Tinea Versicolor Mimicking Pityriasis Rubra Pilaris

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Tinea versicolor is a common noninvasive cutaneous fungal disease. We recount a case of tinea versicolor that mimicked type I (classic adult) pityriasis rubra pilaris. A 54-year-old white man reported a 20-year history of a recurrent pruritic eruption that had marginally improved with use of selenium sulfide shampoo and treatment with oral antihistamines. Results of a skin examination revealed erythematous plaques; islands of spared skin; and follicular erythematous keratotic papules on the trunk, shoulders, and upper arms. A lesion was scraped to obtain skin scales for potassium hydroxide staining. Examination of the stained samples revealed the characteristic "spaghetti and meatballs," confirming the diagnosis.

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Case Report

A 54-year-old white man presented with a 20-year history of a recurrent pruritic eruption that had marginally improved with use of selenium sulfide shampoo and oral antihistamine therapy. Erythematous scaly plaques were noted over the trunk and extremities (Figure 1). Islands of spared skin were most notable on the trunk (Figure 2). Follicular, erythematous, keratotic papules were noted on the shoulders and upper arms (Figure 3). Results of Wood lamp examination revealed a yellow-green fluorescence of the plaques. Results of potassium hydroxide (KOH) staining revealed numerous yeast and hyphae. The patient was diagnosed with tinea versicolor and treated with itraconazole 200 mg/d for 2 weeks. At the follow-up visit, the eruption appeared to be 90% cleared, and the itraconazole was continued for 2 more weeks at the same dosage.

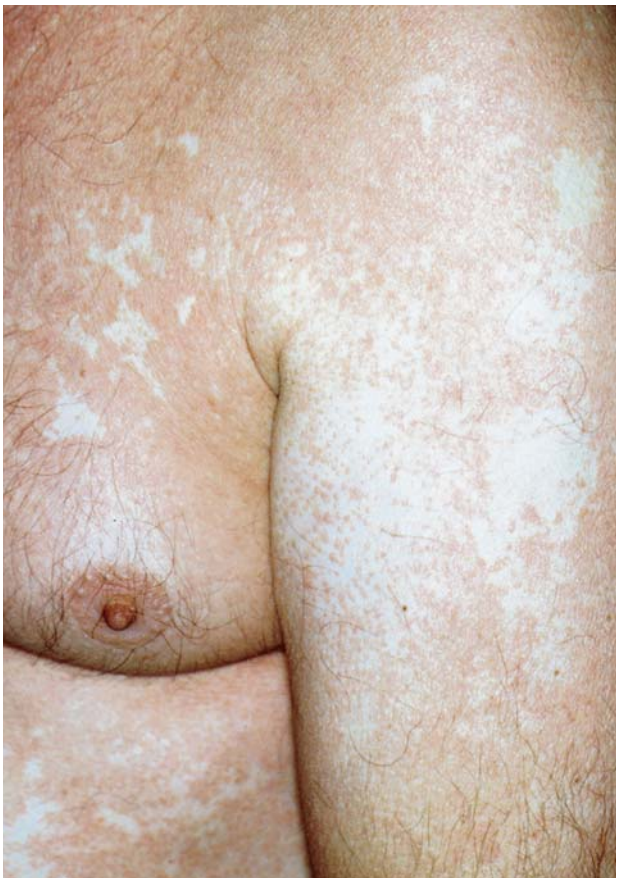

Figure 1. Erythematous scaly plaques and islands of spared skin on the chest.

Comment

Tinea versicolor is a common noninvasive cutaneous fungal disease affecting people worldwide. The incidence is greater in tropical regions with high temperatures and high relative humidity.[1] Tinea versicolor is caused by the fungus *Malassezia furfur*, which is present on the skin of 75% to 98% of healthy individuals.[2,3] M *furfur* is a dimorphic

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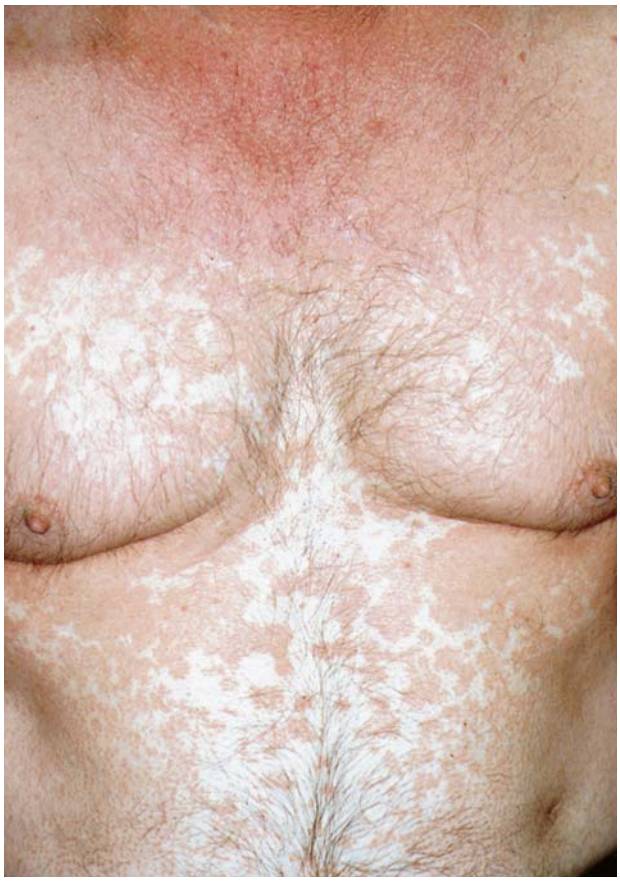

Figure 2. Erythematous scaly plaques with islands of sparing.

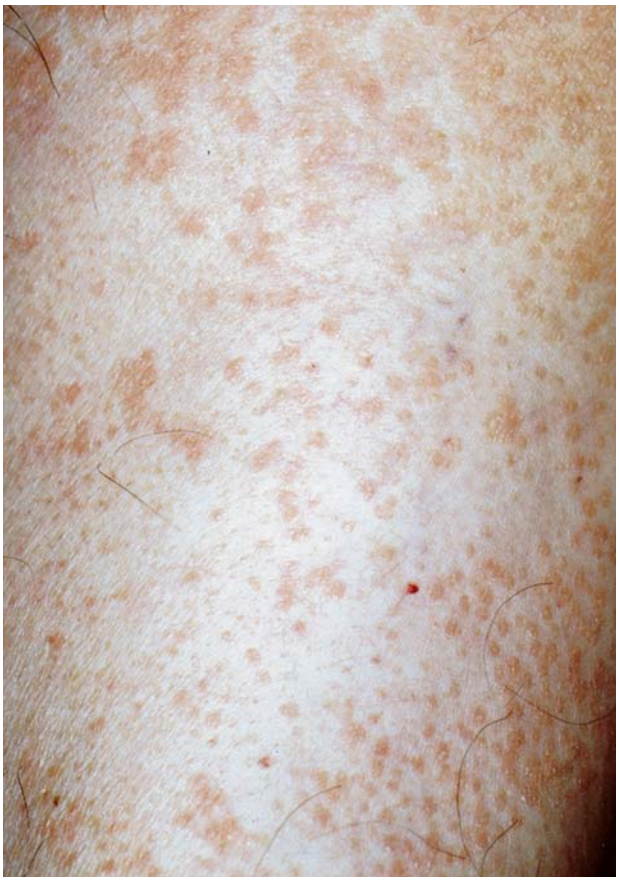

Figure 3. Follicular erythematous keratotic papules.

lipophilic organism that converts from the yeast stage to the mycelian stage under appropriate conditions. Several factors predispose to the development of tinea versicolor, including high temperature, high relative humidity, greasy skin, hyperhidrosis, hereditary factors, immunodeficiency, systemic corticosteroid therapy, and immunosuppressive treatment.[1,2] Because the organisms have a nutritional requirement for fatty acids, colonization starts soon after birth, peaking in late adolescence to early adulthood. M *furfur* is considered part of the skin's normal flora.[1-4] Colonization is likely due to an increase in lipids in sebum-rich areas of the skin and is not a result of poor hygiene.[5] M *furfur* is the generally accepted name, and *Pityrosporum orbiculare* and *Pityrosporum ovale* are synonyms.[3,6]

Tinea versicolor lesions are described as hypopigmented or hyperpigmented scaly macules or patches that vary in color from white to fawn. The lesions typically develop on the neck, chest, back, abdomen, or proximal upper extremities. The pathogenesis of the dyspigmentation is not well understood. Damage to melanocytes, inhibition of monophenol monooxygenase, or both may provide the basis for the hypopigmented lesions, whereas inflammatory stimulation of melanocytes may lead to hyperpigmented lesions.[3,4,7] Patients commonly report that they are unable to tan in the affected areas. Mild pruritus may be present. Similarly, classic adult pityriasis rubra pilaris can present as small, scaly, follicular, keratotic papules with islands of normal skin; however, results of skin biopsy reveal follicular plugs and hyperkeratosis.

Tinea versicolor is primarily a clinical diagnosis that can be confirmed by simply scraping the lesion to obtain skin scales and staining them with KOH. The characteristic appearance of "spaghetti and meatballs" is seen on microscopic examination of the yeast and hyphal forms. Wood lamp illumination causes the skin to fluoresce yellow to yellow-green. Cultures are not necessary. Several diseases must be considered in the differential diagnosis: vitiligo, pityriasis alba, seborrheic dermatitis, secondary syphilis, pityriasis rosea, mycosis fungoides, leprosy, sarcoidosis, and pityriasis rubra pilaris.

Treatment for tinea versicolor includes topical and oral agents. Topical agents such as selenium sulfide 2.5% and pyrroles are the most common therapies. The agents must remain in contact with the skin for a minimum of 5 to 10 minutes before the skin is washed.[3,8] Frequent relapses, extensive disease, or poor compliance with topical regimens may necessitate oral antifungal agents. Exercising one hour after oral administration of ketoconazole or fluconazole treatment and leaving the sweat in contact with the skin for 8 to 12 hours may increase the efficacy of systemic treatment, with reported cure rates of 75% to 100%.[5,8] Itraconazole, which is secreted in the sebum, does not require sweating for effectiveness.[5] Unfortunately, tinea versicolor has a high recurrence rate, and prophylactic or maintenance therapy may be necessary.

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