An unusual ocular presentation of acquired immune deficiency syndrome

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A 50-year-old male who presented with bilateral keratomalacia and on subsequent evaluation was found to be human immunodeficiency virus (HIV) positive is being reported. A MEDLINE search of the literature did not reveal any report of keratomalacia as the initial presenting feature of HIV/ acquired immune deficiency syndrome.

Key words: Chronic diarrhea, conjunctival xerosis, keratomalacia, night blindness, weight loss

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Human immunodeficiency virus (HIV) infection can affect every tissue in the eye - from the eyelids to the optic nerve manifesting most commonly as dry eye, retinal microvasculopathy and cytomegalovirus (CMV) retinitis. Dry eye in HIV infection is due to decreased tear production associated with diminished lactoferrin and lysozyme and is of mild to moderate severity and can be treated with artificial tears. A detailed search on MEDLINE of previously recorded literature of ocular manifestation of acquired immune deficiency syndrome (AIDS) did not reveal keratomalacia as the initial presenting feature of HIV/AIDS.

Case Report

A 50-year-old male reported to the ophthalmic outpatient department (OPD) with complaints of inability to see with the left eye for the past three months and with the right eye for the past one month. Onset was associated with pricking sensation in each eye. He gave history of difficulty in seeing after sunset for the past few years.

He gave history of chronic diarrhea, especially following meals, associated with marked weight loss for the past four years. He was not a known diabetic. He denied history of ocular trauma, fever, cough, hemoptysis, chest pain, breathlessness or any systemic medication.

Patient belonged to poor socioeconomic strata and his diet consisted of only rice with occasional curry. He denied history of alcohol or tobacco abuse. On general examination he was markedly emaciated, weighing only 44 kg [Figure 1]. The vital parameters were stable. The skin was ichthyotic. The hair over the body was sparse and coarse. There was marked pallor, koilonychia and generalized lymphadenopathy. There was no edema, oral thrush or oral hairy leukoplakia. Abdominal examination revealed hepatosplenomegaly. The respiratory, cardiovascular and central nervous systems were essentially normal.
Patient had severe photophobia. There was bilateral madarosis with tylosis [Figure 2]. He had minimal circumcorneal congestion. There was dry keratinization of the palpebral conjunctiva with a corrugated appearance akin to skin. There were Bitot's spots at the temporal limbus with marked xerosis. The visual acuity was reduced in each eye to perception of light with accurate projection of rays.

The cornea of the right eye was reduced to a thin opaque membrane seen in parts only and was covered with mucopurulent strands. There was a gutter involving the whole of the limbus. The anterior chamber was flat with iris visible in areas of absent cornea. The entire iridocorneal mass was protuberant with a whitish/pinkish-appearing patch in the center [Figure 3]. The intraocular pressure could not be assessed.

The cornea of the left eye was shrunken to about 6 mm in diameter, opaque, thinned out and flattened with nasal vascularization. The anterior chamber was flat with iris seen adherent to the cornea. Rest of the details of the anterior chamber could not be made out [Figure 4]. The eye was hypotonous.

Patient was provisionally diagnosed as keratomalacia right eye and atrophic bulbi with xerophthalmia left eye. He was evaluated to determine any contributory causes for loss of weight and chronic diarrhea.
Investigations revealed hemoglobin 9.8 gm/dl. Blood sugar, serum electrolytes, stool examination, renal and liver function tests and chest X-ray were normal. HbsAg and C-reactive protein were negative, electrocardiogram showed sinus bradycardia and abdominal ultrasound showed splenomegaly. enzyme-linked immunosorbent assay (ELISA) for HIV was positive.

He was given intramuscular injection of vitamin A 200,000 IU on the day of admission, the next day and then after one week. In addition he was treated with topical antibiotics and artificial tears. Within a week of the treatment there was a reversal of all changes of conjunctival xerosis and keratinization and the conjunctiva became pink and velvety [Figure 5]. However, there was no change in the corneal condition. The patient sought discharge against medical advice despite being advised about his condition.

Discussion

AIDS caused by HIV is characterized by the deficiency of T helper (CD4) lymphocytes, leading to an inability to combat opportunistic infections.

A provisional diagnosis of AIDS was made based on the world health organization criteria which, was subsequently confirmed in accordance with the National Aids Control Organization guidelines. Besides two major features, weight loss and chronic diarrhea, and one minor feature in the form of generalized lymphadenopathy, the patient was serologically positive too, established by positive ELISA.

He presented with xerophthalmia X3B. The diagnosis of keratomalacia was based on the findings of a melted cornea in a relatively quiet eye associated with clinical features of xerophthalmia with reversal of the conjunctival signs on parenteral vitamin A therapy. The patient could not afford serum retinol levels and CD4 cell count or plasma viral load estimation.

Vitamin A deficiency is the direct cause of xerophthalmia. Hypovitaminosis A is well documented in children, especially following severe measles where vitamin A stores are rapidly depleted due to increased metabolism. Active corneal xerophthalmia is extremely rare in older children and adults, except in severe famines. They are a medical emergency and require urgent treatment by high-dose vitamin A supplementation. Retinol deficiency is quite frequent in the population of HIV-infected individuals. Serum retinol levels of less than 1.05 micromol/L determine a 3.5 to five times higher death risk. The recommended treatment for HIV-positive individuals is similar to their HIV-negative peers.

Liang et al., have found that hypovitaminosis A, E, and B12 accelerated the development of AIDS, whereas their normalization retarded the development of immune dysfunction.

In Ethiopia, vitamin A deficiency is recognized as a serious public health problem among patients with chronic diarrhea, with or without HIV infection. Vitamin A deficiency (VAD, serum retinol <0.70 micromol/L) was observed in 52.7% and 45.5% of diarrheic patients with and without HIV co-infection, respectively. About 13% of healthy controls and 29.3% of asymptomatic HIV-infected blood donors were deficient in vitamin A.

Though hypovitaminosis A is well documented in patients with HIV infection and is aggravated by the associated diarrhea, keratomalacia as presenting ocular manifestation of AIDS was not found on a MEDLINE search of literature. This case is being reported on account of the unusual presenting ocular manifestation of AIDS.

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