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Case Study

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TOPICAL TOFACITINIB: A JAK-STAT INHIBITOR FOR THE TREATMENT OF VITILIGO: A CASE REPORT

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ABSTRACT

Vitiligo is a skin disorder characterized by loss of pigment producing cells, resulting in depigmented patches on the skin. Recent studies have shown that CD8+T cells and interferon-γ play key roles in the destruction of melanocytes in the pathogenesis of vitiligo. We reported improvement after topical tofacitinib in a male patient of vitiligo.

INTRODUCTION

Vitiligo is a skin disorder characterized by loss of pigment producing cells, resulting in depigmented patches on the skin. The exact cause remains unclear. While it is not contagious, it can have a profound impact on an individual's appearance and self-esteem.

CASE REPORT

A 15-year old boy presented to the Dermatology OPD and was diagnosed as a case of vitiligo with acrofacial involvement with a duration of 15 years. There was no family history. It was not associated with any autoimmune condition. At baseline, he had an evident white macule on his nose, scalp and lips. Treatment taken previously were oral corticosteroids for 3 years, antioxidants, topical tacrolimus, topical corticosteroids and oral vitamin D for 4 months. In the past, he had undergone 2 years of phototherapy with very slight improvement, as well as NB-UVB combined with topical tacrolimus and corticosteroids during 2 years with very little improvement.

Baseline investigations were done and the patient was started on topical tofacitinib. After 8 months of therapy, significant re-pigmentation of the nose and lips was observed.

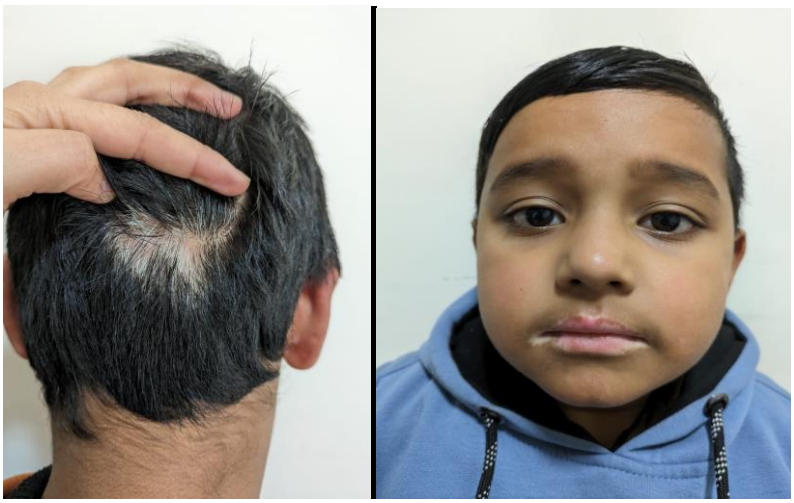

Figure 1 and 2: A white patch on scalp and perioral involvement in a 15-year-old boy.

DISCUSSION

A new hypothesis about the pathogenesis of vitiligo suggests that both genetic and nongenetic factors interfere in melanocytes, which results in their immune-mediated destruction.[1] Recent studies have shown that, CD8+T cells and interferon-γ play key roles in the

destruction of melanocytes in the pathogenesis of vitiligo.[2] Tofacitinib is a JAK1 and JAK3 inhibitor that interferes with interferon-γ signaling, which reduces CXCL10 chemokine expression, blocking the activity of vitiligo. Topical and oral tofacitinib are both forms of JAK inhibitors and share a common mechanism of action. Advantages of topical over systemic tofacitinib are targeted localized treatment, reduced systemic side effects, minimize risk of drug interactions, improved tolerability and less frequent monitoring. The first case suggesting this possible mechanism of action of JAK inhibition was published by Craiglow and King.[3] It has a remarkable impact on quality of life and is emotionally devastating, some patients are at higher risk of social discrimination and stigma; therefore, new possibilities of targeted treatments are welcome. This case suggests that topical tofacitinib might be a safe and effective therapy for vitiligo, especially for pediatric and adolescent patients, for whom systemic therapies are not desired.

BIBLIOGRAPHY

1. Spritz RA. Six decades of vitiligo genetics: genome-wide studies provide insights into autoimmune pathogenesis. *J Invest Dermatol*, 2012 Feb; 132(2): 268–73.
2. Rashighi M, Harris JE. Interfering with the IFN-γ/CXCL10 pathway to develop new targeted treatments for vitiligo. *Ann Transl Med.*, 2015 Dec; 3(21): 343.
3. Craiglow BG, King BA. Tofacitinib citrate for the treatment of Vitiligo a pathogenesis-directed therapy. *JAMA Dermatol*, 2015 Oct; 151(10): 1110–2.