Commentary: Dry eye syndrome and vitamin D deficiency

Dry eye syndrome (DES) is multifactorial with many systemic and ocular associations. The multiplicity of the causative factors makes the treatment of this disease difficult. The magnitude of the problem has only increased in recent times. Technological advances in every field, especially the use of multiple digital display devices have paradoxically caused an increase in dry eye disease as also improvement in its diagnosis.

Among the many etiological factors, an association of Vitamin D deficiency with DES and tear film insufficiency has received attention in recent studies including the study by Watts *et al*. published in this issue of the Indian Journal of Ophthalmology.[1-4] The prevalence of vitamin D deficiency in India has been reported to be 40%–99% (80%–90% in most studies).[5] Most studies define the deficiency as a serum vitamin D level of <20 ng/ml;[4,5] however, some studies[2] have taken <12 ng/ml as the cutoff.

What is the role of Vitamin D in DES? Systemic diseases that are associated with DES like diabetes mellitus, thyroid disease, collagen vascular disorders, depression, anxiety, atopy, and irritable bowel syndrome are all associated with low levels of serum 25-hydroxyvitamin D (25(OH) D) or inadequate sunlight exposure.[2] Vitamin D deficiency causes symptoms associated with dry eyes and its supplementation decreases ocular surface inflammation and improves several tear film parameters such as tear film break up time (TBUT), surface staining with fluorescein dye, eyelid margin hyperemia, and tear secretion.[3] Vitamin D improves corneal epithelial cell barrier function by regulating gap and tight junctions.[2] In mice, vitamin D has been shown to suppress ocular surface inflammation by inhibiting Langerhans cell migration into corneas, and this inhibits corneal neovascularization.[6] Vitamin D induces the production of IL10 and reduces inflammatory cytokines/factors like IL1, IL6, TNF alpha, and C-reactive protein.[4] In addition, vitamin D reduces inflammation by increasing antioxidant cytokines in tears and suppressing both Th1 and Th2 cells.[7] Vitamin D induces D cathelicidin, which promotes the wound healing of conjunctiva and cornea.[8] Vitamin D reduces tear osmolarity and improves the stability of tear film.[7] Lacrimal, salivary, and parotid gland functions may be directly affected by vitamin D.[2]

In the Korean National Health and Nutrition Examination Survey (KNHANES), the association of vitamin D deficiency and sunlight exposure time on DES in a large population was done.[2] This study concluded that deficient sunlight exposure time and inadequate serum 25(OH) D levels were associated with DES in Korean adults. The authors suggested that sufficient sunlight exposure and/or vitamin D supplementation may be helpful in the treatment of DES. In this study, older age, female gender, rheumatoid arthritis, depression, thyroid disorder, history of ocular surgery, urban residence, indoor occupation, inadequate sunlight exposure, and low serum 25(OH)D level were the risk factor for DES.[2]

Synthesis and bioavailability of vitamin D are different in various age groups. The effect of vitamin D supplementation is different in males and females.[3] Vitamin D has an important role to play in estrogen biosynthesis and estrogen signaling. Postmenopausal women have low levels of estrogen which may be a major contributing factor for DES.[2]

Two different forms of vitamin D supplementation are by oral route and by intramuscular injection. Single intramuscular injection of vitamin D has been reported to be an effective way to increase and maintain serum 25(OH) D levels.[2] The use of buccal spray has been studied by Watts *et al*.[4]

In conclusion, Vitamin D opens a new avenue of therapy for the treatment of DES.

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