J Med Allied Sci 2017; 7 (2): 114-117
www.jmas.in
Print ISSN: 2231 1696 Online ISSN: 2231 170X
Journal of Medical & Allied Sciences

Case report

A case report showing coexistence of two autoimmune diseases-psoriasis and vitiligo

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Article history

Received 28 February 2017
Revised 01 April 2017
Accepted 05 May 2017
Early online 17 June 2017
Print 31 July 2017

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Abstract

Psoriasis and vitiligo are autoimmune diseases. Occurrence of both these diseases in the same patient, especially at same sites is uncommon. Here, one such patient having both psoriasis and vitiligo lesions at the same site is being reported. An eight year old male patient presented with asymptomatic flat, white coloured, lesions over the left knee joint and left elbow joint since 3 years, followed by the appearance of multiple, asymptomatic red, scaly, elevated lesions over the pre-existing lesions since 10 days. There was no history of atopy, or drug intake/any applications or joint pains in this patient. There was family history of similar white coloured lesions and hypothyroidism. Cutaneous examination revealed well defined, polysized, depigmented macules measuring around 5x3 cm with leukotrichia affecting the left knee joint and the left elbow joint. There were multiple, well defined, erythematous, plaques measuring around 2x2cm with silvery white scales over the extensor aspect of left elbow joint and left knee joint within the depigmented macules. Auspitz's sign (peeling and pinpoint bleeding spots over the lesion on scrapping with a glass side) was positive. There was sparing of genitalia, palms, soles and scalp. Hair and nail was normal. A provisional diagnosis of vitiligo associated with psoriasis vulgaris was made, which was confirmed on histopathology. Both psoriasis and vitiligo lesions were treated accordingly. Occurrence of both vitiligo and psoriasis in the same patient and at same sites indicates similar etiopathogenesis.

Key words: Coexistence, Koebner's phenomenon, Psoriasis, Vitiligo

DOI: 10.5455/jmas.260021

Psoriasis and vitiligo are fairly common autoimmune skin diseases. Worldwide incidence of Psoriasis is 0.1-3% while that of Vitiligo is 1%[1-3]. Autoimmune skin disease may be associated with a systemic autoimmune disease such as thyroid disease in the same patient. Occurrence of two autoimmune skin diseases in the same patient especially affecting same sites is uncommon. Here we are reporting one such case where there is co-localisation of psoriasis and vitiligo lesions.

Case report

An 8 year old male patient presented with asymptomatic flat, white coloured, lesions over the left knee joint and left elbow joint since 3 years, followed by the appearance of multiple, asymptomat-

ic red, scaly, elevated lesions over the pre-existing lesions since 10 days. There was no history of atopy, or drug intake/any applications or joint pains in this patient. There was family history of similar white coloured lesions and hypothyroidism. There was no apparent aggravating or relieving factors.

Cutaneous examination revealed well defined, polysized, depigmented macules measuring around 5x3 cms with leukotrichia affecting the left knee joint and the left elbow joint (Fig 1 and Fig 2). There were multiple, well defined, erythematous, plaques measuring around 2x2cm with silvery white scales over the extensor aspect of left elbow joint and left knee joint within the depigmented macules (Fig 1 and Fig 2). Auspitz's sign (peeling and pinpoint bleeding spots over the lesion on scrapping with a glass side) was positive. There was sparing of genitalia, palms, soles and scalp. Hair and nail was normal. There was no lymphadenopathy. General physical examination and systemic examination was unremarkable.

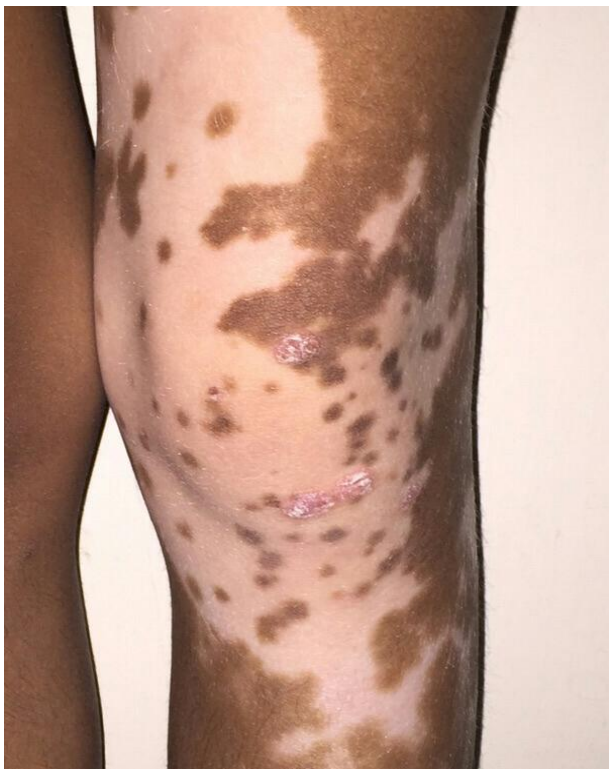

Fig 1. Presence of psoriatic lesions in the centre of depigmented macules over the left knee joint

A provisional diagnosis of vitiligo associated with psoriasis vulgaris was made, which was confirmed on histopathology. Histopathology showed features of uniform hyperkeratosis and parakeratosis with acanthosis and uniform elongation of club shaped rete ridges, along with dilated capillaries in the papillary dermis with suprapapillary thinning of epidermis and complete absence of melanin granules in the vitiliginous areas, confirming the diagnosis of psoriasis and vitiligo. Vitiligo macules were treated with topical Tacrolimus 0.03% ointment once daily in the morning and Decapeptide lotion once daily in the night with sun exposure next day morning and oral betacarotene 30mg (antioxidants) till the resolution of lesions. Psoriasis lesions were treated with topical coal tar 4% ointment and halobetasol propionate cream once daily application till the resolution of the lesions. Psoriasis lesions subsided in 4 weeks time, vitiligo lesions were gradually improving and patient was advised to come for frequent follow up at monthly intervals.

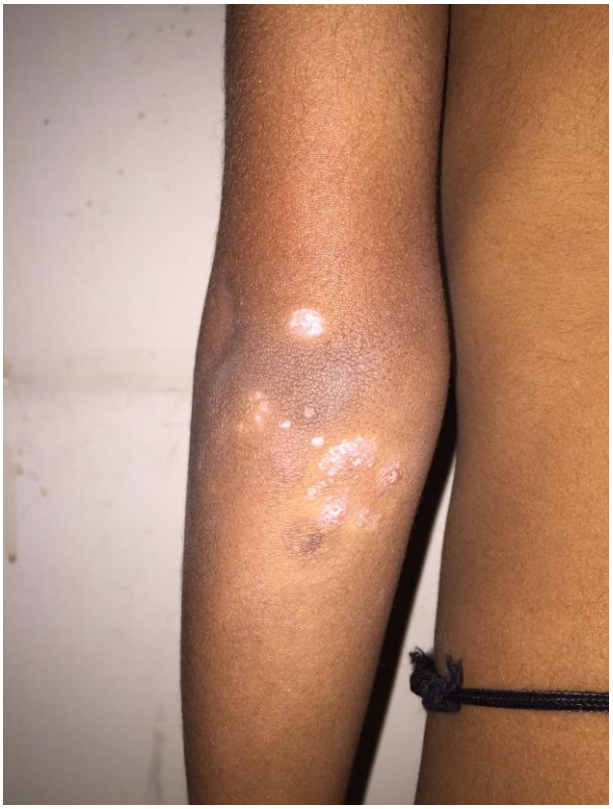

Fig 2. Presence of psoriatic lesions (multiple, erythematous plaque with silvery scale) present over the depigmented macule over the left elbow joint

Discussion

Psoriasis is a chronic relapsing disease characterised clinically by the presence of chronic, bilateral, symmetrical, well defined, erythematosus dry, red, scaly, plaques and papules present over the extensor aspects. The scales are abundant, loose, dry, and silvery white or micaceous. Vitiligo on the other hand is an acquired, progressive disorder of pigmentation characterised by circumscribed hypo or hyperpigmented macules commonly affecting the trauma prone areas / acral areas / lips and often associated with leukotrichia.

Occurrence of both vitiligo and psoriasis in the same patient and at same sites has been reported and this indicates similar etiopathogenesis[4,5]. Various hypotheses have been proposed in the pathogenesis of vitiligo such as-immune response hypothesis, neural hypothesis, autotoxic self-destructive or free radical or antioxidant deficiency hypothesis, composite hypothesis, melanocyte growth factor reduction hypothesis. According to the immune hypothesis theory there is an alteration of immune surveillance that has been proposed as the primary event resulting in the dysfunction and destruction of melanocytes. Biochemical trauma to melanocytes results in release of antigenic components that leads to development of autoantibodies against the same. Alternatively there may be certain immune cells directed against antigenic substance of autologous melanocytes. This hypothesis can be supported by the fact that vitiligo is frequently associated with many autoimmune disorders and the frequent presence of autoantibodies against thyroid, adrenals, malignant melanoma and halo nevus[6,7].

Histopathological examination of perilesional skin of vitiligo patients has suggested the role of lymphocytes in depigmentation process. On examination of peripheral blood T lymphocytes in patients with non-segmental vitiligo, a significant fall in the circulating CD45RO+ and a significant rise in the HLA-DR+ cells was noted, indicating the vital presence of activated peripheral T cell dysregulation. However, no evidence of circulating anti-T lymphocyte antibodies or immune complexes in these patients could be found[8]. All these observations point toward the involvement of an immune mechanism, possibly both humoral and cell mediated, in the pathogenesis of vitiligo.

The persistence of psoriasis can be explained by the ongoing antigen persistence and proliferation of activated T cells after initial sensitization, thus amplifying the process. In addition, a defective functioning of T cells might also contribute to unbridled T-cell activation. Lastly, it could be possible that skin as an organ is capable of initiation and maintenance of inflammation through sustained T-cell activation in patients with psoriasis. This is suggested by the abundant dermal aggregates of activated T cells and mature dendritic cells around modified dermal blood vessels, identified in psoriatic lesions; along with an increased expression of lymphoid organizing chemokines (CCL19, CCL21) making these regions behave like secondary lymphoid organ. These areas have in situ recruitment of both cell types resulting in antigen presentation and T-cell activation; enabling them to function as peripheral non-lymphoid tissue, serving functions normally thought to be performed by peripheral lymph nodes[9,10]. As suggested, the entire normal skin surface contains twice the number of T cells seen in circulation. TNF-α induced endogenous activation of dendritic cells and non-lesional resident T cells also occurs in psoriatic patients, suggesting that even the apparently normal skin of psoriatics already contains a cellular microenvironment that can potentially become self-perpetuating once the disease is initiated by bacterial antigens or superantigens[11]. Another hypothesis suggests that the innate immune system (in response to various environmental stimuli) initiates the psoriatic lesion, but during the process, neo antigens are exposed, inducing an adaptive immune response that maintains the psoriatic lesion. Thus, both the innate and adaptive immune systems triggered by environmental stimuli and/or putative psoriatic antigens could act synergistically to induce psoriasis in a genetically predisposed individual. Dhar et al concluded that, there could be structural similarities between anti stratum corneum antibodies and anti melanocyte antibodies and also a common neuropeptide might be responsible for cohabitation of vitiligo and psoriasis[12].

Another interesting dimension to the etiopathogenesis of psoriasis and vitiligo is initiation or aggravation of the lesions with trauma (Koebner's phenomenon). Occurrence of lesions of both psoriasis and vitiligo predominantly over joints in this patient is due to a shared precipitating factor, i.e. trauma (Koebner's phenomenon). It is also known as isomorphic response and refers to the induction of lesions in the uninvolved skin of patients following cutaneous trauma of any kind. The exact pathogenesis of Koebner phenomenon is not known but true koebner's phenomenon is seen in psoriasis, vitiligo, lichen planus, all of which have autoimmune background. Occurrence of lesions of both vitiligo and psoriasis at same sites predominantly over the extensors of joints is probably due to chronic minor friction/trauma over these sites. Coexistence of both vitiligo and psoriasis lesions over the left knee joint, left shin and left elbow in the present case can be explained by this koebner's phenomenon (isomorphic phenomenon).

Conclusion

Occurrence of multiple autoimmune diseases in the same patient is known to be a common phenomenon. Coexistence of systemic autoimmune diseases such as thyroid disease or diabetes mellitus or pernicious anemia along with

cutaneous autoimmune diseases such as autoimmune urticaria, vitiligo, or psoriaisis are known to occur. Occurrence of two cutaneous autoimmune disorders in the same patient in the same site is also known to occur rarely. However colocalisation of two cutaneous autoimmune disorders such as psoriasis and vitiligo at same sites in the same patient is uncommon[13]. Such occurrence highlights the shared etiopathogenesis of these two diseases.

Acknowledgment: None

Conflict of interest: None

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