



PIGMENTATION

INDUCED VITILIGO: IS MEDICATION OR INFLAMMATION THE REAL CULPRIT?

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Background: Vitiligo's pathogenesis has not been totally discovered, but many theories were proposed. Still, numerous drugs, such as imiquimod, interferon and diphencyprone were described as inducing vitiligo lesions. We report 28 cases of post-inflammatory vitiligo, despite using multiple pharmacologic treatments, and discuss whether skin lesions were induced by inflammatory response or medications. We reviewed clinical, laboratorial, histopathological and photographic records.

Observation: We compared 28 erythrodermic patients (24 men), with ages between 29 and 91 years old. Ten had Sezary Syndrome, of which seven took Interferon, nine had PUVA, two were treated with chlorambucil, 3 with methotrexate and one with radiotherapy, some with more than one treatment. Three patients had mycosis fungoides: one took interferon and two UVB-NB. Six patients had psoriasis: one treated with topical corticosteroids, one with acitretin, two with azathioprine, one with PUVA and the other, cyclosporine. Nine had eczema: 7 treated with corticosteroids, one with methotrexate and one with UVB-NB. All of them developed vitiligo during erythrodermia recovery phase.

Most of vitiligo's pathophysiology theories shows melanocytes depletion as a crucial event. As inducible factors, diphencyprone, imiquimod, interferon, vemurafenib and radiotherapy were already reported. Meanwhile, all these factors have a common key: they stimulate inflammatory responses! Diphencyprone is a synthetic allergen that arouses hypersensitivity responses when used as a topical medication. It modulates epidermal keratinocytes MHC-II expression, induces cytokines mRNA expression and stimulates IL-2, IL-8, IL-10 and TNF α . Imiquimod, however, connects to cellular Toll-like receptor 7, activating cytokines that increase Th1 response and inhibit Th2 response, similar to idiopathic vitiligo.

Key message: We propose that melanocyte depletion is possibly induced by inflammatory cytokines and it may happen during erythrodermia recovery phase and also after taking drugs that stimulate inflammation. Thus, inducible inflammation factors might be responsible for vitiligo lesions.

