



Skin Discolouration on Arms

By Jonathan Liu and Simon Lee, MD, FRCPC

A 50-year-old female of Asian ancestry is referred for management of long-standing skin discolouration. She wanted to enquire about recent advances in the treatment of her skin condition.

On examination, scattered, well-demarcated, white patches are noted on the arms and trunk. The thyroid is unremarkable upon palpation. Wood's lamp evaluation reveals accentuation of loss of colour, with a porcelain-white appearance.

What is your diagnosis?

- a. Tinea versicolor
- b. Tinea incognito
- c. Vitiligo
- d. Neurofibromatosis

Answer: Vitiligo

Vitiligo is an idiopathic, acquired, cutaneous disorder characterized by the loss of pigment production by the melanocytes in the skin. The precise mechanism remains undetermined. Proposed theories include an autoimmune, genetic, oxidative stress, neural, or viral cause.

The world wide incidence of vitiligo is 1 to 2% of the population. The median age of onset is in the second decade of life. It can have devastating consequences in various parts of the world, especially in darker complected races because of the contrast in skin colour. In some parts of India, vitiligo was known as "white leprosy," and patients suffered a similar fate as lepers with social ostracization and abandonment.¹



The onset of vitiligo is characterized by the development of white, well-demarcated macules, which may coalesce to form larger patches. Common sites of involvement include the face, hands, and feet with an acral distribution. Lesions may be localized, but, more commonly, they are widespread, and may involve large areas of the body. The universalis form affects the whole body. Other variants include a segmental and dermatomal type of involvement. Inspection with Wood's lamp reveals accentuation of vitiligo.


There is an association with other autoimmune diseases, such as thyroiditis, diabetes mellitus, pernicious anemia, Addison's disease, and alopecia areata. This lends support to an autoimmune hypothesis. Relevant investigations, depending on clinical findings, include TSH, CBC and antinuclear antibody testing to rule out associated disorders.

Treatment

Effective therapy for vitiligo remains elusive. Topical steroid therapy is often selected as initial treatment because of simplicity of use. Topical calcineurin inhibitors may also be tried, especially for the face and other regions of the body that are susceptible to cutaneous atrophy from prolonged steroid use. Vitamin D analogs, such as calcipotriol, have also been reported to be effective.

Narrow-band ultraviolet therapy (NB-UVB) is the recommended treatment for patients with widespread vitiligo, and it may induce satisfactory response in up to two-thirds of cases. In the past, PUVA (psoralens photochemotherapy combined with UVA light) was the gold standard for therapy; nonetheless, patients required 50 to 300 treatment sessions. Disadvantages of PUVA include gastrointestinal upset with medication, additional cost of psoralens, need for photoprotection, as well as increased risk for squamous cell carcinoma and melanoma. In contrast, NB-UVB treatment appears to have a better safety profile; although large multicentre, long-term follow-up studies are lacking. Patients need to be educated on the risk of attendant ultraviolet light therapy, including burn, cataract formation, and risk of photocarcinogenesis induction.

Other therapeutic options include excimer laser, systemic steroids, intralesional steroid injections and skin graft transplantation. There have also been some reports of success with oral ginkgo supplementation and the use of human placental extracts. Finally, depigmentation with monobenzylether of hydroquinone may be required for patients with extensive involvement.

Vitiligo can be a devastating illness, and its psychological impact on the quality of life in patients should not be underestimated. Supportive measures and the availability of treatment options can offer hope to its sufferers. 

References

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