Is it Tinea Versicolor, Vitiligo, or Both?

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History

An 85-year-old otherwise healthy man with rheumatoid arthritis presented with hypopigmented and dyspigmented patches over the chest and upper and lower back that evolved over several years. In addition, the patient had well-demarcated depigmented patches in the groin area, as well as on the bilateral elbows. Isolated depigmented foci were also noted in the left axilla and right antecubital fossa. The initial lesions were received with a clinical differential of nummular dermatitis vs. Lyme disease vs. cutaneous T-cell lymphoma (CTCL). Eight months later the clinical diagnosis was still suspicious for CTCL with the added clinical differential of vitiligo. The patient was treated with narrow band UVB and topical steroids without changes in the dyspigmentation.

Histopathology

Initial biopsies showed a patchy reduction of melanin at the dermoepidermal junction by Fontana-Masson staining and diminished melanocytes on H&E, Melan-A, and MITF stained slide (**Figure 1**). This pattern was initially diagnosed as compatible with an early, incomplete, or recovering focus of vitiligo. However, subsequent biopsies demonstrated similarly diminished melanocytes and melanin production with the additional finding of intracorneal tinea versicolor (TV) infection. Prior biopsy slides were re-evaluated and TV was identified in one of these by PAS staining.

Histology

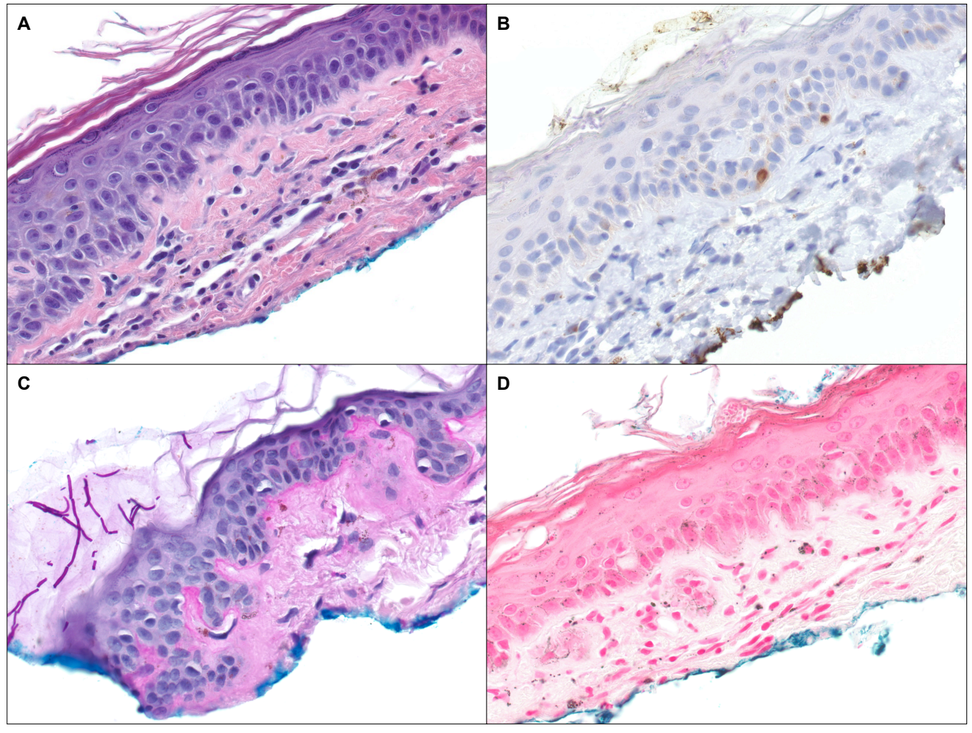

Figure 1.

A. H&E staining. Diminished number of melanocytes and melanin pigments from the basal layer.
B. Melan A immunostaining. Marked loss of melanocytes and their processes.
C. PAS staining. Numerous budding yeasts and heapae are present in the stratum conrenum.
D. Masson-Fontana staining. Patchy reduction of melanin at the dermo-epidermal junction.

Discussion

Because of continued concerns for hypopigmented CTCL, the patient was evaluated by a specialist, who, guided by the compendium of biopsy results, excluded CTCL and drew clinical distinctions between the dyspigmented patches on the patient's body. Lesions of the back and torso were deemed "hypopigmented and dyspigmented in a pattern suggestive of tinea versicolor" and other sites were described as "well-demarcated and depigmented, suggestive of vitiligo."

Ordinarily *Malassezia* (the cause of tinea versicolor) does not result in a marked decrease in the number of melanocytes. Such a pattern (especially with no identifiable intracorneal organisms) would suggest vitiligo in the correct clinical setting. In our case we saw biopsies with distinct TV that had both diminished melanocytes and pigment in the same specimen. We wondered if this pattern was caused by a coincidental overlap of vitiligo and TV, or if the TV might actually be inducing the observed loss of melanocytes.

A possible explanation was found in a literature search. Traditionally, it is believed that *Malassezia* metabolites inhibit the enzyme tyrosinase, blocking the conversion of tyrosine to melanin. This results in diminished melanin production without diminishing the total number of melanocytes. In a 2006 paper (1), Krämer, et al, identified *Malassezia* alkaloids that could induce apoptosis on cultured primary human melanocytes in a dose-dependent manner. Malassezin, the active compound, accomplished this through the induction of cytochrome P450 proteins, with subsequent activation of caspases leading to apoptotic cell death. Due to the complex nature of the metabolites, it has not been possible to detect or extract them from the lesions or dandruff of TV.

This unusual clinical and histological presentation may provide a new insight into the pathogenesis of dyspigmentation in some cases of TV. We postulate that *Malassezia* can produce a vitiligo-like histological pattern *in vivo* by either inducing apoptosis or modulating the expression of melano-genesis-related genes. More investigation is warranted to verify this hypothesis. For now, dermatopathologists confronted with a clinically dyspigmented lesion that presents diminished melanocytes and melanin by histopathology should carefully examine the stratum corneum for the spores and hyphae of TV.

Key Points

- **Both vitiligo and tinea versicolor may occur in the same patient simultaneously**
- **Vitiligo is known to present with diminished melanocytes**
- ***In vitro, Malessezia* can produce metabolites that may induce apoptosis of melanocytes**
- **This case illustrates that melanocyte apoptosis may be a feature of *in vivo* tinea versicolor**
- **Whenever vitiligo is in the clinical differential, tinea versicolor should be sought, even if diminished/absent melanocytes are observed**

References

1. Krämer HJ, et al. Malassezin. A novel agonist of the aryl hydrocarbon receptor from the yeast Malassezia furfur induces apoptosis in primary human melanocytes. *Chembiochem*. 2005 May;6(5):860-5.