

Improvement of Vitiligo after Concurrent Treatment of Hypothyroidism: A Case Report

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Abstract

Vitiligo is a disorder of pigmentation characterized by the development of depigmented macules and patches. The etiology is multifactorial, including immune mediated destruction of melanocytes. Several autoimmune conditions are associated with vitiligo, but thyroid disease is the most common. In this case report, we describe a patient with rapidly depigmenting vitiligo who stabilized on narrowband UVB (NBUVB) phototherapy and oral corticosteroids, and began showing dramatic repigmentation once thyroid replacement was initiated. This highlights the interplay between vitiligo and autoimmune thyroid disease, as well as the need for adequate treatment of both disorders to achieve optimal treatment response.

Keywords: Vitiligo; Hypothyroidism; Treatment; Autoimmune; Phototherapy

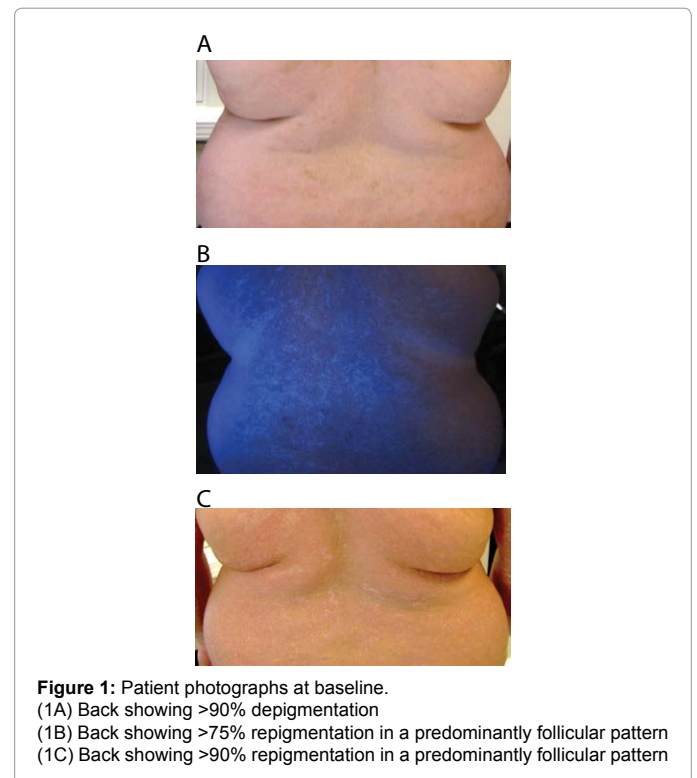
Introduction

Vitiligo is a disorder of pigmentation characterized by the development of depigmented macules and patches over the body. It affects approximately 0.5-1% of the population worldwide and is associated with significant psychosocial impact [1]. The etiology of vitiligo is multifactorial, including an impaired ability to metabolize reactive oxidative species (ROS) as well as the immune mediated destruction of melanocytes [2]. It has been noted that patients with vitiligo are at increased risk of developing other autoimmune diseases including hypothyroidism, alopecia areata, and Type I Diabetes Mellitus (DM). In fact, thyroid dysfunction in people with vitiligo is approximately 20-30%, which is significantly greater than the general population [3-5]. In this case report, we highlight the case of a patient with rapidly depigmenting vitiligo and concurrent hypothyroidism who experienced significant repigmentation after initiating thyroid supplementation coupled with phototherapy.

Case Report

A 25 year old Caucasian female with skin phototype III and rapidly depigmenting generalized vitiligo presented to clinic with greater than 75% of body surface area involvement. Her family history was significant for vitiligo in her maternal great-grandmother and psoriasis in both her mother and brother. Other autoimmune conditions, including thyroid dysfunction in her maternal grandmother and mother and Type I DM in her maternal grandmother, were also noted at the initial encounter. Her review of systems was remarkable for fatigue and weight gain, but she denied any palpitations, cold intolerance, dry skin, menstrual irregularities, or muscle weakness. She was started on prednisone 20 mg daily and narrowband UVB phototherapy for treatment of vitiligo. Thyroid function labs were drawn to rule out potential thyroid dysfunction. At one month follow-up, despite no significant repigmentation, stability of disease was achieved and she was continued on phototherapy, with tapering of prednisone to every other day dosing. TSH levels drawn at the initial visit came back elevated at 7.59 μ IU/mL (normal 0.34-3.00 μ IU/mL) with a T4 in the low normal range at 0.83 ng/dL (normal 0.61-1.35 ng/dL). Thyroid replacement was started by her primary care physician as the patient was symptomatic. Shortly thereafter, the patient began to note repigmentation of affected areas. Her oral prednisone was switched to pulse dosing of dexamethasone and an oral antioxidant was added in addition to continuing phototherapy. Approximately 3

months after treatment was started, depigmented lesions had achieved 50-90% overall re-pigmentation in a predominantly follicular pattern (Figure 1). Now completing the fourth month of treatment, the patient continues to note improvement in pigmentation.



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Discussion

Hypothyroidism is the most common autoimmune disease associated with vitiligo [4]. This case report highlights the interplay between these two conditions, the importance of screening for autoimmune conditions, and the role concurrent treatment plays in achieving optimal repigmentation. A similar case was reported in the literature of an 18 year old female with vitiligo achieving repigmentation while on thyroid replacement, PUVA, oral corticosteroids, and an oral antioxidant [6]. The relationship between autoimmune hypothyroidism and vitiligo was investigated in a paper by Colucci et al. that looked at circulating auto-antibodies against thyroid hormones in vitiligo. These autoantibodies are more prevalent in people with vitiligo, which the authors suggest contributes to an inflammatory milieu, resulting in the formation of both autoimmune thyroid disease and vitiligo (Figure 2). The theory suggests that anti-thyroid hormone antibodies (Anti-TH Ab) cross react with tyrosinase causing inactivation or impairment

and eventual loss of pigment. A potential cross reactivity with acetylcholinesterase, leading to increased acetylcholine levels in the skin, and subsequent inhibition of melanin production may also play a role. Increased levels of ROS also contribute as they initiate a cycle where increased ROS present in people with vitiligo causes thyroid damage and Anti-TH Ab production, which then leads to exacerbation of vitiligo by impairment of melanogenesis [7]. This may be why treatment response improves once the inflammatory milieu is modulated with corticosteroids and phototherapy, and oral antioxidants are used to quench ROS (Figure 3). Currently, the role that thyroid hormone replacement plays in this process is unclear, but it is apparent that concurrent treatment of these two autoimmune conditions creates the optimal environment for repigmentation. However, additional studies are necessary to evaluate this mechanism further.

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