A Case Report on Alopecia Areata Incognita in a 19 Year Old Filipino Female*

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ABSTRACT

Introduction: Alopecia areata incognita is a rare form of alopecia areata which was first reported in 1987. The prevalence of this disease is unknown but it is more common in women. The usual presentation of alopecia areata incognita is acute, diffuse hair thinning. In most cases, it lacks the typical alopetic patches seen in alopecia areata. It may resemble telogen effluvium and androgenetic alopecia. The prognosis of this disease is favorable and recovery is rapid and spontaneous. **Case:** A 19-year-old Filipino female presents with a two-month history of alopecia areata incognita. She initially had a solitary round patch of hair loss on the scalp with proximally tapered hair, rapidly evolving into diffuse hair thinning. CBC, TFTS, FBS, HBA1c, ANA and VDRL were unremarkable. Histopathology demonstrated dense peribulbar lymphocytic infiltrate, miniaturized hair and increased catagen hair consistent with alopecia areata. There was gradual hair growth after treatment with minoxidil 5% lotion and topical betamethasone dipropionate 0.05% lotion.

Keywords: alopecia areata incognita

INTRODUCTION

Alopecia areata incognita (AAI) is a rare type of non-scarring alopecia. The prevalence is unknown and studies show a female predominance. It is more common in patients aged 20 to 40. In AAI, there is abrupt hair loss presenting as rapid hair thinning within weeks to months.[1] In most cases, it lacks the classic presentation of alopecia areata which appears as round to oval bald smooth patches on the scalp. It may be clinically mistaken for telogen effluvium or androgenetic alopecia (Table 1). Since the different diagnoses have different courses and prognosis, it important to confirm the diagnosis thru a scalp biopsy. AAI subtype of alopecia areata has a shorter clinical course followed by rapid recovery. It has a more favorable prognosis than alopecia totalis, universalis and ophiasis, telogen effluvium and androgenetic alopecia.[2]

Table 1. Differential diagnosis of non-scarring alopecia

Non-scarring alopecia	
Diffuse loss	**Patchy loss**
Telogen effluvium	Alopecia, localized type
Alopecia areata incognita	Tinea capitis
Androgenetic alopecia	Trichotillomania
Systemic disease (Thyroid, Iron deficiency, SLE, Dermatomyositis)	Syphilis

CASE REPORT

A 19-year-old female presented with a two month history of diffuse hair loss. History started two months prior to consultation when she noticed a circular patch of hair loss on the vertex of the scalp. A week later, irregular diffuse hair loss was noted on the vertex and parietal areas of the scalp which prompted consult. On physical examination, there was diffuse hair thinning on the scalp with some ill-defined circular patches with proximally tapered hairs at the periphery (Figure 1). The skin mucosa and nails were normal. The hair pull test was positive. The patient had a good general health and she had no history of psychological stress,

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sudden weight loss, pregnancy or intake of any medication.

Histopathology of the scalp biopsy showed decreased follicular density with increased catagen hair and miniaturized hair. There was increased peribulbar infiltrate of lymphocytes and histiocytes (Figure 2a, 2b, 3). The microscopic diagnosis was consistent with a non-scarring alopecia areata. Laboratory examinations were as follows: CBC was normal; thyroid stimulating hormone was slightly decreased (4.69%) with normal thyroid function (FT3 and FT4) for which no medical intervention was given; FBS was normal; HBA1c was slightly elevated at 6.21%, for which patient was advised diet modification; ANA and VDRL were negative. The laboratory examination results were unremarkable. She was treated with minoxidil 5% lotion and betamethasone dipropionate 0.05% lotion. After 2 months, there was a significant response to therapy with remarkable hair growth (Figure 4).

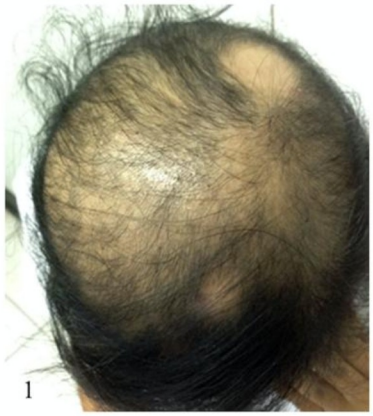

Figure 1. Diffuse hair loss on the scalp with some ill-defined circular patches with proximally tapered hair or "exclamation mark hairs" at the periphery

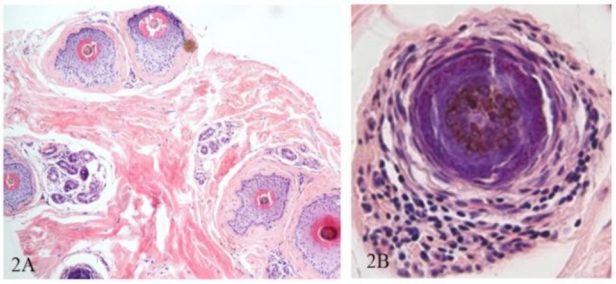

Figure 2. Scalp biopsy specimen from the patient demonstrating (A) non-scarring alopecia with decreased follicular units and increased catagen and miniaturized hairs and (B) heavy lymphocytic infiltrates surrounding the hair bulb.

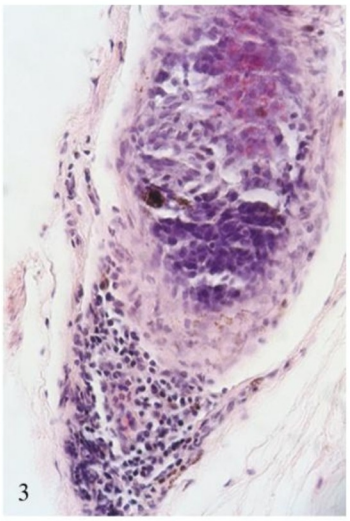

Figure 3. Classic peribulbar "swarm of bees" inflammation in alopecia areata

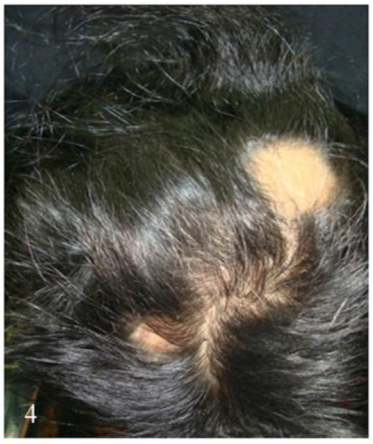

Figure 4. Hair growth after 2-month use of minoxidil 5% lotion and betamethasone dipropionate 0.05% lotion.

DISCUSSION

From 1987 until 2011, there have only been 112 reported cases of AAI. Molina et al reported a strong female predominance comprising 86.6% of the 112 cases.[3] AAI is characterized by an abrupt, diffuse and severe hair thinning. It differs from the classic alopecia areata because in AAI the borders are indistinct and the alopetic patches tend to merge.[1,4,5]

The pathophysiology of AAI is controversial. Rebora et al theorized that AAI occurs when alopecia areata affects patients with a high percentage of telogen hair in the scalp. Early anagen hair is scarce and its high mitotic rate makes it

vulnerable to noxious events resulting in diffuse hair loss rather than patches.[4]

Inui at el examined patients with acute, diffuse and total alopecia and they noted that exclamation mark hairs and broken hairs are specific diagnostic markers for alopecia areata. Sudden follicular miniaturization causes the short broken off hairs and decrease in hair shaft diameter proximally. [6]

Histopathologic findings are similar to the classical forms of alopecia areata despite different clinical presentation. The most consistent finding in AAI biopsy is an inflammatory infiltrate around the terminal hair bulb.[3] A reversal in the anagen-telogen and telogen-vellus ratio is observed.[5]

Treatment includes topical and intralesional corticosteroids, minoxidil, topical immunotherapy, and topical retinoid.[7] In a study by Iorizzo on 50 patients with AAI, treatment with clobetasol propionate 0.05% cream every night for 6 months and intramuscular triamcinolone acetonide 40 mg once a month for two months showed complete hair regrowth in all patients after six months with regrowth maintained after steroid withdrawal.[8] Other treatment options include anthralin, PUVA turban[8], bexarotene[9], topical immunotherapy with diphenylcyclopropenone and squaric acid dibutylester[10]. A properly treated case of AAI has a good prognosis.

In our patient, she initially had an alopetic patch which rapidly progressed to diffuse hair thinning. Despite having a high index of suspicion that it is a case of AAI, it is imperative that other diagnoses which present like AAI are ruled out. Telogen effluvium and androgenetic alopecia also present with diffuse hair thinning like AAI, however the former have different prognosis and management. With inspection of the hair characteristics and skin biopsy, we were able to make an accurate diagnosis. Our patient had proximally tapered hairs which is consistent with alopecia areata. In addition, histopathology of the scalp biopsy showed increased peribulbar infiltrate of lymphocytes and histiocytes, which strengthens our diagnosis. Her response to treatment with topical steroid and minoxidil solution was very good.

CONCLUSION

AAI, a rare type of alopecia areata, clinically resembles other alopetic conditions. In cases with acute and diffuse hair loss, histopathology is helpful in establishing the correct diagnosis. Treatment includes topical and intralesional steroids and minoxidil. Prognosis of AAI is good compared to other types of alopecia, such as alopecia totalis, universalis and ophiasis.

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