Psoriatic Alopecia in a Patient With Crohn Disease: An Uncommon Manifestation of Tumor Necrosis Factor α Inhibitors

Osward Y. Carrasquillo, MD, MPH; Gabriela Pabón-Cartagena, MD; Julián Barrera-Llaurador, MD; Francisco Colón-Fontanez, MD; Rafael F. Martín-García, MD

PRACTICE **POINTS**

- Psoriatic alopecia is a rare nonscarring alopecia that can present as a complication of treatment with tumor necrosis factor α inhibitors.
- This finding commonly is seen in females undergoing treatment with infliximab or adalimumab, usually for Crohn disease.
- Histopathologic findings can show a psoriasiform-pattern, neutrophil-rich, inflammatory infiltrate involving hair follicles or a lichenoid pattern.

Tumor necrosis factor α (TNF-α) inhibitors are used to treat multiple inflammatory diseases including rheumatoid arthritis, inflammatory bowel disease, and psoriasis, among others. This family of medications can cause various side effects, some as common as injection-site reactions and others as rare as the paradoxical induction of psoriasiform skin lesions. Alopecic plaques recently have been described as an uncommon adverse effect of the TNF-α inhibitors adalimumab and infliximab. We present the case of a 12-year-old girl treated with adalimumab for Crohn disease who developed an alopecic crusted plaque on the scalp 6 months after increasing the dose of the medication. Biopsies, special stains, and sterile cultures yielded a diagnosis of psoriatic alopecia secondary to TNF-α inhibitor. A literature review for similar cases found 24 additional patients presenting with similar findings, of which only 6 were part of the pediatric population.

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Tumor necrosis factor α (TNF-α) inhibitor–induced psoriasis is a known paradoxical adverse effect of this family of medications, which includes infliximab, adalimumab, etanercept, golimumab, and certolizumab. In the pediatric population, these therapies recently gained approval for nondermatologic conditions—meaning that this phenomenon is encountered more frequently.[1] In a systematic review of TNF-α inhibitor–induced psoriasis, severe scalp involvement was associated with alopecia in 7.5% of cases.[2] Onset of scalp psoriasis with alopecia in patients being treated with a TNF-α inhibitor should lead to consideration of this condition.

Psoriatic alopecia is an uncommon presentation of psoriasis. Although well described, alopecia as a clinical manifestation of scalp psoriasis is not a well-known concept among clinicians and has never been widely accepted. Adding to the diagnostic challenge is that psoriatic alopecia secondary to TNF-α inhibitor–induced psoriasis rarely has been reported in adults or children.[3-5] Including our case, our review of the literature yielded 7 pediatric cases (≤18 years) of TNF-α inhibitor–induced psoriatic alopecia.[6,7] A primary literature search of PubMed articles indexed for MEDLINE was conducted using the terms *psoriatic alopecia*, *psoriasiform alopecia*, *TNF-α inhibitors*, *infliximab*, *adalimumab*, *etanercept*, *golimumab*, and *certolizumab*.

We present the case of a pediatric patient with psoriatic alopecia secondary to treatment with adalimumab for

From the University of Puerto Rico School of Medicine, San Juan. Drs. Carrasquillo, Barrera-Llaurador, Colón-Fontanez, and Martín-García are from the Department of Dermatology, and Dr. Pabón-Cartagena is from the Transitional Year Program.

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Correspondence: Osward Y. Carrasquillo, MD, MPH, University of Puerto Rico School of Medicine, PO Box 365067, San Juan, PR 00936-5067 (ocarrasquillo5@gmail.com).

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Crohn disease (CD). We also provide a review of reported cases of psoriatic alopecia induced by a TNF-α inhibitor in the literature.

Case Report

A 12-year-old girl presented to our dermatology clinic with erythematous scaly plaques on the trunk, scalp, arms, and legs of 2 months' duration. The lesions involved approximately 15% of the body surface area. The patient's medical history was remarkable for CD diagnosed 4 years prior to presentation of the skin lesions. She had been treated for the past 2 years with adalimumab 40 mg once every 2 weeks and azathioprine 100 mg once daily. Because her CD was poorly controlled, the dosage of adalimumab was increased to 40 mg once weekly 6 months prior to the current presentation.

Our diagnosis was TNF-α inhibitor-induced psoriasis secondary to treatment with adalimumab.

The patient was treated with mometasone lotion 0.1% for the scalp lesions and triamcinolone cream 0.1% for the body lesions. Because of the extent of the psoriasis, we recommended changing adalimumab to ustekinumab, which is approved for CD in adults but is off label in children.

At 1-month follow-up, after receiving the induction dose of ustekinumab, the patient presented with partial improvement of the skin lesions but had developed a large, alopecic, erythematous plaque with thick yellowish scales on the scalp (Figure 1). She also had a positive hair pull test. The presumptive initial diagnosis of the alopecic scalp lesion was tinea capitis, for which multiple potassium hydroxide preparations of scales were performed, all yielding negative results. In addition, histopathologic examination with hematoxylin and eosin staining was performed (Figures 2A and 2B). Sterile tissue cultures for bacteria, fungi, and acid-fast bacilli were obtained and showed no growth. Periodic acid–Schiff staining was negative for fungal structures.

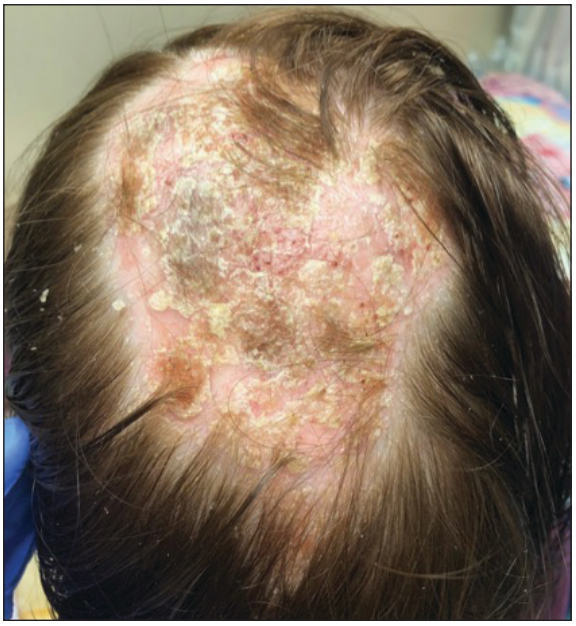

FIGURE 1. Large, alopecic, erythematous plaque on the scalp with yellowish scales.

A second biopsy showed a psoriasiform pattern, parakeratosis, and hypogranulosis, highly suggestive of psoriasis (Figure 2C and 2D). Based on those findings, a diagnosis of psoriatic alopecia was made. The mometasone was switched to clobetasol lotion 0.05%. The patient continued treatment with ustekinumab. At 6-month follow-up, her CD was well controlled and she showed hair regrowth in previously alopecic areas (Figure 3).

Comment

Psoriatic alopecia induced by a TNF-α inhibitor was first reported in 2007 in a 30-year-old woman with ankylosing spondylitis who was being treated with adalimumab.[8] She had erythematous, scaly, alopecic plaques on the scalp and palmoplantar pustulosis. Findings on skin biopsy were compatible with psoriasis. The patient's severe scalp psoriasis failed to respond to topical steroid treatment and adalimumab cessation. The extensive hair loss responded to cyclosporine 3 mg/kg daily.[8]

After conducting an extensive literature review, we found 26 cases of TNF-α–induced psoriatic alopecia, including the current case (Table).[6-16] The mean age at diagnosis was 27.8 years (SD, 13.6 years; range, 7–60 years). The female-to-male ratio was 3.3:1. The most common underlying condition for which TNF-α inhibitors were prescribed was CD (77% [20/26]). Psoriatic alopecia most commonly was reported secondary to treatment with infliximab (54% [14/26]), followed by adalimumab (42% [11/26]). Golimumab was the causative drug in 1 (4%) case. We did not find reports of etanercept or certolizumab having induced this manifestation. The onset of the scalp lesions occurred 2 to 46 months after starting treatment with the causative medication.

Laga et al[17] reported that TNF-α inhibitor–induced psoriasis can have a variety of histopathologic findings, including typical findings of various stages of psoriasis, a lichenoid pattern mimicking remnants of lichen planus, and sterile pustular folliculitis. Our patient's 2 scalp biopsies demonstrated results consistent with findings reported by Laga et al.[17] In the first biopsy, findings were consistent with a dense neutrophilic infiltrate with negative sterile cultures and negative periodic acid–Schiff stain (sterile folliculitis), with crust and areas of parakeratosis. The second biopsy demonstrated psoriasiform hyperplasia, parakeratosis, and an absent granular layer, all typical features of psoriasis (Figure 2).

Including the current case, our review of the literature yielded 7 pediatric (ie, 0–18 years of age) cases of TNF-α inhibitor–induced psoriatic alopecia. Of the 6 previously reported pediatric cases, 5 occurred after administration of infliximab.[6,7]

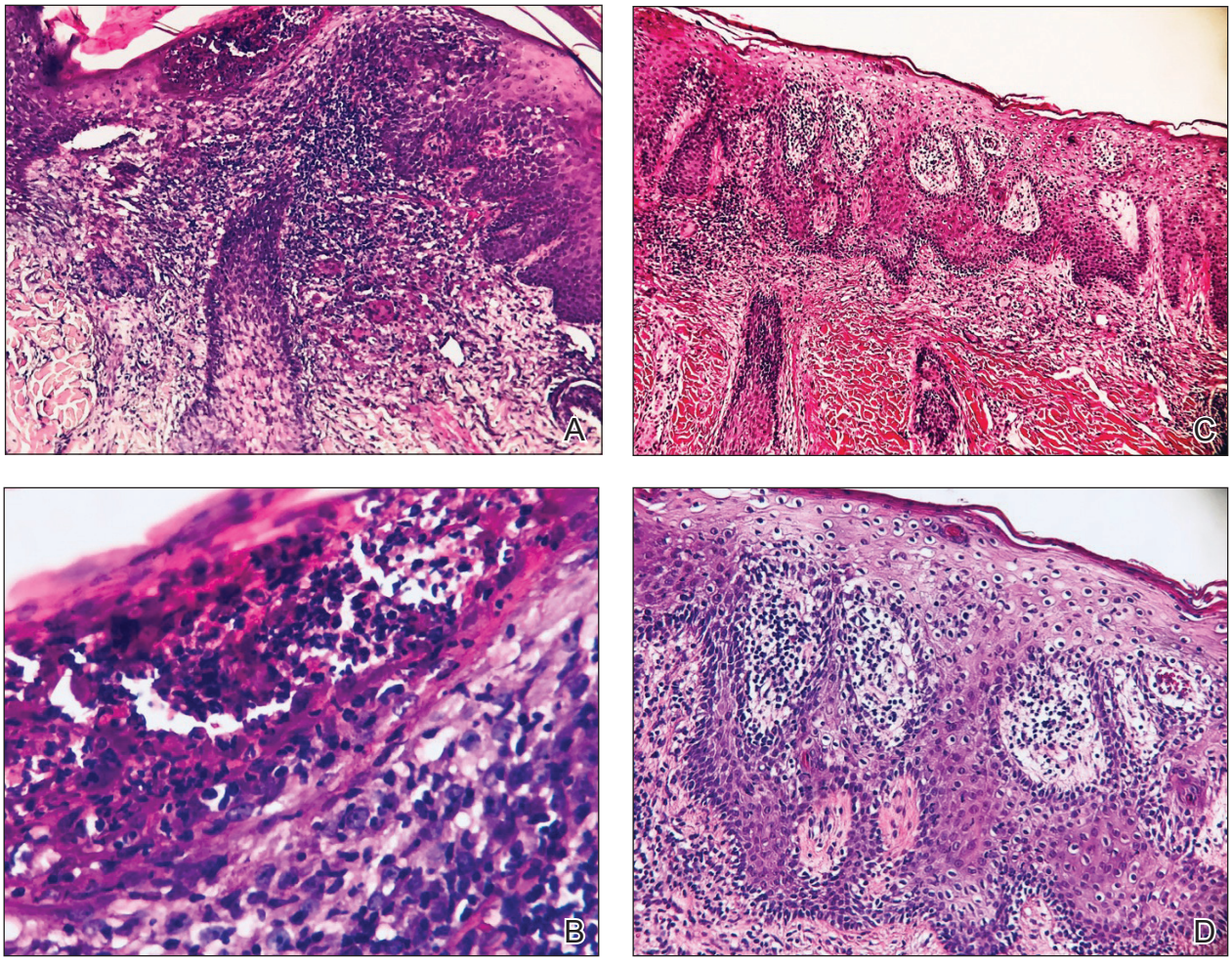

FIGURE 2. A, The first scalp biopsy showed a dense infiltrate of neutrophils in the dermis and perifollicular area (H&E, original magnification ×200). B, The second scalp biopsy showed an intracorneal collection of neutrophils on high magnification (H&E, original magnification ×400). C and D, Psoriasiform hyperplasia with mild parakeratosis and absent granular layer on the second biopsy (H&E, original magnifications ×100 and ×200).

Similar to our case, TNF-α inhibitor–induced psoriatic alopecia was reported in a 7-year-old girl who was treated with adalimumab for juvenile idiopathic arthritis.[6] Nine months after starting treatment, that patient presented with a tender, erythematous, eroded, and crusted alopecic plaque along with scaly plaques on the scalp. Adalimumab was discontinued, and cyclosporine and topical steroids were started. Cyclosporine was then discontinued due to partial resolution of the psoriasis; the patient was started on abatacept, with persistence of the psoriasis and alopecia. The patient was then started on oral methotrexate 12.5 mg once weekly with moderate improvement and mild to moderate exacerbations.

Tumor necrosis factor α inhibitor–induced psoriasis may occur as a result of a cytokine imbalance. A TNF-α blockade leads to upregulation of interferon α (IFN-α) and TNF-α production by plasmacytoid dendritic cells (pDCs), usually in genetically susceptible people.[6,7,9-15] The IFN-α induces maturation of myeloid dendritic cells (mDCs) responsible for increasing proinflammatory cytokines that contribute to psoriasis.[11] Generation of TNF-α by pDCs leads to mature or activated dendritic cells derived from pDCs through autocrine TNF-α production and paracrine IFN-α production from immature mDCs.[9] Once pDCs mature, they are incapable of producing IFN-α; TNF-α then inhibits IFN-α production by inducing pDC maturation.[11] Overproduction of IFN-α during TNF-α inhibition induces expression of the chemokine receptor CXCR3 on T cells, which recruits T cells to the dermis. The T cells then produce TNF-α, causing psoriatic skin lesions.[10,11,13,14]

Although TNF-α inhibitor–induced psoriatic alopecia is uncommon, the condition should be considered in female patients with underlying proinflammatory disease—CD in particular. Perman et al[6] reported 5 cases of psoriatic alopecia in which 3 patients initially were treated with griseofulvin because of suspected tinea capitis.

Reported Cases of TNF-α Inhibitor–Induced Psoriatic Alopecia

Reference (year)	Age, y/ Sex	TNF-α inhibitor	Time to onset of lesions	Clinical presentation	Histopathology	Underlying condition	Treatment and outcome
Papadavid et al[8] (2008)	30/F	Adalimumab	2 mo	Scalp psoriasis with considerable hair loss	Consistent with psoriasis	Ankylosing spondylitis	Cyclosporine; hair regrowth
Manni and Barachini[9] (2009)	30/F	Infliximab	15 wk	Erythematosquamous plaques on the scalp with alopecia	NR	CD	Discontinued infliximab and started cyclosporine; almost complete regrowth of hair after 6 mo
El Shabrawi-Caelen et al[10] (2010)	19/F	Adalimumab	3 mo	Severe alopecia with erythema and exudative discharge	Psoriasis features	CD	Discontinued adalimumab; hair regrowth
	31/F	Adalimumab	2 mo	Confluent areas of scaly psoriatic plaques with shiny and atrophic skin and loss of follicular orifices	Compatible with psoriasis and associated scarring alopecia	CD	NR
Medkour et al[11] (2010)	32/M	Infliximab	10 mo	Erythematous scaly plaques on the scalp with hair loss	Minimal lymphoid perifollicular infiltrate with follicular atrophy	CD	Discontinued infliximab and started topical clobetasol; hair regrowth after 4 wk
Doyle et al[12] (2011)	21/F	Infliximab	>2 mo	Large, scaly, alopecic patch on the scalp	Psoriasis features with a markedly increased number of catagen- and telogen-phase hair follicles; hair miniaturization with areas of peribulbar inflammation	CD	Continued infliximab and started intralesional triamcinolone injections with topical steroids; near-complete resolution of alopecia
	27/F	Infliximab	NR	Erythematous papules and pustules on the scalp	Increased number of catagen- and telogen-phase hair follicles; hair miniaturization with areas of peribulbar inflammation; superficial portion of the specimen showed pustular psoriasis–like changes and increased numbers of eosinophils; negative for fungi on periodic acid–Schiff with diastase staining; negative for spirochetes on Steiner staining	CD	Resistant to NB-UVB phototherapy and topical corticosteroids; switched to certolizumab pegol; resolution of alopecia
	39/F	Adalimumab	NR	Large, scaling, psoriasiform plaques on the scalp associated with considerable alopecia	Increased percentage of hair follicles in the catagen and telogen phases; miniaturization of hairs; superficial aspect of the biopsy showed mild psoriasiform hyperplasia	CD	Topical clobetasol; improvement in scaling and alopecia

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TABLE (continued)

Reference (year)	Age, y/ Sex	TNF-α inhibitor	Time to onset of lesions	Clinical presentation	Histopathology	Underlying condition	Treatment and outcome
Osório et al[13] (2012)	26/F	Infliximab	3 mo	Erythematous and scaly plaques with hyperkeratotic and exudative lesions, inducing nonscarring inflammatory alopecia	Scalp biopsy was not performed	CD	Topical steroids; switched to adalimumab and methotrexate, then switched to infliximab; flare-ups continued
	31/F	Infliximab	9 mo	Erythematous and scaly plaques with hyperkeratotic and exudative lesions, inducing nonscarring inflammatory alopecia	Scalp biopsy was not performed	CD	Started topical steroids and discontinued infliximab; cleared
	23/F	Adalimumab	46 mo	Erythematous and scaly plaques with hyperkeratotic and exudative lesions, inducing nonscarring inflammatory alopecia	Scalp biopsy was not performed	CD	Continued adalimumab and started topical steroids; nearly cleared
	30/M	Adalimumab	11 mo	Erythematous and scaly plaques with hyperkeratotic and exudative lesions, inducing nonscarring inflammatory alopecia	Scalp biopsy was not performed	CD	Discontinued adalimumab and started low-dose oral steroids and methotrexate; resolved
	25/F	Adalimumab	2 mo	Erythematous and scaly plaques with hyperkeratotic and exudative lesions, inducing nonscarring inflammatory alopecia	Scalp biopsy was not performed	CD	Discontinued adalimumab and started topical steroids and methotrexate; prednisolone (for 2 wk); then cyclosporine; mild improvement 4 wk after starting cyclosporine

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TABLE (continued)

Reference (year)	Age, y/ Sex	TNF-α inhibitor	Time to onset of lesions	Clinical presentation	Histopathology	Underlying condition	Treatment and outcome
Perman et al[6] (2012)	7/F	Adalimumab	9 mo	Tender, boggy, erythematous, eroded, hemorrhagically crusted alopecic plaque	Suggestive of early psoriasis	Juvenile idiopathic arthritis	Discontinued adalimumab and started cyclosporine, topical steroids, calcipotriene, and methotrexate; moderate improvement
	11/M	Infliximab	2.2 y	Erythematous, scaly, eroded, crusted, focally alopecic plaques	NR	CD	Topical steroids; cleared after 11 mo
	16/M	Infliximab	5 mo	Erythematous, scaly, eroded, crusted, focally alopecic plaques	NR	Ulcerative colitis	Discontinued infliximab and started adalimumab and topical steroids; cleared after 6 mo
	14/F	Infliximab	2 mo	Erythematous, scaly, eroded, crusted, focally alopecic plaques	NR	CD	Discontinued infliximab and started topical steroids, calcipotriene, and methotrexate; nearly clear after 4 mo
	18/F	Infliximab	7 mo	Erythematous, scaly, eroded, crusted, focally alopecic plaques	NR	CD	Topical steroids and calcipotriene; no improvement
Andrisani et al[14] (2013)	26/F	Infliximab	8 wk	Diffuse erythematous scaly plaque on the scalp; severe hair loss	Characteristic psoriasis with perifollicular atrophy, parakeratosis, and superficial dermal perivascular infiltrates	CD	Switched to ustekinumab; remission of scalp alopecia after 6 mo
Prata Ribeiro et al[7] (2015)	28/M	Infliximab	3 y	Two alopecic plaques in the left parietal region, 1 with erythema and desquamation	Psoriasis features with a mononuclear inflammatory infiltrate involving all levels of intrafollicular and perifollicular structure; intense miniaturization of hair follicles; direct mycologic examination was negative	CD	Started clobetasol gel, coal-tar shampoo, and intralesional steroids on the scalp and continued infliximab; hair regrowth within 3 mo
	14/F	Infliximab	4 mo	Alopecic desquamating plaques detected in the bilateral frontal and parietal region	Hyperkeratosis that extended to the follicular ostia; small foci of parakeratosis; pronounced miniaturization of hair follicles with only 50% of hair terminals in the anagen phase	CD	Started coal-tar shampoo, betamethasone cream on scalp and continued infliximab; hair regrowth within 5 mo

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TABLE (continued)

Reference (year)	Age, y/ Sex	TNF-α inhibitor	Time to onset of lesions	Clinical presentation	Histopathology	Underlying condition	Treatment and outcome
Afanasiev et al[15] (2017)	60/F	Adalimumab (in combination with 6-mercaptopurine)	3 y	15-cm, erythematous, crusted plaque on the scalp with associated alopecia	Mild psoriasiform and spongiotic epidermal hyperplasia with mild hair-follicle keratin plugging and a few neutrophils in the stratum corneum; superficial and mid perivascular lymphocytic inflammation; no evidence of scarring; peribulbar inflammation not seen	CD	Discontinued adalimumab and started 5-aminosalicylic acid; near-complete hair regrowth after 10 mo
	42/F	Infliximab (in combination with leflunomide)	17 mo	Enlarging, crusted, 10-cm plaque of alopecia on the vertex of the scalp	Mild psoriasiform and spongiotic epidermal hyperplasia with mild hair-follicle keratin plugging and a few neutrophils in the stratum corneum	Seronegative inflammatory arthritis	Discontinued leflunomide and infliximab and started methotrexate; alopecia resolved after 10 mo
	56/F	Adalimumab (in combination with leflunomide)	2 y	Erythematous pruritic area with thick, yellow, adherent scale–crust and patchy alopecia on the temporal scalp	Mild psoriasiform and spongiotic epidermal hyperplasia with mild hair-follicle keratin plugging and a few neutrophils in the stratum corneum; superficial and mid perivascular lymphocytic inflammation	Seronegative inflammatory arthritis	Discontinued adalimumab and started clobetasol foam and added abatacept; alopecia resolved 1 mo later
Helm et al[16] (2018)[a]	53/M	Golimumab	3 mo	5×6-cm, well-demarcated plaques with obliteration of follicular ostia and fine scaling	Scalp biopsy was not performed	Ankylosing spondylitis	Recommended sun protection; started clobetasol solution and intralesional steroids; notable improvement
Current case	12/F	Adalimumab (in combination with azathioprine)	6 mo[b]	Large, alopecic, erythematous plaque with thick yellowish scales	First biopsy: Compatible with sterile pustular folliculitis; second biopsy: compatible with psoriasis	CD	Switched to ustekinumab and topical clobetasol lotion; hair regrowth

Abbreviations: TNF-α, tumor necrosis factor α; F, female; NR, not reported; CD, Crohn disease; M, male; NB-UVB, narrowband UVB.

[a]Although the authors reported this case as alopecia areata and scarring alopecia, they asserted that the clinical presentation fell within the spectrum of findings seen in psoriasiform dermatitis associated with alopecia.

[b]Lesions appeared 6 months after the dosage of adalimumab was increased to 40 mg once weekly. The patient had been treated with adalimumab 40 mg once every 2 weeks for 4 years before the dosage of adalimumab was increased.

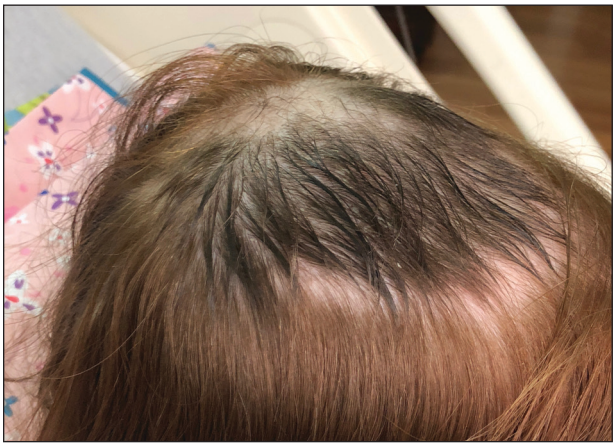

FIGURE 3. Considerable hair regrowth was noted at 6 months' follow-up.

Conditions with similar clinical findings should be ruled out before making a diagnosis of TNF-α inhibitor–induced psoriatic alopecia. Although clinicopathologic correlation is essential for making the diagnosis, it is possible that the histologic findings will not be specific for psoriasis.[17] It is important to be aware of this condition in patients being treated with a TNF-α inhibitor as early as 2 months to 4 years or longer after starting treatment.

Previously reported cases have demonstrated various treatment options that yielded improvement or resolution of TNF-α inhibitor–induced psoriatic alopecia. These include either continuation or discontinuation of the TNF-α inhibitor combined with topical or intralesional steroids, methotrexate, or cyclosporine. Another option is to switch the TNF-α inhibitor to another biologic. Outcomes vary from patient to patient, making the physician's clinical judgment crucial in deciding which treatment route to take. Our patient showed notable improvement when she was switched from adalimumab to ustekinumab as well as the combination of ustekinumab and clobetasol lotion 0.05%.

Conclusion

We recommend an individualized approach that provides patients with the safest and least invasive treatment option for TNF-α inhibitor–induced psoriatic alopecia. In most reported cases, the problem resolved with treatment, thereby classifying this form of alopecia as noncicatricial alopecia.

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