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Case Presentation

Alopecia areata with white hair regrowth: case report and review of poliosis

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Abstract

Alopecia areata is thought to be a T-cell mediated and cytokine mediated autoimmune disease that results in non-scarring hair loss. Poliosis has been described as a localized depigmentation of hair caused by a deficiency of melanin in hair follicles. A 57-year-old man with a history of alopecia areata developed white hair regrowth in areas of previous hair loss. We retrospectively reviewed the medical literature using PubMed, searching: (1) alopecia areata and (2) poliosis. Poliosis may be associated with autoimmune diseases including alopecia areata, as described in our case. However, it is also reported in patients who have cutaneous lesions, genetic syndromes, infections, medication use, and trauma. Hair regrowth following alopecia areata may be associated with poliosis. We hypothesize that the incidence of poliosis in areas of previous alopecia areata-related hair loss may be greater than reflected in the published literature.

Keywords: alopecia, areata, poliosis

Introduction

Alopecia areata is a T-cell mediated and cytokine mediated autoimmune disease in which the loss of protection provided by immune privileged normal hair follicles causes non-scarring hair loss [1]. Poliosis describes a focal patch of white hair. Although most commonly referred to as a “white forelock” when located on the anterior scalp, poliosis can involve a patch of white hair anywhere on the body including the eyebrows, eyelashes, and beard [2]. We describe a man with alopecia areata who developed white hair regrowth in areas of previous hair loss.

Case synopsis

A 57-year-old man presented with recurrent alopecia areata. He had a past medical history of ulcerative colitis that was diagnosed in 1996 and controlled on mesalamine 4 grams rectally daily. His alopecia areata had been diagnosed more than a year ago; the 2 previous sites of hair loss demonstrated white hair regrowth (Figure 1).



Figure 1 (a and b). Distant (a) and closer (b) views of a 57-year-old man with two patches of white hair regrowth in areas of previous alopecia areata-related hair loss at the left occipital and left posterior scalp.

Cutaneous exam noted new hair loss at the left frontal scalp (1.5 x 1.5 cm) (Figure 2) and adjacent to a previous area of alopecia areata-related hair loss on the occipital scalp (3 x 2 cm). Lab results including complete blood count, chemistry panel, thyroid stimulating hormone, free thyroxine hormone, total triiodothyronine, anti-thyroglobulin antibody, antiperoxidase antibody, antiparietal cell antibody, and vitamin B12 were negative or at normal levels.



Figure 2 (a and b). Distant (a) and closer (b) views of new areas of alopecia areata related hair loss at the left frontal scalp (1.5 x 1.5 cm).

The patient was given intralesional injections of triamcinolone (2.5 milligrams/milliliters x 0.8 milliliters) into multiple sites within the two areas of new hair loss during the initial visit. He was subsequently seen for follow-up in 4 weeks and was given additional intralesional injections of triamcinolone (2.5 milligrams/milliliters x 0.8 milliliters) at the same sites of hair loss. After the patient's third cycle of intralesional triamcinolone 1 month later, his occipital and left frontal areas of alopecia had nearly resolved, with white hair regrowth (Figure 3).



Figure 3 (a and b). Posterior (a) and side (b) views of the occipital and left frontal scalp showing white hair regrowth after 3 intralesional injections of triamcinolone (2.5 milligrams/milliliters x 0.8 milliliters).

Discussion

Alopecia areata occurs as a patchy, confluent or diffuse pattern of non-scarring hair loss [1, 3]. This disease has been suggested to cause dystrophic anagen hair follicles, increased frequency of telogen state follicles or both [4]. Immune cells involved in the pathogenesis of alopecia include both CD4+ lymphocytes and CD8+ lymphocytes. Cytokines such as TNF-alpha, interleukin, and IFN-gamma may also be inhibited [3].

Alopecia areata is associated with other autoimmune diseases including allergic rhinitis, anemia, bronchial asthma, diabetes, hypertension, systemic lupus erythematosus, thyroid disorders, and ulcerative colitis [1]. There is also a genetic susceptibility to the development of alopecia areata involving specific alleles of the HLA region [4]. Environmental factors, including exposure to proinflammatory agents and other modulators such as stress and diet may also trigger symptoms. In addition, medication-associated alopecia areata has also been observed in patients receiving antineoplastics, antitumor necrosis factor drugs, antiviral therapies, immunosuppressants, and psychiatric drugs [3].

The diagnosis of alopecia areata can be established based upon clinical examination. Dermoscopy evaluation or a skin biopsy of the affected scalp can also aid in confirming the diagnosis [5]. Histologically, there is a perifollicular lymphocytic infiltrate [6].

Current treatment for alopecia areata depends on the age of the patient and the extent of scalp involvement. Therapies may include anthralin, minoxidil, psoralen and UV-A therapy (PUVA), and topical or systemic corticosteroids [5-8]. Hair regrowth typically recurs once the inflammatory response is inhibited.

Poliosis is a localized patch of white hair in a group of hair follicles. It can involve any hairy area on the body including the scalp, eyebrows, eyelashes, or beard [2]. Poliosis may occur owing to an inherited defect in melanization, secondary to an autoimmune destruction of the pigment cells, or as a result of hair follicle damage. In addition, a defect in melanin transfer caused by an immune response directed at the cortical keratinocytes may contribute to the development of poliosis [9].

Poliosis occurs in the setting of several genodermatoses (Table 1) [10-18]. In addition, poliosis has also been associated with other autoimmune conditions, such as alopecia areata. Acquired insults including medications, neoplastic lesions, and inflammatory conditions can also result in poliosis (Table 2) [12,19-33]. The histopathology of poliosis shows a decrease or absence of either melanin, melanocytes, or both in the hair bulbs of the affected hair follicles [34].

Table 1. Genodermatoses associated with poliosis

Genodermatoses	References
Alezzandrini syndrome	10
Marfan syndrome	11
Neurofibromatosis type 1	12
Piebaldism	13
Prolidase deficiency	14
Tietze syndrome	15
Tuberous sclerosis	16
Vogt-Koyanagi-Harada syndrome	17
Waardenburg syndrome	18

Table 2. Non-genodermatoses associated with poliosis

Autoimmune Diseases	References
Alopecia areata	19
Sarcoidosis	20
Vitiligo	21
Medications	
Acitretin	22
Cetuximab	23
Chloramphenicol	24
Chloroquine	25
Econazole	26
Imiquimod	27
Prostaglandin analogues	28
Other Causes	
Blepharitis	29
Melanocytic lesions	30
Neurofibroma	12
Postherpetic	31
Trauma	32
Trigeminal autonomic cephalalgia	33

The treatment of poliosis is directed toward its etiology. In children, in whom several hereditary syndromes are the instigating factor, the white hair regrowth may be more resistant to treatment. However, in adults, in whom poliosis may be secondary to benign or malignant melanocytic lesions, management should be directed toward treating the underlying lesions. Additionally, medications, viral infections, or trauma-induced poliosis should prompt discontinuing the causative drug or treating the poliosis-associated condition.

When poliosis is associated with inflammatory conditions such as alopecia areata, sarcoidosis, vitiligo, and Vogt-Koyanagi-Harada syndrome, the treatment is focused on decreasing the autoimmune or inflammatory insult. Recent studies have reported the role of interleukin-1-beta and alpha-melanocyte stimulating hormone (MSH)-related tripeptide in stimulating human hair pigmentation under pro-inflammatory conditions [35]. These new targets may be valuable for patients with recalcitrant poliosis secondary to alopecia areata [35].

Repigmentation of poliosis has also been reported after epithelial grafting in areas of vitiligo [36]. It has also been reported that patients with graying hair have regained hair pigmentation after radiation therapy for head cancer or resolution of certain inflammatory events such as erythrodermic eczema and erosive candidiasis of the scalp [37,38]. Using ultraviolet light to mature amelanotic melanocytes in the outer root sheath has also been reported to repigment hair [39,40]. Repigmentation in these individuals has been suggested to be related to repopulation of the hair bulb melanogenic zone [41].

Poliosis may be part of the clinical presentation of regrowing hairs in alopecia areata [2]. Alopecia areata associated with white hair regrowth has been suggested to be a result of an inflammatory or autoimmune mechanism of melanocytes in the hair cells targeted in alopecia areata [42]. Hair bulb melanocytes are initially targeted in acute alopecia areata. Subsequently, poliosis develops in vitiliginous patches because of the loss of melanocytes from hair bulbs after the primary immunologic destruction of epidermal melanocytes [43].

Previously reported patients with white hair regrowth associated with alopecia areata include a series of patients with alopecia areata and ocular and testicular abnormalities in addition to white hair regrowth [44]. Mosaic hair color changes have also been described in two patients with alopecia areata [45]. Additionally, a patient with rapid whitening of scalp hair and diffuse hair loss has also been observed [46]. In summary, although patients with alopecia areata-associated poliosis have been recorded, we have noticed that the published literature is short in reporting such individuals.

Conclusion

Alopecia areata results from an immunologic cause of hair loss and has a multifactorial etiology. Poliosis describes a localized depigmentation of hair resulting from a deficiency of melanin in hair follicles [30]. Although not fully understood, postulated pathogenesis of poliosis suggest an inflammatory destruction of the melanocytes in the hair follicle, apoptosis of the follicular melanocytes, or a targeted autoimmune response [34,47]. The pathogenesis of alopecia areata-associated poliosis suggests that the delay of repigmentation could be attributed to the damaged hair pigmentary unit, which may have a reduced number of available melanocytes in the hair follicle. Our patient provides an example of poliosis associated with alopecia areata. We hypothesize that alopecia areata-associated poliosis may be greater than reflected in the published literature.

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