A young man presented with asymptomatic, overlapping, hyperpigmented patches on his back.

What is your diagnosis?
Photo Quiz Discussion

The Diagnosis: Trichrome Tinea Versicolor

Tinea versicolor (TV) is a common cutaneous fungal infection of the stratum corneum caused by the dimorphic lipophilic fungus Malassezia furfur.1 The infection is commonly characterized by hyperpigmented, hypopigmented, or pink, irregular, scaly macules occurring most often on the trunk. Rarely, overlapping lesions produce striking trichromatic patterns of pigmentation. The associated rash generally is asymptomatic, and the cosmetically displeasing pigment alteration is the presenting complaint of most patients. Pityrosporum ovale and Pityrosporum orbiculare represent the yeast phase of M furfur.2 P ovale appears to play an etiologic role in seborrheic dermatitis and Pityrosporum folliculitis.2,3 Under appropriate conditions, the saprophytic yeast converts to the mycelial morphology associated with the
characteristic scaling macules. Conditions associated with the mycelial transition are a warm humid environment, oily skin or an inherited predisposition for it, Cushing disease, depressed cellular immunity, or malnutrition.4

M furfur is readily identified by examining a simple potassium hydroxide mount of skin scrapings. The characteristic “spaghetti and meatballs” appearance of the hyphal and yeast elements confirms the diagnosis of TV, which makes culture unnecessary. Wood’s lamp examination reveals a yellowish to yellowish-green fluorescence and helps exclude erythrasma and tinea corporis from the differential diagnosis. The clinical differential diagnosis includes other entities that result in dyspigmentation, such as vitiligo, pityriasis alba, psoriasis, seborrheic dermatitis, confluent and reticulate (Gougerot-Carteaud) papillomatosis, erythrasma, and dermatophytosis.5

Several theories have been proposed to explain the dyspigmentation seen in pityriasis versicolor. Hypopigmentation may result from dicarboxylic acids such as azelaic acid produced by the fungus.5 Azelaic acid produces strong inhibition of the dopa-tyrosine reaction in vitro. These same acids also exhibit a cytotoxic effect on melanosomes.6,8 Another possibility is that the scale of TV prevents tanning in these areas.9 Hyperpigmentation in TV is not fully understood. An increased thickness of the keratin layer in hyperpigmented individuals7 and an inflammatory response resulting in stimulation of melanocytes are possible mechanisms.4

Treatment regimens include 2.5% selenium sulfide shampoo applied liberally and allowed to remain for 10 minutes to overnight before washing off. Ten-minute applications may be repeated daily for 2 weeks and then weekly to monthly to prevent recurrence. Overnight applications generally are performed monthly as needed. Topical azole antifungals, such as clotrimazole and ketoconazole are effective in treating TV, and topical terbinafine has been shown to be effective in both cream and gel forms.10,11 Terbinafine spray has been marketed for the treatment of TV. It is unclear how large a contribution the vehicle makes to the therapeutic effect of the product. Although topical treatment is ideal for cases with limited involvement, systemic therapy offers greater convenience when treating widespread disease. Systemic therapy with a single dose of ketoconazole (400 mg) was shown to be as effective as 200 mg daily for 10 days.12 Pityriasis versicolor often is recurrent, and repeat or maintenance treatment may be necessary. This can be accomplished with weekly or monthly topical selenium sulfide treatments, weekly washing with benzoyl peroxide or zinc pyrithione soap, or periodic systemic therapy.

REFERENCES