

Localized vitiligo occurring in an old biopsy scar: A case report



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INTRODUCTION

Vitiligo is a depigmentation disorder of the skin, hair, or mucosa, which reveals white macules or patches caused by destruction of pigment-producing melanocytes.¹ A complex interaction between genetic, environmental, and self-destructive factors generates this form of response.² An initiating environmental event such as severe sunburns is thought to cause substantial stress in an already susceptible melanocyte, which elicits an autoimmune response in genetically predisposed individuals.³

Vitiligo's increased prevalence in immediate relatives has been established. Vitiligo affects 1% of the general population, whereas the risk of an affected patient's sibling and monozygotic twin for developing the disease is 6.1% and 23%, respectively.⁴ There is an increased risk of developing other autoimmune diseases as well, such as type 1 diabetes, autoimmune thyroiditis, pernicious anemia, and Addison disease. These frequencies suggest a nongenetic component plays an important role in the pathogenesis of the disease, in addition to a genetic predisposition.⁵

Oxidative stress is an environmental risk factor that could play a role in causing vitiligo. Stress can result from overproduction of pro-oxidant species or creation of antioxidant chemicals via a reduction reaction.⁶ Melanocytes are constantly being exposed to stressors, including ultraviolet radiation and harmful chemicals and pathologic conditions such as inflammation and cancer, which increase production of reactive oxygen species (ROS). Healthy melanocytes are capable of lessening these stressors, whereas melanocytes from patients with vitiligo are more vulnerable. Decreased catalase, a key enzyme that degrades hydrogen peroxide and catalyzes the

Abbreviation used:

ROS: reactive oxygen species

creation of other antioxidant agents, glutathione peroxidase and glucose-6-phosphate dehydrogenase, protects melanocytes from self-destructive ROS. Decreased levels of catalase have been found in melanocytes of patients with vitiligo.⁷ This finding proposes a present oxidation-reduction defect involved in the disease. Studies found patients with vitiligo experience forms of stress, triggering the immune system to attack the body's own melanocytes.

The pathogenesis of role of the Koebner phenomenon in the development vitiligo is still unknown, but its presence has been established.⁸ It has been suggested that trauma to the skin may cause hydrogen peroxide to accumulate. This accumulation may cause proteins such as heat shock protein, that are designated to protect the cell from damage, to then become immunogenic, therefore eliciting an autoimmune response.⁹ Many people report first noticing their vitiligo after traumatic events, periods of stress, or severe sunburns.¹⁰ We report the case of localized vitiligo occurring in an old biopsy scar from 25 years prior.

CASE REPORT

A 33-year old man presented with tenderness and rapid depigmentation over several weeks of an old biopsy scar on his back. At age 9, the patient had a mole biopsied in the exact location, which led to scarring. The mole was determined to be benign. The patient denied a change in scar pigmentation until recently when the area turned white. The

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Fig 1. Clinical photo of a nonscaly depigmented patch occurring in an old biopsy scar in the medial scapular back.

change in pigmentation and pain concerned him. He denied picking or scratching of the area, and denied pruritus. The patient reported a history of excessive sun exposure and chronic sunburns in the area but denied any recent sunburns. The patient denied a medical history of autoimmune diseases. The patient also denied a family history of autoimmune disease, but reported malignant melanoma in an aunt and basal cell carcinoma in his mother.

Physical examination found a nonscaly depigmented patch occurring in an old biopsy scar involving medial scapular back. No halo nevi or evidence of vitiligo was noted in the area. A careful inspection and palpation of the area was performed, and skin was of normal temperature, turgor, and texture. There were no suspicious skin lesions, rashes, or depigmented areas noted on a total skin examination.

A differential diagnosis of halo nevus was made initially, and a shave biopsy of the patch was performed. Bacitracin and a bandage were applied to the site. The patient was instructed to apply Bacitracin twice a day for 2 to 3 days and to begin clobetasol propionate cream twice a day once the biopsy site healed. The patient was lost to follow-up.

The shave biopsy showed a proliferation of fibroblasts aligned parallel to the skin surface interposed among linearly arranged thickened collagen bundles and small blood vessels. Most notably, there was an absence of melanocytes and melanin pigment within the epidermis extending over and

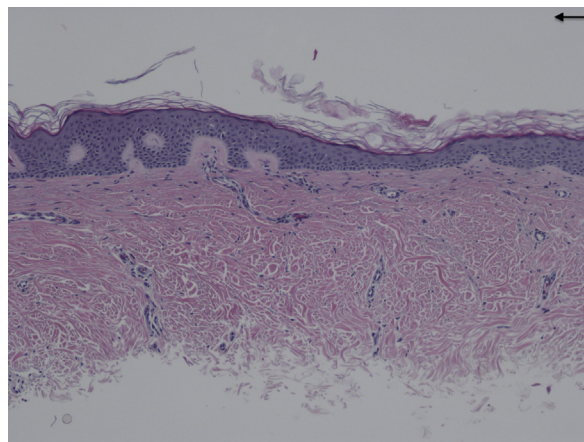


Fig 2. Absence of melanin pigment and melanocytes within the epidermis overlying a zone of fibrosis. (Hematoxylin-eosin stain; original magnification: $\times 10$.)

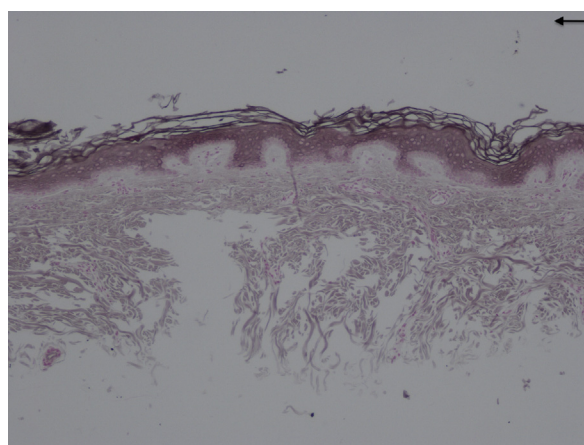


Fig 3. The absence of melanin pigment within the epidermis is highlighted with a Fontana-Masson stain for melanin pigment. (Original magnification: $\times 10$.)

slightly beyond a zone of fibrosis. The clinical diagnosis was most consistent with recent vitiligo occurring in an old biopsy scar. See [Figs 1 through 4](#).

DISCUSSION

Current evidence suggests vitiligo is an autoimmune disease with a genetic predisposition that can be triggered by nongenetic environmental risk factors. Increased prevalence among immediate relatives has been established. In addition, risk factors including ultraviolet radiation and harmful chemicals have been found to increase the production of harmful ROS, when combined with decreased catalase in susceptible melanocytes, and have been found in patients with vitiligo. A study on vitiligo by Deo et al¹⁰ found that many patients first notice their vitiligo following severe sunburns. This finding supports the belief that the vitiligo could be caused by decreased catalase and severe sunburns. A severe

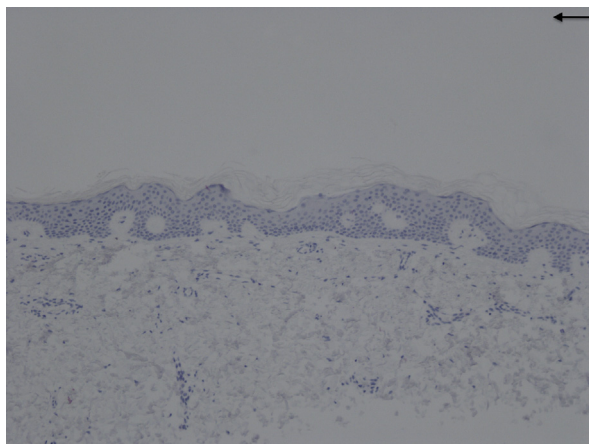


Fig 4. The absence of melanocytes within the epidermis is highlighted with a Melan-A immunoperoxidase stain. (Original magnification: $\times 10$.)

sunburn can cause local trauma to the skin, and the development of vitiligo could be explained by the Koebner phenomenon. However, in contrast, an old biopsy from 25 years prior to the incidence of vitiligo has not been reported, to our knowledge, as a possible triggering environmental event. Furthermore, a family history of autoimmune disease was not established in this patient.

Although a genetic predisposition of autoimmune disease or catalase levels could not be established because of a history of excessive sunburns in the

area and excessive scar tissue resulting from the biopsy scar, a local autoimmune attack on melanocytes could have occurred from a combination of these factors.

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