Comments on the article: "Update on therapy for superficial mycoses: review article part I"*

Fabiane Mulinari-Brenner[1]
Carla Cristina Marques[1]
Luiz Eduardo Fabricio de Melo Garbers[2]
Patrícia Kiyori Watanabe[1]

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To the authors of the article: "Update on therapy for superficial mycoses: review article part I"[1].

After going over the aforementioned article together with Dermatology Professors of the Hospital das Clínicas - UFPR, 3 items of this paper have provoked discussion, namely:

1) Perifolliculitis capitis abscedens et suffodiens, currently called dissecting cellulitis of the scalp, is classified as primary scarring alopecia since the 2000 consensus statement. Recent publications do not associate this condition with fungal colonization. [2] The 2013 paper "What's new in cicatricial alopecia?" indicates an inflammatory process that attacks and destroys the stem cells of hair follicles as the cause of this primary alopecia.[3] Tchernev has described a disordered keratinization - which leads to the occlusion and accumulation of keratin at the hair follicle, followed by its dilation and rupture - as its main cause. This is justified not only by the induction of a granulomatous inflammatory process accompanied by the attraction of gigantic cells, partially phagocyting the keratin masses, but also by an inflammatory bacterial process derived from a superinfection, most frequently caused by Staphylococcus aureus and Staphylococcus epidermidis (which are considered to be the main factors in the chemotaxis of neutrophils).[4] This alopecia is irreversible even with appropriate therapy, due to the partial or complete destruction of the hair follicle caused by neutrophil infiltration and infiltration of giant cells. The association with other skin diseases resulting from follicular occlusion, such as hidradenitis suppurativa and acne conglobata suggests a common pathogenic mechanism based on follicular retention.

2) The black dots clinically observed in tinea capitis correspond to the comma or spiral-shaped hair seen in dermoscopy, rarely presenting as exclamation mark hair, a feature more suggestive of alopecia areata.[5]

3) Table 9 of the article shows griseofulvin as a therapeutic option for chronic mucocutaneous candidiasis, at a dose of 25 mg (initial dose) diluted in 500ml of glycosylated solution, and addition of 25 mg of hydrocortisone sodium succinate to the intravenous solution. This is probably a misprint. The correct drug would be amphotericin B. Other intravenous options include fluconazole and caspofungin. [5]

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[1] Universidade Federal do Paraná (UFPR) – Curitiba (PR), Brazil.
[2] Faculdade Evangélica do Paraná (FEPAR) – Curitiba (PR), Brazil.

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MAILING ADDRESS:
Fabiane Mulinari-Brenner
Hospital de Clínicas da Universidade Federal do Paraná
Departamento de Clínica Médica - Dermatologia.
Rua General Carneiro, 181 - Alto da Glória
80060-900 - Curitiba – PR - Brazil.
E-mail: fmbrenner@ufpr.br

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