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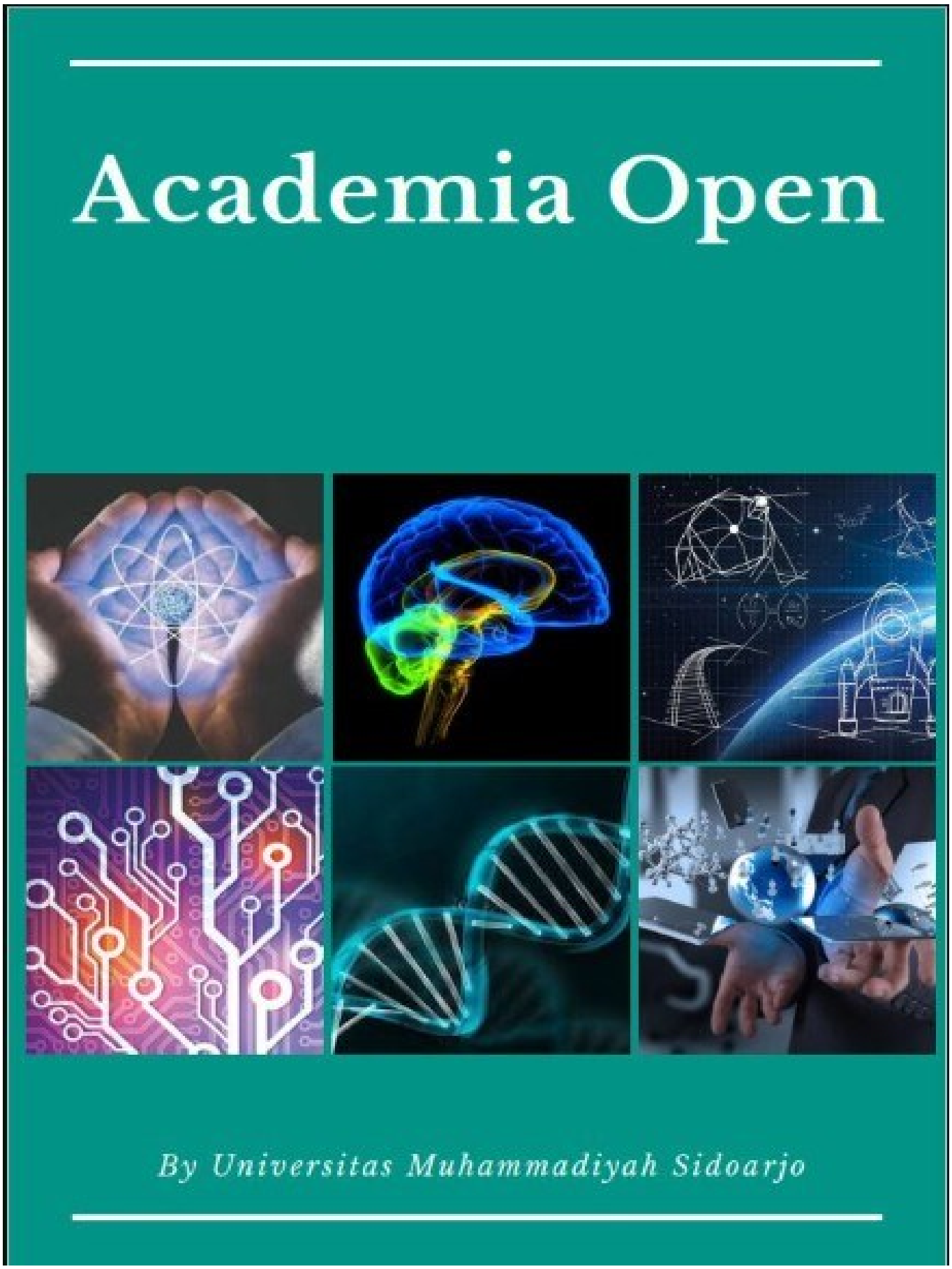


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# Table Of Contents

**Academia Open**

Vol. 11 No. 2 (2026): December
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# Early Hair Regrowth With Adjunctive Secretome Therapy in Severe Patchy Alopecia Areata: Pertumbuhan Rambut Dini dengan Secretome Adjuvan pada Alopecia Areata Berat

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## Abstract

**General Background:** Alopecia areata is a non-scarring immune-mediated hair loss disorder with an unpredictable course and frequent relapse. **Specific Background:** Corticosteroids remain standard treatment for active disease, but responses may be inconsistent when scalp involvement is extensive, while clinical evidence for mesenchymal stem cell-derived secretome remains limited.**Knowledge Gap:** Reports combining MSC-derived secretome with standard corticosteroid therapy in severe patchy alopecia areata while objectively tracking Severity of Alopecia Tool scores remain scarce. **Aims:** This case report described the early clinical course of a 23-year-old man with severe patchy alopecia areata and an estimated baseline SALT score of 73 who received intralesional triamcinolone, oral methylprednisolone, topical mometasone, and adjunctive secretome. **Results:** After one month, hair density increased with visible vellus and early terminal hairs, while the estimated SALT score decreased from 73 to 55, representing approximately 25% less scalp involvement. No new patches, local complications, or reported systemic adverse effects were observed. **Novelty:** The case provides objectively tracked early SALT changes during a corticosteroid-based multimodal regimen incorporating cell-freesecretome. **Implications:** The findings warrant controlled prospective studies with standardized preparations and longer follow-up because the concurrent treatments prevent attribution of the observed response to secretome alone.

**Highlights:**

- Estimated scalp-loss scoring declined from 73 to 55 within one month.
- Vellus and terminal hairs became visible across previously bald scalp regions.
- No treatment-related local or systemic adverse effects were observed during follow-up.

**Keywords:** Alopecia Areata, Secretome, Intralesional Triamcinolone, Corticosteroids, Regenerative Medicine

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## Introduction

Alopecia areata is a chronic, immune-mediated condition causing non-scarring hair loss on the scalp and, in some patients, other hair-bearing sites. The disease arises from a breakdown of the immune privilege that normally protects anagen-phase follicles. Cytotoxic CD8+ NKG2D+ T cells appear central to this process, acting through interferon-γ (IFN-γ), interleukin-15 (IL-15), and JAK-STAT signaling. The resulting perifollicular inflammation disrupts the hair cycle and can force follicles out of anagen prematurely.[1] [2] [3] [4]

Presentation ranges from a few isolated patches to total scalp involvement (alopecia totalis) or loss of all scalp and body hair (alopecia universalis). The clinical course is notoriously unpredictable: some patients regrow hair spontaneously, while others face chronic or relapsing disease. Factors linked to a worse prognosis include more widespread scalp involvement, longer disease duration, younger age at onset, nail changes, and coexisting autoimmune conditions.[1] [3] [5]

Treatment choice depends on how active and extensive the disease is, along with individual patient factors, prior treatment history, and what options are realistically available. For localized patchy disease, intralesional corticosteroids are the usual first choice. Systemic corticosteroids may be appropriate for select patients with widespread, fast-moving, or highly active disease, and potent topical steroids are often added as a local adjunct. Even so, relapse and partial response remain persistent challenges with these approaches.[5] [6]

MSC-derived secretome is a cell-free product composed of the soluble factors and extracellular vesicles that these cells secrete. Preclinical work suggests it can modulate inflammation, angiogenesis, and tissue repair through pathways relevant to hair follicles, but these laboratory findings have not yet translated into demonstrated clinical benefit in AA, and no standardized secretome formulation, dose, delivery route, or treatment schedule currently exists.[7] [8] [9] [10] Here I document the early clinical course of a patient with severe patchy AA treated with a corticosteroid-based multimodal regimen to which secretome was added as an adjunct. Reports specifically evaluating MSC-derived secretome alongside standard corticosteroid therapy in severe patchy AA, with an objectively tracked change in SALT score, are still scarce; much of the existing literature is mechanistic or preclinical, or describes cellular MSC administration rather than a cell-free secretome product.[11] This report adds to that limited descriptive literature without claiming priority as the first such case.

## Case Presentation

A man, 23 years old, came to the dermatology clinic because of scalp hair loss that had been slowly enlarging over the previous 3-6 months. It began as a single small patch before spreading to several areas of the scalp. He reported no pain, itching, burning, prior scalp injury, fever, recent illness, significant psychological stress, medication use, or chemical exposure preceding onset. This was his first episode of hair loss, and he had no personal or family history of autoimmune disease.

General physical examination was normal. Scalp examination revealed several confluent, well-demarcated, smooth, non-scarring bald patches over the frontal, bilateral temporal, parietal, and posterior vertex regions (Figure 1), follicular openings remained intact and there was no erythema, scaling, pustules, crusting, or atrophy. Exclamation-mark hairs were visible along several patch margins, indicating active disease. Eyebrows, eyelashes, beard, and body hair were unaffected, and the nails showed no pitting, trachyonychia, or other changes.

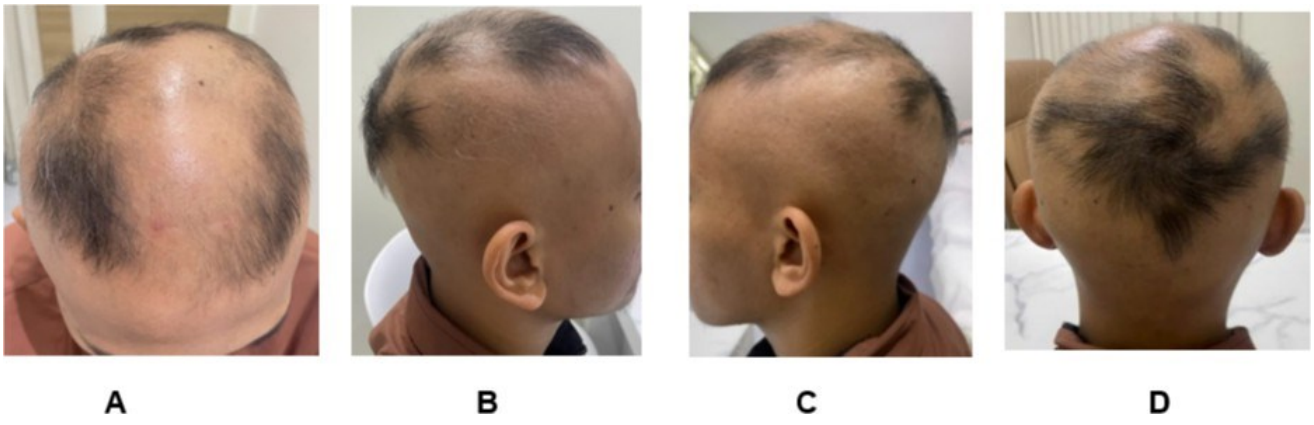


**Figure 1.** Baseline clinical photographs showing extensive non-scarring alopecic patches: (A) frontal scalp, (B) right temporal scalp, (C) left temporal scalp, and (D) posterior vertex scalp.

These clinical features were consistent with severe patchy AA. Disease severity was estimated with the SALT score, a widely used tool for quantifying scalp hair loss, giving a baseline value of 73 and indicating substantial scalp involvement.[12] [13]

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Because this figure was derived from standardized photographs rather than a hands-on, quadrant-by-quadrant assessment, it is referred to throughout as an "estimated" SALT score to reflect how it was obtained.

The extent of hair loss and the signs of active inflammation supported a multimodal approach. Treatment consisted of intralesional triamcinolone acetonide injected into the active patches, oral methylprednisolone 4 mg once daily for one month, and topical mometasone furoate in a petrolatum base applied daily. A secretome injection was added on top of this regimen as a supportive measure for follicular recovery, not as a substitute for the established anti-inflammatory therapy.

By the one-month visit, the previously bald areas showed noticeably denser hair, with diffuse fine vellus and early terminal hairs (Figure 2), and there was nothing to suggest the disease was still progressing or that new patches had formed.

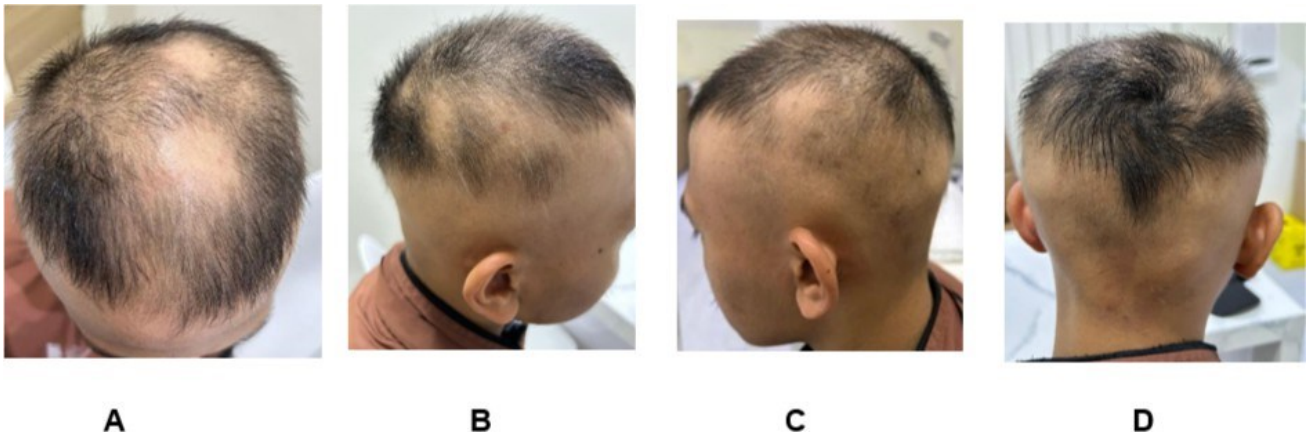


**Figure 2.** One-month follow-up photographs showing early regrowth: (A) frontal scalp, (B) right temporal scalp, (C) left temporal scalp, and (D) posterior vertex scalp. Diffuse vellus hairs and early terminal hairs are visible

Over that month, the estimated SALT score dropped from 73 to 55, a reduction of roughly 25% in scalp involvement. None of the local complications sometimes seen with intralesional therapy, such as skin thinning, telangiectasia, pigment change, or infection, occurred, and the patient reported no systemic side effects from the short course of oral corticosteroid.

**Table 1.** Clinical characteristics

| Variable | Finding |
|---|---|
| Age | 23 years |
| Sex | Male |
| Main complaint | Progressive patchy scalp hair loss |
| Symptom duration | Approximately 3-6 months |
| Diagnosis | Severe patchy alopecia areata |
| Clinical subtype | Patchy alopecia areata |
| Exclamation-mark hairs | Present |
| Nail involvement | Absent |
| Scarring | Absent |
| Estimated baseline SALT score | 73 |
| Estimated one-month SALT score | 55 |
| Follow-up duration | One month |

**Table 2.** Clinical timeline

| Time point | Clinical findings and management |
|---|---|
| 3-6 months before presentation | Patchy scalp hair loss appeared and gradually enlarged |
| Initial assessment | Extensive non-scarring alopecic patches with exclamation-mark hairs; estimated SALT 73 |
| Day 0 | Intralesional triamcinolone, methylprednisolone 4 mg/day, topical mometasone in petrolatum, and adjunctive secretome started |
| One-month follow-up | Increased density with vellus and early terminal hairs; estimated SALT 55; no reported treatment-related adverse effects |

**Table 3.** Change after treatment

| Clinical Parameter | Baseline | One-Month Follow-Up |
|---|---|---|
| Estimated SALT score | 73 | 55 |
| Hair density | Markedly reduced | Improved |
| Alopecia distribution | Extensive confluent patches | Persistent but reduced patchy alopecia |
| Vellus hairs | Not observed | Present |
| Early terminal hairs | Not observed | Present |
| Disease activity | Active | Clinically decreased |
| Adverse effects | None observed | None observed |

# Result and Discussion

## A. Pathogenesis and Clinical Features

AA is fundamentally autoimmune: the immune system mistakenly attacks anagen-phase hair follicles. Under normal circumstances, follicles are shielded from immune attack by low expression of MHC molecules and local immunoregulatory signals. Once this shield breaks down, autoreactive immune cells can recognize follicular tissue and sustain ongoing inflammation.[1] [2] [3] [4]

One key pathway implicated in AA runs through CD8+ NKG2D+ cytotoxic T cells alongside IFN-γ and IL-15. IFN-γ appears to heighten immune recognition of follicles, while IL-15 feeds a self-perpetuating inflammatory cycle via JAK-STAT signaling. Blocking parts of this pathway in experimental models has been shown to halt or even reverse AA-like disease, the biological rationale behind JAK-inhibitor therapies.[2] [4]

Typical AA lesions are well circumscribed, smooth, non-scarring patches with follicular openings still visible. Exclamation-mark hairs are a hallmark of active disease, and trichoscopy can reveal additional clues, including yellow dots, black dots, broken hairs, exclamation-mark hairs, and short regrowing vellus hairs, that help confirm the diagnosis and track progress over time.[14] [15]

The present patient had clinical findings typical of active patchy AA. Extensive involvement of several scalp regions and an estimated SALT score of 73 placed him in a severe category. A SALT score of 50 or greater is commonly used as a threshold for severe scalp involvement in clinical trials and contemporary therapeutic studies.[13] [16] While diagnosis was clinical in this case, the preserved ostia and absence of scale, pustules, marked erythema, or scarring made tinea capitis and primary cicatricial alopecia less likely. The morphology and exclamation-mark hairs also favored AA over trichotillomania and androgenetic alopecia.

## B. Rationale for Combination Treatment

Treatment of AA should reflect its severity and activity. Contemporary guidelines support intralesional corticosteroids as a standard option for localized patchy AA. For selected patients with extensive, rapidly progressive, or highly active disease, systemic corticosteroids may be considered, recognizing the risks of adverse effects and relapse. Potent topical corticosteroids are frequently used as local adjuncts.[5] [6]

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In this patient, intralesional triamcinolone was chosen to provide local anti-inflammatory activity in the most active patches. Systemic methylprednisolone was added because involvement was widespread, and topical mometasone was used to maintain local treatment across affected scalp areas. Corticosteroids can reduce inflammatory activation around hair follicles, but a favorable early response cannot be attributed to one component of a combined regimen.

Targeted JAK inhibitors have since expanded the treatment options available for severe AA. In two phase 3 randomized trials, baricitinib produced significantly greater scalp regrowth than placebo by week 36.[16] This finding underscores the centrality of JAK-STAT signaling in AA, though it does not follow that every patient requires a JAK inhibitor, nor that other therapies are obsolete; the appropriate choice still depends on clinical guidelines, individual patient factors, access, contraindications, and a considered balance of benefit against risk.[5] [6]

## C. Secretome as an Adjunct

MSC-derived secretome comprises biologically active soluble molecules and extracellular vesicles. It has been investigated as a cell-free approach intended to retain some paracrine effects of MSCs while avoiding administration of living cells. General regenerative-medicine literature reports immunomodulatory and tissue-repair activity in experimental and preclinical settings.[7] [8]

In hair research, extracellular vesicles and MSC-related paracrine factors have been studied for possible effects on dermal papilla cells, follicular signaling, and hair-cycle regulation. These observations provide a plausible scientific rationale for research, but they should not be presented as evidence that secretome is clinically effective in AA. Preparations differ by cell source, culture conditions, isolation process, composition, dose, and route of administration, making comparisons difficult.[8] [9] [10]

A systematic review of adipose-derived stem cell-based interventions and secretome in reversible alopecias (a mixed review of cases with androgenetic alopecia or alopecia areata) identified limited and heterogeneous clinical evidence, with only one randomized controlled trial and insufficient data to establish standardized protocols or condition-specific efficacy.[10] Importantly, evidence across reversible alopecias cannot be assumed to apply directly to AA, which has a distinct autoimmune pathogenesis. The available clinical literature is insufficient to define standardized protocols or establish additive benefit beyond conventional AA therapy.

In the present case, secretome was administered as an adjunct to corticosteroids. The subsequent presence of vellus and early terminal hairs is compatible with early follicular recovery. That said, the intralesional, oral, and topical corticosteroids were all started at the same time as the secretome, so the improvement observed here could stem from the corticosteroids themselves, natural fluctuation of AA, some added effect from the secretome, or a mix of all three; this case simply cannot pin the improvement on secretome alone.

## D. Comparison with Previous Clinical Reports

The one-month regrowth observed in the present case can be considered against previously reported treatment approaches for alopecia areata. Reports of intralesional triamcinolone acetonide monotherapy for patchy AA generally describe visible regrowth emerging over several weeks to a few months of repeated sessions rather than within the first month.[17] [18] Similarly, time-to-event analyses of intramuscular corticosteroid therapy suggest that meaningful regrowth typically requires more than one month of treatment.[19] Case reports using minimally manipulated umbilical cord-derived mesenchymal stem cells administered as cellular therapy have also described favorable regrowth, but over longer follow-up periods and with a different (cellular, rather than cell-free secretome) intervention.[20] Reports combining platelet-rich plasma with corticosteroids in difficult-to-control AA have likewise reported clinical improvement, though again over a longer observation window.[21] [22] Relative to these prior reports, the regrowth observed here over one month appears comparatively rapid; however, because corticosteroids, secretome, and other components of the regimen were introduced concurrently, causal attribution of this apparent acceleration to secretome specifically cannot be isolated from the present case alone.[1] [3]

## E. Clinical Outcome and Limitations

The estimated SALT score fell from 73 to 55 within one month, and the concurrent emergence of vellus and early terminal hairs is consistent with early reactivation of follicular growth. Regrowth in AA often begins as fine, hypopigmented hair that later thickens and repigments; a one-month window is too short, however, to know whether this will progress to sustained terminal-hair regrowth or lasting remission.[13] [14]

No local adverse effects of intralesional therapy or patient-reported systemic effects of short-term methylprednisolone were identified during follow-up. While reassuring, this brief observation period says nothing about longer-term safety, and relapse remains possible even after an encouraging initial response; ongoing follow-up is essential.[5] [6]

Several limitations should be acknowledged. As a single case, this report cannot establish that the treatment is effective. Because the interventions were started together, their individual contributions cannot be separated. Follow-up was limited to one month, and since the SALT score was derived from standardized photographs rather than a direct bedside examination, some measurement variability cannot be excluded. Future work on secretome as an AA adjunct should, where possible, confirm severity through in-person assessment, use independent or blinded evaluators or validated computer-assisted scoring, and extend follow-up duration.[23] [24] Trichoscopic monitoring and histopathologic confirmation were also not performed in this case.

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## Conclusion

This case describes early improvement in severe patchy AA following intralesional triamcinolone acetonide, oral methylprednisolone, topical mometasone furoate, and adjunctive secretome injection. One month on, the patient had visibly denser hair, with vellus and early terminal hairs present, and his estimated SALT score had dropped from 73 to 55.

This case demonstrates that a multimodal treatment approach can be documented in a single patient, but it does not establish that secretome itself was responsible for the improvement. Because corticosteroids and secretome were administered concurrently, the response cannot be attributed to either intervention alone. Before secretome can be considered for routine use in AA, controlled prospective studies with standardized manufacturing, clearly reported dosing, appropriate comparator groups, and longer follow-up will be needed.

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