular leishmaniasis. Aspirate from the eyelid swelling was done that revealed a high load (+ 6) of Leishman Donovan bodies (Figure 2).



**Figure 2:** Direct Smear was positive for Leishman Donovan bodies (Giemsa stain).

Based on this result, the patient was referred to the Leishmaniasis Research and Treatment Center of our hospital where he was treated with sodium stibogluconate 20mg/kg/day for 45 days with complete clinical cure (Figure 3).



Figure 3: Appearance of the patient on completion of treatment.

# Discussion

Encompassing a complex group of disorders, leishmaniasis is caused by a unicellular eukaryotic obligatory intracellular protozoa of the genus *Leishmania* that is endemic in over 98 countries in the world-most of them developing including Ethiopia. The organisms are transmitted by phlebotomine sandflies of the genus Phlebotomus in the "Old World" (Asia, Africa, and Europe) and the genus Lutzomyia in the "New World" (the Americas) [1].

To date, more than 20 species of leishmaniasis have been identified including *L. donovani, L. major, L. tropica, L. aethiopica, L. braziliensis* and they produce widely varying clinical syndromes ranging from self-healing cutaneous ulcers to fatal visceral disease. These syndromes fall into three broad categories: visceral leishmaniasis (VL), cutaneous leishmaniasis (CL), and mucosal leishmaniasis (ML). The eye may be affected in cutaneous, mucocutaneous and Post Kala–Azar Dermal Leishmaniasis (PKDL) [2].

Cutaneous leishmaniasis generally involves eyelids in 2-5% of the cases, most often on the external corner. The pathologic pattern is comparable to what can be observed on the rest of the skin but the fragility of the eyelid gives an increased risk of local spreading. The eyelid infection may result in eyelid edema, ulceration, and scarring leading to ectropion, with occasional spread to conjunctiva and lacrimal ducts. In mucocutaneous leishmaniasis, the lesions are more destructive and may lead to loss of normal ocular anatomy [3,4].

Further involvement of ocular structures may result in granular or nodular conjunctivitis, episcleritis, interstitial keratitis, nodular keratitis with heavy pannus formation, and ulcerative keratitis [5,6]. In some patients, phlyctenular and nummular keratitis have been observed, which appears to be because of a hypersensitivity reaction to the Leishmania antigens [7]. Other ocular manifestations include anterior uveitis, glaucoma, retinal hemorrhages, central retinal vein thrombosis, optic neuritis, chronic dacryocystitis, scleromalacia, epidermoid carcinoma and ptosis [8,9].

Clinical diagnosis of ocular lesions is difficult because it has nonspecific characteristics and can simulate other conditions like chalazion, eyelid or conjunctival tumors, dacryocystitis or nonspecific interstitial keratitis and uveitis [10,11].

Demonstration of amastigotes (Leishman-Donovan bodies) in material obtained from a lesion remains the diagnostic gold standard. Microscopic examination of eyelid skin smears, aspirates of eyelid or conjunctival lesions, or biopsies of the lesion is used for detection of parasites. Culture of smear or biopsy material may yield leishmania. PCR is more sensitive than microscopy and culture and allows identification of leishmania to the species level.

Although lesions in cutaneous leishmaniasis heal spontaneously in the majority of cases, their potential blinding complications in ocular involvement indicates that treatment is needed. The most common medications currently used for treatment of leishmaniasis include Pentavalent antimonials, amphotericin B, Liposomal Amphotericin B, Miltefosine and Paromomycin sulfate.

# Conclusion

If left untreated, ocular leishmaniasis is a potentially blinding disease. Delays in diagnosis and treatment, as well as the patient's own immune reaction to the pathogen can cause irreversible damage to the eye or its adnexae. Therefore, maintaining a high level of suspicion amongst physicians who work in endemic areas can lead to early diagnosis and treatment, hence preventing blinding complications.

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