Case of Persistent Regrowth of Blond Hair in a Previously Brunette Alopecia Areata Totalis Patient

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Abstract

We present a case of a brunette, 64-year-old female with no previous history of alopecia areata who presented to our clinic with diffuse hair loss over the scalp. She was treated with triamcinolone acetonide intralesional injections and experienced hair re-growth of initially white hair that then partially re-pigmented to blond at the vertex. Two years following initiation of therapy, she continued to have blond hair growth on her scalp with no dark hair re-growth and no recurrence of alopecia areata.

Introduction

Alopecia areata (AA) is a fairly common autoimmune disorder of non-scarring hair loss. The disease commonly presents as hair loss from any hair-bearing area of the body. Following hair loss, it is not rare to see initial growth of depigmented or hypopigmented hair in areas of regrowth in the first anagen cycle. However, sustained and widespread hypopigmented hair regrowth in a patient with alopecia areata totalis is a rare phenomenon.

Case Report

A 64-year-old Caucasian, brunette female presented to our office complaining of two weeks of diffuse loss of hair from her scalp. The patient denied previous history of alopecia areata, autoimmune disease, recently beginning new medications, anemia, or a precipitating adverse event. The patient denied any history of dermatological diseases and had no family history of AA or autoimmune disease. She admitted to attempting treatment with OTC treatment regimens for her hair loss but was unable to recall specific details. Her past medical history was significant for hypertension and hypercholesterolemia, treated with atenolol and simvastatin, respectively.

Physical exam revealed diffuse thinning of hair over the entire scalp without scarring. No other body areas were affected. Over the next six months, her alopecia worsened to involve complete hair loss over her scalp (**Figure 1**).

Initial workup included a complete blood count (CBC), comprehensive metabolic panel (CMP), thyroid stimulating hormone (TSH) test and antinuclear antibody (ANA) test. All values were unremarkable, and the ANA was negative. The patient declined a biopsy.

A clinical diagnosis of alopecia areata was made. The patient was treated with 5.0 mg/mL intralesional triamcinolone injections that were repeated every four to six weeks for 24 months. She concurrently used OTC minoxidil 5% solution as well as B12 and biotin supplements. She reported no side effects of treatment.

During the course of treatment, she began to see steady scalp regrowth of white hair within six months. Following initial growth of completely depigmented hair, she began to see growth of blond hair. Two years after treatment was initiated, complete scalp-hair regrowth had occurred, with blond-colored hair on the scalp vertex. Visual inspection demonstrated no demarcation line of color change, but blond hair was observed down to the root of the hair in a patchy distribution (**Figure 2**). She denied application or use of chemical colorants or dyes.

Figure 1

Figure 2

Discussion

Alopecia areata is a fairly common autoimmune disease. It can affect any hair-bearing area but typically presents on the scalp in well-circumscribed, circular or ovoid patches.[1] It can present with total scalp hair loss (alopecia totalis), loss of all scalp and body hair (alopecia universalis), in an ophiasis pattern (loss of hair along the periphery of the occipital, parietal and temporal scalp), sisaipho pattern (loss of hair in the frontal parietotemporal scalp), patchy hair loss (reticular variant) and a diffuse thinning variant.[2] Often, "exclamation point hairs" can be seen in and around the margins of the hair loss. The distal ends of these hairs are thicker than the proximal ends, and they are a marker of active inflammation.[1]

A high percentage of patients experience remission of the disease and have hair re-growth. It is common to have initial hypopigmentation or de-pigmentation of hair re-growth during the first anagen phase. Most patients experience re-pigmentation to original hair color or even slight hypopigmentation of original hair color with subsequent growth.

Epidemiology

Alopecia areata is one of the most common autoimmune diseases, with a lifetime risk of 1.7 percent. AA affects both sexes equally. It is commonly encountered by dermatologists, representing from 0.7 percent to 3.8 percent of dermatological patient visits.[3]

As with many autoimmune diseases, there tends to be a higher predilection of occurrence in patients afflicted with other autoimmune disease. In particular, thyroid disease, including Grave's, Hashimoto's thyroiditis and simple goiter has a high disease association with AA, with a co-presence of 8 percent to 28 percent.[4] Vitiligo is also seen in a higher percentage of AA patients compared to the general population.[5] It should be noted that there is often no concurrent vitiligo in distinct areas affected by alopecia areata because melanocytes within the epidermis express different antigens than those expressed by melanocytes within the hair follicle.[6]

In patients with alopecia areata, there is also a high association with psychiatric morbidity, especially anxiety and depression. AA patients have a lifetime risk of 74 percent of developing one or more psychiatric illnesses.[7]

Etiology

Hair color is determined by the type and amount of melanin within the keratinocytes of the hair.

Table 1. Alopecia Areata Therapies

First-line Therapies	Second-line Therapies	Third-line Therapies
Intralesional corticosteroids	Sulfasalazine	Systemic corticosteroids
Topical corticosteroids	Photochemotherapy	Methotrexate
Minoxidil	Excimer laser	Cyclosporine
Anthralin	Fractional photothermolysis laser	Azathioprine
Topical immunotherapy		Biologics
Prostaglandin analogs		
Topical retinoids		
Capsaicin		

Melanocytes situated in close proximity to the hair bulb transfer melanosomes containing melanin to newly formed keratinocytes within the hair bulb. The amount, type and density of pigment found within the keratinocytes in the cortex of the hair determine the color and tone of the hair.[1] Pigmented hair in alopecia areata is targeted over depigmented hair, often with characteristic sparing of white and grey hair.[1,2]

The underlying etiology and pathophysiology are still unclear, but it is known that alopecia areata occurs due to an autoimmune assault to the hair follicle that results in hair loss.[8] Hair follicles under non-pathological conditions have immune privilege, meaning there is an environment around the hair follicle that protects it from the immune system. One of the first steps in the development of alopecia areata is loss of hair-follicle immune privilege. Some individuals are genetically predisposed to loss of hair-follicle immune privilege due to expression of specific HLA class II alleles.[3] Genetic studies have linked HLA-DQ3 to alopecia areata.[8] Once immune privilege is lost, inflammatory cells attack pigment-producing anagen hair bulbs, and autoantigens are produced through autoreactive T cells with a TH1 cytokine profile (both CD4+ and CD8+). That pigmented hairs are targeted in alopecia areata suggests that melanocytes or melanogenesis-associated proteins within melanocytes around the hair follicle are the target of these autoantigens.[9]

Decreased numbers of melanoblasts and abnormal melanogenesis have been demonstrated in areas of hair regrowth.[10] This promotes hypopigmented regrowth. It is possible that in patients with permanent hypopigmentation, such as our patient, a cytotoxic event may have occurred that either partially or fully destroyed the melanocytes responsible for hair color. Therefore, the continued hypopigmentation of hair in the patient in this case may be explained by a persistent decrease of pigment transfer by melanocytes produced by damaged melanoblasts to the hair-fiber keratinocytes. This may account for the re-pigmentation, though decreased, that occurred in a patchy distribution on the vertex of the scalp in this patient.

It is unlikely that an acute episode of the rapid, diffuse form of AA affecting only pigmented hairs is the underlying etiology of our patient's presentation. In the "overnight whitening" phenomenon, a patient loses all pigmented hairs rapidly, leaving behind only hairs that were already white or grey. Historical figures such as Mary, Queen of Scotts and Marie Antoinette are rumored to have experienced "overnight whitening."[11] This phenomenon only describes the underlying etiology of total scalp whitening. Our patient suffered alopecia areata totalis of the scalp and then grew back depigmented hair that eventually gained some pigmentation, resulting in blond hair.

Histopathology

Histologically, the acute phase of AA is characterized by an infiltrate of mononuclear T-cells (both CD8+ and CD4+) and eosinophils around the lower portion of anagen follicles. It has been described histopathologically as a "swarm of bees."[1,3]

Chronically, there is a decrease in the number of mononuclear cells with miniaturization of hair follicles within the superficial dermis. Melanocytes are found within the hair bulb in decreased numbers, and they contain a decreased amount of melanin.[12]

Differential Diagnosis

The differential diagnosis for alopecia areata includes other non-scarring alopecias such as androgenic alopecia, trichotillomania, telogen effluvium, loose anagen syndrome, secondary syphilis (consider RPR) and non-active cicatricial alopecia.[1]

Treatment/Management

Several therapeutic modalities have been utilized in the treatment of alopecia areata. To date, there is no cure or prevention method for AA, and current therapies are aimed at ceasing hair loss and inducing hair re-growth. Therapy should be tailored and adjusted to the patient's response. It is not uncommon for practitioners to utilize multiple therapies concurrently.

First-, second- and third-line therapies are listed in **Table 1**. Our patient responded well to intralesional corticosteroid injections, so we will discuss this treatment modality in detail.

The injection of intralesional corticosteroids, namely triamcinolone acetonide, is one of the most commonly used therapies for alopecia areata, resulting in hair regrowth in 64 percent to 97 percent of treated areas.[4] To date, this is considered a first-line therapy in adult AA patients with less than 50% scalp involvement.[10,14,15] The most commonly utilized concentration is 5 mg/ml, with no more than 3 mL injected into the scalp every four to six weeks to minimize side effects.[13] Common side effects are atrophy of tissue at injection sites, skin hypopigmentation at injection sites, and telangiectasias. Relapse rates vary based on the type of AA being treated. In limited alopecia areata, the reported relapse rate at three months following therapy is 29%, while the reported rate in alopecia totalis is 72%.[13]

Patients with AA often experience significant psychological and psychosocial impacts. Treatment should be aimed at alleviating these effects. As mentioned previously, AA patients tend to have high rates of psychological comorbidities such as anxiety and depression, and practitioners should be sure to screen for and address these issues.

Conclusion

Alopecia areata is a common autoimmune condition that causes non-scarring hair loss in any hair-bearing area. Most patients experience hair regrowth, and it is common for hair growth in the first anagen cycle to be hypopigmented or depigmented. Our patient demonstrated an unusual case of AA totalis in which previously dark-pigmented hair regrew as blond hair. The exact etiology of this rare occurrence remains unknown, but we speculate a loss of hair-follicle autoimmune privilege and autoantibody production against melanocytes may be responsible. This may have led to reduced numbers of melanoblasts, incomplete melanogenesis and partial destruction of the mechanism of melanin production and/or transfer of melanin to keratinocytes within the hair follicle.

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