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CASE REPORT

Alopecia Universalis as the First Clue to Sjögren's Disease: A Case of Complete Reversible Alopecia

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Abstract

Alopecia areata (AA) is an autoimmune disorder characterized by non-scarring hair loss and a highly variable clinical course. Increasing evidence suggests an association between AA and systemic autoimmune diseases, although its occurrence in Sjögren's disease (SjD) remains uncommon and poorly characterized.

We report the case of a 36-year-old woman who presented with progressive generalized alopecia and sicca symptoms with minor salivary gland biopsy that confirmed SjD. Treatment with hydroxychloroquine and a low dose of systemic corticosteroids resulted in complete hair regrowth.

This case highlights the importance of considering underlying systemic autoimmune disease in patients with severe or atypical alopecia and demonstrates that appropriate immunomodulatory therapy can lead to full reversibility of hair loss.

Keywords: alopecia areata; alopecia universalis; Sjögren's disease; autoimmune diseases; hydroxychloroquine.

Introduction

Alopecia areata (AA) is an autoimmune disorder characterized by perifollicular and intrafollicular T-cell infiltration, presenting with sharply demarcated, non-scarring patches of hair loss [1]. Alopecia areata affects almost 2% of the population at some point in their lifetime [2].

The clinical expression of AA is highly variable, and its course is often unpredictable.

Severe manifestations include alopecia totalis (AT), characterised by complete scalp hair loss, and alopecia universalis (AU), defined by the absence of hair on the scalp and body [3]. Alopecia ophiasis (AO) is an additional subtype that is often difficult to treat [4]. AA is considered acute when spontaneous regrowth occurs within 12 months, whereas persistence beyond 12 months defines chronic disease [3].

The diagnosis of AA is challenging as it depends on clinical presentation and the exclusion of other causes of hair loss. The absence of a specific biomarker for AA further complicates both diagnosis and disease monitoring. Although scalp biopsies and dermoscopy can provide valuable diagnostic information, they are invasive procedures and are not routinely used in clinical practice [5].

The pathogenesis of AA is not fully understood, but evidence strongly supports an autoimmune basis with genetic susceptibility. Environmental and stress-related epigenetic factors may contribute to disease onset [6]. The pathophysiology involves disruption of the hair follicle's immune privilege, activation and infiltration of autoreactive CD8+ T lymphocytes, and the development of autoantibodies targeting follicular antigens [7].

Recent evidence indicates that patients with AA have a clinically meaningful increase in both atopic and autoimmune comorbidities. AA has been associated with multiple autoimmune diseases, including autoimmune thyroid disease, vitiligo, rheumatoid arthritis, systemic lupus erythematosus and other autoimmune diseases [8].

Although AA may show spontaneous remission, regrowth is often slow and can take months to years. Early onset of disease, family history, extensive scalp involvement, ophiasis pattern, nail changes, atopy, and associated autoimmune disorders are recognized predictors of poorer treatment response [9].

Treatment remains challenging, as no single therapeutic option has demonstrated consistent superiority, and responses vary widely among patients. Multiple treatments have been proposed for

AA, yet current guidelines do not clearly identify a superior therapeutic option [4]. Traditional medical therapies include the use of topical or systemic glucocorticosteroids, topical minoxidil, and light therapy. Other, less frequently employed therapeutic options include ultraviolet A phototherapy or excimer laser treatment, as well as various systemic immunomodulatory agents [9].

The development of immunotherapeutic agents such as JAK inhibitors is promising for AA, but current evidence is still limited, relying mostly on small studies and case reports [5].

These factors highlight the importance of individualized therapeutic strategies and the continued search for reliable treatment options in alopecia areata.

Case presentation

A 36-year-old female, with a history of pulmonary tuberculosis treated in 2020, presented to the rheumatology department in August 2024 with xerostomia, xerophthalmia, dry cough, and generalized alopecia. Sicca symptoms had emerged in March 2024. According to the medical history, alopecia initially appeared in 2022 following prolonged ultraviolet exposure during a seaside vacation, despite the patient reporting adequate photoprotective measures (Figure 1).

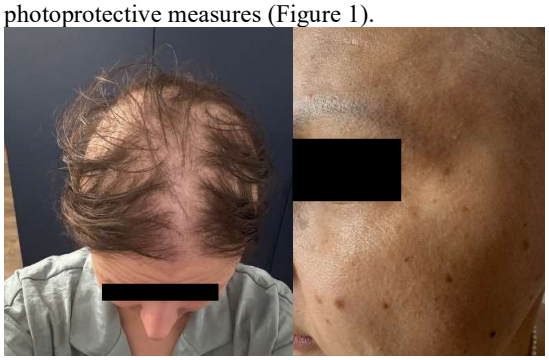

Figure 1. *Patchy alopecia involving the scalp and eyebrows (2022)*

The hair loss subsequently progressed to a diffuse pattern and failed to improve with topical corticosteroid or minoxidil therapy.

A skin biopsy performed in April 2022 demonstrated minimal histopathological changes compatible with chronic or tumid cutaneous lupus. Biological investigations at that time revealed leukopenia, positive ANA, positive anti-Ro antibodies, negative anti-La, low complement C3, negative anti-double-stranded DNA and anti-Sm antibodies, normal TSH levels, and no evidence of inflammatory syndrome. The patient remained without rheumatologic follow-up until August 2024.

At the time of the examination, the patient was in good general condition, without arthralgias or clinical signs of arthritis, and continued to report sicca symptoms, dry cough, vaginal dryness, and diffuse alopecia. No cutaneous lesions were observed. Laboratory tests showed normal complete blood count, normal hepatic and renal function, elevated IgG levels, and no complement consumption. Rheumatoid factor was negative, and there was no evidence of inflammatory syndrome. Immunological testing revealed ANA at 1:160 (negative threshold 1:80), negative anti-double-stranded DNA and anti-Sm antibodies, and positive anti-Ro antibodies. The Schirmer test was positive.

A dermatology consultation confirmed the diagnosis of alopecia universalis.

A minor salivary gland biopsy showed lymphocytic sialadenitis with a focus score >1. (Figure 2).

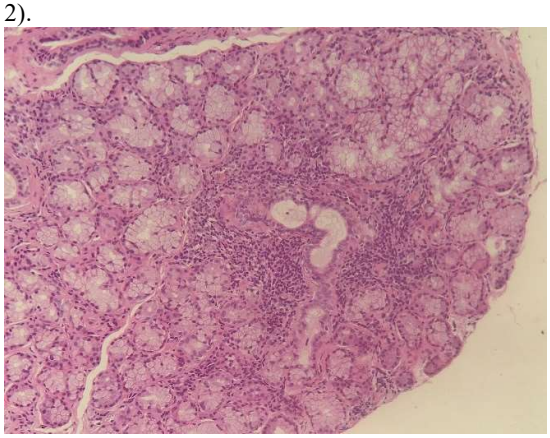

Figure 2. *Salivary gland showing marked inflammatory infiltrate consisting of aggregates of small lymphocytes focus >1*

The diagnosis of Sjögren's disease was established. The alopecia was interpreted, according to the dermatology evaluation, as autoimmune-associated alopecia. Treatment was initiated with hydroxychloroquine 200 mg/day and methylprednisolone 16 mg/day, with a subsequent gradual tapering of the corticosteroid dose.

The clinical and laboratory evolution was favorable under treatment during periodic follow-up visits. At the most recent evaluation in October 2025, alopecia had completely resolved. (Figure 3).

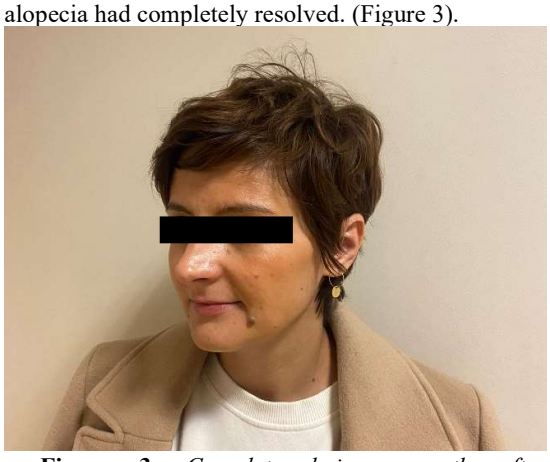

Figure 3. *Complete hair regrowth after immunomodulatory treatment*

Laboratory findings showed no evidence of active disease, with normal complete blood count, negative rheumatoid factor, and no complement consumption.

In view of the favorable clinical course and complete remission of alopecia, continuation of hydroxychloroquine therapy was recommended.

Discussions

This case highlights the complex relationship between alopecia and systemic autoimmune diseases, particularly SjD. Although alopecia is not classically recognized as a hallmark of SjD, several studies indicate that hair loss may occur more frequently in autoimmune diseases than previously appreciated, with most available data originating from case reports and small series [10]. The coexistence of alopecia AU and SjD in our patient underscores the potential overlap between follicular autoimmunity and systemic immune dysregulation.

From a pathophysiological perspective, AA and AU result from loss of the immune privilege of the hair follicle, recruitment of autoreactive $CD8^+$ T lymphocytes, and interferon-γ-driven JAK-STAT signaling [7,11]. Systemic autoimmune activation characteristic of SjD- marked by B-cell hyperactivity, interferon signature, and autoreactive T-cell infiltration- may facilitate or exacerbate these follicular autoimmune mechanisms [12]. This pathogenic overlap may explain the occurrence of severe hair loss in selected patients with SjD.

The diagnostic evaluation was challenging, particularly given the initial biopsy findings suggestive of chronic cutaneous lupus, reflecting the well-recognized clinical and histopathological overlap between lupus-specific alopecias and alopecia areata, which can closely mimic discoid lupus erythematosus and complicate the differential diagnosis [13]. This case reinforces the importance of a multidisciplinary approach when evaluating patients with severe, diffuse, or treatment-refractory alopecia. Identifying an underlying autoimmune disease is crucial, especially when hair loss presents atypically or progresses rapidly.

The complete regrowth observed after treatment with hydroxychloroquine and systemic corticosteroids aligns with previous reports describing improvement of autoimmune-associated alopecia following systemic immunomodulatory therapy [14]. Although emerging targeted treatments such as JAK inhibitors have shown promise in AA and AU, current evidence remains limited and derived largely from small studies [15]. Our case illustrates that traditional immunomodulators can be highly effective when alopecia is part of a broader systemic autoimmune process such as SjD.

Overall, this case contributes to the limited but growing body of literature describing AU associated with SjD and highlights the need for further investigation into the prevalence, mechanisms, and optimal management of alopecia in systemic autoimmune diseases.

Conclusions

Alopecia areata may represent an uncommon but clinically significant manifestation of SjD. This case underscores the importance of evaluating patients with severe or atypical alopecia for underlying systemic autoimmune disorders. Effective treatment of the autoimmune disease with systemic immunomodulatory therapy can result in complete hair regrowth. Multidisciplinary collaboration and early recognition are essential for optimizing patient outcomes. Further studies are needed to clarify the incidence and pathophysiology of alopecia in SjD and to establish evidence-based therapeutic strategies.

Conflicts of Interest: The authors declare no conflicts of interest.

Informed Consent: Written informed consent was obtained from the patient for publication of this case report and accompanying images. The identity of the patient has been kept anonymous in accordance with ethical standards.

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