Case Report

Vitiligo in association with vernal keratoconjunctivitis

Sunil Kumar *; Wajeeha Al Khars

Abstract

Vernal keratoconjunctivitis (VKC) is a chronic allergic inflammation of ocular surface involving the tarsal and/or bulbar conjunctiva. Signs of VKC are confined to the conjunctiva and cornea. The skin of the lid remains uninvolved. Here we report a case of 17-year-old male suffering from VKC who develops vitiligo of lid skin and lash poliosis. All ocular and systemic causes of localized skin and lash depigmentation were excluded in our patient by thorough clinical examination and investigations. During regular follow-up for two-year patient did not develop any ocular and systemic illness presenting as vitiligo and poliosis. We believe that VKC was the most possible etiology of Vitiligo of lid and lash poliosis in this patient.

Keywords: Vernal keratoconjunctivitis, Poliosis, Vitiligo

Introduction

Vernal keratoconjunctivitis (VKC) is a chronic, bilateral, at times asymmetrical, seasonally exacerbated, allergic inflammation of the ocular surface, involving tarsal and/or bulbar conjunctiva.1 The skin of the lid remains uninvolved. Vitiligo of lid and lash Poliosis is the depigmentation of skin and hair and has been described previously in association with several inflammatory conditions including idiopathic uveitis, Vogt–Koyanagi–Harada syndrome, sarcoidosis, Alezzandrini syndrome, Marfan’s syndrome and tuberous Sclerosis. We present a case of vitiligo of lid skin and lash poliosis in a patient suffering from VKC.

Case report

A 17-year-old male presented with complaints of depigmentation of lid skin and white eyelashes for one year. He was suffering from recurrent itching, redness and watering for last two years. His past medical and family history was non-remarkable. The patient denied any hearing abnormality and alopecia. He was using anti-allergic eyedrops intermittently for the presumed diagnosis of allergic conjunctivitis. His visual acuity was 20/20 in each eye.

Specialist Eye Hospital, Dhahran, Al Khobar, Saudi Arabia

* Corresponding author. Tel.: 966530129683.
e-mail address: Sunkaru79@hotmail.com (S. Kumar).
On examination he had vitiligo localized to both lids and poliosis of the eyelashes (Fig. 1). Ocular examination was remarkable for giant papillae of the palpebral conjunctivae (Fig. 2). There was no evidence of uveitis. Both fundi were normal. Systemic examination was unremarkable. Full blood count, erythrocyte sedimentation rate, Antinuclear antibody, liver and renal function tests were within normal limits. Fluorescein angiography showed no abnormality. HLA-DR4 serology was assigned on Human leukocyte antigen (HLA) typing.

The patient was diagnosed as suffering from VKC and treated with topical rimexolone, olopatadine and lubricating eyedrops. During two year follow-up the patient did not develop uveitis or other systemic symptoms. Poliosis and vitiligo remained localized to lids.

Discussion

Vitiligo is an idiopathic condition characterized by destruction of the melanocytes in circumscribed areas of the skin, resulting in patches of depigmentation. Depigmentation of the lid skin and poliosis of the eyelash is commonly seen in vitiligo. Vitiligo affects all age groups with no sex predilection. It affects approximately 1–4% of the world population. Vitiligo is found to be associated with several autoimmune diseases such as thyroid disease, Addison’s disease and pernicious anemia. Association of vitiligo and poliosis with several uveitic syndromes is well established. Vitiligo is characterized by incomplete penetrance, multiple susceptibility loci, and genetic heterogeneity. Human Leukocyte Antigens (HLA) are genetic markers for general susceptibility to vitiligo with HLA-DR4, B13 and B35 reported to be increased in different ethnic groups.2

T helper type 2 (Th2) cells and their cytokines, along with various growth factors involved in ocular inflammation of VKC may affect the eyelashes as well. A positive correlation between eyelash length and severity of VKC has been reported with an unknown chemical mediator postulated as the cause of increased lash growth. Increased expression of mRNA encoding Th2-type interleukins was observed in allergic tissue from VKC.3 Tear levels of Th2 Interleukin (IL) 4 and 5 were higher in patients with VKC compared to controls.

VKC is proposed to be a phenotypic expression of upregulation of the cytokine genes. The cytokine genes on chromosome 5q, through production of IL 3, 4 and 5 regulate the prevalence of Th2 in VKC.4 Recently, polymorphisms of the IL4 gene were shown to be a genetic risk factor for susceptibility toward vitiligo and upregulation of the IL-4 transcript, protein and IgE levels is proposed to play a crucial role in pathogenesis in vitiligo.5

To our knowledge there have been no prior reports of an association between VKC and vitiligo in the literature. We suggest that higher levels of IL-4 in VKC in susceptible haplotypes may be responsible for the pathogenesis of vitiligo.

Conflict of Interest

The authors declare that there are no conflicts of interest.

References

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