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PITYRIASIS VERSICOLOR: A FEW NEW ASPECTS ON ETIOLOGY, PATHOGENESIS, AND TREATMENT

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ABSTRACT

Pityriasis versicolor (PV), also known as tinea versicolor, is caused by *Malassezia* species. Clinical features of pityriasis versicolor include either hyperpigmented or hypopigmented finely scaly macules. The most frequently affected sites are the trunk, neck, and proximal extremities. In cases of pathogenicity, Malassezia can directly harm the host via virulence factors or toxins, or indirectly by triggering damaging host responses. The diagnosis typically relies on recognising characteristic clinical features. Due to the wide variability in its clinical presentation, recognising the differential diagnosis is critical. Topical therapies are the primary treatment for PV, encompassing nonspecific antifungal agents like sulphur with salicylic acid, selenium sulphide 2.5%, and zinc pyrithione. Additionally, specific topical antifungal medications with either fungicidal or fungistatic properties may also be incorporated into the topical treatment regimen, such as imidazoles, allylamines, and ciclopirox olamine. Patient education and the promotion of good personal hygiene are pivotal to reduce the risk of recurrence. In this paper, we discuss the pathophysiology, risk factors clinical features, diagnosis, and management as well as prevention of *Tinea versicolor.*

Keywords: *Tinea versicolor*, *Malassezia* species, Zinc pyrithione, scaly macules, pathophysiology

INTRODUCTION:

Tinea versicolor also known as dermatomycosis furfuracea, Pityriasis versicolor (PV) and Tinea flava. It manifests as poorly to well-demarcated discoloured or light pink scaly patches, usually affecting the trunk and arms. The disease occurs worldwide but is most prevalent in humid and warm tropical regions. PV tends to be more active in summer seasons **[1-4]**. These fungi are common parts of the cutaneous organisms on humans as well as warm-blooded animals **[5]**. On the trunk and upper arms, patients with tinea versicolor tend to display asymptomatic hypopigmented or hyperpigmented, finely scaled round or oval macules/patches **[6]**. However, even receiving proper treatment, the disease can usually occur, which worsen the impact on PV those suffering' quality of life **[7]**. Presence of itching has been suggested to be based on the type of lesion, severity of condition, site of the lesions, and other associated factors such as sweat or exposure to sunlight. The disease is most commonly observed in young adult people. Many species of the fungus *Malassezia* are observed in skin of healthy people as part of normal microbiota. In, its yeast phase, the organism shows two morphologically distinct forms: an ovoid form and a spherical form. The fungus is designated by its given names Pityrosporum ovale and Pityrosporum orbiculare, in that direction **[8, 9]**. Additional, non-invasive work-up (including dermatoscopy, UV-induced fluorescence dermatoscopy, Wood's light examination, or direct microscopy) helps with diagnostic with cases which may be clinically vague. A rise in temperature, humidity, and carbon dioxide tension are significant risk factors **[10]**. These fungi are a common component of the cutaneous microbiota on humans and other warm-blooded animals **[11]**. Although modest pruritus is experienced by some people, PV is typically asymptomatic **[12]**. The most common issue among patients who are seeking therapy is the unsightly appearance of their skin **[13]**. Skin scrapings from the edges of lesions are used for microscopy to confirm the diagnosis of PV **[14]**.

Pathophysiology

A fungus called Malassezia resembles yeast and is dimorphic. It only produces PV when it is in the pathogenic, hyphal, filamentous form. It is the most prevalent type of fungus found in every part of the human body other than the feet **[15]**. Patients with dandruff have higher trans epidermal water loss, which is indicative of a disturbance of the skin barrier. Itching, flaking, and erythema—symptoms that are easily associated with seborrhoeic dermatitis and dandruff—are directly caused by this phenomenon **[16]**. Hot and muggy conditions, hyperhidrosis, greasy emollient

application, wearing face masks for protection, seborrhea, endocrine and neuropathic disorders, pregnancy, oral contraceptive use, corticosteroid use, malnourishment, poor general health, and genetic predisposition are all factors that favour the conversion from the yeast-like form **[17]**. Toxins and virulence factors produced by Malassezia add to its pathogenicity. Malassezia, on the other hand, obtains nutrients from the human host in a commensal relationship without harming it. Moreover, the idea of mutualism emerges when Malassezia, a fungus, colonises the epidermis and offers defence against potentially harmful microorganisms like *S. aureus* **[18]**. Malassezia interacts with skin in two ways: first, by direct contact, where some metabolites of the fungus, such as those that cause skin irritation, can irritate the skin. Indirect touch is the other method, which causes skin inflammation by stimulating immunological or allergy pathways **[19]**. By cleaving the extracellular proteins of the host and other microbes, these enzymes alter the external environment directly **[20]**. Moreover, these proteases function as virulence agents, particularly in skin with weaker defences. Breakdown of Free fatty acids (FFAs) of the skin are the most likely source of epidermal hyperproliferation. FFAs from sebaceous triglycerides are essential to Malassezia since they are lipid-dependent fungus. Malassezia's lipases extract different FFAs from sebum; some fatty acids are retained while others are discarded. The scalp's epidermal barrier is breached by these leftover FFAs as they enter the stratum corneum. Studies conducted in vitro revealed that aspartyl proteases produced from Malassezia are capable of hydrolyzing *S. aureus* protein A, a crucial virulence component involved in immune evasion and biofilm formation. The synthesis of phospholipase, lipase, acid sphingomyelinases (which break down sebum lipids), haemolysin, and the capacity to form a biofilm are additional significant virulence factors of Malassezia **[21]**.

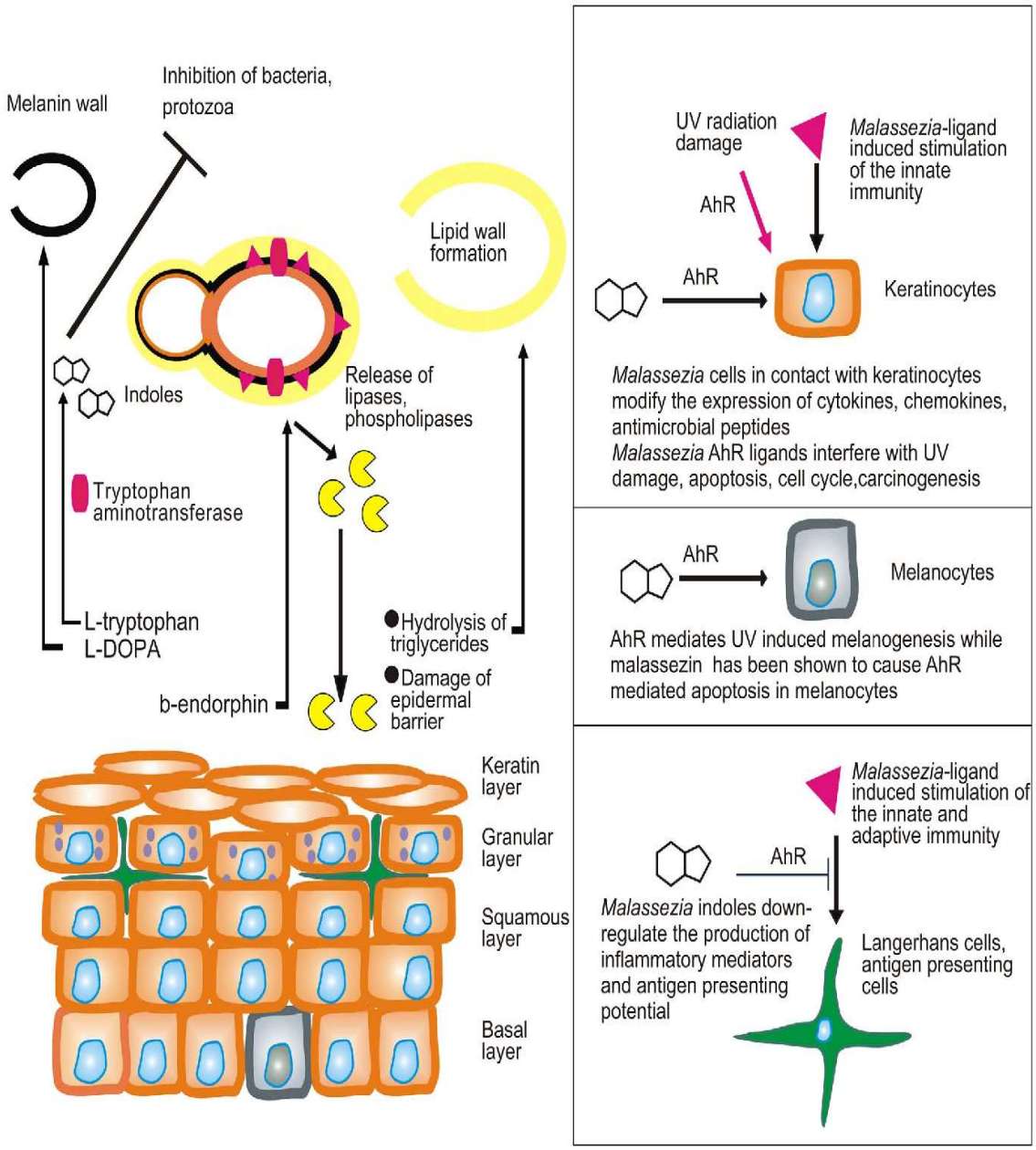

Figure 1: Model showing the putative interactions of Malassezia yeasts with the skin

Risk Factors:

Patients under total parenteral nutrition (TPN) and immunocompromised patients with increased length of stay (LOS) in intensive care units are at risk for Malassezia infections. Risk for Malassezia infections is also high in very-low-birth-weight infants and highest in premature infants. The mechanism of transmission to the infant is vertical or horizontal. After host exposure, the degree of prematurity, the corresponding skin condition, endotracheal intubation, central vascular access, diseases such as necrotizing enterocolitis or focal bowel perforation, and abdominal surgery contribute to colonization. Colonization is

further enhanced by the pathogen's virulence factors, including adherence properties that favour colonization and proliferation followed by biofilm formation in central vascular catheters. Compromised or immature host immunity, delayed diagnosis followed by persistent Malassezia fungemia and subsequent delayed vascular catheter removal, tissue or valve injury, insufficient antifungal dosing, or coinfection may lead to dissemination and occasionally result in poor prognosis.

Sign and Symptoms:

Tinea versicolor is characterized by mildly scaly hypopigmented or hyperpigmented macules/patches, most commonly affecting areas of skin that are rich in sebum production such as the trunk (especially the upper part), neck, shoulders, and upper arms **(Figure 2)**. Facial involvement is less common in adults. On the other hand, facial involvement is common in children and may be the only site involved. The forehead is the usual site of facial involvement. Other sites of involvement, such as forearms and thighs, are less common **(Figure 3)**. Unusual sites of involvement include the scalp, eyelid, axilla, areola, periareolar area, antecubital fossae, popliteal fossa, pubis, groin, perineum, penile shaft and vulva. Smaller macules may have a powdery appearance because of flaking. Over time, the macules enlarge radially and coalesce into patches or very superficial plaques. The lesions are covered with a fine scale, which is often difficult to appreciate upon clinical examination. On the other hand, the scale becomes more apparent when the lesion is stretched or scraped (the 'evoked scale sign'). In patients with tinea versicolor, when the affected skin is wiped with a piece of wet cloth and scraped, it yields a considerable amount of dirty brown keratin. In general, hyperpigmented lesions tend to occur in fair-skinned patients whereas hypopigmented lesions tend to occur in dark skinned individuals. When hyperpigmented lesions occur in dark-skinned individuals, they are often grey-black, dark brown or black whereas these are often tan, light brown, red or pink in fair-skinned individuals. Lesions may become more apparent following exposure to the sun and are thus more noticeable during the summer months. Mixed hyperpigmented and hypopigmented lesions may be found, especially in the axilla and groin. A Wood lamp examination reveals folliculocentric fluorescence in hypopigmented areas. Papular tinea versicolor presents with multiple, asymptomatic, monomorphic, red brown papules (2–3 mm), which may or may not show the fine overlying scale. They are usually found on the trunk. Confetti-like tinea versicolor presents with asymptomatic confetti-like spots with slightly scaly surfaces. The spots are usually bilateral and symmetrically distributed.

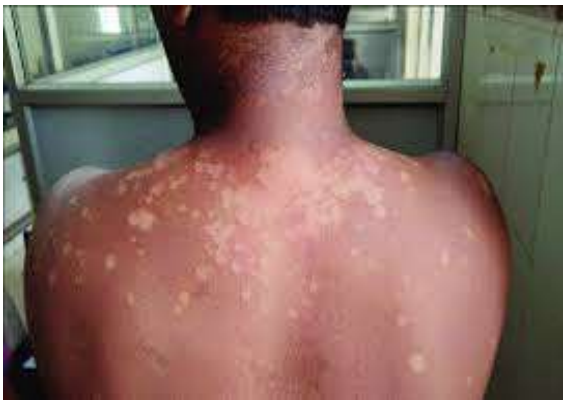

Figure 2: Hyperpigmented confluent roundish erythemo-desquamative PV macules on the back of a young man

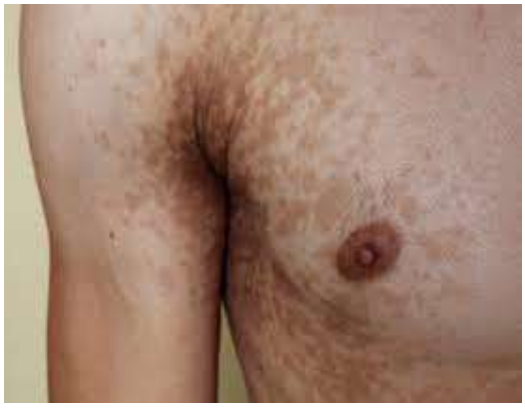

Figure 3: Depigmented PV spots near the armpit

Diagnosis and treatment

There are many other possible diagnosis, particularly for conditions with atypical appearances. Pityriasis alba, nevus anemicus, nevus depigmentosus, idiopathic guttate hypomelanosis, eruptive hypomelanosis, progressive macular hypomelanosis, hypomelanosis of Ito, vitiligo, ash-leaf spot in tuberous sclerosis, corticosteroid-induced hypopigmentation, arsenicosis, leprosy, hypopigmented mycosis fungoides, and post-inflammatory hypopigmentation are among the differential diagnosis of tinea versicolor's hypopigmented lesions **[22]**.

1.Culture

In PV, microbiological cultivation is not advised. Due to the necessity of using synthetic mycological media enhanced with olive oil and requiring 1-4 weeks of incubation at 32 °C, the procedure is challenging **[23]**.

2. Direct Microscopic Examination

When a patient exhibits the typical clinical appearance of PV but does not exhibit the characteristic fluorescence in the Wood's lamp examination (e.g., after using shampoos containing ketoconazole), direct microscopic examination is very useful. Skin scrapings are analysed using phase

contrast and light microscopy after being dissolved in a solution of potassium hydroxide (KOH) and dimethyl sulfoxide (DMSO). Roundish spores and elongated fungal hyphae have a distinctive look that is similar to spaghetti and meatballs. Another method for gathering material appropriate for staining and further microscopic examination is the basic scotch test, which involves the attachment of cellophane foil to the scales **[24]**.

3.Histopathology

A skin biopsy is only useful for ruling out a differential diagnosis in the great majority of PV patients; it is not a routine part of medical practice. Therefore, as options are discussed below, the authors suggest thinking about those that are quicker, less costly, and less invasive. On histology, there are acanthosis, mild hyperkeratosis, and modest epidermal alterations. Spores and hyphae are typically more abundant in hyperpigmented lesions compared to hypopigmented ones. Additionally, hyperpigmented PV shows a single, atypically big melanosome **[25]**.

4.Wood's Light

Wood's light has a wavelength range of 320–450 nm with a peak wavelength of 365 nm, making it an ultraviolet (UV) emitter. A dimly lit area is necessary for the examination. Many compounds, referred to as chromophores, including those found in or on the surface of the skin, can become excitedly fluorescent when exposed to UV radiation. As with porphyrin-dependent stimulated fluorescence in Corynebacterial dermatoses, one of the things that appears to be responsible for this number could be washing out the chromophores with a shower or bath before the consultation **[26]**.

5.Reflectance confocal microscopy

The reflectance confocal microscopy (RCM) characteristics of PV are poorly understood. At the level of the horny layer, clusters of rounded, bright structures and tortuous, hyperreflective structures can be detected. These correspond to the look of spaghetti and meatballs in conventional direct microscopy **[27]**.

6.Dermatoscopy

Clinical characteristics pertaining to the distinctive appearance and distribution of skin lesions are typically used to diagnose PV. On the other hand, situations with unusual shape or distribution could be difficult to diagnose and need to be distinguished from other pigmentation diseases. The gold standard for inflammoscopy is non-contact polarized dermatoscopy. It has been observed that the pigmentation in lesions that are hyperpigmented or hypopigmented is typically irregular. Because these locations are more humid than other areas of the skin, scales usually appear in the skin's furrows and around hair follicle openings. Whereas wrinkled scaling is more common in

dermatoscopy of hyperpigmented lesions, scaling is more commonly found in hypopigmented lesions.

7.Ultraviolet-Induced Fluorescence Dermatoscopy

UVFD, or ultraviolet-induced fluorescence dermatoscopy, is a new dermatoscopic technique that makes use of UV light. Similar to Wood's lamp, UVFD uses excited fluorescence released by chromophores to create colorful visuals. Bright blue keratin and blue background elastin and collagen are the primary sources of luminescence in PV. Hypopigmented lesions can be identified by light greenish structureless areas, whereas hyperpigmented lesions have dark greenish structureless areas, however the lack of published data on UVFD hints to PV.

Pharmacological treatment

1.Antifungals

Generally speaking, oral antifungals are only used to treat severe, persistent, stubborn, or recurring tinea versicolor. There are several benefits to oral antifungal therapy, such as better patient compliance, a shorter course of treatment, more convenience, a shorter recovery period, and a lower recurrence rate. Fatigue, malaise, headache, cutaneous eruption, pruritus, dyspepsia, nausea, vomiting, abdominal pain, diarrhoea, hypertension, congestive cardiac failure, thrombocytopenia, hypokalaemia, albuminuria, hypertriglyceridemia, and abnormal liver function are among the adverse events linked to the use of oral antifungals.

2.Itraconazole

Triazole antifungal Itraconazole inhibits cytochrome P450-dependent ergosterol production, which modifies fungal cell activity in a manner similar to ketoconazole. For PV to be treated successfully, a mycological response of at least 1000 mg itraconazole must be produced during the course of treatment.

3. Fluconazole

Fluconazole is a triazole antifungal that, like itraconazole and ketoconazole, inhibits cytochrome P450-dependent ergosterol production. As a triazole antifungal, oral fluconazole inhibits cytochrome P450-dependent ergosterol synthesis, which makes it an extremely efficient treatment for tinea versicolor. Orally administered fluconazole can stay in the stratum corneum for around two weeks after the dose.

4. Pramiconazole

The formation of ergosterol in fungal cells is inhibited by the relatively new triazole called promiconite. Its efficacy against Candida species, Malassezia species, and dermatophytes has been demonstrated in vitro. Pramiconazole was 10 times more active than ketoconazole against Malassezia species and twice as active as itraconazole against Candida species at doses less than 1 µg/mL. Clinical signs and symptoms,

including erythema, itching, and desquamation, were graded on a five-point scale for a global clinical evaluation and significantly decreased from baseline during the course of the trial The most frequent treatment-emergent adverse events (AEs) were diarrhea and nausea, with hydroxypropyl-β-cyclodextrin, the formulation of the study medication, probably playing a role.

5. Ketoconazole

The first broad-spectrum antifungal used to treat systemic and superficial mycoses was ketoconazole, an imidazole. Ketoconazole limits cell development and function by inhibiting the enzyme lanosterol 14α-demethylase, which impairs ergosterol production. It's been demonstrated that ketoconazole cream works just as well as 1% clotrimazole and 1% terbinafine cream, while ketoconazole shampoo works just as well as 2.5% selenium sulphide and 1% flutrimazole shampoo.

Laser and photodynamic therapies:

A limited number of studies reported the successful treatment of tinea versicolor with 308-nm excimer laser, narrow-band ultraviolet (UV)-B phototherapy, 5-aminolevulinic acid photodynamic therapy and methylene blue photodynamic therapy. Well-designed, large-scale, multicentre, randomized, placebo-controlled trials are needed to confirm or refute these findings.

Alternative therapies:

A wide variety of alternative medicines have been shown to have some therapeutic effects on tinea versicolor. In some cultures, alternative therapies are popular for the treatment of tinea versicolor. These include topical application of beeswax and honey,essential oils of Cymbopogon citratus, quince seed mucilage hydrogel decorated with essential oils of Nigella sativa, Citrus sinensis and Cinnamon verum, polyherbal Unani formulation, Pentas longiflora leaf extract, Acalypha wilkesiana leaf extract, Artemisia sieberi shrub extract, nitric oxide-liberating cream and irradiated human amniotic membrane in combination with tea tree oil.

Prophylactic treatment:

The relapse rate is high because Malassezia species are normal commensals on the skin surface. Good personal hygiene may limit recurrences to a certain extent. Long-term intermittent prophylactic therapy should be considered for patients with frequent recurrence of the disease who desire treatment, especially during the warmer months of the year. Unfortunately, research studies evaluating the efficacy of prophylactic antifungal treatment are scarce. Prophylactic administration of topical ketoconazole 2%, clotrimazole 1% or selenium sulfide 2.5% shampoo applied to the whole body for 10 minutes once a month may lead to decreased relapse rate of tinea versicolor.

CONCLUSION:

PV is a common, chronic, and relapsing dermatosis with various clinical manifestations Although the diagnosis is usually made on the basis of the clinical presentation, the additional diagnostic methods described above might be useful for confirmation and differentiating it from other differential diagnosis, especially in atypical presentations. The early initiation of treatment and prompt identification of recurrence help to avoid complications, especially the social burden of pigmentation disorders, significantly affecting the patients' quality. A wide range of antifungal agents are effective in the treatment of tinea versicolor. In general, topical antifungal agents are the first-line treatment of tinea versicolor as there are fewer adverse events associated with their use. Oral antifungal agents are usually reserved for severe, widespread, recalcitrant, or recurrent disease. This review helps to gain information regarding PV which can help to know how PV spreads, signs and symptoms of PV, Treatment thereby enhancing patient care, improving the outcomes, and contributing to the overall well-being of individuals affected by PV.

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