

Influence of systemic corticotherapy on the triggering of pityriasis versicolor

Clarissa Matarangas Moreira da Fraga,¹ Rita de Cássia Birschiner,² Alice Pignaton Naseri³ and Lucia Martins Diniz¹

¹Center for Health Sciences, Universidade Federal do Espírito Santo, Vitória, Espírito Santo, Brazil, ²Maruípe Health Unit, Vitória, Espírito Santo, Brazil and ³Medical Clinic Department (Nephrology), Cassiano Antonio Morais Hospital, Vitória, Espírito Santo, Brazil

Summary

Pityriasis versicolor is a frequent mycosis and the use of systemic corticotherapy is one of its predisposing factors. This is an observational, cross-sectional, analytical and comparative study, conducted from January 2012 to January 2013 in the following outpatient clinics: Dermatology Service, Cassiano Antonio Moraes Hospital (HUCAM), Vitória, ES, Brazil; Nephrology Service, HUCAM; and Leprosy Department, Maruípe Health Unit, Vitória, ES, Brazil. Patients, undergoing long-term systemic corticotherapy (or not), were assessed with respect to the presence of pityriasis versicolor. If there was mycosis, a direct mycological examination would be carried out. The spss 17.0 software was used for the statistical analysis. From the total of 100 patients, nine had pityriasis versicolor, being eight from the corticotherapy group and one from the group with no use of corticosteroids. Regarding the patients with mycosis, the prevalent age ranged from 20 to 39 years, with six patients; six were women; seven mixed race; eight were undergoing long-term systemic corticotherapy; seven were taking low-dose systemic corticosteroids; four had leucocytosis; five had normal total cholesterol and triglycerides; and four had normal glycaemia. There was increased frequency of pityriasis versicolor in the group undergoing systemic corticotherapy with statistical significance, corroborating the only study on the topic (1962).

nd Prophylaxis of Fungal Diseases

Key words: Pityriasis versicolor, tinea versicolor, Malassezia, corticosteroids, oral administration, risk factors.

Pityriasis versicolor

Pityriasis versicolor is a frequent superficial mycosis, determined by *Malassezia* yeast, found in normal skin biota of 90% of adults. From the world occurrence, it is most common in tropical regions, due to the hot and humid climate, without a distinction of sex or race.^{1,2}

Correspondence: C. M. M. da Fraga, Mestrado Profissionalizante em Medicina, Centro de Ciências da Saúde, Universidade Federal do Espírito Santo, Av. Marechal Campos, Santa Cecília, Vitória, Espírito Santo 29042-715, Brazil. Tel.: 55 27 3335 7324. E-mail: clarissa.matarangas@gmail.com

Submitted for publication 28 November 2013 Revised 13 March 2014 Accepted for publication 23 March 2014 The presence of lipids in the skin favours *Malassezia* during postpuberty due to the apex of sebaceous glands development stimulated by sex hormones from this time of life.^{3–8} In areas rich of sebaceous glands, *Malassezia* occurs in the yeast phase (saprophytic form) and, under stimulation of trigger factors, it becomes a mycelium (opportunistic phase), causing mycosis.^{2.9}

The triggering of mycosis is due to a set of factors: use of skin moisturisers; high humidity and temperature of the climate; hyperhidrosis; use of systemic corticosteroids; and genetic predisposition, among others.^{9–11} Systemic corticosteroids would increase the frequency of mycosis by changing the lipid composition of the cutaneous surface or by immunocompromising the individual.^{12–14}

Clinically, the lesions are limited spots, initially rounded, confluent, of varied colours, with furfuraceous scales, observed by stretching the skin distally (Zileri's sign), and they predominate in the upper portion of the trunk and proximal areas of the upper limbs. Most cases are asymptomatic.^{1,2} The diagnosis is clinical and obtained through direct mycological examination.² For the localised forms, the treatment is topical with antifungal imidazole derivatives. In diffuse forms, 2.5% selenium sulphide in shampoo, 25% sodium hyposulphite, or imidazole derivatives orally administered can be used.¹⁵

Glucocorticoids

Glucocorticoids are effective in the management of inflammatory and autoimmune diseases, including dermatologic diseases, due to their anti-inflammatory and immunosuppressive effects. However, their use can trigger complications.^{16–18} There are several corticosteroids actions: regulation of carbohydrates, lipids and proteins metabolism; preservation of the normal function of the cardiovascular, immunological and endocrine systems, etc.^{16,19}

Glucocorticoids promote hyperglycaemia because they increase hepatic gluconeogenesis and peripheral resistance to insulin. They also alter the lipid metabolism, increasing the lipoproteins of low and high densities and, more commonly, the triglycerides.^{16,18} Treatments undergone for more than 30 days are considered long term, forcing gradual discontinuation of the drug.²⁰

The use of oral corticosteroids in high doses and for long periods can lead to complications.¹⁸ The risk of infections, including fungal, is caused by the inhibition of the inflammatory response and the immunosuppressive effects of these drugs.^{18,20,21} In 1962, Boardman *et al.* [22] observed the occurrence of pityriasis versicolor in patients taking the medication.

Therefore, this study was conducted to assess whether long-term use of systemic corticosteroids predisposes to increased frequency of pityriasis versicolor and to search how this influence occurs, whether by mechanism of immunosuppression or by change in glucose and lipid metabolism, through white blood cell (WBC) count, lipidogram and glucose levels analyses among patients using systemic corticotherapy or not.

Materials and method

This is an observational, cross-sectional, analytical and comparative study, conducted from January 2012 to January 2013 in the following outpatient clinics: Dermatology Service, Cassiano Antonio Moraes Hospital (HUCAM), Vitória, ES, Brazil; Nephrology Service, HUCAM; and Leprosy Department of Maruípe Health Unit, Vitória, ES, Brazil.

Patients with certain underlying disease undergoing long-term corticotherapy or not were selected and they composed two groups: patients using systemic corticosteroids (oral prednisone) for more than 30 days and patients who were not using this therapy. In case they agreed, a dermatological exam of the integument would be performed to verify the existence of pityriasis versicolor and subsequently a standard record would be filled out with identification data and latest results of laboratory exams (WBC count, lipidogram and blood glucose).

In patients with mycosis, scales were collected from the spots with a sterile scalpel blade to confirm the diagnosis through the direct mycological examination. This procedure consisted of applying a drop of 20% potassium hydroxide to the material collected on a glass slide to observe it under an optical microscope. The examination result was positive, observing thick and sinuous pseudofilaments and rounded blastospores, arranged as a bunch of grapes.

In the occurrence of mycosis, the following data of lesions were recorded: colour; location; symptoms; previous history of the disease; family history; and use of skin moisturisers. Based on the research conducted by Framil *et al.* [23], the location of lesions was categorised as follows: mild (affecting only one region of the body), moderate (affecting more than one to three regions of the body) and disseminated (more than three regions of the body affected).

The variable 'corticosteroid dose' was categorised on the basis of the study conducted by Zonana-Nacach *et al.* [24], who assessed the effects of corticotherapy in patients with systemic lupus erythematosus, whose oral doses administered were divided into two categories: high doses (above or equal to 60 mg day⁻¹); and low doses (less than 60 mg day⁻¹).

All the patients with mycosis under study had been treated appropriately. Exclusion criteria were as follows: (a) use of glucocorticoids for less than 30 days; (b) use of other immunosuppressive drug; (c) patients with HIV infection; and (d) patients under 18 years of age.

The spss version 17.0 software (SPSS Inc., Chicago, IL, USA) was used for the statistical analysis, performing the *chi-square test* to calculate odds ratio (OR) and 95% confidence interval (95% CI), taking the significance level of $P \leq 0.05$ as a basis for qualitative variables. The absolute and relative frequencies of the following variables were determined: demographic

(age, sex, occupation and race); clinical (colour of the lesions, location, presence/absence of symptoms, prior personal history of mycosis, family history and use of skin moisturisers), with respect to patients with pityriasis versicolor, using systemic corticotherapy or not and their doses and laboratory exams (WBC count, lipidogram and blood glucose).

The study was approved by the Ethics Committee of the Health Sciences Center, Federal University of Espírito Santo, keeping the privacy of patients who consented in writing to participate in the research.

Results

In the study period, 100 patients with nephrological, rheumatological and dermatological diseases of various aetiologies were cared for. From this group, 50 patients were undergoing long-term systemic cortico-therapy and 50 were not. The median age was 40 years, the average was 40 years and 9 months (standard deviation: 13 years and 7 months), the minimum age was 18 years and the maximum 74 years.

Pityriasis versicolor occurred in nine cases out of a total of 100 patients, confirmed by direct mycological examination. Eight patients belonged to the cortico-therapy group and one to the group with no treatment, representing a statistically significant difference (OR = 9.33; 95% CI: 1.12-77.7; P = 0.031).

With respect to colour, the analysis of the characteristics of pityriasis versicolor lesions showed predominance of hypochromic spots in eight cases (88.9%) and one (11.1%) with light-brown lesions. Regarding the topography, there were two cases (22.2%) characterised as mild, four moderate cases (44.4%) and three disseminated (33.3%). Four patients (44.4%) had symptoms (itching) and two (22.2%) confirmed prior personal history of mycosis. Four patients (44.4%) have had positive family history for the disease and three (33.3%) commonly applied moisturisers.

The group of patients with pityriasis versicolor was compared with the group without the mycosis with respect to the demographic variables, as shown in Table 1. Table 2 shows the absolute and relative frequencies of patients with and without pityriasis versicolor according to the use or non-use of systemic corticotherapy and their doses. Laboratorial variables are described in Table 3, comparing the groups of patients with and without mycosis with those who had the values of WBC count, lipidogram and fasting blood glucose entered in the medical records.

With respect to WBC count and corticotherapy, the group taking oral corticosteroid had the following

Table 1 Absolute and relative frequencies of demographic char-
acteristics between the groups with and without pityriasis
versicolor.

	Pityria	Pityriasis versicolor					
	Yes	Yes					
	N	%	N	%	Р		
Age (years)							
18–19	1	11.1	3	3.3	0.167		
20–29	3	33.3	14	15.4			
30–39	3	33.3	25	27.5			
40–49	1	11.1	21	23.1			
50–59	0	0	22	24.9			
60–69	1	11.1	3	3.3			
70–79	0	0	3	3.3			
Sex							
Male	3	33.3	37	40.7	0.480		
Female	6	66.7	54	59.3			
Race							
White	1	11.1	38	41.8	0.069		
Mixed	7	77.8	50	54.9			
Black	1	11.1	3	3.3			
Total	9	100	91	100			

Table 2 Absolute and relative frequencies with respect to the use and non-use of corticotherapy and its doses between the groups with and without pityriasis versicolor.

	Pityr				
	Yes		No		
	N	%	N	%	Ρ
Corticotherapy					
Yes	8	88.9	42	46.9	0.031
No	1	11.1	49	53.8	
Dose					
Low	7	77.8	35	38.5	0.622
High	1	11.1	7	7.7	
Does not apply	1	11.1	49	53.8	
Total	9	100	91	100	

results: 27 (60%) patients had values between 4000 and 10 000 leucocytes (normal); two (4.4%) had values less than 4000 leucocytes (leucopenia); and 16 (35.6%) had values greater than 10 000 leucocytes (leucocytosis). In the group of patients who were not undergoing corticotherapy, the results were: 34 (77.3%) patients had values between 4000 and 10 000 leucocytes; one (2.3%) had smaller value than 4000 leucocytes; and nine (20.5%) had values greater than 10 000 leucocytes (Fig. 1).

The lipidograms were analysed with respect to the use/non-use of oral corticosteroid and, considering normal values up to 200 mg dl^{-1} and increased values above this level. The analyses revealed the

	Pityriasis versicolor				
	Yes		No		
	N	%	N	%	Р
WBC count					
Leucopenia	0	0	3	3.7	0.186
Normal	3	42.9	58	70.7	
Leucocytosis	4	57.1	21	25.6	
Total	7	100	82	100	
Total cholesterol					
Normal	5	71.4	43	57.3	0.694
Hypercholesterolaemia	2	28.6	32	42.7	
Total	7	100	75	100	
Triglyceridaemia					
Normal	5	71.4	36	51.4	0.438
Hypertriglyceridaemia	2	28.6	34	48.6	
Total	7	100	70	100	
Glucose					
Normal	4	80	59	77.6	1.000
Glucose intolerance	1	20	17	22.4	
Total	5	100	76	100	

Table 3 Absolute and relative frequencies of laboratorial characteristics between the groups with and without pityriasis versicolor.



Figure 1 Number of patients undergoing corticotherapy or not with respect to WBC count.

following results: the group undergoing corticotherapy had 22 (52.4%) patients with total cholesterol less than 200 mg dl⁻¹ and 20 (47.6%) with total cholesterol greater than this value. The group that was not using corticosteroids had: 26 (65%) patients with normal values of total cholesterol and 14 (35%) with increased total cholesterol, without statistical



Figure 2 Distribution of cholesterolemia between the groups of patients undergoing corticotherapy or not.

significance (OR = 0.592; 95% CI: 0.244–1.440; P = 0.270). The distribution is shown in Fig. 2.

Still, with regard to the lipidograms, the results obtained for triglycerides – considering normal values up to 150 mg dl⁻¹ and increased values above this level – were: 16 (43.2%) patients with normal values of triglycerides; and 21 (56.8%) with increased values in the group taking systemic corticosteroid; and 25 (62.5%) patients within the normal range of triglycerides; and 15 (37.5%) with increased levels in the group that was not using corticosteroids, without statistical significance (OR = 0.457; 95% CI: 0.184–1.139; P = 0.112). The distribution of the variable is shown in Fig. 3.

Regarding the glucose metabolism, values up to 100 mg dl^{-1} were considered normal fasting blood glucose and values above this level were considered increased (glucose intolerance). Thus, it was observed that from the individuals taking systemic corticosteroid, 32 (82.1%) had normal fasting blood glucose and seven (17.9%) had it increased. In the group with no treatment, 31 (73.8%) patients were within the range considered normal and 11 (26.2%) within the altered range, without statistical significance (OR = 1.622; 95% CI: 0.557–4723; P = 0.431).

Discussion

Even though the relationship between systemic corticosteroids and fungal infections have been studied in the past, only a systematic and controlled research²²



Figure 3 Distribution of cholesterolemia between the groups of patients undergoing corticotherapy or not.

was intended to assess the effects of corticotherapy on pityriasis versicolor.

In this study, 16% cases of pityriasis versicolor were found in patients undergoing long-term systemic corticotherapy, compared to the group that was not using corticosteroid (2%), with statistical significance (P = 0.031), corroborating the data obtained by Boardman et al. (1962), who found 10 (9.4%) individuals with pityriasis versicolor among 106 patients undergoing corticotherapy and no case in the group of 82 patients that were not undergoing the therapy. Experimentally, another study conducted by Burke et al. (1961) assessed the relationship between tinea versicolor and corticosteroids. Scales of mycoses or cultures of Pityrosporum orbiculare were inoculated in 21 individuals, six of which had endogenous or exogenous hypercortisolism. These six patients developed, at least, microscopic evidence of the presence of Malassezia furfur, whereas only one patient from the other 15 without hypercortisolism developed mycosis.^{22,25}

With respect to the colour of the fungal lesions, there was a predominance of hypochromic spots, corroborating the results obtained by Chetty *et al.* [12], Morais *et al.* [26], Ghosh *et al.* [27] and Santana *et al.* [28].

Most patients had more than one topography affected by mycosis, with a prevalence of moderate forms, followed by the disseminated form. This result is similar to that found by Framil *et al.* [23], whereas Morais *et al.* [26] found predominance of the

disseminated form. The three studies reinforce the data in the literature, which state that confluence of lesions until they reach large areas of skin is common.^{3,12,29}

The results for the variable 'symptoms' were balanced, resembling the results obtained by Morais *et al.* [26], Ghosh *et al.* [27] and Rao *et al.* [30]. However, the results of this study are in contrast with those in the literature, which describes the disease as asymptomatic or oligosymptomatic, in most cases.^{1,29}

The previous personal history of pityriasis versicolor was negative in 77.8% of the cases; however, Morais *et al.* [26], Ghosh *et al.* [27] and Santana *et al.* [28] found balanced results, with 52.6%, 48.1% and 55.2% respectively. The result of this study differed from that of the literature, which mentions the recurrences of mycoses as common, contributing to its recidivating characteristic and occurring in 60% patients in the first year after treatment and 80% in the second year.^{31,32}

The family history was positive in almost half the cases. This result is comparable to that obtained by Rao *et al.* [30] (38.3%), Hafez *et al.* [33] (39%) and Terragni *et al.* [34] (43.8%) and in disagreement with Chetty *et al.* [12] (21%), Burke *et al.* [25] (17%), Ghosh *et al.* [27] (25.5%), Faergmann *et al.* [35] (18.8%) and He *et al.* [36] (21.2%).

Hafez *et al.* [33] and He *et al.* [36] studied the influence of genetics on mycoses and they found a likely multifactorial inheritance pattern for pityriasis versicolor, following a polygenetic additive model. The factors precipitated the disease in genetically predisposed individuals.

Regarding the use of skin moisturisers, the results (relative frequencies) were similar to those found by Morais *et al.* [26]. Roed-Petersen *et al.* [37] argued that the use of lotions in the integument would favour *Malassezia* and pityriasis versicolor, by making the skin more oily and due to the fact that long-chain fatty acids favour fungal proliferation by serving as substrates.

Mycosis predominated within the range from 20 to 39 years of age, corroborating with data found by Burke *et al.* [24], Rao *et al.* [30], He *et al.* [35] and Framil *et al.* [38]. In other studies, such as those conducted by Chetty *et al.* [12], Morais *et al.* [26] Ghosh *et al.* [27] and Arenas *et al.* [39] the first decade was the predominant group. The results reinforce the literature that describe tinea versicolor as more frequent in adolescents and young adults.²

Females were predominant in the group with pityriasis versicolor, as observed by Santana *et al.* [28], Faergmann *et al.* [34], Furtado *et al.* [40] Belém *et al.* [41] and Miranda *et al.* [42]. This result disagrees with those found by Chetty *et al.* [12], Burke *et al.* [25], Morais *et al.* [26], Ghosh *et al.* [27], Rao *et al.* [30], He *et al.* [36] Framil *et al.* [38] and Kyriakos *et al.* [43], in which males were prevalent.

Kyriakos *et al.* [43] attributed the predominance of male cases to excessive sweating due to increased exposure of men to physical efforts, whereas He *et al.* [35] suggested that there was greater activity of the sebaceous glands in men due to increased production of sex hormones. There was a predominance of females in other studies and He *et al.* [35] attributed the fact to women's aesthetics concern.

Mycosis prevailed in mixed race patients, corroborating data found by Morais *et al.* [26] and differing from Burke *et al.* [25] and Belém *et al.* [41], who found predominance of white patients. These divergent results may be explained by the epidemiological differences between populations of each sample. Chetty *et al.* [12] observed high incidence of tinea versicolor in Madras (India), a region with tropical climate, and proposed that this fact was a result of the largest number of sebaceous glands of black inhabitants of tropical areas.

The region of the study has hot and humid climate and racial variation, with significant share of browns and blacks. The tropical climate favours for hyperhidrosis, which in turn contributes to the outbreak of *tinea* versicolor. This relationship, however, is difficult to measure and still, some authors reported no racial differences in sweating.^{44,45}

To assess how the influence of corticotherapy on the triggering of pityriasis versicolor occurs, the variables WBC count, lipidogram and blood glucose were analysed in relation to the use or non-use of systemic corticosteroid and the absolute and relative frequencies of these variables among patients with and without mycosis were described.

Systemic corticotherapy caused little leucopenia, keeping a normal number of leucocytes in most cases. Regarding the group without corticotherapy, the treatment led to more cases of leucocytosis. These data also corresponded to the results of WBC count in patients with pityriasis versicolor. Therefore, the mechanism of immunosuppression caused by the drug was not due to leucopenia.^{16,18}

Indeed, corticotherapy causes leucocytosis, mainly at the expense of neutrophilia. Thus, the results obtained allow concluding that the mechanism by which the corticosteroids increase the frequency of pityriasis versicolor might even be due to an immunosuppressant effect, in this case qualitative, but not measurable in the study. The hypothesis that immunosuppression is due to the overall decrease of leucocytes (quantitative) has been discarded.^{16,18}

The distribution of the variable 'dose' between the groups with and without pityriasis versicolor showed predominance of lower doses in the group with mycosis, suggesting that high doses (immunosuppressive) were not required to cause fungal infection and that, therefore, it may not be caused by immunosuppression.

The effect of systemic corticosteroid on the lipidogram showed a greater number of cases in which there was hypercholesterolaemia and hypertriglyceridaemia when compared to the group without corticotherapy. These results corroborate with the literature that describes increased LDL cholesterol and triglycerides in patients undergoing corticotherapy. However, in the description of the lipidogram frequencies, most patients with pityriasis versicolor showed normal cholesterolaemia and triglyceridaemia, hindering the hypothesis that while altering the lipid metabolism, glucocorticoids would change the cutaneous lipid composition, thus favouring mycosis.^{16,18}

Burke *et al.* [25] suggested that a biochemical or functional change on the cutaneous surface would make an individual susceptible to tinea versicolor and this change in the lipid film of the integument would be genetically predetermined and precipitated by other factors, such as the systemic corticotherapy. On the other hand, Boardman *et al.* [22] studied the composition of the skin of patients with and without pityriasis versicolor and found no significant differences between the groups.

Finally, normal fasting blood glucose was prevalent among patients with mycosis. This result is consistent with those obtained by comparison between levels of fasting blood glucose and use/non-use of corticosteroids, which in turn revealed no significant number of patients with hyperglycaemia in the group taking systemic corticosteroids. This fact reinforces the suggestion by Mandel *et al.* [46], who stated that increased blood glucose levels would not function as an isolated precipitating factor for the development of mycosis. It also corroborates the findings of García-Ilumbría *et al.* [47] who did not observe greater susceptibility to pityriasis versicolor in diabetic patients when compared to non-diabetic patients.

It is concluded that long-term systemic corticotherapy increased the frequency of pityriasis versicolor, but the distribution of laboratorial variables among patients with pityriasis versicolor showed predominance of normal WBC count, lipidogram and fasting blood glucose.

Pityriasis versicolor and systemic corticotherapy

Acknowledgments

The authors are thankful to: Cassiano Antonio Morais University Hospital; Maruípe Health Unit; and the participating patients enrolled in the sample.

Conflict of interest

There is no conflict of interest.

References

- 1 Hu SW, Bigby M. Pityriasis versicolor: a systematic review of interventions. *Arch Dermatol* 2010; **146**: 1132–40.
- 2 Crespo-Erchiga V, Gómez-Moyano E, Crespo M. Pityriasis versicolor and the yeasts of genus *Malassezia*. Actas Dermosifiliogr 2008; **99**: 764–71.
- 3 Zaitz C, Ruiz LRB, Souza VM. Dermatoses associadas às leveduras do gênero *Malassezia*. *An Bras Dermatol* 2000; **75**: 129–42.
- 4 Di Silverio A, Zeccara C, Serra F, Ubezio S, Mosca M. Pityriasis versicolor in a newborn. Mycoses 1995; 38: 227–8.
- 5 Jena DK, Sengupta S, Dwari BC, Ram MK. Pityriasis versicolor in the pediatric age group. Indian J Dermatol Venereol Leprol 2005; 71: 259–61.
- 6 Nagata R, Nagano H, Ogishima D, Nakamura Y, Hiruma M, Sugita T. Transmission of the major skin microbiota, *Malassezia*, from mother to neonate. *Pediatr Int* 2012; **54**: 350–5.
- 7 Guyton AC, Hall JE. Tratado de fisiologia médica, 10th edn. Rio de Janeiro: Guanabara Koogan, 2002.
- 8 Delemarre EM, Felius B, Delemarre-van de Waal HA. Inducing puberty. Eur J Endocrinol 2008; **159**(Suppl. 1):S9–15.
- 9 Mendez-Tovar LJ. Pathogenesis of dermatophytosis and tinea versicolor. Clin Dermatol 2010; 28: 185–9.
- 10 Gaitanis G, Magiatis P, Hantschke M, Bassukas ID, Velegraki A. The Malassezia genus in skin and systemic diseases. Clin Microbiol Rev 2012; 25: 106–41.
- 11 Park HJ, Lee YW, Choe YB, Ahn KJ. Skin characteristics in patients with pityriasis versicolor using non-invasive method, MPA5. Ann Dermatol 2012; 24: 444–52.
- 12 Chetty GN, Kamalam A, Thambiah AS. Pityriasis versicolor-a study of 200 cases in a tropical skin clinic. *Mycoses* 1979; **22**: 234–6.
- 13 Sodré CT, Assis TL, Azulay RD. Pitiríase versicolor. An Bras Dermatol 1984; 59: 275–80.
- 14 Saadatzadeh MR, Ashbee HR, Cunliffe WJ, Ingham E. Cell-mediated immunity to the mycelia phase of *Malassezia* spp. In patients with pityriasis versicolor and controls. *Br J Dermatol* 2001; **14**: 77–84.
- 15 Sampaio SAP, Rivitti EA. Micoses superficiais. In: Sampaio SAP, Rivitti EA (eds), *Dermatologia*, 3rd edn. São Paulo: Artes Médicas Ltda, 2007; 703–22.
- 16 Williams LC, Nesbitt LTJR. Update on systemic glucocorticosteroids in dermatology. Dermatol Clin 2001; 19: 63–77.
- 17 Finamor LP, Finamor FJR, Muccioli C. Corticoterapia e uveítes. Arq Bras Oftalmol 2002; **65**: 483–6.
- 18 Freitas THP, Souza DAF. Corticosteroides sistêmicos na prática dermatologica. Parte I – Principais efeitos adversos. An Bras Dermatol 2007; 82: 63–70.
- 19 Gupta P, Bhatia V. Corticosteroid physiology and principles of therapy. Indian J Pediatr 2008; 75: 1039–44.
- 20 Aulakh R, Singh S. Strategies for minimizing corticosteroid toxicity: a review. Indian J Pediatr 2008; 75: 1067–73.
- 21 Seguro LP, Rosario C, Shoenfeld Y. Long term complications of past glucocorticoid use. *Autoimmun Rev* 2013; **12**: 629–32.
- 22 Boardman CR, Malkinson FD. Tinea versicolor in steroid-treated patients. Incidence in patients with chronic ulcerative colitis and regional enteritis treated with corticotropin and corticosteroids. *Arch Dermatol* 1962; 85: 44–52.

- 23 Framil VMS, Melhem MSC, Szeszs W, Corneta EC, Zaitz C. Pitiríase versicolor: isolamento e identificação das principais espécies de Malassezia. An Bras Dermatol 2010; 85: 111–4.
- 24 Zonana-Nacach A, Barr SG, Magder LS, Petri M. Damage in systemic lupus erythematosus and its association with corticosteroids. *Arthritis Rheum* 2000; **43**: 1801–8.
- 25 Burke RC. Tinea versicolor: susceptibility factors and experimental infection in human beings. *J Invest Dermatol* 1961; **36**: 389–402.
- 26 Morais PM, Cunha MGS, Frota MZM. Aspectos clínicos de pacientes com pitiríase versicolor atendidos em um centro de referência em Dermatologia Tropical na cidade de Manaus (AM), Brasil. An Bras Dermatol 2010; 85: 797–803.
- 27 Ghosh SK, Dey SK, Saha I, Barbhuiya JN, Ghosh A, Roy AK. Pityriasis versicolor: a clinicomycological and epidemiological study from a tertiary care hospital. *Indian J Dermatol* 2008: 53: 182–5.
- 28 Santana JO, Azevedo FLA, Filho Campos PC. Pitiríase versicolor: caracterização clínico-epidemiológica em pacientes da área urbana de Buerarema-BA, Brasil. An Bras Dermatol 2013; 88: 218–24.
- 29 Gupta AK, Batra R, Bluhm R, Faergemann J. Pityriasis versicolor. Dermatol Clin 2003; 21: 413–29.
- 30 Rao GS, Kuruvilla M, Kumar P, Vinod V. Clinico-epidermiological studies on tinea versicolor. *Indian J Dermatol Venereol Leprol* 2002; 68: 208–9.
- 31 Faergemann J. Pityriasis versicolor. J Am Acad Dermatol 1994; 31: S18–20.
- 32 Ingordo V, Naldi L, Colecchia B, Licci N. Prevalence of pityriasis versicolor in young Italian sailors. *Br J Dermatol* 2003; **149**: 1270–2.
- 33 Hafez M, El-Shany S. Genetic susceptibility in pityriasis versicolor. Dermatologica 1985; 171: 86–88.
- 34 Terragni L, Lasagni A, Oriani A, Gelmetti C. Pityriasis versicolor in the pediatric age. *Pediatr Dermatol* 1991; 8: 9–12.
- 35 Faergemann J, Fredriksson T. Tinea versicolor with regard to seborrheic dermatitis. An epidemiological investigation. *Arch Dermatol* 1979; **115**: 966–8.
- 36 He SM, Du WD, Yang S et al. The genetic epidemiology of tinea versicolor in China. Mycoses 2008; 51: 55–62.
- 37 Roed-Petersen J. Tinea versicolor and body lotions. Acta Derm Venereol 1980; 60: 439–40.
- 38 Framil VMS, Melhem MSC, Szeszs W, Saitz C. Novos aspectos na evolução clínica da pitiríase versicolor. An Bras Dermatol 2011; 86: 1135–40.
- 39 Arenas R, Isa-Isa R, Cruz AC. Pityriasis versicolor in Santo Domingo, Dominican Republic. In vivo morphological data of *Malasezzia* spp. in 100 cases. *Rev Iberoam Micol* 2001; 18: 29–32.
- 40 Furtado MSS, Cortêz ACA, Ferreira JA. Pitiríase versicolor em Manaus, Amazonas – Brasil. An Bras Dermatol 1997; 72: 349–51.
- 41 Belém LF, Lima EO, Andrade DA et al. Estudo epidemiológico da pitiríase versicolor no estado da Paraíba, Brasil. Rev Bras Anal Clin 2001; 33: 66–67.
- 42 Miranda KC, De Araujo CR, Soares AJ, De Aquino Lemos J, Souza LK, Do Rosário RSM. Identificação de espécies de Malassezia em pacientes com pitiríase versicolor em Goiânia-GO. Rev Soc Bras Med Trop 2006; 39: 582–3.
- 43 Kyriakos KP, Terzoudi S, Palamaras I, Pagana G, Michailides C, Emmanuelides S. Pityriasis versicolor prevalence by age and gender. *Mycoses* 2006; **49**: 517–8.
- 44 La Ruche G, Cesarini JP. Histology and physiology of black skin. Ann Dermatol Venereol 1992; 119: 567–74.
- 45 Herrmann F, Prose PH, Sulzberger MB. Studies on sweating. V. Studies of quantity and distribution thermogenic sweat delivery to the skin. J Invest Dermatol 1952; 18: 71–86.
- 46 Mandel EH, Ores RO, Siragusa RJ. The incidence of diabetes mellitus in patients with tinea versicolor. J Natl Med Assoc 1974; 66: 198– 200.
- 47 García-Ilumbría L, Richard-Yegres N, Pérez-Blanco M, Yegres F, Mendoza M, Acosta A. Superficial mycoses: comparative study between type 2 diabetic patients and a non-diabetic control group. *Invest Clin* 2005; 46: 65–74.