

Alopecia Areata Incognita: A Case Report

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INTRODUCTION

Alopecia areata incognita, also known as diffuse alopecia areata, is an uncommon subtype of alopecia areata primarily observed in young females. Alopecia Areata Incognita is characterized by sudden and intense hair loss, differing from the distinctive bald patches commonly associated with Alopecia Areata. Importantly, AAI generally carries a more favorable prognosis compared to alopecia areata totalis, universalis, and ophiasic areata¹.

CASE REPORT

A 68-year-old female patient, resident of Mexico City, with a history of sympathectomy performed 20 years ago and a mitral valve replacement, with four cardiac surgeries, presents with a dermatosis located on the scalp, affecting the frontal, temporal, parietal, and occipital regions, characterized by decreased hair density and thinning over a 10-year period (Images 1 and 2). Trichoscopy reveals yellow dots, pigtail hairs, dystrophic hairs, and short vellus hairs throughout the scalp (Images 3 and 4). Histopathological findings were consistent with alopecia areata incognita.



Image 1



Image 2

Images 1 and 2: Decreased density and thinning of hair

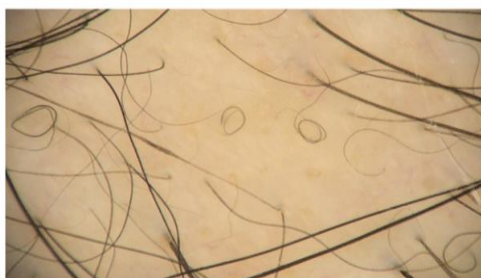


Image 3



Image 4

Images 3 and 4: Trichoscopy shows yellow dots, pigtail hairs, dystrophic hairs and short vellus hairs throughout the scalp

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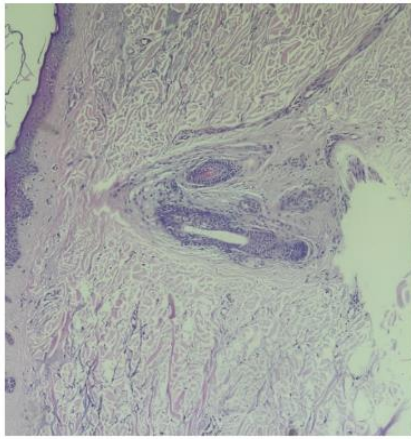


Image 5

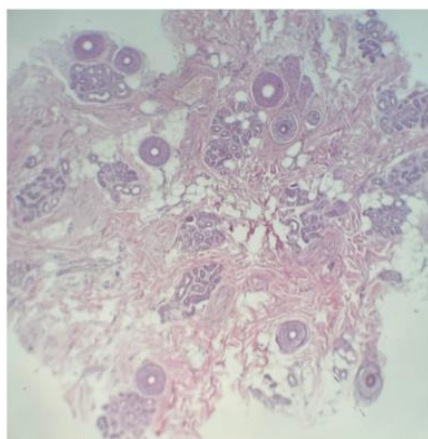


Image 6

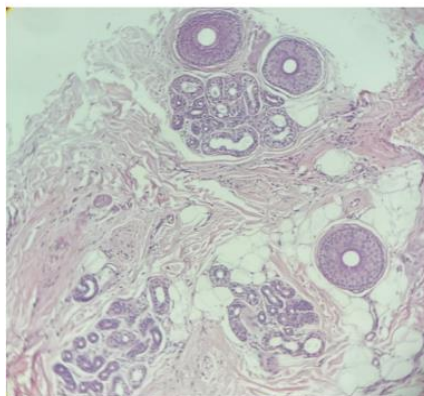


Image 7

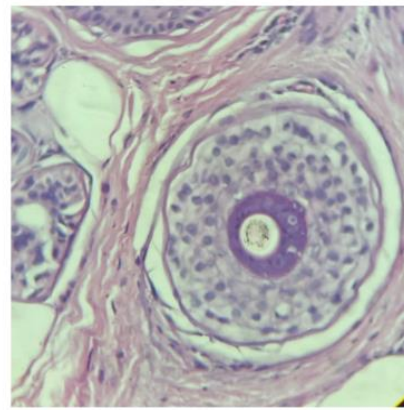


Image 8

Images 5 to 8: Five hair follicles are observed, of which four are terminal and one is vellus, all in the catagen-telogen phase. There is a hair follicle with a discrete perifollicular inflammatory infiltrate by lymphocytes, as well as a follicular trail (Images 6 and 7).

DISCUSSION

Alopecia areata incognita (AAI), initially documented by Rebora in 1987, a subtype of alopecia areata, clinically resembling telogen effluvium², characterized by acute and intense hair loss without bald patches that are typical of alopecia areata (AA)^{1,3}.

AA affects up to 2% of the global population and can occur at any age. However, its prevalence is higher in children (1.92%) compared to adults (1.47%). Additionally, a higher incidence has been reported in females than in males, particularly in those with late-onset disease, which is defined as onset after the age of 50⁴.

Dysregulation of several immune-mediated pathways has been implicated in the development of AA in susceptible patients with multigenetic predisposition and environmental triggers. Normally, hair follicles have immune privilege that protects against autoimmunity, but in AA patients, this privilege is disrupted, leading to an immune attack on the hair follicle, targeting autoantigens related to melanocytes and keratinocytes. Th1 and Th17 cytokines are involved in this immune response and correlate with disease activity. Genetic predisposition is supported by family history and genome-wide association studies identifying polymorphisms, notably

in PTPN22. Potential triggers include viral infections and various vaccines⁴.

Recent findings indicate that trichoscopy can enhance diagnostic accuracy in hair disorders. In cases of typical alopecia areata, trichoscopy allows for the assessment of disease activity by identifying dystrophic hairs, exclamation point hairs, and cadaverized hairs. The distinctive presence of yellow dots within the follicular ostia of both empty and hair-bearing follicles is particularly valuable for diagnosis².

AAI is incredibly difficult to diagnose⁵, and may be erroneously diagnosed as androgenic alopecia, requiring scalp biopsy for definitive confirmation¹. Histopathological characteristics of AAI differ clinically from other types of alopecia areata but share similarities with the classical forms of the disease, exhibiting variations based on the stage of the condition. In acute AAI scalp biopsies, a consistent finding is an inflammatory infiltrate surrounding the terminal hair bulb. This infiltrate decreases as the condition becomes chronic and becomes more concentrated around miniaturized follicles. Additionally, a reversal in the anagen-telogen and terminal-vellus ratios is consistently observed, providing crucial evidence for diagnosing long-standing cases. Although follicular density remains preserved during the acute and subacute stages, it may decline over time⁶.

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Treatment options for AA vary widely, from non-medical interventions to medical treatments. Treatment is recommended when spontaneous resolution is unlikely, or if hair loss significantly impacts the patient's life. Effective treatments include topical and intralesional steroids, topical immunotherapies, JAK inhibitors, and systemic corticosteroids. Other less frequently used treatments with variable success include topical calcineurin inhibitors, latanoprost, bimatoprost, cryotherapy, methyl-aminolevulinic acid photodynamic therapy, 308-nm excimer laser, pulsed infrared diode laser, and antihistamines. Choosing the right treatment depends on disease severity, patient-perceived severity, distress, and social impact. Patient involvement is crucial in treatment decisions⁷.

Severity at first consultation is a crucial prognostic factor, factors associated with progression to extensive disease included younger age of onset, concurrent autoimmune or atopic diseases, atopic dermatitis, and thyroid disease⁴. AAI has a favorable prognosis compared with alopecia aerata totalis, universalis and ophiasis alopecia areata³.

CONCLUSION

Alopecia areata incognita (AAI) presents unique diagnostic challenges due to its resemblance to other hair loss conditions, necessitating careful assessment through scalp biopsies and trichoscopy. Understanding the immune and genetic factors involved in AA can aid in better diagnosis and treatment. Despite various treatment options, the involvement of patients in their treatment decisions is crucial, and the initial severity of the condition significantly influences the prognosis. AAI generally has a better prognosis compared to more extensive forms of alopecia areata. In our patient's case, we observed numerous typical signs, including yellow dots, pigtail hairs, dystrophic hairs, and short vellus hairs across the entire scalp. These findings were confirmed through histopathological examination.

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