

Answer to Dermacase continued from page 1557

4. Tinea versicolor

Tinea versicolor, also known as pityriasis versicolor, is a common superficial fungal infection of the skin caused by yeast species of the *Malassezia* genus. It affects nearly 1% of the general population and has an incidence of up to 50% in some tropical climates.¹ In temperate climates, eruptions occur more commonly in the summer than in the winter months.¹ The disease primarily affects adolescents and young adults, although it can occur at any age.

Clinically, tinea versicolor presents as multiple well-demarcated, scaly, oval-to-round hypo- or hyperpigmented macules that frequently coalesce into larger patches. The scale on the patches is usually subtle and is often best visualized by gently scraping the skin with a scalpel blade, the edge of a glass slide, or a fingernail.² As the term *versicolor* implies, the lesions can be of varying colours, such as white, pink, tan, light brown, and dark brown.^{2,3} The lesions are typically distributed on the upper trunk, upper arms, and neck.⁴ A follicular variant and an inverse form that involves the face, flexural regions, and extremities, have also been described.⁴ Although the disease is generally asymptomatic, patients might complain of pruritus.

Tinea versicolor is caused by dimorphic, lipophilic *Malassezia* organisms (*Malassezia furfur* being the most common), which are part of the normal skin flora. Clinical disease occurs when these organisms convert from their saprophytic yeast form to their pathogenic mycelial or hyphal form.⁴ This conversion can be triggered by a variety of exogenous and endogenous factors, including heat and moisture, occlusion of the skin by clothing or cosmetics, use of body oils, use of systemic corticosteroids, immunosuppression, Cushing disease, malnutrition, pregnancy, and hyperhidrosis. Hereditary factors might also play a role.²

The underlying mechanisms of hypo- and hyperpigmentation in tinea versicolor are not fully understood. Hypopigmentation might result from yeast production of azelaic acid, which inhibits tyrosinase, an enzyme responsible for melanin synthesis. Another possible explanation suggests that the thickened epidermis and scale characteristic of tinea versicolor blocks ultraviolet light and prevents tanning. Hyperpigmentation might result from a pronounced inflammatory response to the infection.²

Diagnosis

Diagnosis of tinea versicolor is made clinically and is confirmed by direct microscopic examination of scale prepared with 10% potassium hydroxide solution. The presence of both hyphae and spores in the characteristic "spaghetti and meatballs" pattern is diagnostic. A Wood lamp examination can also be useful, as affected



areas might fluoresce a bright yellow to coppery orange colour. This finding, however, only occurs in around one-third of cases.⁵ Skin biopsy and culture are not generally required to confirm diagnosis.

The differential diagnoses for tinea versicolor include vitiligo, pityriasis alba, postinflammatory hypo- and hyperpigmentation, seborrheic dermatitis, pityriasis rosea, guttate psoriasis, tinea corporis, nummular eczema, secondary syphilis, confluent reticulated papillomatosis of Gougerot and Carteaud, and mycosis fungoides.

Treatment

Tinea versicolor responds well to a variety of topical and systemic treatments. Topical therapy is generally reserved for patients with limited skin disease whereas systemic therapy is preferred for patients with more extensive or recurrent disease.

Topical treatment options include both nonspecific and specific antifungal agents. Nonspecific agents act by physically or chemically removing the infected stratum corneum without having direct antifungal activity.⁵ Examples include selenium sulfide, propylene glycol, benzoyl peroxide, and sulfur with salicylic acid. The most commonly used is 2.5% selenium sulfide. Available as a lotion, cream, or shampoo, it is applied to affected areas for 10 to 15 minutes daily, then washed off. The typical course of therapy is 7 days. Although it is effective, patients might complain about its unpleasant odour and a stinging sensation after application.⁶

Topical antifungal agents include the azoles and allylamines. A number of topical azoles have demonstrated efficacy in treating tinea versicolor, including ketoconazole, fluconazole, clotrimazole, miconazole, econazole, sertaconazole, and flutrimazole.^{1,5,7} Ketoconazole is the agent most commonly used. It is available as a cream or shampoo. Ketoconazole 2% shampoo has been shown to be effective when applied daily for 1, 3, or 14 days.^{7,8}

Terbinafine, an allylamine that is offered as a 1% cream or spray, has also been successful when applied twice daily for 7 days.⁵

Systemic therapy involves the use of oral antifungal agents. Ketoconazole is used most widely, although other options include fluconazole and itraconazole. Therapeutic efficacy with ketoconazole has been demonstrated at a daily dose of 200 mg for 5, 10, 14, or 28 days; and at a dose of 400 mg when administered once daily for 3 days, once weekly for 2 weeks, or 3 times every 12 hours or 7 days.⁵ A single 400-mg dose can also be effective, although relapse rates are thought to be higher with this regimen.⁹ The typical regimen for itraconazole is 200 mg/d for either 5 or 7 days, and fluconazole is given at a dose of 150 to 300 mg once weekly for 2 or 4 weeks. Many azoles are excreted by the eccrine sweat glands, so exercise and sweating after ingesting the oral medication might increase efficacy. Oral terbinafine and griseofulvin are generally ineffective in treating tinea versicolor.⁵

While a mycological cure can be achieved, normal pigmentation of the affected areas might not return for months after treatment cessation. Patients should be advised to avoid prolonged sun exposure during this time, as tanning will enhance the contrast between affected areas and normal skin.

Recurrence of tinea versicolor is not uncommon. For patients with frequent relapses, prophylactic treatment might be required. Effective options include monthly treatments of oral ketoconazole (a single 400-mg dose or 200 mg/d for 3 days) and itraconazole (a single 400-mg dose).⁵

Dr Prajapati is a first-year dermatology resident at the University of Alberta in Edmonton.

Dr Mydlarski is an Assistant Professor in the departments of medicine and medical genetics at the University of Calgary in Alberta.

Competing interests

None declared

References

- Gupta AK, Bluhm R, Summerbell R. Pityriasis versicolor. *J Eur Acad Dermatol Venereol* 2002;16(1):19-33.
- Sunenshine PJ, Schwartz RA, Janniger CK. Tinea versicolor. *Int J Dermatol* 1998;37(9):648-55.
- Gupta AK, Batra R, Bluhm R, Boekhout T, Dawson TL Jr. Skin diseases associated with *Malassezia* species. *J Am Acad Dermatol* 2004;51(5):785-98.
- Gupta AK, Ryder JE, Nicol K, Cooper EA. Superficial fungal infections: an update on pityriasis versicolor, seborrheic dermatitis, tinea capitis, and onychomycosis. *Clin Dermatol* 2003;21(5):417-25.
- Gupta AK, Batra R, Bluhm R, Faergemann J. Pityriasis versicolor. *Dermatol Clin* 2003;21(3):413-29.
- Faergemann J. Management of seborrheic dermatitis and pityriasis versicolor. *Am J Clin Dermatol* 2000;1(2):75-80.
- Rigopoulos D, Gregoriou S, Kontochristopoulos G, Ifantides A, Katsambas A. Flutrimazole shampoo 1% versus ketoconazole shampoo 2% in the treatment of pityriasis versicolor. A randomised double-blind comparative trial. *Mycoses* 2007;50(3):193-5.
- Lange DS, Richards HM, Guarnieri J, Humeniuk JM, Savin RC, Reyes BA, et al. Ketoconazole 2% shampoo in the treatment of tinea versicolor: a multicenter, randomized, double-blind, placebo-controlled study. *J Am Acad Dermatol* 1998;39(6):944-50.
- Sadeque JB, Shahidullah M, Shah OR, Kamal M. Systemic ketoconazole in the treatment of tinea versicolor. *Int J Dermatol* 1995;34(7):504-5.