Medscape

Tinea Versicolor

Updated: May 22, 2018
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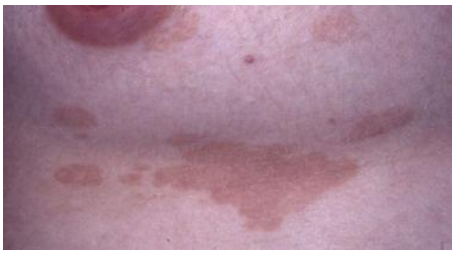
Overview

Background

Tinea versicolor is a common, benign, superficial cutaneous fungal infection usually characterized by hypopigmented or hyperpigmented macules and patches on the chest and the back. In patients with a predisposition, tinea versicolor may chronically recur. The fungal infection is localized to the stratum corneum. Note the image below.



In patients with lighter skin color, lesions frequently are light tan or salmon colored.

Pathophysiology

Tinea versicolor is caused by the dimorphic, lipophilic organisms in the genus Malassezia, formerly known as Pityrosporum. Fourteen species are recognized within this classification of yeasts, of which Malassezia globosa, Malassezia sympodialis, and Malassezia furfur are the predominant species isolated in tinea versicolor.[1, 2, 3, 4, 5, 6, 7, 8] Malassezia is extremely difficult to propagate in laboratory culture and is culturable only in media enriched with C12- to C14-sized fatty acids. Malassezia is naturally found on the skin surfaces of many animals, including humans. Indeed, it can be isolated in 18% of infants and 90-100% of adults.[9]

The organism can be found on healthy skin and on skin regions demonstrating cutaneous disease. In patients with clinical disease, the organism is found in both the yeast (spore) stage and the filamentous (hyphal) form. Factors that lead to the conversion of the saprophytic yeast to the parasitic, mycelial morphologic form include a genetic predisposition; warm, humid environments; immunosuppression; malnutrition; pregnancy; and Cushing disease. Human peptide cathelicidin LL-37 plays a role in skin defense against this organism.

Even though Malassezia is a component of the normal flora, it can also be an opportunistic pathogen. The organism is considered to be a factor in other cutaneous diseases, including Pityrosporum folliculitis, confluent and reticulate papillomatosis, seborrheic dermatitis, psoriasis, and some forms of atopic dermatitis. Malassezia species have also been shown to be a pulmonary pathogen in patients with immunosuppression due to stem cell transplantation.[10]

Etiology

Most cases of tinea versicolor occur in healthy individuals with no immunologic deficiencies. Nevertheless, several factors predispose some people to develop this condition. These factors include genetic predisposition; warm, humid environments; immunosuppression; malnutrition; application of oily preparations; corticosteroid usage; and Cushing disease.[11, 12, 13] The use of bath oils and skin lubricants may increase the risk of developing tinea versicolor.[14]

The reason why this organism causes tinea versicolor in some individuals while remains as normal flora in others is not entirely known. Several factors, such as the organism's nutritional requirements and the host's immune response to the organism, are significant.

The organism is lipophilic, and lipids are essential for growth in vitro and in vivo. Furthermore, the mycelial stage can be induced in vitro by the addition of cholesterol and cholesterol esters to the appropriate medium. Because the organism more rapidly colonizes humans during puberty when skin lipids are increased more than that of adolescent levels and tinea versicolor is manifested in sebum-rich areas (eg, chest, back), individual variations in skin surface lipids are hypothesized to play a major role in disease pathogenesis. However, patients with tinea versicolor and control subjects do not demonstrate any quantitative or qualitative differences in skin surface lipids. Skin surface lipids are significant for the normal presence of M furfur on human skin, but they probably play little role in the pathogenesis of tinea versicolor.

Evidence has been accumulating to suggest that amino acids, rather than lipids, are critical for the appearance of the diseased state. In vitro, the amino acid asparagine stimulates the growth of the organism, while another amino acid, glycine, induces hyphal formation. In vivo, the amino acid levels have been shown to be increased in the uninvolved skin of patients with tinea versicolor in two separate studies.

Another significant causative factor is the patient's immune system. Although sensitization against M furfur antigens is routinely present in the general population (as proven by lymphocyte transformation studies), lymphocyte function on stimulation with the organism has been shown to be impaired in patients who are affected. This outcome is similar to the situation of sensitization with Candida albicans. In short, cell-mediated immunity plays some role in disease causation.

Oxidative stress as shown by expression of reduced glutathione contributes to the pathogenesis of this condition.[15]

In steroid-associated atrophying tinea versicolor, the Malassezia infecting the epidermis may impair the barrier function of skin, thus allowing improved penetration of topical corticosteroids. This may lead to enhanced corticosteroid-induced atrophy of the affected skin.[16, 17, 18] However, as atrophying tinea versicolor has been reported in patients who have not used topical steroids, it has been proposed that a T-cell–mediated immune response to the Malassezia organism may be responsible for the atrophy noted. This theory suggests that Th1 cytokines recruit histiocytes to the site of epidermal infection. These histiocytes serve as a source of elastases and they up-regulate the metalloproteinase activity, ultimately leading to atrophy of the affected epidermis.[17, 19, 20]

Epidemiology

Frequency

United States

Tinea versicolor occurs more frequently in areas with higher temperatures and higher relative humidities. The national prevalence of this condition is 2-8% of the population. The exact incidence in the United States is difficult to assess because many individuals who are affected may not seek medical attention.

International

Tinea versicolor occurs worldwide, with prevalences reported to be as high as 50% in the humid, hot environment of Western Samoa and as low as 1.1% in the colder temperatures of Sweden.

Race

Although the alteration in skin pigmentation is more apparent in darker-skinned individuals, the incidence of tinea versicolor appears to be the same in all races.

Sex

Several studies have addressed the frequency of tinea versicolor based on sex, and no dominance of either sex is apparent.

Age

In the United States, tinea versicolor is most common in persons aged 15-24 years, when the sebaceous glands are more active. The occurrence of tinea versicolor before puberty or after age 65 years is uncommon.[21] In more tropical countries, age frequency varies; most cases involve people aged 10-19 years who live in warmer, humid countries, such as Liberia and India.

Prognosis

Tinea versicolor is a benign skin disease that causes scaly macules or papules on the skin. As the name implies (versi means several), the condition can lead to discoloration of the skin, with colors ranging from white to red to brown. The condition is not considered contagious because the causative fungal pathogen is a normal inhabitant of the skin. Treatment leads to cessation of scaling within a few days, but discoloration may last for weeks to months. If scale cannot be provoked and new lesions are not developing, then there is no need to repeat treatment and the patient can be reassured that ongoing infection in unlikely.

Although tinea versicolor is recurrent for some patients and, therefore, a chronic disease, the condition remains treatable with the available remedies (see Medical Care and Medication). Thus, the prognosis is excellent and new treatments continue to be developed.[22]

Patient Education

Patients need to realize that tinea versicolor is caused by a fungus that is normally present on the skin surface; thus, it is not considered a contagious disease. Sequelae from the disease are not permanent, and any pigmentary alterations resolve entirely 1-2 months after treatment is initiated. Treatment is needed to remedy the condition and for prophylaxis to prevent recurrences.

Presentation

History

Most individuals with tinea versicolor report cosmetically disturbing, abnormal pigmentation. The involved skin regions are usually the trunk, the back, the abdomen, and the proximal extremities. The face, the scalp, and the genitalia are less commonly involved. In patients with fair skin, the color of each lesion varies from almost white to reddish-brown or fawn colored. In darker skin types, involved areas can have varying degrees of either hypopigmentation or hyperpigmentation. A fine, dustlike scale covers the lesions.

Tinea versicolor patients often report that the involved skin lesions fail to tan in the summer and cause the affected areas to become more apparent. Conversely, affected areas may become subtler in winter months as background tan fades.

Occasionally, a tinea versicolor patient also reports mild pruritus. In most instances, the condition is asymptomatic.

Greater than 20% of tinea versicolor patients report a family history of the condition. This subset of patients records a higher rate of recurrence and longer duration of disease.[23]

Physical Examination

Although tinea versicolor is often diagnosed by the observation of the classic hypopigmented-tohyperpigmented, centrally coalescing, oval-to-round patches with mild scale, it can occasionally be difficult to distinguish from other dermatoses. The scale on these lesions is not always immediately evident and may require scratching or stretching of the skin surface.[14]

A dermatoscope is also a useful diagnostic tool in examining affected skin. In hypopigmented lesions, dermoscopy shows a well-demarcated, white area with fine scales found largely in skin furrows. Hyperpigmented lesions under dermoscopy show fine scale in skin furrows overlying brown pigmentation.[24, 25]

Tinea versicolor can present in four forms in addition to a rare, atrophic form.

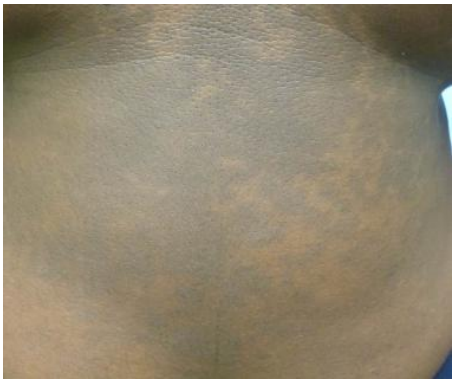
Tinea versicolor - Form 1

The most common appearance of the disease is as numerous, well-marginated, finely scaly, oval-to-round macules scattered over the upper trunk, with occasional extension to the lower trunk, neck, and proximal extremities.

The macules tend to coalesce, forming irregularly shaped patches of pigmentary alteration. As the name versicolor implies, the disease characteristically reveals a variance in skin hue. The involved areas can be either darker or lighter than the surrounding skin.

The condition is more noticeable during the summer months when the discrepancy in color from the normal skin becomes more apparent.

Fine, powdery scale may be readily apparent and is uniformly provocable with light scraping of the involved skin with a scalpel blade or the edge of a glass slide. See the image below.



Hyperpigmented macules forming some confluent patches on the abdomen. While scale is not readily apparent, it is easily provoked with light scratching. On dark skin, affected areas may be hypopigmented or hyperpigmented.

Tinea versicolor - Form 2

An inverse form of tinea versicolor also exists in which the condition has an entirely different distribution, affecting the flexural regions, the face, or isolated areas of the extremities. This form of tinea versicolor is more often seen in hosts who are immunocompromised.

This form of the disease can be confused with candidiasis, seborrheic dermatitis, psoriasis, erythrasma, and dermatophyte infections.

Tinea versicolor - Form 3

The third form of Malassezia infections of the skin involves the hair follicle. This condition is typically localized to the back, the chest, and the extremities.

This form can be clinically difficult to differentiate from bacterial folliculitis. The presentation of Pityrosporum folliculitis is a perifollicular, erythematous papule or pustule.

Predisposing factors include diabetes, high humidity, steroid or antibiotic therapy, and immunosuppressant therapy. Additionally, several reports reveal that M furfur also plays a role in seborrheic dermatitis.

Tinea versicolor - Form 4

Another clinical presentation is multiple firm, 2- to 3-mm, monomorphic, red-brown, inflammatory papules. These lesions may, or may not also demonstrate a fine white scale.

The lesions are usually found on the torso and are asymptomatic.

Histologically, the rash demonstrates not only fungal hyphae and spores in the stratum corneum, but also an interface dermatitis in the superficial dermis.[26]

Atrophying tinea versicolor

Atrophying tinea versicolor is a rare form that presents as atrophic, ivory-colored–to–erythematous lesions that are oval-to-round in shape. These lesions can have a wrinkled surface, and the atrophy is limited to the areas of skin affected by tinea versicolor.[16, 27]

Histologic features of these lesions include epidermal atrophy, vascular ectasia, and rarefaction of collagen and elastic fibers.[16, 19]

These lesions are often reported in patients who are either misdiagnosed or have a comorbid condition requiring an extended use of topical corticosteroids.[16, 17] However, cases of atrophying tinea versicolor have been described in patients who do not have a history of using topical corticosteroids.[19]

DDx

Differential Diagnoses

- Confluent and Reticulated Papillomatosis
- Erythrasma
- Guttate Psoriasis
- Pityriasis Alba
- Seborrheic Dermatitis
- Tinea Corporis
- Vitiligo

Workup

Workup

Laboratory Studies

The clinical presentation of tinea versicolor is distinctive, and the diagnosis is often made without any laboratory documentation.

The ultraviolet black light (Wood lamp) can be used to demonstrate the coppery-orange fluorescence of tinea versicolor. However, in some cases, the lesions appear darker than the unaffected skin under the Wood lamp, but they do not fluoresce.

The diagnosis is usually confirmed by potassium hydroxide (KOH) examination, which demonstrates the characteristic short, cigar-butt hyphae that are present in the diseased state. The KOH finding of spores with short mycelium has been referred to as the spaghetti and meatballs or the bacon and eggs sign of tinea versicolor. For better visualization, ink blue stain, Parker ink, methylene blue stain, or Swartz-Medrik stain can be added to the KOH preparation. Contrast stain containing 1% Chicago sky blue 6B and 8% KOH (as the clearing agent) achieves the greatest sensitivity and specificity.[28]

Special media are required for culture. Because the diagnosis is usually clinically suspected and can be confirmed with a KOH preparation, cultures are rarely obtained.

With blood examination, no definitive deficiencies of normal antibodies or complement are present in patients with tinea versicolor, but research continues in this area. For example, although individuals who are affected reveal no specific antibody levels above those of age-matched controls, M furfur antigens do elicit a specific immunoglobulin G response in patients with seborrheic dermatitis and tinea versicolor detected by enzyme-linked immunosorbent assay and Western blotting assays. M furfur does induce immunoglobulin A, immunoglobulin G, and immunoglobulin M antibodies, and it can activate complement via both the alternate pathway and the classical pathway.

Various studies have found defects in lymphokine production, natural killer T cells, decreased phytohemagglutinin and concanavalin A stimulation, interleukin 1, interleukin 10, and interferon gamma production by lymphocytes in patients.

Although these tests do not suggest an immunologic disorder, they do suggest a reduced body response to the specific fungal elements that produce tinea versicolor. Further assessment is warranted.

Histologic Findings

The organism that causes tinea versicolor is localized to the stratum corneum. M furfur can be detected by hematoxylin and eosin (H&E) alone, although periodic acid-Schiff (PAS) or methenamine silver staining are more confirmatory. On rare occurrences, the organism can approach the stratum granulosum, and it can even be found inside keratinocytes.[29] The epidermis reveals mild hyperkeratosis and acanthosis, and a mild perivascular infiltrate is present in the dermis. An acanthosis nigricans–like epidermal change is noted in the papular variety, with dilated blood vessels observed in erythematous lesions.

Treatment

Medical Care

Patients should be informed that tinea versicolor is caused by a fungus that is normally present on the skin surface and is therefore not considered contagious. The condition does not leave any permanent scar or pigmentary changes, and any skin color alterations resolve within 1-2 months after treatment has been initiated. Recurrence is common, and prophylactic therapy may help reduce the high rate of recurrence.

Tinea versicolor can be successfully treated with various agents.[30] Effective topical agents include selenium sulfide, zinc-pyrithione, sodium sulfacetamide, ciclopirox olamine,[31] , tacrolimus,[32] as well as azole and allylamine antifungals.[33, 34, 35, 36, 37] Even if a small area of skin is involved, treating with topicals from the neck to the knees may make treatment more successful.[14] Various regimens can be used. Selenium sulfide lotion is liberally applied to affected areas of the skin daily for 2 weeks; each application is allowed to remain on the skin for at least 10 minutes prior to being washed off. In resistant cases, overnight application can be helpful. Topical azole antifungals can be applied every night for 2 weeks. Weekly application of any of the topical agents for the following few months may help prevent recurrence. In patients with widespread disease, some topical antifungal therapy can be expensive. Over-the-counter shampoo formulations of selenium sulfide, zinc-pyrithione, and ketoconazole are low-cost options that are widely available and can easily be used to cover large surface areas. Topical allylamines have been demonstrated to be clinically and mycologically effective. Tacrolimus 0.03% applied topically has been shown to provide a mycologically effective treatment; however, it is not effective in speeding the reduction in appearance of hypopigmentation associated with tinea versicolor.

While oral ketoconazole is contraindicated for the treatment of tinea versicolor, the topical foam may be useful in some patients.[38] The risk of serious liver damage, adrenal gland problems, and harmful drug interactions with use of oral ketoconazole outweighs it benefit for fungal skin infections.[39]

Oral therapy with other systemic antifungals is effective for tinea versicolor and is often preferred by patients because of convenience and oral administration is less time consuming than topical treatment. Of course, oral therapy can be used in consort with topical regimens. Fluconazole, and itraconazole are the preferred oral agents.[40, 41, 42] Various dosing regimens have been used. Fluconazole has been offered as a single 150- to 300-mg weekly dose for 2-4 weeks and is the safest oral agent. Itraconazole is usually given at 200 mg/d for 7 days. Pramiconazole and sertaconazole have also been used in the management of tinea versicolor.[43, 44] A review suggested the following dosing regimens: 200 mg/d for 5 or 7 days of itraconazole, 300 mg/wk for 2 weeks of fluconazole, and 200 mg/d for 2 days of pramiconazole.[45]

Oral therapy does not prevent the high rate of recurrence, and treatment with an oral or topical agent may need to be repeated intermittently throughout the year. Because tinea versicolor is a benign condition and oral therapy is not without risk, the decision to treat with an oral agent should be made only after a complete discussion of the risks involved.[46] In the case of oral terbinafine, some subgroups of M furfur apparently are not clinically responsive, although in vitro studies suggest fungistatic activity.[47] Also, a regimen of 1 tablet a month of fluconazole or itraconazole has been used successfully to prophylactically prevent recurrences.[48]

Reports describe successful treatment of tinea versicolor with photodynamic therapy.[49, 50]

Diet

Dietary alterations have not proved successful in the treatment of tinea versicolor.

Long-Term Monitoring

Tinea versicolor has a high rate of recurrence, and prophylactic treatment with topical or oral therapy on an intermittent basis is necessary to prevent recurrences in most cases.

Medication

Medication Summary

Tinea versicolor responds well to both topical and oral antimycotic therapies. Many patients prefer oral therapy because of its convenience.

Antifungals

Class Summary

Topical antifungals temporarily eradicate the condition, although treatment may need to be intermittently repeated to prevent recurrence. Oral therapy for tinea versicolor is convenient and effective, but it does not prevent recurrences. A once-monthly (for 6 mo) oral dose of fluconazole is a popular alternative.

Terbinafine topical (Lamisil)

Terbinafine topical inhibits squalene epoxidase, which decreases ergosterol synthesis, causing fungal cell death. Use this medication until symptoms significantly improve. The duration of treatment should be greater than 1 week but not greater than 4 weeks.

Clotrimazole topical (Mycelex, Lotrimin-AF)

Clotrimazole topical is a broad-spectrum antifungal agent that inhibits yeast growth by altering cell membrane permeability, causing fungal cell death. Reevaluate the diagnosis if no clinical improvement is seen after 4 weeks.

Ketoconazole topical (Extina, Ketozole)

Ketoconazole is an imidazole broad-spectrum antifungal agent. It inhibits the synthesis of ergosterol, causing cellular components to leak, resulting in fungal cell death. Ketoconazole achieves excellent skin levels with minimal oral dosing, but oral treatment with this agent for skin infections is contraindicated because of safety concerns. M furfur is eradicated by the presence of ketoconazole in outer skin layers.

Ciclopirox (Loprox)

Ciclopirox interferes with the synthesis of DNA, RNA, and protein by inhibiting the transport of essential elements in fungal cells.

Butenafine (Mentax)

Butenafine damages fungal cell membranes, causing fungal cell growth to arrest.

Naftifine (Naftin)

Naftifine is a broad-spectrum antifungal agent and synthetic allylamine derivative; it may decrease the synthesis of ergosterol, which, in turn, inhibits fungal cell growth. If no clinical improvement is seen after 4 weeks, reevaluate the patient.

Econazole topical (Ecoza)

Econazole is effective in cutaneous infections. It interferes with RNA and protein synthesis and metabolism. It disrupts fungal cell wall membrane permeability, causing fungal cell death.

Oxiconazole (Oxistat)

Oxiconazole damages the fungal cell wall membrane by inhibiting the biosynthesis of ergosterol. Membrane permeability is increased, causing nutrients to leak out and resulting in fungal cell death.

Questions & Answers

Overview

What is tinea versicolor (pityriasis versicolor)?

What causes tinea versicolor (pityriasis versicolor)?

What factors increase the risk of developing tinea versicolor (pityriasis versicolor)?

In addition to tinea versicolor (pityriasis versicolor), what other diseases are caused by Malassezia infection?

Which factors predispose to tinea versicolor (pityriasis versicolor)?

When does Malassezia infection cause tinea versicolor (pityriasis versicolor)?

What is the role of lipids in the pathogenesis of tinea versicolor (pityriasis versicolor)?

What is the role of amino acids in the pathogenesis of tinea versicolor (pityriasis versicolor)?

What is the role of the immune system in the development of tinea versicolor (pityriasis versicolor)?

What is the role of oxidative stress in the pathogenesis of tinea versicolor (pityriasis versicolor)?

What is the role of topical steroids in the pathogenesis of tinea versicolor (pityriasis versicolor)?

What is the prevalence of tinea versicolor (pityriasis versicolor) in the US?

What is the international prevalence of tinea versicolor (pityriasis versicolor)?

Does the incidence of tinea versicolor (pityriasis versicolor) vary by race?

Is tinea versicolor (pityriasis versicolor) more common in men or women?

Is tinea versicolor (pityriasis versicolor) more common in certain age groups?

What is the prognosis of tinea versicolor (pityriasis versicolor)?

What information should be given to reassure patients with tinea versicolor (pityriasis versicolor)?

Presentation

How does tinea versicolor (pityriasis versicolor) affect different areas and types of skin?

Do tinea versicolor (pityriasis versicolor) skin lesions tan?

Does tinea versicolor (pityriasis versicolor) cause pruritus?

How common is family history of tinea versicolor (pityriasis versicolor)?

What are the physical exam findings of tinea versicolor (pityriasis versicolor) and what is the role of a dermatoscope in diagnosis?

What are the most common physical findings of tinea versicolor (pityriasis versicolor)?

What are the physical findings of the inverse form of tinea versicolor (pityriasis versicolor)?

What are the physical findings of tinea versicolor (pityriasis versicolor) of the hair follicles?

What are the physical findings of papular tinea versicolor (pityriasis versicolor)?

What is the typical presentation of atrophying tinea versicolor?

DDX

What are the differential diagnoses for Tinea Versicolor?

Workup

Is an ultraviolet black light (Wood lamp) used to diagnose tinea versicolor (pityriasis versicolor)?

What is the role of a potassium hydroxide (KOH) exam in the diagnosis of tinea versicolor (pityriasis versicolor)?

What is the role of culture in the diagnosis of tinea versicolor (pityriasis versicolor)?

What is the role of testing in the diagnosis of tinea versicolor (pityriasis versicolor)?

What are the histologic findings of tinea versicolor (pityriasis versicolor)?

Treatment

What should a patient with tinea versicolor (pityriasis versicolor) be informed about its potential for contagiousness, permanence, and recurrence?

Which topical agents are effective for the treatment of tinea versicolor (pityriasis versicolor)?

What are the risks of oral ketoconazole for the treatment of tinea versicolor (pityriasis versicolor)?

Which oral antifungal agents are effective in the treatment of tinea versicolor (pityriasis versicolor)?

Does oral therapy for tinea versicolor (pityriasis versicolor) prevent recurrence?

What should be considered before selecting oral antifungal agents for the treatment of tinea versicolor (pityriasis versicolor)?

Are dietary changes beneficial in the treatment of tinea versicolor (pityriasis versicolor)?

How can tinea versicolor (pityriasis versicolor) recurrence be prevented?

Medications

What types of medications are effective in the treatment of tinea versicolor (pityriasis versicolor)?

Which medications in the drug class Antifungals are used in the treatment of Tinea Versicolor?

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Disclosure: Nothing to disclose.

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Disclosure: Nothing to disclose.

Acknowledgements

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Disclosure: Nothing to disclose.

References

1. Crespo-Erchiga V, Florencio VD. Malassezia yeasts and pityriasis versicolor. Curr Opin Infect Dis. 2006 Apr. 19(2):139-47. [Medline].
2. Gaitanis G, Velegraki A, Alexopoulos EC, Chasapi V, Tsigonia A, Katsambas A. Distribution of Malassezia species in pityriasis versicolor and seborrhoeic dermatitis in Greece. Typing of the major pityriasis versicolor isolate M. globosa. Br J Dermatol. 2006 May. 154(5):854-9. [Medline].
3. Morishita N, Sei Y, Sugita T. Molecular analysis of malassezia microflora from patients with pityriasis versicolor. Mycopathologia. 2006 Feb. 161(2):61-5. [Medline].
4. Rincon S, Celis A, Sopo L, Motta A, Cepero de Garcia MC. Malassezia yeast species isolated from patients with dermatologic lesions. Biomedica. 2005 Jun. 25(2):189-95. [Medline].
5. Krisanty RI, Bramono K, Made Wisnu I. Identification of Malassezia species from pityriasis versicolor in Indonesia and its relationship with clinical characteristics. Mycoses. 2009 May. 52(3):257-62. [Medline].
6. Prohic A, Jovovic Sadikovic T, Krupalija-Fazlic M, Kuskunovic-Vlahovljak S. Malassezia species in healthy skin and in dermatological conditions. Int J Dermatol. 2016 May. 55 (5):494-504. [Medline].
7. Nakabayashi A, Sei Y, Guillot J. Identification of Malassezia species isolated from patients with seborrhoeic dermatitis, atopic dermatitis, pityriasis versicolor and normal subjects. Med Mycol. 2000 Oct. 38 (5):337-41. [Medline].
8. Tarazooie B, Kordbacheh P, Zaini F, Zomorodian K, Saadat F, Zeraati H, et al. Study of the distribution of Malassezia species in patients with pityriasis versicolor and healthy individuals in Tehran, Iran. BMC Dermatol. 2004 May 1. 4:5. [Medline].
9. Prohić A, Jovović Sadiković T, Kuskunović-Vlahovljak S, Baljić R. Distribution of Malassezia Species in Patients with Different Dermatological Disorders and Healthy Individuals. Acta Dermatovenerol Croat. 2016 Dec. 24 (4):274-281. [Medline].
10. Blaes AH, Cavert WP, Morrison VA. Malassezia: is it a pulmonary pathogen in the stem cell transplant population?. Transplant Infectious Disease. August, 2009. 11:313-317. [Medline].
11. Burkhart CG, Dvorak N, Stockard H. An unusual case of tinea versicolor in an immunosuppressed patient. Cutis. 1981. 27(1):56-8. [Medline].
12. Gulec AT, Demirbilek M, Seckin D, et al. Superficial fungal infections in 102 renal transplant recipients: a case-control study. J Am Acad Dermatol. 2003 Aug. 49(2):187-92. [Medline].
13. Mendez-Tovar LJ. Pathogenesis of dermatophytosis and tinea versicolor. Clinics in Dermatology. 2010. 28:185-188. [Medline].
14. Fungal Diseases. Bolognia J, Jorizzo JL, Schaffer JV, eds. Dermatology. 3rd ed. Philadelphia, Pa: Elsevier Saunders; 2012. Vol 2: 1251-55.
15. Kilic M, Oguztuzum S, Karadag S, Cakir E, Aydin M, Ozturk L. Expression of GSTM4 and GSTT1 in patients with Tinea versicolor, Tinea inguinalis, and Tinea pedis infections: a preliminary study. Clinical Dermatology. 2011. 36:590-594. [Medline].
16. Haiduk J, Treudler R, Ziemer M. Atrophying tinea versicolor with epidermal atrophy. J Dtsch Dermatol Ges. 2016 Jul. 14 (7):740-3. [Medline].
17. Cullingham K, Hull PR. Atrophying pityriasis versicolor. CMAJ. 2014 Jul 8. 186 (10):776. [Medline].
18. Tatnall FM, Rycroft RJ. Pityriasis versicolor with cutaneous atrophy induced by topical steroid application. Clin Exp Dermatol. 1985 May. 10 (3):258-61. [Medline].
19. Crowson AN, Magro CM. Atrophying tinea versicolor: a clinical and histological study of 12 patients. Int J Dermatol. 2003 Dec. 42 (12):928-32. [Medline].
20. Levy JM, Magro C. Atrophying pityriasis versicolor as an idiosyncratic T cell-mediated response to Malassezia: A case series. J Am Acad Dermatol. 2016 Nov 2. [Medline].
21. Muhammad N, Kamal M, Islam T, Islam N, Shafiquzzaman M. A study to evaluate the efficacy and safety of oral fluconazole in the treatment of tinea versicolor. Mymensingh Med J. 2009 Jan. 18(1):31-5. [Medline].
22. Carreira A, Ferreira JB, Pereira I, Ferreira J, Filipe P, Ferreira RB, et al. Blad-containing oligomer: a novel fungicide used in crop protection as an alternative treatment for tinea pedis and tinea versicolor. J Med Microbiol. 2018 Feb. 67 (2):198-207. [Medline].
23. He SM, Du WD, Yang S, et al. The genetic epidemiology of tinea versicolor in China. Mycoses. 2008 Jan. 51(1):55-62. [Medline].
24. Zhou H, Tang XH, De Han J, Chen MK. Dermoscopy as an ancillary tool for the diagnosis of pityriasis versicolor. J Am Acad Dermatol. 2015 Dec. 73 (6):e205-6. [Medline].
25. Errichetti E, Stinco G. Dermoscopy in General Dermatology: A Practical Overview. Dermatol Ther (Heidelb). 2016 Dec. 6 (4):471-507. [Medline].
26. Suwattee P, Cham PM, Solomon RK, Kaye VN. Tinea versicolor with interface dermatitis. J Cutan Pathol. 2009 Feb. 36(2):285-6. [Medline].
27. Romano C, Maritati E, Ghilardi A, Miracco C, Mancianti F. A case of pityriasis versicolor atrophicans. Mycoses. 2005 Nov. 48 (6):439-41. [Medline].
28. Lim SL, Lim CS. New contrast stain for the rapid diagnosis of pityriasis versicolor. Arch Dermatol. 2008 Aug. 144(8):1058-9. [Medline].
29. Janaki C, Sentamilselvi G, Janaki VR, Boopalraj JM. Unusual observations in the histology of Pityriasis versicolor. Mycopathologia. 1997. 139(2):71-4. [Medline].
30. Gupta AK, Lyons DC. Pityriasis versicolor: an update on pharmacological treatment options. Expert Opin Pharmacother. 2014 Aug. 15(12):1707-13. [Medline].
31. Gupta AK, Skinner AR. Ciclopirox for the treatment of superficial fungal infections: a review. Int J Dermatol. 2003 Sep. 42 Suppl 1:3-9. [Medline].
32. Sepaskhah M, Sadat MS, Pakshir K, Bagheri Z. Comparative efficacy of topical application of tacrolimus and clotrimazole in the treatment of pityriasis versicolor: A single blind, randomised clinical trial. Mycoses. 2017 Jan 25. [Medline].
33. Hull CA, Johnson SM. A double-blind comparative study of sodium sulfacetamide lotion 10% versus selenium sulfide lotion 2.5% in the treatment of pityriasis (tinea) versicolor. Cutis. 2004 Jun. 73(6):425-9. [Medline].
34. Vermeer BJ, Staats CC. The efficacy of a topical application of terbinafine 1% solution in subjects with pityriasis versicolor: a placebo-controlled study. Dermatology. 1997. 194 Suppl 1:22-4. [Medline].
35. Carrillo-Munoz AJ, Giusiano G, Ezkurra PA, Quindos G. Sertaconazole: updated review of a topical antifungal agent. Expert Rev Anti Infect Ther. 2005 Jun. 3(3):333-42. [Medline].
36. Gold M, Bridges T, Avakian E, Plaum S, Pappert EJ, Fleischer AB, et al. An open-label study of naftifine hydrochloride 1% gel in the treatment of tinea versicolor. Skin Med. Sept 2011. 9:283-6. [Medline].
37. Croxtall J, Plosker G. Sertaconazole: a reviw of its use in the management of superficial mycoses in dermatology and gynacology. Ingenta/Adis International. Jan 2009. 69:339-359. [Medline].
38. Cantrell WC, Elewksi BE. Can pityriasis versicolor be treated with 2% ketoconazole foam?. J Drugs Dermatol. 2014 Jul 1. 13(7):855-9. [Medline].
39. U.S. Food and Drug Administration. Nizoral (ketoconazole) oral tablets: Drug Safety Communication - Prescribing for unapproved uses including skin and nail infections continues; linked to patient death. FDA Medwatch. May 19, 2016. Available at http://www.fda.gov/safety/medwatch/safetyinformation/safetyalertsforhumanmedicalproducts/ucm502073.htm.
40. Hickman JG. A double-blind, randomized, placebo-controlled evaluation of short-term treatment with oral itraconazole in patients with tinea versicolor. J Am Acad Dermatol. 1996 May. 34(5 Pt 1):785-7. [Medline].
41. Karakas M, Durdu M, Memisoglu HR. Oral fluconazole in the treatment of tinea versicolor. J Dermatol. 2005 Jan. 32(1):19-21. [Medline].
42. Partap R, Kaur I, Chakrabarti A, Kumar B. Single-dose fluconazole versus itraconazole in pityriasis versicolor. Dermatology. 2004. 208(1):55-9. [Medline].
43. Wahab MA, Ali ME, Rahman MH, Chowdhury SA, Monamie NS, Sultana N, et al. Single dose (400mg) versus 7 day (200mg) daily dose itraconazole in the treatment of tinea versicolor: a randomized clinical trial. Mymensingh Med J. 2010 Jan. 19(1):72-6. [Medline].
44. Faergemann J, Todd G, Pather S, et al. A double-blind, randomized, placebo-controlled, dose-finding study of oral pramiconazole in the treatment of pityriasis versicolor. J Am Acad Dermatol. 2009 Dec. 61(6):971-6. [Medline].
45. Gupta AK, Lane D, Paquet M. Systematic review of systemic treatments for tinea versicolor and evidence-based dosing regimen recommendations. J Cutan Med Surg. 2014 Mar-Apr. 18(2):79-90. [Medline].
46. Mellen LA, Vallee J, Feldman SR, Fleischer AB Jr. Treatment of pityriasis versicolor in the United States. J Dermatolog Treat. 2004 Jun. 15(3):189-92. [Medline].
47. Leeming JP, Sansom JE, Burton JL. Susceptibility of Malassezia furfur subgroups to terbinafine. Br J Dermatol. 1997 Nov. 137(5):764-7. [Medline].
48. Faergemann J, Gupta AK, Al Mofadi A, Abanami A, Shareaah AA, Marynissen G. Efficacy of itraconazole in the prophylactic treatment of pityriasis (tinea) versicolor. Arch Dermatol. 2002 Jan. 138(1):69-73. [Medline].
49. Kim YJ, Kim YC. Successful treatment of pityriasis versicolor with 5-aminolevulinic acid photodynamic therapy. Arch Dermatol. 2007 Sep. 143(9):1218-20. [Medline].
50. Qiao J, Li R, Ding Y, Fang H. Photodynamic therapy in the treatment of superficial mycoses: an evidence-based evaluation. Mycopathologia. 2010. 170:339-343. [Medline].
51. Fernandez-Nava HD, Laya-Cuadra B, Tianco EA. Comparison of single dose 400 mg versus 10-day 200 mg daily dose ketoconazole in the treatment of tinea versicolor. Int J Dermatol. 1997 Jan. 36(1):64-6. [Medline].
52. Burkhart CG. Tinea versicolor. J Dermatol Allergy. 1983. 6:8-12.