Green Pityriasis Versicolor – A Novel Presentation

Vijay Zawar[1,*] and Antonio Chuh[2]

[1]*Department of Dermatology, Godavari Foundation Medical College, India*

[2]*School of Public Health, The Chinese University of Hong Kong and The Prince of Wales Hospital, Hong Kong*

Abstract: Pityriasis versicolor may present as hypopigmented, hyperpigmented, concomitantly hypo- and hyperpigmented, erythematous, and atrophic subtypes. We report a 15-year-old boy with pruritic greenish plaques on his anterior abdominal wall. Examination under Wood's light and skin scrapings for KOH smear examination substantiated pityriasis versicolor. We found no extraneous cause for the greenish pigmentation. The eruptions and the discolouration subsided four weeks after systemic fluconazole treatment. We believe that this represents the first case of such presentation of pityriasis versicolor. The mechanism of the strange colouration is unknown.

Keywords: *Malassezia spp*, *M. globosa*, *M. furfur*, pigmentation, Wood's light, yeast.

BACKGROUND

Pityriasis versicolor (PV) is caused by *Malassezia spp*, lipophilic yeasts found in the sebaceous glands, the commonest species being *M. globosa*, *M. sympodialis*, *M. restricta*, and *M. furfur* [1-4]. PV is classically described as pink, coppery brown, reddish brown, tan, pale, or while patches which might be scaly or non-scaly. Under Wood's light, a golden yellow fluorescence is characteristic. However, the lesions being greenish under normal room light have not been reported. We report the first case of microscopically-confirmed PV for which a greenish colour led to convolutions in the diagnostic workup.

CASE REPORT

A 15-year-old boy student was brought to consult us for mildly pruritic, non-painful lesions over his trunk for three weeks. The rash was first noticed by the boy himself, and was progressively becoming more intensely-coloured and larger over three weeks. His school life was affected by the pruritus, which was aggravated on exercise and on profuse sweating. His parents were concerned as to whether this rash could be contagious to the siblings, and the school teachers were concerned if the rash was contagious to other classmates of the patient.

His past history for major illnesses was unremarkable. There was no indication of any systemic disease or immunodeficiency. He has no history of clinically diagnosed PV. The duration and strength of sun exposure over past few weeks were similar to such exposure in the past. His mother and elder sister had history of PV several months ago, being treated by other medical practitioners. The family had no history of dermatophytic infections over the past two years. Our patient was not a swimmer. He and his mother specifically denied the use of any topical or systemic medications, including over-the-counter remedies and herbal remedies, before and during eruption of the skin rash.

Our examination revealed multiple well-demarcated pale-green-coloured scaly plaques with fine scaling on his anterior abdominal wall (Figure **1**). The lesions were multiple, annular and oval in shape, some plaques were irregular. The finger nail sign revealed the typical furfuraceous scales.

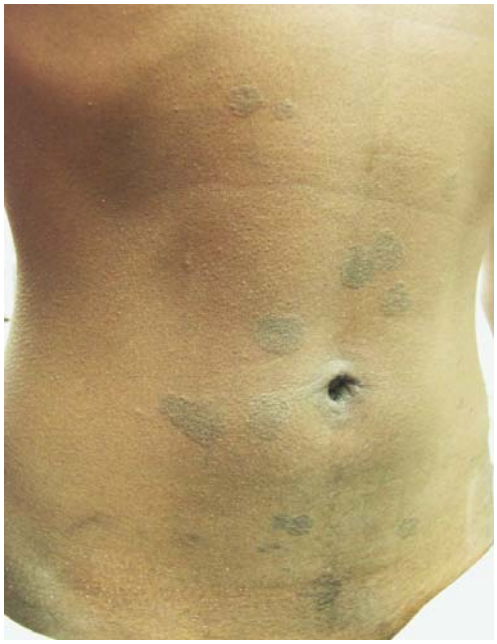

Figure 1: Multiple well-demarcated, pale-green plaques with fine scaling on the anterior abdominal wall.

*Address correspondence to this author at the Shreeram Sankul, Opp. Hotel Panchavati, Vakilwadi, Nashik-422002, Maharashtra State, India;
Tel: 0253-2571820; Fax: 0253-2312090; E-mail: vijayzawar@yahoo.com

The orientations of the patches seemed to be random, and were not along the skin lines of Langer or Blaschko's lines. No peripheral collarette scaling configuration was noted. His scalp showed mild seborrhoeic dermatitis. However, the other skin regions including extremities and palmoplantar surfaces were unaffected. Mucosal surfaces were normal. Fingernails and toenails were normal. His throat was not inflammed. There was no regional or generalised lymphadenopathy.

Examination under Wood's light revealed yellow fluorescence over the plaques. Unaffected skin areas showed no fluorescence. His inner garments did not fluoresce under Wood's light. Re-examination one-day later upon a nice bath with adequate soaps revealed the same fluorescence and no change in the colour or configuration of the lesions was noted after cleansing the lesions. We also examined this patient for a possibility of green chromhidrosis but this was not seen even after active exercise to the point of profuse sweating, wearing bright white thin cotton inner truncal clothing.

His complete blood count, fasting and post-prandial blood glucose, liver function tests, and creatinine were normal. HIV antibodies and VDRL were negative. Skin scraping from one of the most prominent lesions for potassium hydroxide smear revealed multiple slender non-septate hyphae with clumps of spores demonstrating a "meatball-and-spaghetti' appearance (Figure **2**). Skin scrapings were also submitted for fungal culture on Sabouraud's medium enriched with olive oil revealed tiny growth at the end of six weeks. The colony sample also confirmed spherical yeast like forms, further confirmed the diagnosis of PV, but there was no evidence of pathogenic dermatophytic fungal infection. As the patient did not consent for the skin biopsy, we could not study the histopathological features in this case.

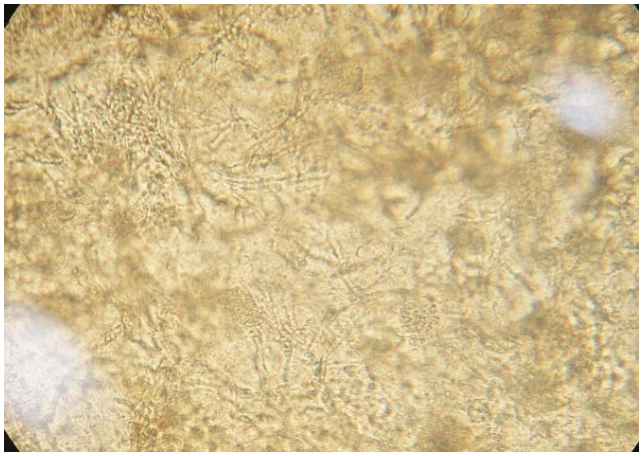

Figure 2: KOH study revealed several slender, non-septate hyphae with spores, typical "meatball-and-spaghetti" configuration.

Our clinical diagnosis substantiated by laboratory findings was thus PV. We prescribed oral fluconazole 400 mg once a week for three weeks and topical ketoconazole cream applied twice daily for four weeks.

We saw the patient at the end of weeks two and four. His liver function remained unaffected. Pruritus disappeared, colour became dull and the scales reduced by two weeks. The plaques, scaling and discolouration completely subsided with slight post-inflammatory hypopigmentation. The fears of the parents and school teachers were allayed. We saw the patient again 12 weeks later. Complete resolution of the plaques was seen. By then, we had examined his household members. There was no evidence of green pityriasis versicolor in any of them. There was no recurrence until six months after the complete resolution. Later, the patient was lost for the follow up.

DISCUSSION

PV is named such because of its wide variations in hyper- and hypopigmentation. It is caused by *Malassezia spp*, lipophilic yeasts found in the sebaceous glands. The distributions of various *Malassezia spp* causing PV in different geographic regions can be different. The commonest species are *M. globosa*, *M. sympodialis*, *M. restricta*, and *M. furfur* [1-4]. In patients with extensive PV, the presence of more than one species is common [5]. Owing to constraints in resources, we could not undertake species identification in our patient.

Several coloured morphological variants are reported in PV, including hypopigmented (alba) [6], hyperpigmented (black, brown or fawn), red-tan or erythematous appearances [7, 8], and atrophic subtype [9]. There is no strict correlation between the malassezia species concerned and whether the lesions are hyper- or hypopigmented [10].

The classical fluorescence of PV under Wood's light is believed to be due to a bisindolyl compound known as pityrialactone [11]. This is however unrelated to the colour of rash under normal sunlight or room lighting.

Proposed mechanisms for hypopigmentation in PV include screening effect of scales, release of metabolites toxic to melanocytes, and inhibition of

tyrosinase by dicarboxylic acids including azelaic acid. Recently, pityriacin, an UV absorbing substance, has been implicated in the causation of hypopigmentation. This and other tryptophan induced fluorochromes are postulated to render yeasts less sensitive to ultraviolet light [12].

As for hyperpigmentation, cytokine production induced by *Malassezia spp* may lead to pigmentary changes in hyperpigmented variants. *M. furfur* is known to be able to produce melanin or melanin-like compounds both *in vitro* and *in vivo*, thus causing hyperpigmentation [13]. Vascular dilatation has also been shown to be implicated in hyperpigmented PV and PV rubra [14, 15].

To our best knowledge, there has been no previously reported case with green coloration in PV. This coloration was starkly different from other patients with PV within and beyond our locality. The cause of such appearance is unknown. Having ruled out artefact- related reasons of the green colour of the lesions, we believe that the skin colour change was intrinsically related to the malassezia infection, possibly with the immunopathological changes. The mechanism for such is yet unknown to us. We urge our readers to look out for patients with this variant of PV to elucidate more information regarding its occurrence, susceptibility factors, pathogenesis for the specific colouration, and outcomes of this variant of PV.

REFERENCES

[1] Shah A, Koticha A, Ubale M, Wanjare S, Mehta P, Khopkar U. Identification and speciation of malassezia in patients clinically suspected of having pityriasis versicolor. Indian J Dermatol 2013; 58: 239. http://dx.doi.org/10.4103/0019-5154.110841

[2] Sugita T, Zhang E, Tanaka T, Nishikawa A, Tajima M, Tsuboi R. Recent advances in research on Malassezia microbiota in humans. Med Mycol J 2013; 54: 39-44. http://dx.doi.org/10.3314/mmj.54.39

[3] Dutta S, Bajaj AK, Basu S, Dikshit A. Pityriasis versicolor: Socioeconomic and Clinico Mycological Study in India. Int Dermatol 2002; 41: 823-4. http://dx.doi.org/10.1046/j.1365-4362.2002.01645.x

[4] Kindo AJ, Sophia SK, Kalyani J, Anandan S. Identification of Malassezia species. Indian J Med Microbiol 2004; 22: 179-81.

[5] Romano C, Mancianti F, Nardoni S, Ariti G, Caposciutti P, Fimiani M. Identification of Malassezia species isolated from patients with extensive forms of pityriasis versicolor in Siena, Italy. Rev Iberoam Micol 2013 Feb 19.

[6] Thoma W, Krämer HJ, Mayser P. Pityriasis versicolor alba. J Eur Acad Dermatol Venereol 2005; 19: 147-52. http://dx.doi.org/10.1111/j.1468-3083.2004.01085.x

[7] Maeda M, Makimura KC, Yamaguchi H. Pityriasis versicolor rubra. Eur J Dermatol 2002; 12: 160-4.

[8] 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. http://dx.doi.org/10.5021/ad.2012.24.4.444

[9] Tellechea O, Cravo M, Brinca A, Robalo-Cordeiro M. Pityriasis versicolor atrophicans. Eur J Dermatol 2012; 22: 287-8.

[10] Mayser P, Stapelkamp H, Krämer HJ, Podobinska M, Wallbott W, Irlinger B, *et al*. Pityrialactone – a new fluorochrome from the tryptophan metabolism of Malassezia furfur. Antonie Van Leeuwenhoek 2003; 84: 185-91. http://dx.doi.org/10.1023/A:1026042903354

[11] Zawar VP, Joshi PB, Patil DJ. Clinical and mycological studies in pityriasis versicolor. Med J Western India 1993; 342: 64-7.

[12] Gaitanis G, Velegraki A, Mayser P, Bassukas ID. Skin diseases associated with Malassezia yeasts: Facts and controversies. Clin Dermatol 2013; 31: 455-63. http://dx.doi.org/10.1016/j.clindermatol.2013.01.012

[13] Youngchim S, Nosanchuk JD, Pornsuwan S, Kajiwara S, Vanittanakom N. The role of L-DOPA on melanization and mycelial production in Malassezia furfur. PLoS One 2013; 8: e63764. http://dx.doi.org/10.1371/journal.pone.0063764

[14] Dotz WI, Henrikson DM, Yu GS, Galey CI. Tinea versicolor: a light and electron microscopic study of hyperpigmented skin. J Am Acad Dermatol 1985; 12: 37-44. http://dx.doi.org/10.1016/S0190-9622(85)70006-0

[15] Cui F, She XD, Li XF, Shen YN, Lü GX, Liu WD. Effects of Malassezia isolates on cytokines production associated with melanogenesis by keratinocytes. Zhongguo Yi Xue Ke Xue Yuan Xue Bao 2007; 29: 196-200.

Received on 29-07-2013 Accepted on 11-08-2013 Published on 30-11-2013