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Folliculocentric tinea versicolor

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Abstract

Tinea versicolor (TV) is typically an asymptomatic fungal infection of the stratum corneum owing to *Malassezia* overgrowth. It presents as hypo- or hyperpigmented macules with fine scale that coalesce into patches on the trunk, neck, and/or arms. Presented in this report is a 34-year-old man with an interesting case of folliculocentric tinea versicolor manifesting as perifollicular hypopigmented macules on the lower back.

Keywords: folliculocentric, tinea versicolor, pityriasis versicolor, Malassezia

Introduction

Tinea versicolor is caused by *Malassezia* yeasts, which are common flora to the seborrheic areas of human skin [1]. The hair infundibula within these sebaceous areas is considered the yeast cell niche because it contains sebum, a lipid-rich energy source that is needed by *Malassezia* yeasts because of their absence of a fatty acid synthase gene [2-5]. The predominant clinically relevant species of *Malassezia* are *M. globosa* and *M. furfur*. The etiology of the hypopigmented skin lesions commonly seen in this disorder is not clear. Azelaic acid produced by *Malassezia* is theorized to act as a competitive inhibitor to tyrosinase activity thus diminishing melanin production [6,7]. It is also postulated that the thickened scale of the stratum corneum inhibits ultraviolet rays from contacting melanocytes, thereby decreasing pigmentation [8]. Host factors

involved in TV are poorly understood. He et al. state that up to 21% of patients report a positive family history implying a possible genetic predisposition [9]. Alternative risk factors for TV include heat, humidity, excessive perspiration, Cushing syndrome, long-term immunosuppressive corticosteroid use, pregnancy, diabetes mellitus, and poor nutrition [10-12]. Under the conditions associated with these risk factors, the yeast form of *Malassezia* changes to the mycelial form leading to clinical disease.

Case Synopsis

A 34-year-old man with a family history of vitiligo was evaluated in clinic for asymptomatic hypopigmented spots on his back and chest, which had been present for years. The rash flared 1-2 months before presentation and spread to involve his arms and lower back. He had not sought treatment for this problem. This patient was otherwise healthy and denied a history of diabetes mellitus. Physical exam showed hypopigmented perifollicular macules on the lower back that gradually coalesced into ill-defined hypopigmented patches on the mid and upper back, chest, and arms (**Figure 1**). Scale was not appreciated and KOH examination was not performed. Wood's lamp examination was negative. A 4mm punch biopsy obtained from a hypopigmented macule on the lower back revealed budding yeast and hyphae within the stratum corneum consistent with a diagnosis of tinea versicolor (**Figure 2**).

Case Discussion:

Tinea versicolor typically manifests as hypo- or hyperpigmented macules coalescing into patches on the trunk, neck, and arms. Fine scale is typically

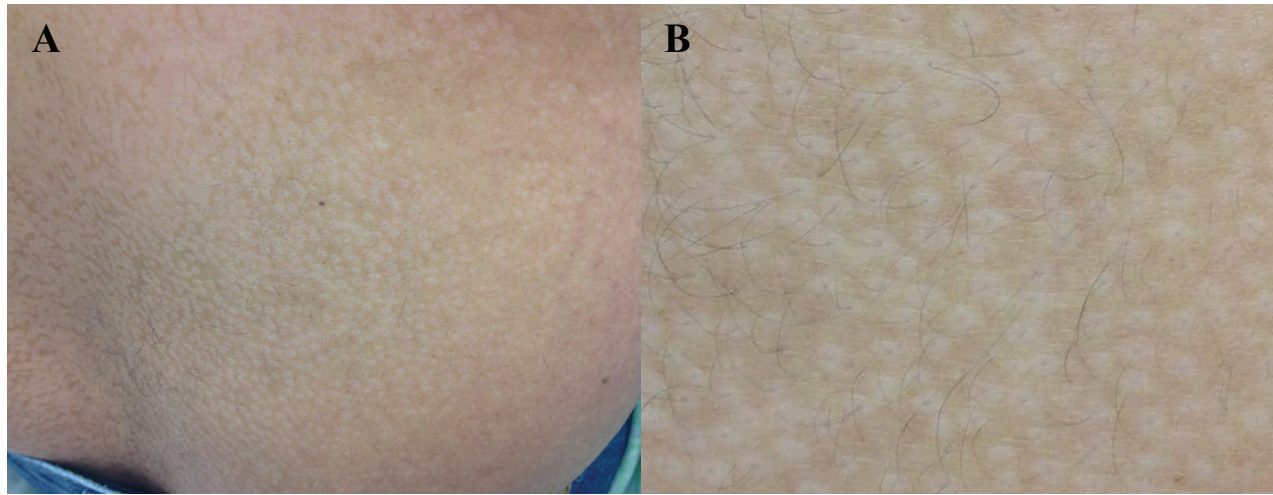


Figure 1. (A) Folliculocentric hypopigmented macules on the lower back coalescing into patches on the upper back. (B) Striking perifollicular loss of pigment within the lower back lesions.

appreciated within these lesions. However, if scale is not visible, the “evoked scale” sign should be sought wherein appreciable scale can be generated by stretching or scraping a TV lesion [13,14]. Microscopic examination of skin scrapings with potassium hydroxide shows short hyphae and yeast forms reminiscent of “ziti and meatballs.” Histologic examination is often unnecessary but shows hyphae and yeast forms in the stratum corneum with minimal inflammation. Wood’s lamp is positive in fewer than 50% of TV cases but can aid in diagnosis if yellow fluorescence is present [15].

Various clinical presentations of TV have been described including hypopigmented, hyperpigmented, combination hyper- and hypopigmented, erythematous, circinate, acral, and atrophic variants. To the best of our knowledge, cases of folliculocentric tinea versicolor have been rarely mentioned in the literature. Some authors believe it is an uncommon manifestation of TV [16]. Other authors believe tinea versicolor initially presents as small folliculocentric macules that eventually progress into the larger hypopigmented macules and patches where the folliculocentric quality is lost [17,18]. Folliculocentric TV with papules has been seen in immunocompromised transplant patients [19]. Mostafa et al. recently found an increase in hair loss within tinea versicolor lesions which they attributed to *Malessezia* species within the hair follicles [20].

Tinea versicolor should be distinguished from other hypopigmented conditions such as progressive macular hypomelanosis (PMH), vitiligo, and pityriasis alba. Progressive macular hypomelanosis was the leading condition in the differential diagnosis in our case. PMH manifests as asymptomatic non-scaly hypopigmented macules that merge into patches on the trunk. Wood’s light reveals folliculocentric red fluorescence in hypopigmented areas. *Propionibacterium acnes*, which has been cultured from follicles within hypopigmented areas of PMH, is believed to be responsible for the fluorescence and hypopigmentation [21,22]. Vitiligo presents with completely depigmented macules and patches with perifollicular retention of pigment, which is distinct from TV, which shows hypopigmentation and folliculocentric loss of pigment. Pityriasis alba

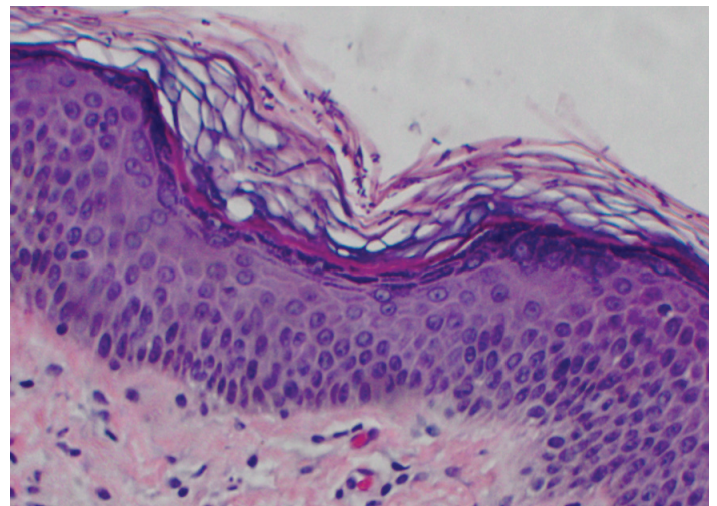


Figure 2. Budding yeast and hyphae in the stratum corneum with minimal inflammation.

can present with superficial scale similar to tinea versicolor but primarily affects children, favors the cheeks, and lacks the evoked scale sign [23].

Malassezia overgrowth affecting hair follicles is more commonly associated with pityrosporum folliculitis than tinea versicolor [24]. Pityrosporum folliculitis manifests as erythematous, often pruritic, follicular papules on the upper trunk. Papule scrapings of pityrosporum folliculitis reveal budding yeast similar to TV but with an absence of hyphae that is seen in TV lesions [25].

We suggest that follicular hyperpigmentation or hypopigmentation could be an adjunct criterion for tinea versicolor. Because this finding may not be apparent in the centers of larger coalesced lesions, one should look to the periphery of the lesions for this clue to the diagnosis of tinea versicolor.

Conclusion

Tinea versicolor should be included in the differential diagnosis when patients present with perifollicular hypopigmented or hyperpigmented macules. Scaling of the skin can be suggestive of tinea versicolor and should prompt microscopic examination with potassium hydroxide or chlorazole black E. When there is no obvious scale, the evoked scale sign could be helpful to guide the diagnosis.

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