

Case report

Pediatric alopecia universalis with poliosis post-psychosocial stress: a case report on methotrexate and adjunctive managementKaren Paola David,¹ Carlos Solórzano Flores,^{2✉} Andrea Pineda,³ Abner Baquedano²¹Department of Pediatric Dermatology, San Felipe Hospital, Tegucigalpa, Honduras²Faculty of Medical Sciences, National Autonomous University of Honduras (UNAH), Tegucigalpa, Honduras³Faculty of Health Sciences, Central American Technological University (UNITEC), Tegucigalpa, Honduras**Abstract**

Alopecia areata (AA) is an immune-mediated non-scarring hair loss disorder. Severe forms, including alopecia totalis and alopecia universalis (AU), are rare in children and often refractory. Psychosocial stress has been implicated as a trigger, and pigmentary changes may occur during regrowth. We report a 9-year-old girl from Honduras with AU involving the scalp, eyebrows, eyelashes, and forearms, with nail pitting following a significant psychosocial stressor. Laboratory studies showed normal thyroid function and low vitamin D levels. Initial treatment included topical clobetasol propionate 0.05% for 6 months, topical minoxidil 5% for 12 months, and oral vitamin D supplementation for 6 months, with minimal response. Systemic therapy was initiated with oral corticosteroids (1 mg/kg/day) tapered over 4 months, followed by oral methotrexate (12 mg/m²/week) for 1 year; topical therapies were maintained concurrently throughout systemic treatment. Hair regrowth began at 3 months of methotrexate, initially as diffuse poliosis, followed by complete terminal hair regrowth and normalization of nail changes. Psychological support was provided throughout management. This case highlights psychosocial stress as a potential contributor in pediatric AU and supports a multidisciplinary approach. Methotrexate was effective and well tolerated, and poliosis may be a marker of follicular recovery.

Keywords: pediatric dermatology, alopecia, autoimmune, hair disorder, therapy treatment**Received:** 14 January 2026 | **Returned for modification:** 3 June 2026 | **Accepted:** 2 July 2026**Introduction**

Alopecia areata (AA) is a chronic immune-mediated disorder characterized by non-scarring hair loss (1–5). It results from autoimmune disruption of the hair follicle cycle, leading to shedding that may affect the scalp, beard, eyebrows, eyelashes, and other hair-bearing areas (6–9). In some cases, hair regrowth occurs with altered pigmentation (poliosis), reflecting involvement of the follicular melanocyte unit; poliosis has been reported in approximately 29.4% of patients with severe AA and is most commonly observed during the early stages of hair regrowth (10, 11).

The clinical course of AA is highly variable. Severe subtypes include alopecia totalis (AT), defined by complete scalp hair loss, and alopecia universalis (AU), characterized by complete loss of scalp and body hair (12–14). Epidemiological studies estimate that 7% to 30% of patients with AA progress to AT or AU (6). In addition to its cutaneous manifestations, AA has been associated with several systemic comorbidities, including cardiovascular disease and arterial hypertension, supporting routine clinical monitoring in affected individuals (15).

The psychosocial burden of AA is considerable, particularly in pediatric populations. Studies utilizing the Children's Dermatology Life Quality Index (CDLQI) have demonstrated that quality-of-life impairment in children with AA is comparable to, and in some cases exceeds, that observed in severe atopic dermatitis

and psoriasis (16, 17). Children frequently experience peer stigmatization, school-related anxiety, diminished self-esteem, and emotional distress. Psychosocial stress may further contribute to disease activity through activation of neuroendocrine pathways involving mediators such as substance P and corticotropin-releasing hormone, which disrupt hair follicle immune privilege and promote local inflammation (7).

Therefore, we report the case of a child with alopecia universalis following a significant psychosocial stressor that achieved complete hair regrowth, including pigmentary recovery, under methotrexate-based multimodal therapy.

Case report

A 9-year-old female from Marcala, La Paz, Honduras, with no relevant past medical or family history, presented with a 1-year history of progressive hair loss. She had no allergies or systemic illnesses, and her growth and development were appropriate for her age. The onset coincided with a significant psychosocial stressor—namely, maternal separation due to employment in another department, which was considered a potential precipitating factor. Hair shedding initially affected the parietotemporal scalp and progressively extended over the following months, resulting in near-complete scalp hair loss with associated eyebrow and eyelash involvement by the time of presentation.

At evaluation, the patient weighed 28 kg. Vital signs were stable, and arterial blood pressure remained consistently within normal age- and sex-adjusted percentiles throughout follow-up. Physical examination revealed disseminated non-scarring alopecia involving the scalp, eyebrows, eyelashes, and forearms, with only approximately 10 sparse terminal hairs remaining on the frontal scalp (Fig. 1). Nail involvement was evident as multiple pits. No additional systemic abnormalities were identified.

Laboratory investigations, including thyroid-stimulating hormone (TSH) and free thyroxine (fT4), were within normal limits, whereas serum vitamin D levels were insufficient at 23 ng/ml. Trichoscopy of the parietal scalp demonstrated marked hair rarefaction with extensive areas devoid of terminal hairs, preserved follicular ostia, and multiple empty follicles without evidence of scarring (Fig. 2). Exclamation mark hairs, black dots, broken hairs, yellow dots, and perifollicular inflammatory signs were not observed. These findings were consistent with advanced non-scarring alopecia universalis.

The diagnosis of alopecia universalis was established clinically. Scalp biopsy was not pursued because of the characteristic clinical presentation and the cultural and economic barriers limiting access to this procedure.

The patient was referred for psychological support as part of a multidisciplinary management plan. On psychological evaluation, she demonstrated marked separation anxiety related to her mother's absence and reported school avoidance. Initial treatment consisted of topical clobetasol propionate 0.05% foam applied nightly for 6 months, topical minoxidil 5% for 1 year, and oral vitamin D supplementation for 6 months. Because of the extensive nature of the disease and limited response to topical therapy, oral corticosteroid therapy was initiated at 1 mg/kg/day (28 mg/day) and tapered by approximately 25% every 4 weeks until discontinuation after 4 months.

Early improvement was characterized by the appearance of white vellus hairs, followed by partial terminal regrowth in the occipital region. Two weeks after initiation of corticosteroid therapy, oral methotrexate was introduced at a dose of 12 mg/m²/week and maintained for 1 year, with good tolerance and no reported adverse effects. Topical clobetasol and minoxidil were continued concomitantly throughout systemic treatment.

Progressive hair regrowth became evident by the 3rd month of methotrexate therapy. By the end of treatment, complete restoration of scalp, eyebrow, and eyelash hair had been achieved, including areas that initially exhibited poliosis (Fig. 3). Nail pitting also improved substantially. In parallel with the clinical response, the patient experienced marked psychological improvement, reflected by reduced appearance-related distress, improved self-confidence, and enhanced social interaction, as reported by caregivers. Methotrexate was discontinued after 1 year because complete clinical remission had been achieved and maintained. At 1-year post-treatment follow-up, sustained remission without clinical recurrence was confirmed.

Discussion

The temporal relationship between the onset of maternal separation and the progressive development of AU in our patient supports psychosocial stress as a precipitating factor. This is consistent with evidence that acute emotional stressors can precede disease onset or trigger rapid progression in pediatric AA, mediated through neuroendocrine pathways involving substance P and corticotropin-releasing hormone, which disrupt hair follicle immune privilege and amplify local inflammation (7, 18). Psychological evaluation in our patient revealed marked separation anxiety and school avoidance, findings that aligned with the documented psychosocial burden of the disease. CDLQI-based studies have shown that quality-of-life impairment in children with AA is comparable to, and in some cases exceeds, that seen in severe atopic dermatitis and psoriasis (16, 17), underscoring the importance of integrating psychological support from the outset of management, as was done in this case.

A relevant clinical question is whether resolution of the psychosocial trigger alone, without systemic intervention, might have led to spontaneous remission. Although localized AA can remit spontaneously, this is substantially less likely in extensive variants such as AU (1, 13). The extent of hair loss, multi-site involvement including the forearms and eyelashes, nail pitting, and insufficient vitamin D levels in our patient collectively indicated a severe phenotype with a poor prognosis, justifying early escalation to systemic therapy rather than a watchful waiting approach. Notably, thyroid function was normal and no autoimmune comorbidities were identified, demonstrating that severe AU can occur in the absence of other systemic autoimmune disease.

Regarding treatment, topical clobetasol and minoxidil produced minimal benefit over 6 months, consistent with the well-recognized limited efficacy of topical agents in extensive disease (19, 20). The addition of systemic corticosteroids at 1 mg/kg/day resulted in early vellus regrowth, and methotrexate at 12 mg/m²/week, introduced 2 weeks later and maintained for 1 year, was associated with progressive and ultimately complete hair restoration. This response is in line with reports supporting methotrexate as an effective immunomodulatory option in refractory pediatric AU (9). Importantly, the drug was well tolerated with no adverse effects over the full treatment year, and complete remission was sustained at 1-year post-discontinuation follow-up. In a resource-limited setting in which Janus kinase inhibitors remain financially or logistically inaccessible, this outcome reinforces methotrexate as a practical and cost-effective first-line systemic option (8, 13, 20).

The appearance of widespread poliosis during the regrowth phase is a notable feature of this case. Poliosis has been reported in approximately 29.4% of patients with severe AA and is thought to reflect autoimmune-mediated melanocyte destruction, oxidative stress, or impaired follicular melanocyte stem cell function (10, 11, 21). In our patient, poliosis was not limited to a single region but involved the scalp, eyebrows, and eyelashes, a more extensive distribution than described in previously reported adult cases. The subsequent full repigmentation following sustained methotrexate therapy suggests that immunomodulatory treatment can restore melanocyte function alongside follicular recovery. This supports the role of poliosis not merely as a cosmetic finding but as a dynamic marker of follicular immune activity and therapeutic response.

This report has several limitations. Scalp biopsy and advanced immunological testing were not performed due to cultural and economic barriers. However, the characteristic clinical presentation, trichoscopic findings, and durable treatment response provide strong support for the diagnosis. These constraints also reflect the real-world conditions under which many pediatric patients with severe AA are managed globally, and reporting them adds practical value for clinicians practicing in similar settings.

Conclusions

This case demonstrates that complete clinical resolution may be achievable even in the presence of poor prognostic indicators, including extensive alopecia universalis and nail involvement. It further emphasizes the importance of integrating psychological assessment and support into the management of pediatric AA when significant emotional stressors are identified. In resource-limited settings, a multimodal approach

incorporating topical therapy, systemic corticosteroids, methotrexate, and psychological support may be an effective and accessible therapeutic strategy for severe pediatric disease.

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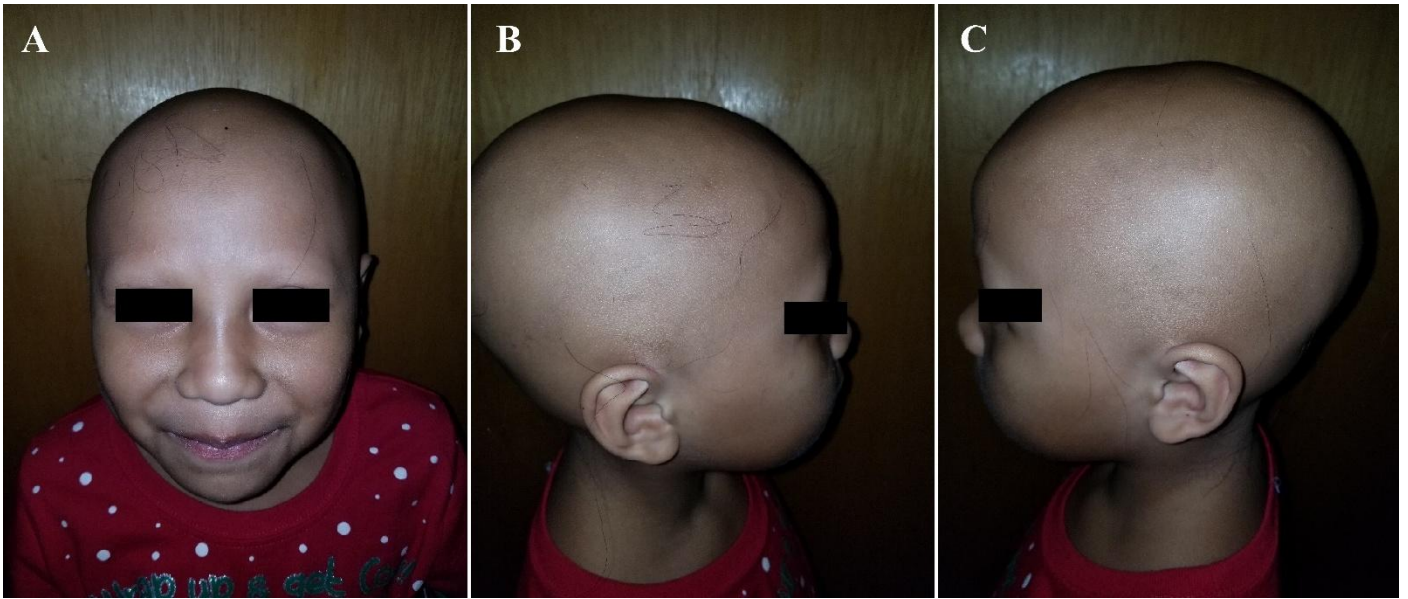


Figure 1. Clinical photos of a patient with alopecia universalis: A) frontal view revealing complete loss of scalp hair, eyelashes, and eyebrows; the skin appears smooth with no signs of inflammation or scarring, B–C) lateral views demonstrating total denudation of the scalp, including the occipital and temporal regions.

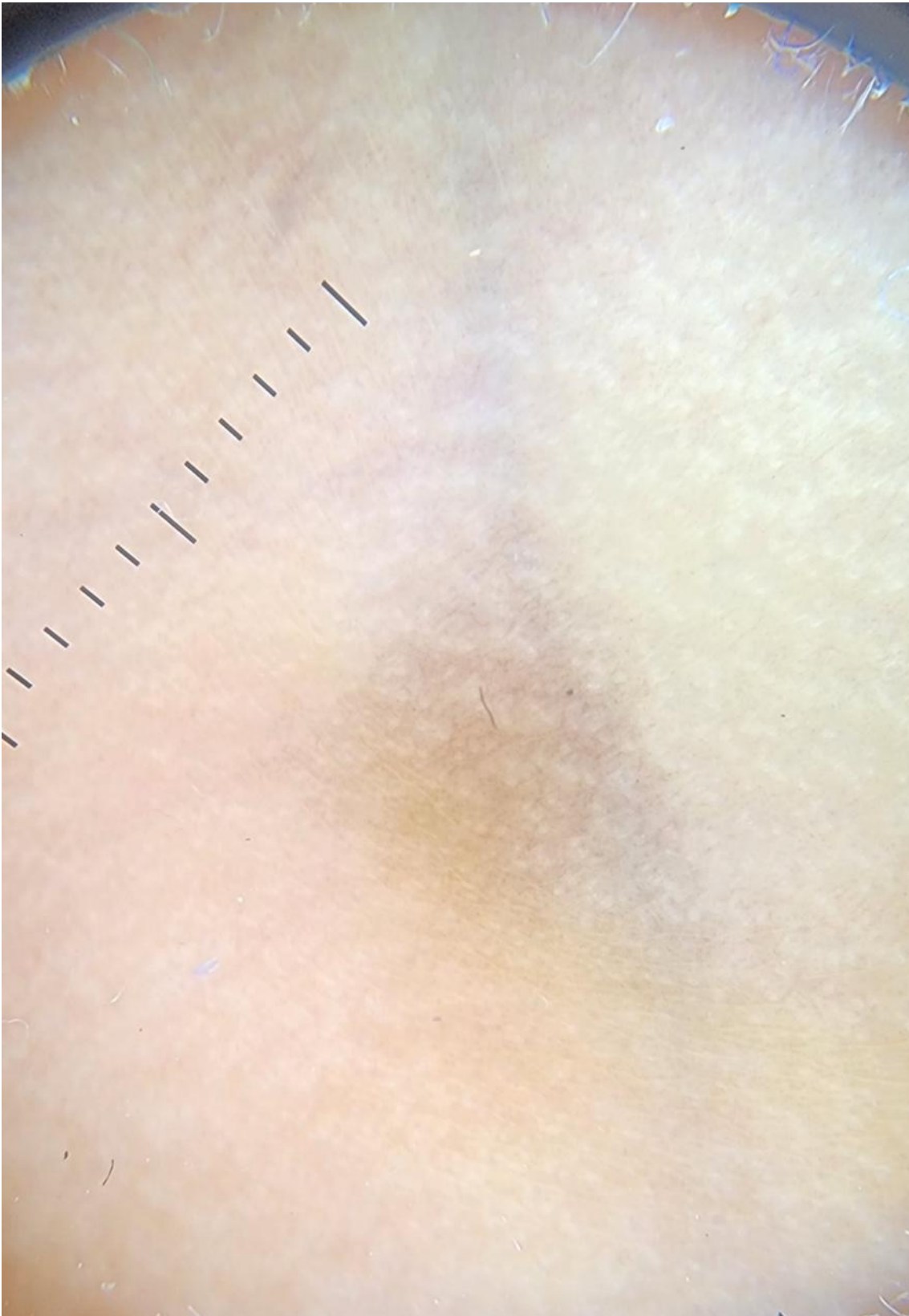


Figure 2. Trichoscopy of the parietal scalp. It demonstrates a hairless field with preserved follicular ostia. No definitive black dots, exclamation mark hairs, or dystrophic hairs are appreciable within the limits of image resolution.



Figure 3. Clinical photos demonstrating scalp hair repigmentation with poliosis: A) frontal view and (B–C) lateral views of the scalp, showing partial regrowth of terminal scalp hair following treatment. Notably, there are multiple well-demarcated strands of depigmented (white) hair consistent with poliosis, predominantly involving the parietotemporal regions, interspersed among normally pigmented dark hair.