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FUNGAL INFECTIONS OF SKIN AND SUBCUTANEOUS TISSUE (A BONIFAZ AND M PEREIRA, SECTION EDITORS)

Pityriasis Versicolor in Children and Adolescents: an Update

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Abstract

Purpose of Review Pityriasis versicolor (PV) is a superficial mycosis that it can occur at any age, even in newborns. In this review, we will describe epidemiological data, mycological characteristics of yeast, pathogenesis and clinical characteristics of the disease, different diagnostic resources, and the current recommendations for treatment.

Recent Findings The typical morphology and topography of PV allow us to make a quick diagnosis, but atypical presentations have been described. Diagnostic tools, such as dermoscopy, can also reveal patterns that allow the evaluation of characteristics of scales and pigment in lesions. The discovery of new species and new mechanisms of interaction with the host has broadened the panorama of aetiological possibilities.

Summary Although PV is a common disorder, extensive research is necessary to better understand the pathophysiology of the disease, immunological characteristics of the pathogen-host relationship and resources needed to precisely diagnose the disease, treat the disease, and avoid its chronic and recurrent course.

Keywords Pityriasis versicolor · *Malassezia* spp. · children · adolescents · tinea versicolor · superficial mycoses · hypopigmented lesions

Introduction

Superficial mycoses are frequent entities in daily medical practice, so their recognition is important. Pityriasis versicolor (PV) is one of them and its most common manifestation facilitates diagnosis; however, there are new clinical forms described that extend the differential diagnoses of the disorder, new species of *Malassezia* as etiological agents and mechanisms of interaction with the host, as well as new treatment opportunities. In this review, we will focus on these findings and also describe the concepts regarding epidemiology, diagnosis, treatment and prognosis.

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Concept

PV is a superficial (restricted to the stratum corneum), chronic and relapsing mycosis caused by yeasts of the genus *Malassezia* [1••]. The name PV derives from the fine desquamation, appearing similar to bran and being present in the lesions that characterize this condition, which can be hypopigmented or hyperpigmented. PV has also been called tinea versicolor, mainly in Anglo-Saxon literature, because the aetiological agent is called *Microsporon furfur* [2–4].

Epidemiology

PV is a cosmopolitan entity but is predominately found in tropical regions where it affects up to 40–50% of individuals

and tends to recur in summer, unlike regions such as Sweden where a frequency of up to 0.5% has been reported [5, 6]. Although no recent studies have been performed on the disease, Heidrich et al. reported a 0.3% decrease in the prevalence per year [1••].

Age

PV can occur at any age and some reports indicate its emergence even in the neonatal period in association with factors such as prematurity, hospitalization in intensive care units, high humidity in incubators, direct contact with affected family members and persistence of maternal hormones. PV is more common in adolescents and young adults. The predominant age group varies in different studies, with some patients aged older than 30 years [1••, 7–11]. In infants, frequencies ranging from 3.7 to 31% have been described [12, 13].

Gender

PV is more common in men, which has been attributed to increased sebaceous activity [1••]. However, some studies have reported a higher frequency in women, most likely because women seek medical care more often than men [9, 14, 15].

Phototype

PV usually presents in dark phototypes, but this link is considered to correspond to a more prominent appearance of the lesions; however, some studies do not support the tendency towards hypopigmented lesions in individuals with darker skin or that the predisposition is dependent on age or gender [1••, 16].

Table 1 shows the characteristics of patients with PV in studies published in the last 5 years.

Aetiology (Mycological Characteristics)

The genus *Malassezia* includes yeasts of the division *Basidiomycota,* subphylum *Ustilaginomycotina,* class *Malasseziomycetes* and family *Malasseziaceae,* most of which are dependent on lipids (except *M. pachydermatis)*. These yeasts are part of the microbiota of nearly the entire surface of the human skin, except for the soles of the feet and are mainly found on areas such as the scalp, face and trunk. Under certain conditions, these yeasts are associated with a broad spectrum of clinical entities, although PV is the most common disease caused by such agents and the only disease for which *Malassezia* has been established as a causative agent [1••, 26, 27].

The genus *Malassezia* currently includes 18 species that are present on the skin, 10 of which have been found on human skin, while the remaining 8 species were isolated from homeothermic animals, as shown in Table 2 [4, 28, 29].

Although *M. furfur* was previously considered a unique aetiological agent of PV, we currently know that the frequency of *M. furfur* as a causative agent varies across different species based on reports from multiple studies according to the region studied, the year of the study and the methods used to identify *Malassezia* species; for example:

- In Argentina in 2012, *M. sympodialis* and *M. globosa* were reported to have the highest frequencies at 51% and 40%, respectively [26].
- In Adana, Turkey, in 2009, *M. globosa* was documented in 47.7% of PV cases, *M. furfur* was documented in 36.4% of PV cases and *M. slooffiae* was documented in 15.9% of PV cases [31].
- In 2014, in Iran, *M. globosa* was isolated in 65.1% of PV cases and *M. obtusa* was isolated in 17.4% of PV cases [17].
- In 28 students in northeast Iran, *M. restricta* was the most frequently detected species [20].
- In recurrent disseminated PV, a newly proposed form of the disease, *M. japonica* and *M. furfur* were isolated more frequently [32]. However, in 2013, in patients with extensive PV, species other than those previously reported were not detected [28].

Notably, as shown in Table 2, *M. pachydermatis* and *M. nana*, which were initially isolated in animals, have also been isolated in PV lesions, as indicated in Table 1, where we show the *Malassezia* species isolated from PV lesions in studies conducted in the last 5 years.

To differentiate between *Malassezia* species, a combination of biochemical, morphological, biological and molecular tests is required. Morphologically, *Malassezia* yeasts measure 1.5–10 μm, exhibit asexual reproduction with monopolar budding, have shapes varying from spherical to ovoid, and have a thick wall compared to other yeasts (measuring 0.12 μm), which accounts for up to 26–37% of the cell volume. Cultures should be performed in special media, such as Sabouraud-dextrose supplemented with sterile olive oil, Dixon medium or its modified formula, Leeming and Notman agar and Ushijima medium, except for the species *M. pachydermatis*, which is capable of growing in conventional media. The saprophytic (yeast) phase requires the presence of fatty acids for its progression and yields mycelium (the opportunistic phase) under favorable conditions. To achieve this, temperatures between 30 and 35 °C and humidity must be supplied to the cultures, which should be placed in plastic bags to prevent desiccation. Biochemical and physiological tests and reagents, such as the catalase reaction, β-glucosidase, polysorbate requirements 20, 40, 60, and 80

Table 1 Epidemiological characteristics and species of *Malassezia* in PV

Author	Country	Year	Number of patients	Gender (%)	Age group with higher frequency (years)	Most frequent clinical variant	Most frequent anatomical location	Identified species	Identification method
Rodoplu et al. [17]	Turkey	2014	146	M=82.8 F=17.1	18–34	NR	More than 1 area	*M. globosa* 65.1% *M. obtusa* 17.4% *M. japonica* 7.4% *M. nana* 3.7%	Catalase activity and β-glucosidase, SDA culture, assimilation of cremophor EL, utilization of Tweens 20, 40, 60, and 80, precipitate production in mDixon
Xie et al. [18]	China	2014	24	NR	NR	NR	NR	*M. globosa* 95.8% *M. restricta* 91.7% *M. dermatis* 33.3%	RT-PCR
Ibekwe et al. [19]	Nigeria	2015	304	M=38.8 F=61.1	12–21	Hypopigmented	Face	*M. furfur* 81.7% *M. restricta* 5.1% *M. sympodialis* 2.3%	PCR-RFLP
Heidrich et al. [1]	Brazil	2015	2239	M= 42 F= 58	21–30	NR	Trunk	NR	NR
Sharma et al. [10]	India	2016	262	M=65.6 F=34.4	21–30	Hypopigmented	Neck	*M. furfur* 77.3% *M. globosa* 12.4%	PCR-RFLP
Moallaei et al. [20]	Iran	2017	42	M=59.5 F=40.5	18–22	NR	Neck	*M. restricta* (8.4%) *M. globosa* (6.2%) *M. sympodialis* (4.2%) *M. furfur* 3%	PCR- RFLP
Yahya et al. [15]	Nigeria	2017	265	M=40.2 F=59.8	20–29	NR	NR	NR	NR
Elshabrawy et al. [21]	Egypt	2017	137	M=51 F=49	20–29	Hypopigmented	Back and neck	*M. furfur* 44% *M. globosa* 24.5% *M. sympodialis* 12.2% *M. restricta* 10.2%	PCR-RFLP
Diongue et al. [22]	Senegal	2018	100	H=100	31–60	NR	Trunk	*M. furfur* 1oo% **	MALDI-TOF MS
Honnavar et al. [23]	India	2018	77*	NR	NR	NR	NR	*M. furfur* 44.7% *M. globosa* 25.7% *M. restricta* 21.5%	MALDI-TOF MS
Cam et al. [24]	Vietnam	2019	271	NR	NR	NR	NR	*M. globosa* 42.4% *M. dermatis* 17.3% *M. furfur* 14.4%	mDixon, SDA, Tweens 20, 40, 60, and 80, chromatase .
Awad et al. [25]	Iraq	2019	27	M=74 F=26	15–22	NR	NR	*M. furfur* 32% *M. globosa* 14% *M. pachydermatis* 8%	Gel electrophoresis

*Only included 6 patients with PV

**n = 39, the rest did not grow in culture

M male, *F* female, *NR* not reported, *SDA* Sabouraud dextrose agar, *RT-PCR* reverse transcriptase-polymerase chain reaction, *PCR-RFLP* polymerase chain reaction restriction fragment length polymorphism, *MALDI-TOF MS* matrix-assisted laser desorption ionization time-of-flight mass spectrometry

Table 2 The distribution of *Malassezia* species isolated from humans and animals and the associated clinical entities (taken from reference [60])

Malassezia species in humans		*Malassezia* species in animals	
M. furfur	PV, SD, SI, F	*M. pachydermatis*	Dogs, cats, birds, pachyderms
M. sympodialis	PV, SD, SI, AD, F	*M. nana*	Cats, dogs, cattle
M. globosa	PV, SD, AD, F	*M. equina*	Horses, cattle
M. slooffiae	SD	*M. caprae*	Goats, horses
M. restricta	SD, AD, F	*M. cuniculi*	Rabbits
M. obtusa	SD, AD	*M. brasiliensis*	Parrots
M. dermatis	AD	*M. psittaci*	Parrots
M. japonica	AD,	*M. vespertilionis*	Bats
M. yamatoensis	SD, AD		
M. arunalokei	SD		

PV pityriasis versicolor, *SD* seborrheic dermatitis, *AD* atopic dermatitis, *SI* systemic infection, *F* folliculitis

(Tweens), cremophor EL (as a source of lipids for growth) and pigment and fluorochrome production in the presence of tryptophan, help with identification [34–36]. Table 3 shows the characteristics of the *Malassezia* species that affect the human skin.

Definitive identification of *Malassezia* species requires molecular analysis using various tools, such as direct DNA sequencing, real-time PCR, analysis of single-strand polymorphism conformations based on PCR, random amplification of polymorphic DNA, pulsed-field electrophoresis, Raman spectroscopy and proteomics by laser desorption/ionization mass spectrometry (MALDI-TOF MS), among others [21, 43].

Pathogenesis

Malassezia, which is part of the cutaneous microbiota in pilosebaceous units, requires certain factors that favor its proliferation, such as endogenous or exogenous and environmental, genetic and immunological factors. Accordingly, *Malassezia* is detected at a higher frequency in adolescents and young adults or those with endocrine disorders due to excessive lipid conditions caused by hormonal changes; in individuals using contraceptives; pregnant women; hot and humid climates; hyperhidrosis; those that use fatty products for skin care, such as creams and sunscreens; and also in those with poor hygiene, immunosuppressed, or malnourished individuals. Other predisposing factors included treatment with topical or systemic corticosteroids, solid organ transplant recipients, treatment with anti-TNF-α monoclonal antibodies (adalimumab, etarnecept and infliximab) and hereditary predisposition, which causes greater susceptibility in certain individuals as a positive family history has been reported in 5 to 39% of cases in different series [34, 42–45].

The pathophysiology of *Malassezia* associated with cutaneous disorders is unknown, but some mechanisms of interaction between *Malassezia*, the host and the immune response triggered have been described [3].

Due to its location on the skin, *Malassezia* interacts directly with keratinocytes. Hydrophobicity is a factor that allows *Malassezia* to adhere to tissues and even confers the ability to form biofilms. Another important characteristic is the conformation of its wall; this characteristic contains an external laminar layer connected to pattern recognition receptors, mainly the type C lecithin receptor, present in myeloid cells with which it communicates under inflammatory conditions, as well as dendritic cells [37–39, 46•, 47, 48]. The union of the fungal wall to the type C lecithin receptor results in activation of signaling pathways such as the MAPK, NF-κB and NFAT pathways. Other receptors for *Malassezia* exist, such as dectin-2, Mincle and Toll-2 type receptors, all of which induce the release of cytokines and antimicrobial peptides. In addition, the lipid layer of the fungus induces the release of stromal thymic lymphopoietin. *Malassezia* is also capable of triggering inflammatory cascades through the products of its metabolism by enzymes such as lipases and phospholipases, which capture unsaturated free fatty acids such as oleic acid, an irritant with a desquamative effect on keratinocytes and arachidonic acid that produces proinflammatory eicosanoids that damage the stratum corneum, subsequently altering the barrier function as confirmed by observed increases in transepidermal water loss [49]. *Malassezia* also converts tryptophan into alkaloids such as malassezin, indirubin and indole 3,2-b-carbazol, which can promote apoptosis of melanocytes or inhibit the respiratory burst in neutrophils [48].

In atrophic variety of PV, which was initially considered to be related to prolonged use of corticosteroids, telangiectasia and atrophy in the lesion are currently considered to be potentially related to angiogenesis and flattening of epidermal ridges secondary to a Th1 hypersensitivity response triggered by antigens of the fungus, resulting in the release of TNF-α, IFN-γ and IL-1β and a subsequent decrease in the mitotic activity of

Table 3 Morphological, physiological, and biochemical characteristics of 12 *Malassezia* species [36–40]

Species	Morphology of the colony and texture	Color of the colony	Cellular morphology	Budding	SDA 32 °C	Tween				Cremophor EL	Catalase	Tryptophan	β-glucosidase	Dixon agar		
						20	40	60	80					32 °C	37 °C	40 °C
M. yamatoensis	Folded or semi-folded, with a complete lobed margin	White-yellowish, semi-glossy	EO 2–4.5 x 2–7.5 μm	Narrow base	.	+	+	+	+	?	+	?	?	+	+	−
M. restricta	Smooth, hard, friable	Cream, opaque	EO 4–6 μm	Broad base	−	−	−	−	−	−	−	−	−	+	+	
o−	−															
M. nana	Smooth, convex, soft, viscous	Cream or yellow, bright to opaque	EO 1–3 x 2–5 μm	Narrow base	−	v	+	+	w	−	+	?	−	+	+	+
o−																
M. pachydermatis	Convex	Cream, opaque.	2-2.5x4.5 μm	Broad base	+	+	+	+	+	+	+ (w)	−	+ (−)	+	+	+
M. japonica	Folded, a lobed full edge	Pale yellow, semi-bright to opaque	EO 2–5 x 2–7 μm	Sympodial	−	−	w	+	−	?	+	?	?	+	+	−
M. obtusa	Smooth, flat, viscous	Cream	OC 4–6 μm	Broad base	−	−	−	−	−	−	+	−	+	+	−ow	−
M. arunalokei	Flat, soft, moderately convex	White-cream	EO 4–5.5 μm	Narrow base	?	−ppt	−ppt	−ppt	de	−ppt	−	?	−	+	+	−
M. dermatis	Convex, a continuous or lobed margin	White-yellowish	EO 2–8 x 2–10 μm	Broad base	−	+	+	+	+	wo +	+	?	−	+	+	+
M. globosa	Coarse, rough, raised, folded	Cream, opaque	A 6–8 μm	Narrow base	−	−	−	−	−	−	+	−	−	+	−ow	−
M. slooffiae	Finely folded	Cream, opaque	OC 1.5–3.5 μm	Broad	−	−ow				(−)	+	+	−	−	+	−
−	+	+	+													
M. sympodialis	Flat, smooth, soft	Cream, opaque	A 2.5–5 μm	Sympodial	−	−ow	−	−	−	−	−	−	−	+	+	+
M. furfur	Smooth, soft, friable, with central convex elevation	Cream	EOC 6 μm	Broad base	−	+ (−)	+			(−)	+ (−)	+ (−)	+ (−)	+		
(−)	+	−ow	+	+	+											

SDA Sabouraud dextrose agar, *E* spherical, *O* oval, *C* cylindrical, *w* weakly positive, *( )* rare deviation of the usual pattern, *?* Unknown, *v* variable, *ppt* appearance of precipitate or an opaque zone without yeast growth

keratinocytes. In addition, the recruitment and activation of histiocytes by interferon generate the elastases responsible for the observed dermal elastolysis. A Th2 cell response favoring fungal overgrowth has also been described [50–52, 53•, 54].

The coloration changes observed in PV were previously thought to be pseudoleucoderma as a result of the effect of light filtration by scales and the abundance of fungal structures; however, we now know that such changes are true changes in coloration whose mechanisms have not been well elucidated. The presence of dicarboxylic acids, lipoperoxidation products, azelaic acid and metabolites derived from tryptophan (pityriacitrin and pityrialactone), which decrease the activity of the tyrosinase enzyme, has been described. In addition, ultrastructural evaluation of PV lesions shows degenerative damage to melanocytes, with mitochondrial and cytoplasmic vacuoles, a visible cytoskeleton, and a decrease in the number of melanosomes and their size in keratinocytes in the stratum spinosum. Even less is known about hyperpigmented lesions, but reports suggest that they are due to the thick corneum layer, a greater number of microorganisms and denser inflammatory infiltrate, as well as inflammatory response-induced stimulation of melanocytes, which result in increased pigment production [55–57].

Recently, the expression of the gene *MGL_3741* in *M. globosa* has been shown to be overexpressed. This gene codes for the dihydroxy acid dehydratase enzyme, a key enzyme in the pathway of isoleucine and valine synthesis, which are the amino acids required as carbon and nitrogen sources for yeast. Accordingly, this pathway may serve as a target in the development of new therapies [58].

Clinical Characteristics

The distribution of PV lesions is related to the nutritional requirements of the yeast because its lipophilic nature predominates in the skin with increased sebum production; thus, the yeast more frequently affects the anterior and posterior thorax, neck and proximal regions of the extremities of adolescents and adults. PV is less common on the face (particularly the eyelids), armpits, groin, buttock, penis and perineum, the latter two of which may be less frequently affected due to extreme humidity and heat [59, 60]. PV is less frequent in children, but the most common location is the face, possibly due to contact of the forehead, interciliary region and nasolabial folds during breastfeeding (Fig. 1a, b). PV can occur in the legs, mainly affecting women due to excessive application of lotion and disseminated cases have been reported to be associated with the use of steroids; no involvement of the palms and soles has been documented [30, 42].

Regarding the morphology, the typical lesions are oval or round macules, which can be dotted, lenticular, nummular, reticular or follicular, and sometimes form polycyclic plates with a fine scale (furfuraceous) on the surface, which becomes more evident when scraping the skin with a curette or with the nail (Besnier's sign) and after skin stretching due to its detachment (Zireli's sign) [61, 62••]. Cutaneous lesions are usually asymptomatic or associated with mild itch.

The name PV is derived from the clinical characteristics of the cutaneous lesions because such lesions may have different pigment tones. The clinical classification is established according to the pigmentation of the lesions or their characteristics:

1. Hypochromic variety. It is characterized by hypochromic lesions covered with fine scales from 2 to 2 cm length, with irregular shapes, tending to converge forming patches with cartographic aspects. This is the most frequent variety and predominates in brown skin (Fig. 1a, b).
2. Hyperchromic variety. It has hyperchromic lesions with a light brown color and scales on the surface. They may be preceded by the erythematous variant and are more often described in light phototypes; however, some authors report that these lesions may be light brown in people with light skin and dark brown or dark grey in people with brown skin [63].
3. Hypo/hyperchromic variety. Most patients have lesions of a single hue; however, hyper- and hypopigmented lesions can coexist, especially in brown skin or frequently tanned patients.
4. Erythematous variety. Erythematous lesions with a pink color and a fine scale on the surface that are more evident in people with fair skin after sun exposure and may later evolve into the hyperchromic variety.
5. Vitiligo-like variety, parasitic achromia or PV alba. Some authors consider this type as a very accentuated expression of the hypochromic variety [40]; however, evidence indicates that it is a distinct entity: the lesions are practically achromic, depigmented, and resemble vitiligo [40]. Two stages have been described, early stage with scales, yeast and fluorescence under Wood light and late stage; in this phase, the pathogen is not detectable, and no desquamation is evident and the fluorescence under a black light is negative. Moreover, especially in the late stage, this variety does not respond adequately to antimycotic therapy, further supporting its distinction as a unique entity [56].
6. Dermatophytoid (tinea-like) or circinate variety. The lesions are delimited by a well-established "pseudo" active edge, as tinea corporis.
7. Atrophic or pseudo-atrophic variety. This variety is characterized by erythaematous or hypopigmented lesions with mild desquamation and depression. This variety was

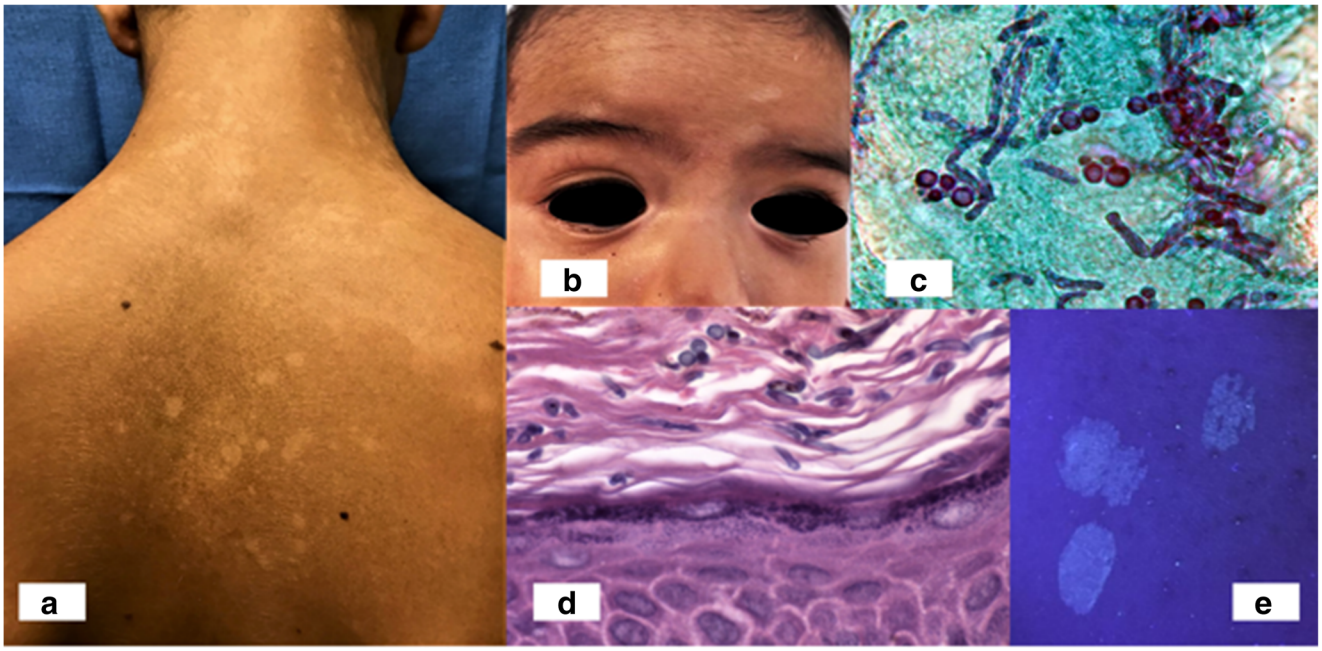

Fig. 1 **a** Hypopigmented PV in a teenager. **b** Hypopigmented PV in a children. **c** Direct examination that shows short hyphae and blastoconidia, image in "meatballs and spaghetti." **d** Histopathology (PAS staining) that exhibits hyphae and blastoconidia in stratum corneum. **e** Hypopigmented lesions with wood light greenish-yellow fluorescence

originally described by De Gracianski and Mery as a complication after the application of corticosteroids considering that *Malassezia* breaks the skin barrier because it is confined to the stratum corneum and thus increases the absorption of the steroid, which leads to atrophy as a side effect [64]. However, cases with atrophic lesions have been described without this association and atrophy has been proposed to be secondary to the antigens of *Malassezia* in the epidermis causing a type IV hypersensitivity reaction, a Th1 response with the release of cytokines causing apoptosis, inadequate proliferation of keratinocytes and elastolysis, which explain the atrophy. Whether this type is a variety of pityriasis versicolor or only a consequence of inadequate management with topical steroids remains controversial [51, 53•, 62••]. This variety has not been described in pediatric patients and is more frequent at the end of adolescence and in adulthood [65].

8. *Imbricata* variety. The term "imbricata" or "imbrex" derives from Latin and translates to superposition of tiles or layers. This variety is characterized by hypopigmented lesions with superimposed scales on each other, reminiscent of tinea imbricata or *Tokelau.* Few reports of this variety are available. There are few reports of this variety, it has been described in adolescents and it is still necessary to conduct studies that allow to determine if, like tinea imbricata, it can be associated with conditions with immunodeficiency such as hyperIgE syndromes [66].

9. Intertriginous variety. This is a rare variety that affects the antecubital, axillary and inguinal folds. It is characterized by erythematous or hyperpigmented asymptomatic lesions and is sometimes difficult to distinguish from erythrasma and intertrigo by *Candida,* with which it can coexist [62••].

10. Papule-like variety: This is a rare variety that we have found in our clinical experience, which appears as slightly elevated lesions [30].

Differential Diagnosis

The entities that should be considered as a differential diagnosis are different according to the clinical variety. For the hypochromic variant: pityriasis alba, hypocromic solar dermatitis, hypochromic syphilids, residual leukodermia, eczematides and indeterminate leprosy should be considered. In the hyperchromic variant, melanocytic nevi should be considered; for the acromic variant: vitiligo; erythaematous variant: Gibert pityriasis rosea and syphilitic roseola should be considered; for the dermatophytoid variety, tinea corporis should be considered; for the imbricata variety, tinea imbricata or *Tokelau* should be considered; the intertriginous variety: erythrasma and intertrigo by *Candida* should be considered; and the atrophic variant can be confused with mycosis fungoides, anetoderma and atrophoderma [61, 67].

Diagnosis

The diagnosis is basically clinical and can be facilitated with auxiliary methods.

Wood light

This technique has great utility for detecting lesions that are not extensive or defined; it is also useful for monitoring after treatment. The characteristic fluorescence is greenish-yellow. This test can be negative in up to 3.6% of cases, and some gels or hair lacquers can yield false positives (Fig. 1e) [30, 61].

Dermoscopy

Recently, the dermatoscopic findings of some of the PV variants have been described using this non-invasive diagnostic tool, which may be useful. In the hyperchromic variety, poorly limited diffuse brown pigment has been described and in the hypochromic variety, non-uniform whitish areas with perilesional hyperpigmentation have been described; in both types, the presence of scale and desquamation in patches or in grooves have been described as the most consistent findings [61, 68•].

Mycological Study

A sample can be collected by scraping lesions to obtain scales with two slides or with transparent adhesive tape, which is stuck on a scaly lesion and then placed on a slide. A direct examination should be performed with 20% potassium hydroxide (KOH), which is stained with Parker blue, methylene blue or Albert staining; Albert staining is superior to the other two stains because it facilitates observation of structures that are stained purple. Under light microscopy, 4 to 8-μm blastoconidia clusters and 2 to 4-μm short fragmented filaments or hyphae in an italic «s» form will be observed; the association of the above findings yields the typical image that has been described as "meatballs and spaghetti" (Fig. 1c). This pattern is enough to reach a diagnosis.

Culture is not necessary for routine diagnosis but is indispensable to identify the species for research and epidemiological purposes.

Histopathological Study

Biopsies of lesions are performed only in cases of diagnostic uncertainty or to rule out other causes. A mild inflammatory process and fungal structures (hyphae and blastoconidia) in the stratum corneum with PAS staining (periodic acid Schiff stain) are observed (Fig. 1d) [61].

Treatment

The treatment of PV should aim to reduce the number of relapses as much as possible through effective and safe therapy that includes patient education and topical and/or systemic treatments because no modality can minimize the chronicity of the disease.

Education of the Patient

Education is important under all conditions, including education regarding the presence of *Malassezia* as part of the cutaneous microbiota, the mechanism and its potential triggers, and the course of the disease. The persistence of the cutaneous pigmentary changes should be explained to the patient and his/her family. Patients must understand the chronic evolution and high frequency of relapse despite treatment, the importance of appropriate treatment, and the treatment objectives.

Possible predisposing factors should be eliminated, such as excessive sun, the use of oils, creams, bronzers, or other oily products for the care of the skin, and we must indicate treatment for hyperhidrosis with dryings, talc, topical aluminium chloride, or botulinum toxin and also detection and treatment of hormonal disorders and improvement in hygienic measures.

Topical Treatment

Topical antifungals are the first line of treatment, and existing systematic reviews have concluded that most antifungals are better than placebo if used properly [68•, 69]. They can be not specific (they act by physically or chemically removing the infected tissue and preventing stratum corneum invasion) or specific (they have fungicidal and/or fungistatic effects). Table 4 shows the available topical drugs, which are usually prescribed 1–2 times per day for 1–4 weeks [71•].

Topical drugs are found in various forms, such as lotions, mousses, shampoos, creams, ointments, gels and solutions. This aspect should be considered when selecting a medication because the choice depends on the site to be treated and the absorption in the affected region. Moreover, creams and ointments are oily, and their use can perpetuate the proliferation of *Malassezia*. We should also consider that commercial availability varies according to geographic region.

The most common adverse effects with the use of antifungals are cutaneous: burning, mild itching and redness [70].

Systemic Treatment

Systemic treatments are recommended for severe, extensive or recalcitrant cases [69]. Table 4 shows the systemic drugs used in the treatment of PV, as well as the recommended doses.

Ketoconazole has long been considered the gold standard, is a cheap drug, and has been proven to be effective in the

Table 4 Topical and systemic treatment options for PV

Topical	Non-specific	Selenium sulfide 2.5% [72]	
		Zinc pyririonate 1% [71•]	
		Propylene glycol 50% [71•]	
		Benzoyl peroxide 10% [73]	
		Whitfield ointment (3% salicylic acid + 6% benzoic acid) [74]	
		Adapalene 0.1% [75]	
		Tacrolimus 0.03% [76]	
	Specific	Bifonazole 1% [77]	
		Clotrimazole 1% [76]	
		Ciclopirox Olamine 1% [71•]	
		Econazole 1% [78]	
		Fenticonazole 2% [30]	
		Fluconazole 2% [71•]	
		Ketoconazole 1% or 2% [70]	
		Luliconazole 1% [79]	
		Miconazole 2% [71•]	
		Oxiconazole 1% [71•]	
		Sertaconazole 1% or 2% [71•]	
		Tioconazole 1% [80]	
		Terbinafine 1% [70]	
Systemic	Drug	Adult dose	Pediatric dose
	Ketoconazole [69]	200 mg/day for 10 days 200 mg every 12 h x 3 doses 400 mg single dose	3–5 mg/kg/day
	Itraconazole [69]	200 mg/day for 5–7 days 400 mg single dose	
	Fluconazole [81]	300 mg/week x 2–4 weeks 400 mg single dose	
	Pramiconazole [82]	200 mg/day for 2–3 days 400 mg single dose	Not described

prophylaxis against PV. However, due to the risk of hepatotoxicity, adrenal insufficiency and other frequent adverse effects and drug interactions, other therapies such as itraconazole, fluconazole, or pramiconazole are preferred. Although terbinafine is an effective option in other superficial mycoses, such as dermatophytosis, it is not useful in PV because the metabolite excreted is not active and does not reach the stratum corneum where the yeast resides [69].

The most common adverse effects of systemic drugs are gastrointestinal (nausea, vomiting, and diarrhea), followed by headache and fatigue [70].

Prognosis

The evolution of the disease is chronic and recurrent and varies from a few weeks to 30 years; it is more prolonged in warm and humid regions. Three groups of patients have been described according to the evolution of the disease: in the first group, the disease resolves clinically and mycologically with conventional treatment without relapses; the second group includes patients who present up to 4 relapses in a year and respond to conventional treatment and relapse is clearly related to predisposing factors; finally, patients who present more than 4 relapses in a year, have a poor response to clinical and mycological treatment and have no clear risk factors are included in group 3. Further studies are necessary to determine the relationship between *Malassezia* spp. and host immunity in the latter group of patients [83].

Relapse frequencies range from < 60% to 90% in the first 2 years, and maintenance therapy is therefore suggested in addition to improvement or elimination of the identified predisposing factors [4, 71•].

Different prophylactic regimens with topical and systemic drugs have been described:

- 2.5% selenium disulfide shampoo or 2% zinc pyririonate used 2–3 days a month [30].
- Shampoo with 2% selenium or 2% ketoconazole applied to the full body for 10 min once a month [63].
- Itraconazole 200 mg applied twice a day, 1 day a month, for 6 months [70, 84].

Unfortunately, hyperpigmentation persists for several months after mycological cure.

Conclusions

Although pityriasis versicolor is a common cutaneous infectious disease and it is hardly wrong in its diagnosis, its chronic and relapsing course makes necessary more research that allows understanding the physiopathogenic mechanisms that are targets of treatment options to avoid relapses or recurrences, such as the use of other tools for non-invasive diagnosis such as dermatoscopy that facilitate differentiating them from other dermatological diseases.

Compliance with Ethical Standards

Conflict of Interest Karen Adilene Camargo-Sánchez, Mirna Toledo-Bahena, Carlos Mena-Cedillos, Erika Ramirez-Cortes, Sonia Toussaint-Caire, Adriana Valencia-Herrera, Marcela Salazar-García, and Alexandro Bonifaz declare no conflicts of interest relevant to this manuscript.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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- • Of importance
- •• Of major importance

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