What Is Your Diagnosis?

A 54-year-old man presented with a rash on his arms and chest. The rash had been present for several years and remained unchanged. It was slightly pruritic and had been previously treated with hydrocortisone cream without improvement. On physical examination, hyperpigmented follicular papules with fine scale were noted over his chest, upper back, bilateral shoulders (left), and antecubital fossae (right). A potassium hydroxide preparation of a scraping from a lesion on his chest was positive for spores and hyphae.

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The Diagnosis: Folliculocentric Tinea Versicolor

Tinea versicolor (TV) is a superficial mycosis caused by various yeasts and lipophilic fungi of the genus *Malassezia*. There are 3 dominant species: *Malassezia globosa*, *Malassezia sympodialis*, and *Malassezia furfur*.[1] In the United States, the prevalence of TV is 2% to 8%.[2] Tinea versicolor usually presents as scaly hypopigmented or hyperpigmented macules on the chest, back, or proximal extremities.[3] An inverse variant manifests as lesions in flexural areas and more frequently is seen in immunocompromised individuals.[2,4] Tinea versicolor also may present as 2- to 3-mm, erythematous, perifollicular papules or pustules.[2,4,5]

Although *Malassezia* is a component of the normal flora of the skin, it also can be an opportunistic pathogen. In patients with clinical disease, the organism is found in both the yeast (spore) and filamentous (hyphal) form.[2] Besides TV, *Malassezia* organisms play a role in *Pityrosporum* folliculitis, seborrheic dermatitis, neonatal cephalic pustulosis, and some forms of atopic dermatitis.[2]

Tinea versicolor usually is found in young adults but can present in patients of all ages. Because the yeast of the genus *Malassezia* are lipophilic, the presence of fatty acids on the skin favors their growth.[6] In adolescence, hormonal stimulation causes the release of lipids by sebaceous glands to increase and *Malassezia* species develop in large quantities.[6] The most important predisposing factors that lead to the conversion of the saprophytic yeast to the parasitic mycelial forms and thus the development of TV include heat, humidity, and the use of oily lotions or creams and corticosteroids.[1,6-8] Genetic predisposition and malnutrition also may play a role.[6,9]

Malassezia species contain lipases, which are able to metabolize fatty acids, such as arachidonic acid or vaccenic acids, and azelaic acid is released as one of the metabolites. Azelaic acid inhibits the action of the tyrosinase enzyme and blocks the conversion of tyrosine to melanin, which results in the appearance of hypochromic macules.[6] Histologically, the skin within the hypopigmented macules contains melanosomes that are smaller than those found in normal skin.[6,10,11] It is not known how *Malassezia* species stimulate melanin production in hyperpigmented TV, though hyperkeratinization may play a role.[6]

Our patient had a folliculocentric variant of TV, which is an unusual presentation. Although folliculocentric TV has been reported,[12] it seems to be exceedingly rare. It should be distinguished from the more common *Pityrosporum* folliculitis. Other entities in the differential diagnosis of folliculocentric TV include follicular atopic dermatitis, folliculotropic mycosis fungoides, and pityriasis rubra pilaris.

Potassium hydroxide preparation readily distinguishes TV from these other diagnoses.

As with all other forms of TV, folliculocentric TV is treated with topical antifungal agents such as ketoconazole,[13] terbinafine hydrochloride,[14] ciclopirox,[15] and selenium sulfide. Systemic treatment with oral ketoconazole, itraconazole, or fluconazole may be necessary for patients with extensive disease or for patients who have been treated with topical agents without improvement.[3,13] Most topical and systemic treatments are effective, though trials comparing relative efficacy among different antifungal agents and regimens are lacking.[16]

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