

Exuberant clinical presentation of probable *Malassezia* folliculitis in a young nonimmunosuppressed patient *

Apresentação clínica exuberante de provável foliculite por *Malassezia* em jovem imunocompetente

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Abstract: *Malassezia* folliculitis is an inflammatory disorder observed in both immunocompetent and immunosuppressed patients. The authors describe an unusual and exuberant presumed case affecting the face, trunk and upper limbs of a 12-year-old nonimmunosuppressed patient. Although the agent was not identified by culture, the clinical and histopathological aspects plus the response to specific treatment support the diagnosis of *Malassezia* folliculitis. The only possible predisponent cause observed on the patient was greasy skin. Repetitive cultures were negative. Treatment with itraconazol promoted apparent cure, however, the patient relapsed twelve months later.

Keywords: Folliculitis; *Malassezia*; Skin; Skin manifestations; Spores, fungal

Resumo: Foliculite por *Malassezia* é processo inflamatório observado em pacientes imunocompetentes e imunossuprimidos. Os autores relatam um provável caso exuberante e incomum comprometendo a face, tronco e membros superiores de paciente de 12 anos de idade, não imunossuprimido. Embora o agente não tenha sido cultivado, os achados clínicos e histopatológicos aliados à resposta terapêutica sugerem o diagnóstico de foliculite por *Malassezia*. A única possível causa predisponente demonstrada no paciente foi a pele oleosa. Tentativas de cultivo do agente foram negativas. O tratamento com itraconazol promoveu cura aparente, entretanto, houve recaída após 12 meses.

Palavras-chave: Esporos fúngicos; Foliculite; *Malassezia*; Manifestações cutâneas; Pele

INTRODUCTION

Malassezia folliculitis (*M.folliculitis*), previously known as *Pityrosporum* folliculitis, was recognized as a specific disease by Potter in 1973.¹ It has been described by some authors as a common disease in both

young and middle-aged patients.² It has been interpreted as an infection of the hair follicle or as an inflammatory process according to different authors.¹⁻⁴

⁴ It occurs as an acneiform eruption presenting mild

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to moderate pruritus predominantly on the chest, upper back, and shoulders.¹ Many patients with *M. folliculitis* have been following anti-acne regimens before correct diagnosis. Therefore, patients presenting an acneiform eruption not improving with specific treatment must be investigated for *Malassezia* folliculitis. The authors report an exuberant and unusual presumed clinical case in a nonimmunosuppressed patient without any other predisponent causes than his greasy skin.

CASE REPORT

A 12-year-old male patient with a two-month history of severe acneiform eruption was seen at our hospital unit. He had been treated with tetracyclines and systemic corticosteroid after a presumptive diagnosis of acne vulgaris. As no improvement was observed, the patient was referred to our service and complained of pruritus, worse in the warm days. His past medical history was unremarkable and no medications had been taken previously. On clinical examination monomorphic acneiform skin lesions were observed on his face, trunk and upper limbs (Figures 1 and 2). A greasy skin plus few pustular and comedo-like lesions were also observed. Two punch-biopsies were performed and showed the same picture of a suppurative folliculitis. The pilosebaceous follicles appeared dilated, with keratinous plug, containing amorphous cellular debris, many inflammatory cells and globular structures (Figures 3 and 4). PAS and silver methenamine stain were positive to oval, simple budding yeast-like cells, consistent with *Malassezia* spp. (Figure 5). Specimens from additional punch biopsy collected for culture were negative. The diagnosis of the patient was supported by histopathologi-



FIGURE 1: *Malassezia* folliculitis. Multiple papules, pustules and rare comedones on the face



FIGURE 2: *Malassezia* folliculitis. Multiple papules and pustules on the upper thorax

cal analyses, which did not reveal mycelial forms and showed abundant *Malassezia* yeasts in the follicles.

The patient was treated with itraconazole 200mg daily for two months with almost complete disappearance of all lesions. However, there was a relapse 12 months later when, once again, itraconazole 200mg/daily proved efficient.

DISCUSSION

Malassezia folliculitis has been considered as an inflammatory skin disorder for some authors and as an infection of the hair follicle by others and reported more frequently in young and middle-aged male patients.¹⁻⁴ As *Malassezia* yeasts are commensals in normal skin the positive cultures do not necessarily

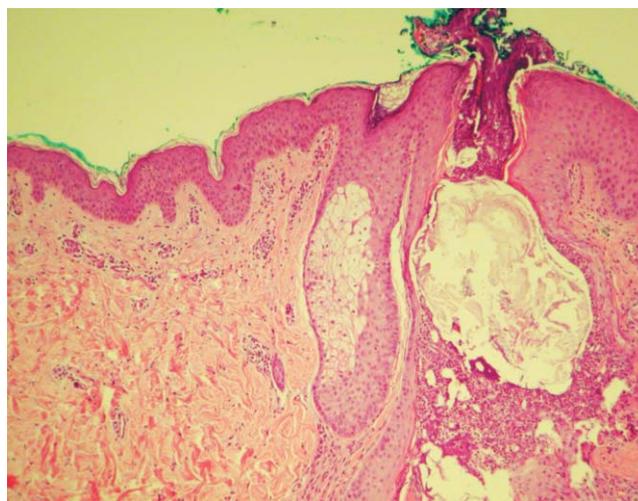


FIGURE 3: *Malassezia* folliculitis. Hyperkeratosis and cornel plug in the dilated follicular infundibulum. Inflammatory cells and granular debris are showed in the dermis suggesting rupture of the hair follicle H.E.40X

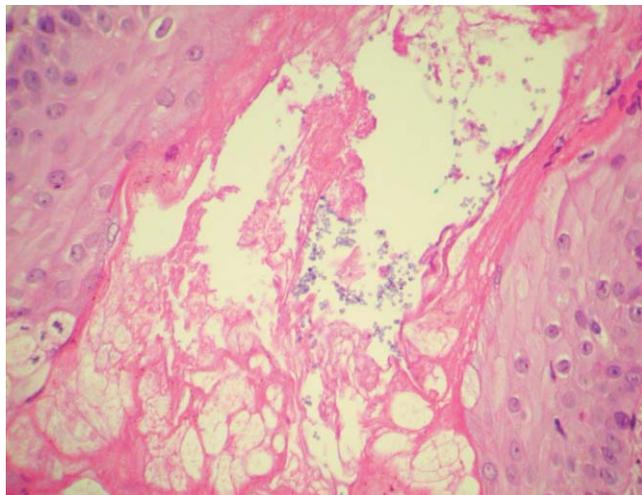


FIGURE 4: *Malassezia* folliculitis. Follicular isthmus with numerous tiny yeast-like organisms. H.E.400X

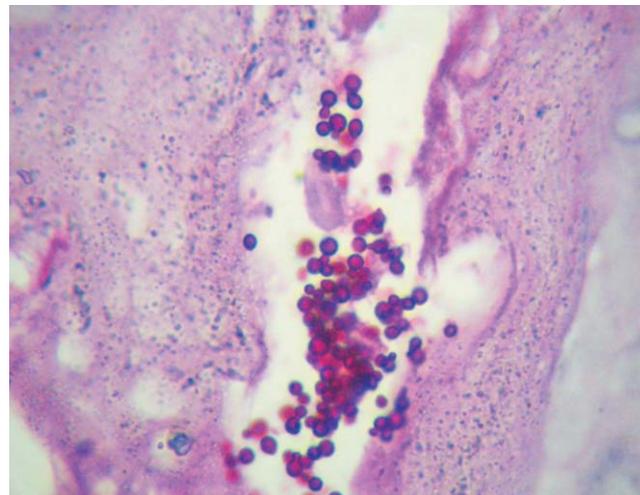


FIGURE 5: *Malassezia* folliculitis - PAS stain reveals spherical to oval yeast-like organisms. PAS 1000X

imply that the fungus is the etiological agent. However, the presence of large numbers of yeasts in the follicle and around it associated with the rich presence of inflammatory cells, sometimes promoting the formation of small localized abscesses and response to antifungal treatment has provided sufficient elements to consider the disorder as an individualized disease.^{1,2} It is interesting that in cases of *Malassezia* folliculitis mycelia elements are not observed.¹ This may suggest that the conversion to mycelia form may not be necessary to promote inflammation and clinical disease.¹ In our case the exuberant clinical aspect associated with the intensity of the inflammatory process, not responsive to the previous corticosteroid treatment, suggested that the *Malassezia* yeasts were promoting a real infectious process. Comorbidities have been associated with *M. folliculitis*, such as seborrheic dermatitis in 40% of cases, acne vulgaris in 27% and pityriasis versicolor in 6%.² The differential diagnosis varies according to the presence of comorbidities like HIV-infection; in these patients the differential diagnosis would be among *M. folliculitis* and other papular eruptions such as the papulopuritic eruption of HIV, HIV-associated eosinophilic folliculitis and suppurative folliculitis of bacterial etiology.³ In this particular patient, the first hypothesis was a corticosteroid induced acneiform eruption, despite the clinical history indication that the lesions already existed before the corticosteroid treatment.

Although the pathophysiological phenomena in *Malassezia* folliculitis are unknown, Hill et al.⁴ observed that the overgrowth of *Malassezia* yeasts in the follicle is a secondary event caused by occlusion by hyperkeratosis. The inflammatory reaction observed in the follicle may be the result of the ability of a *Malassezia* lipase to hydrolyse triglycerides into free

fatty acid, as well as a demonstration that *Malassezia* species can induce the production of inflammatory cytokines in human epidermal keratinocytes via Toll-like receptor 2 observed in vitro.⁵

It has been recently shown that the *Malassezia* species identified in the follicle causing *M. folliculitis* were the same species identified on healthy skin of cases and controls. According to Azaka et al.⁶ the species identified in 32 cases of *M. folliculitis* were *M. globosa*, *M. restricta* and *M. sympodialis* - the most common. The same study showed that the composition of *Malassezia* microbiota on apparently healthy skin of patients with *Malassezia* folliculitis was the same as that identified in the skin of healthy patients. Previous studies have shown that the predominant species in healthy skin of normal individuals are *M. globosa* and *M. sympodialis*.⁷ Framil et al, working with samples of patients with pityriasis versicolor in Brazil identified *M. sympodialis* as the most frequent (39.0%), followed by *M. furfur* (19.3%), *M. globosa* (26.8%) and *M. slooffiae* (4.9%).⁸ It must be highlighted that it is difficult to isolate the causative agents, even using the Dixon agar medium; the molecular methods could be an option when available.

A seven-day course of itraconazole 200mg/daily proved, in a double blind placebo-controlled study, to be efficient in treating 13 patients with *M. folliculitis*.⁹ Based on this previous trial in addition to recognizing the potential hepatotoxic adverse effect when using ketoconazole, the authors chose itraconazole as their treatment option. In the literature, topical ketoconazole, oral ketoconazole alone or in combination with topical ketoconazole promoted cure in respectively 12%, 75% and 75% of 26 patients, but recurrence was observed within 3 to 4 months after the end of treatment with these regimens.¹⁰ Alternative treatments

such as photodynamic therapy for those with poor response or adverse effects of antifungal drug have been proposed.

Although this case report is based on a single patient, we suggest that skin biopsies should be done in cases of atypical acneiform eruption occurring in pre-adolescent, nonimmunosuppressed patients. □

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