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Adalimumab-related alopecia in a patient affected by psoriasis.

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Abstract

Alopecia induced by biological therapy is a rare side effect of this type of drugs. A total of 23 patients of psoriasiform eruptions with severe scalp involvement that induced alopecia during anti-tumor necrosis factor (anti-TNF) treatment of non-dermatological conditions have been previously reported. We present a 50-year-old man affected by plaque psoriasis that developed psoriasiform patches with alopecia over his scalp 10 months after initiating treatment with adalimumab. Punch biopsy of the alopecic area on the scalp revealed psoriasiform epidermal changes and alopecia areata-like dermal changes. Along with these findings, there was a dermal inflammatory infiltrate made up of eosinophils and plasma cells. In conclusion, scalp psoriasiform lesions with alopecia in patients treated with anti-TNF agents have been rarely reported. We describe a patient with anti-TNF therapy-related alopecia affected by psoriasis. Our patient has a peculiar histology with features of psoriasis and alopecia areata in addition to eosinophils and plasma cells. This entity may respond to topical treatment. However in patients of severe scalp involvement anti-TNF suspension should be considered.

Keywords: anti-TNF, adalimumab, alopecia areata, psoriasis, psoriatic alopecia

Introduction

Adverse reactions to anti-tumor necrosis factor (anti-TNF) biologic drugs are ever-rising due to their increase in the treatment of autoimmune diseases such as rheumatoid arthritis (RA), inflammatory bowel disease (IBD) and psoriasis. Anti-TNF drug-induced alopecia is a less well-known effect of this type of drugs and clinically resembles primary psoriatic alopecia or alopecia areata. To our knowledge, a total of 23 patients of psoriasiform eruptions with severe scalp involvement that induced alopecia during anti-TNF treatment have been previously reported. Similar histopathological features to our patient have only been described in 4 women with Crohn disease [1, 2], in which the alopecia may clinically and histologically mimic psoriatic alopecia and alopecia areata but can be histologically distinguished from primary psoriasis by the presence of plasma cells and eosinophils and from alopecia areata by epidermal psoriasiform changes and dermal plasma cells.

Case Synopsis

A 50-year-old man affected by plaque psoriasis developed scaly psoriasiform patches associated with alopecia on the scalp 10 months after initiating treatment with adalimumab (**Figure 1**). In addition, psoriasiform patches appeared on his upper extremities, previously without lesions. Punch biopsy of the alopecic area on the scalp revealed psoriasiform epidermal changes including hyperplasia, parakeratosis, and neutrophil infiltration. We could also observe alopecia areata-like dermal changes such as an increased catagen/telogen ratio and miniaturized hairs as well as peribulbar lymphocytic inflammation. Along with these findings, there was a dermal inflammatory infiltrate made up of lymphocytes, eosinophils and plasma cells (**Figure 2**). Periodic acid-Schiff (PAS) staining was negative for fungi. The culture showed growth of saprophytic fungi of no significance. The patient was diagnosed with anti-TNF-related alopecia. Treatment with adalimumab was stopped because the lesions continued progressing despite topical clobetasol. The alopecic area and the psoriatic patches significantly

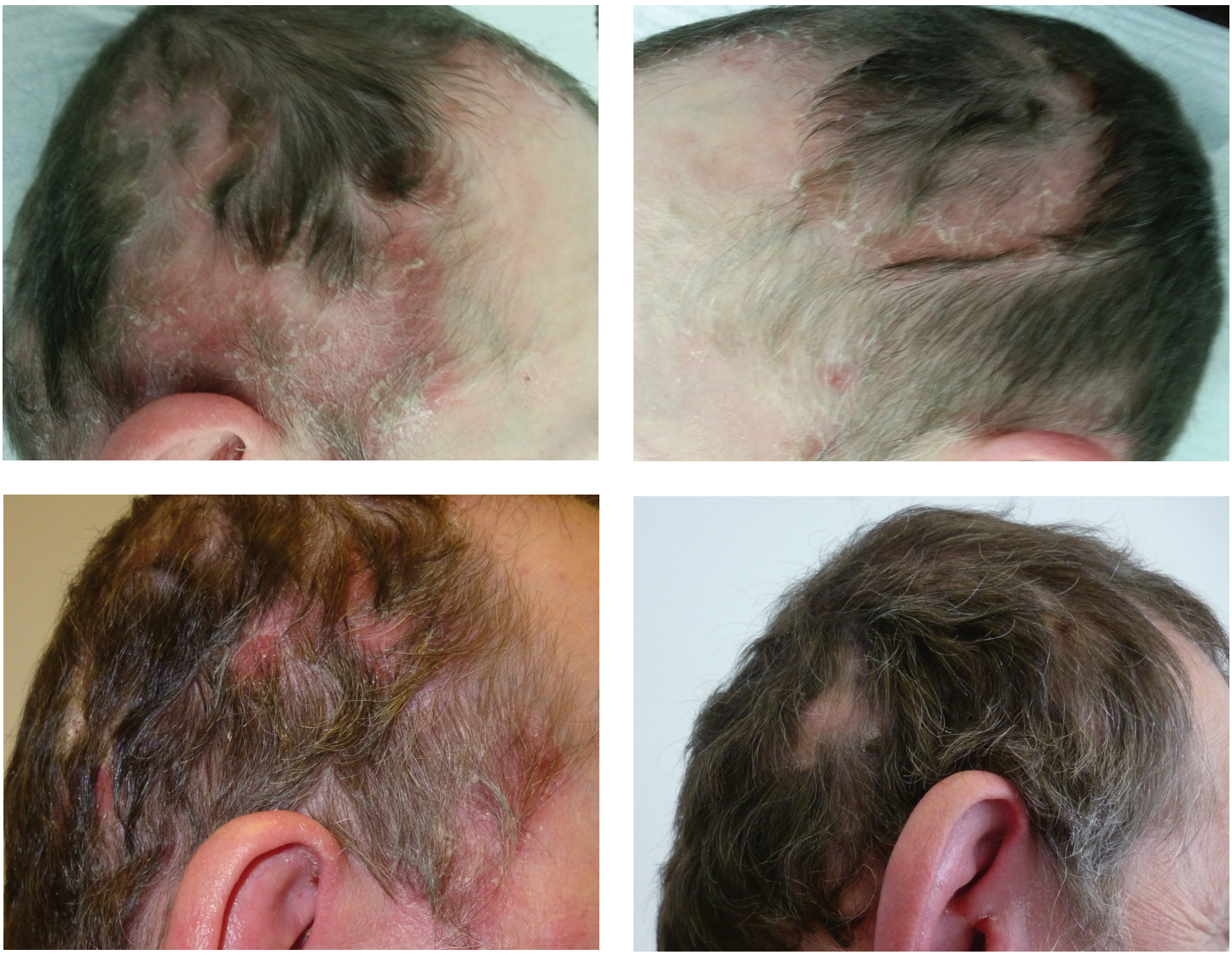

Figure 1. *Anti-TNF-related alopecia. Top) Scaly psoriasiform patches associated with alopecia on the scalp 10 months after initiating treatment with adalimumab. Bottom) The alopecic area significantly improved after stopping adalimumab and initiating topical clobetasol at 3 months (bottom left and at 6 months (bottom right).*

improved at 6 months (**Figure 1**, **bottom right**).

Case Discussion

As the use of anti-TNF drugs increases, an expanding repertoire of adverse effects is becoming evident. In addition to adverse systemic effects on the immune system, anti-TNF agents are also recognized as causing a wide range of cutaneous psoriasifom, granulomatous, lichenoid and eczematous reactions [1,3], cutaneous lymphomas, herpes simplex, bacterial infections, and acute generalized exanthematous pustulosis [3]. A well-recognized adverse effect is alopecia areata [4] and another distinctive cutaneous reaction to anti-TNF therapy is the development of psoriasiform lesions. Adalimumab has been reported to induce a significantly higher rate of this type of lesions, compared to etanercept or infliximab [5].

New-onset psoriasis in patients being treated with anti-TNF agents usually presents as guttate or chronic plaque psoriasis on the trunk, extremities, and scalp, but can also show features of palmoplantar pustulosis [6, 7]. Patients with history of plaque psoriasis tend to develop lesions with a different morphology and located in previously unaffected skin areas [3].

No statistically significant predisposing factors for development of new-onset psoriasis have so far been found, but genetic and environmental triggers could be involved [8, 9]. There are speculations on the mechanism of this paradoxical effect, and one concerns the interplay between TNF and interferon-

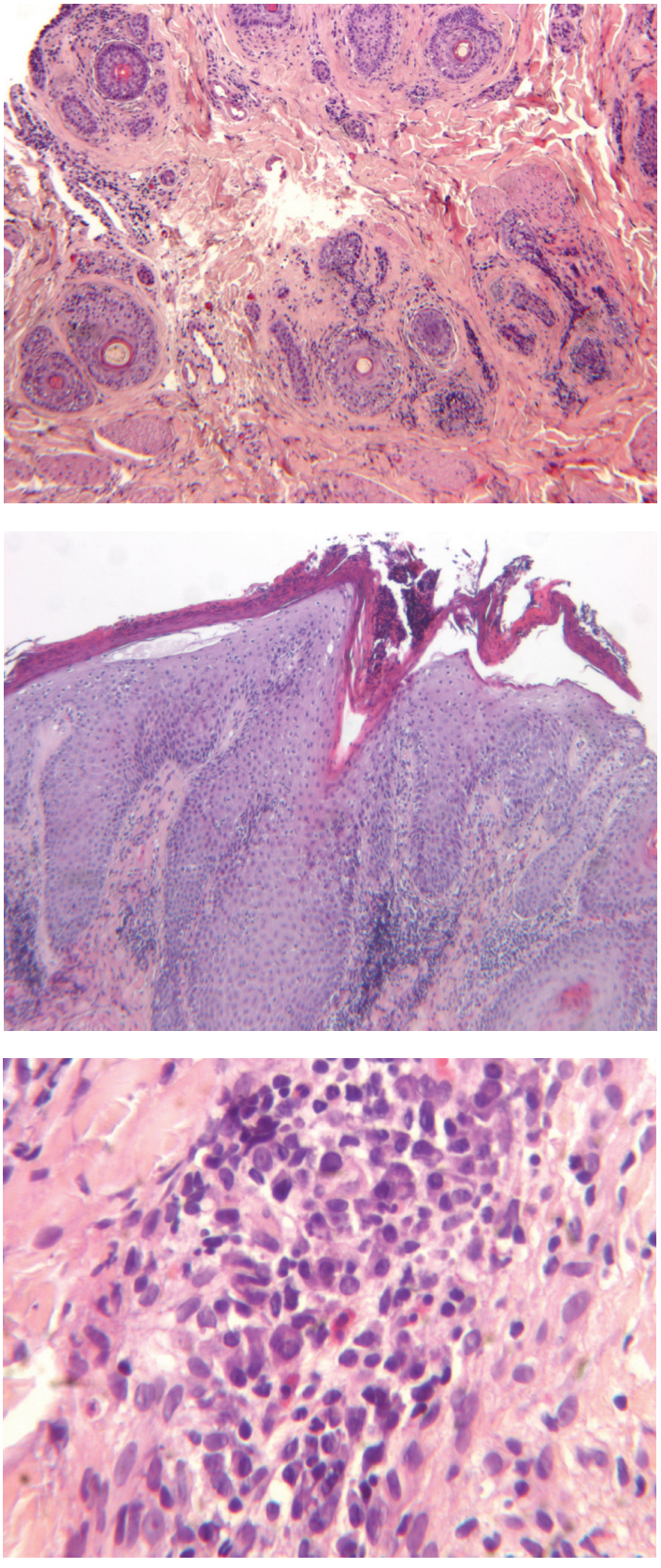

Figure 2. *Anti-TNF-related alopecia histology, H&E. Top) Alopecia areata-like dermal changes: increased catagen/telogen ratio and miniaturized hairs as well as peribulbar lymphocytic inflammation, 10x. Middle) Psoriasiform epidermal changes: hyperplasia, parakeratosis and neutrophil infiltration, 10x. Bottom) Dermal inflammatory infiltrate made up of lymphocytes, eosinophils and plasma cells, 40x.*

alpha (IFNα). Plasmacytoid dendritic cells, the main source of IFNα, are found increased in early psoriatic skin lesions and seem to initiate psoriasis via IFNα production. IFNα subsequently upregulates the expression of CXCR3 on T cells, which leads to recruitment of T cells into the skin. IFNα also activates the secretion of proinflammatory cytokines by myeloid dendritic cells. On the other hand, TNF is known to decrease IFNα production by inhibiting the maturation of plasmacytoid dendritic cells. Therefore, by blocking TNF, IFNα levels rise, explaining the onset of psoriasis in patients with rheumatologic disorders and underlying the complexity of the TNF and IFNα interplay [5, 10]. Other cytokines could also be involved because new-onset psoriasis has also been described as an adverse effect of other biological therapies [3].

The reason why only a small group of patients being treated with anti-TNF therapy develop psoriasiform eruptions, palmoplantar pustulosis, or alopecia is another challenging question, which might involve TNF receptor polymorphisms [7]. Genetic variations in the TNF pathway may also predispose to psoriasis or unmask the disease when susceptible individuals use these agents [10]. Anti-TNF drug-induced alopecia is an adverse effect of this type of drugs and clinically resembles primary psoriatic alopecia or alopecia areata.

The differential diagnosis of this type of alopecia includes telogen effluvium, alopecia areata, primary psoriatic alopecia, and drug reaction. Telogen effluvium is characterized by increased telogen hairs and an absence of inflammation and this is usually easily differentiated by histopathological features [11]. Primary psoriatic alopecia refers to alopecia arising in patients with known primary psoriasis and usually shows classic psoriatic changes in the epidermis features of alopecia areata, as well as scarring alopecia in the dermis. However, eosinophils and plasma cells are rarely present in primary psoriatic alopecia and so their presence is helpful in distinguishing an anti-TNF-induced alopecia from a true primary psoriatic alopecia. Classic alopecia areata shows increased catagen/telogen hairs, increased miniaturized hairs, and a variable degree of peribulbar lymphocytic inflammation. Eosinophils are only occasionally seen scattered in

the inflammatory infiltrate and plasma cells are rarely present. Furthermore, alopecia areata is not usually associated with clinically inflamed lesions or surface psoriasiform changes within the epidermis. When the epidermal psoriasiform changes are less pronounced, the presence of plasma cells within the periadnexal and dermal inflammatory infiltrate seems to a be a helpful feature in distinguishing anti-TNF-induced alopecia from alopecia areata.

To our knowledge, a total of 23 patients of psoriasiform eruptions with severe scalp involvement that induced alopecia during anti-TNF treatment have been previously reported in the English language literature (**Table 1**). All patients were affected by rheumatologic diseases or IBD and most were women. The same histopathological characteristics as our patient had been only described by Doyle et al. [1]; the scalp biopsies of 3 women with Crohn disease revealed epidermal psoriasifom changes and alopecia areata-like changes in the dermis, but can be histologically distinguished from primary psoriasis by the presence of plasma cells and eosinophils, and from alopecia areata by epidermal psoriasiform changes and dermal plasma cells. Recently in this Journal, Toda-Brito et al. [2] described a new similar diagnosis in a woman with Crohn disease. We describe a new patient of anti-TNF-related alopecia in a patient previously affected by psoriasis (Table I). The clinical diagnostic criteria for anti-TNF-related alopecia proposed by Doyle et al. [1] are:

1. Recent initiation of anti-TNF treatment
2. No prior history of psoriasis
3. Flare of psoriasis after starting the anti-TNF treatment
4. Alopecic plaque(s) on the scalp
5. Often, erythematous scaly patches and/or pustular lesions on the scalp and elsewhere on the body

We propose a change in these clinical criteria. The second clinical criterion should not be required because anti-TNF-related alopecia may also appear in patients with psoriasis treated with this type of drugs, as in this patient we describe.

It should be pointed out that recently, Ribeiro et al. [12] described two similar patients with Crohn disease. They had the following discrepancies in the histological findings of the 4 previous patients and our patient: the lymphocytic infiltrate was not restricted to the peribulbar region and eosinophils and plasmocytes were not identified in the inflammatory infiltrate.

This entity may respond to topical treatment. However, in patients with severe scalp involvement, anti-TNF suspension and systemic treatment should be considered in order to avoid scarring alopecia [5], of which there are two patients published. On the other hand, switching from one anti-TNF inhibitor to another may not improve psoriatic lesions, with recurrence rates varying among the series; 48% for Collamer et al. [9], 85% for Cullen et al. [8], and 96% for Rahier et al. [13]. Some authors consider that switching to an alternative anti-TNF treatment may be helpful [8], whereas others do not [13].

Conclusion

In summary, anti-TNF therapy-related alopecia may closely mimic psoriatic alopecia and alopecia areata but can be histologically distinguished from alopecia areata by epidermal psoriasiform changes and dermal plasma cells and from primary psoriasis by the presence of plasma cells and eosinophils [1]. The importance of recognizing this entity lies in the implications it carries for patient management. Specialists who manage patients treated with anti-TNF should be aware of this drug-associated complication in order to ensure faster diagnosis, prevent needless treatments, and initiate an appropriate therapy rapidly [10].The decision whether to continue anti-TNF therapy depends on the severity of the psoriasis, the effect of treatment modification on the primary disease, and the risk-benefit ratio of alternative forms of therapy [10].

References

1. Doyle LA, Sperling LC, Baksh S, et al. Psoriatic alopecia/alopecia areata-like reactions secondary to anti-tumor necrosis factor-α therapy: a novel cause of noncicatricial alopecia. Am J Dermatopathol. 2011;33(2):161-166. [PMID: 21317611].
2. Toda-Brito H, Lopes L, Soares-Almeida L, et al. Adalimumab-induced psoriatic alopecia/alopecia areata-like reaction in a patient with Crohn's disease. Dermatol Online J. 2015;21(11):6-8.[PMID: 26632930].
3. Collamer AN, Guerrero KT, Henning JS, et al. Psoriatic skin lesions induced by tumor necrosis factor antagonist therapy: a literature review and potential mechanisms of action. Arthritis Rheum. 2008;59(7):996-1001. [PMID: 18576309].

4. Tauber M, Buche S, Reygagne P, et al. Alopecia areata occurring during anti-TNF therapy: a national multicenter prospective study. J Am Acad Dermatol. 2014;70(6):1146-1149. [PMID: 24831323].
5. Osório F, Magro F, Lisboa C. Anti-TNF-alpha induced psoriasiform eruptions with severe scalp involvement and alopecia: report of five cases and review of the literature. Dermatology. 2012;225(2):163-167. [PMID: 23052429].
6. Lee HH, Song IH, Friedrich M, et al. Cutaneous side-effects in patients with rheumatic diseases during application of tumour necrosis factor-alpha antagonists. Br J Dermatol. 2007;156(3):486-491. [PMID: 7300238].
7. El Shabrawi-Caelen L, La Placa M, Vincenzi C, et al. Adalimumab-induced psoriasis of the scalp with diffuse alopecia: a severe potentially irreversible cutaneous side effect of TNF-alpha blockers. Inflamm Bowel Dis. 2010;16(2):182-183. [PMID: 19462433].
8. Cullen G, Kroshinsky D, Cheifetz AS, et al. Psoriasis associated with anti-tumour necrosis factor therapy in inflammatory bowel disease: a new series and a review of 120 cases from the literature. Aliment Pharmacol Ther. 2011;34(11-12):1318-1327. [PMID: 21957906].
9. Collamer AN, Battafarano DF. Psoriatic skin lesions induced by tumor necrosis factor antagonist therapy: clinical features and possible immunopathogenesis. Semin Arthritis Rheum. 2010;40(3):233-240. [PMID: 20580412].
10. Perman MJ, Lovell DJ. Five cases of anti-tumor necrosis factor alpha-induced psoriasis presenting with severe scalp involvement in children. Pediatr Dermatol. 2012;29(4):454-459. [PMID: 21995665].
11. Weedon D. Skin Pathology. 3rd ed. London. UK: Churchill Livingstone; 2010;421-422.
12. Ribeiro LB, Rego JC, Estrada BD, Bastos PR, Piñeiro Maceira JM, Sodré CT. Alopecia secondary to anti-tumor necrosis factor-alpha therapy. An Bras Dermatol. 2015;90(2):232-5. [PMID: 25830994].
13. Rahier JF, Buche S, Peyrin-Biroulet L, et al. Severe skin lesions cause patients with inflammatory bowel disease to discontinue anti-tumor necrosis factor therapy. Clin Gastroenterol Hepatol. 2010;8(12):1048-1055. [PMID: 20728573].
14. Papadavid E, Gazi S. Palmoplantar and scalp psoriasis occurring during anti-tumour necrosis factor-alpha therapy: a case series of four patients and guidelines for management. J Eur Acad Dermatol Venereol. 2008;22(3):380-382. [PMID: 18269613].
15. Manni E, Barachini P. Psoriasis induced by infliximab in a patient suffering from Crohn's disease. Int J Immunopathol Pharmacol. 2009;22(3):841-844. [PMID: 19822070].
16. Medkour F, Babai S, Chanteloup E, et al. Development of diffuse psoriasis with alopecia during treatment of Crohn's disease with infliximab. Gastroenterol Clin Biol. 2010;34(2):140-141. [PMID: 20138448].
17. Andrisani G, Marzo M, Celleno L. Development of psoriasis scalp with alopecia during treatment of Crohn's disease with infliximab and rapid response to both diseases to ustekinumab. Eur Rev Med Pharmacol Sci. 2013;17(20):2831-2836. [PMID: 24174369].
18. Udkoff J, Cohen PR. Severe Infliximab-Induced Alopecia and Scalp Psoriasis in a Woman with Crohn's Disease: Dramatic Improvement after Drug Discontinuation and Treatment with Adjuvant Systemic and Topical Therapies. Dermatol Ther (Heidelb). 2016;6(4):689-695. [PMID: 27844446].

Table 1. *Summary of patients of psoriasiform eruptions with severe scalp involvement that induced alopecia during anti-TNF treatment.*

	Reference	Sex	Age	Disease	Anti-TNFα	Concomitant IT	Psoriasiform eruptions	Alopecia	Latency months	Personal history psoriasis	Family history psoriasis	Manage-ment	Stop anti-TNFα	Outcome after management	Histopath of the scalp
1	Papadavid *et al*., 2008 [14]	F	30	AS	ADA	Salazopyrina	palmoplantar pustulosis, scalp, trunk	significant hair loss	NM	NM	NM	stop ADA, topical CTC, CyA	yes	improvement	features of pso-riasis
2	El Shabra-wi-Caelen *et al*., 2010 [7]	F	19	CD	ADA	no	scalp, trunk, abdomen, extremities	difussed alopecia	3	NM	NM	stop ADA	yes	clearance	two biopsies: fea-tures of psoriasis, without any evi-dence of scarring alopecia
3	El Shabra-wi-Caelen *et al*., 2010 [7]	F	31	CD	ADA	NM	scalp	pro-gressive alopecia, confluent areas of scaly psoriatic plaques with shiny and atro-phic skin and loss of follicular orifices	2	NM	NM	NM	NM	scarring alo-pecia	scarring alopecia
4	Manni *et al*,. 2009 [15]	F	30	CD	IFX	NM	palmoplantar pustulosis, trunk, arms, scalp	alopecia	3	no	no	topical treatment, stop IFX and CyA	yes	nearly clear	NM
5	Medekour *et al*., 2010 [16]	M	32	CD	IFX	AZA	palmoplantar and scalp pus-tulosis, diffuse plaque-type	inflam-matory pustulosis on the scalp with extensive alopecia	10	NM	NM	topical treatment and stop IFX	yes	clearance	two biopsies consistent with psoriasis: minimal lymphoid perifol-licular infiltrate with follicular atrophy and a parakeratosis and superficial dermic perivascular infiltrates.

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	Reference	Sex	Age	Disease	Anti-TNFα	Concomitant IT	Psoriasiform eruptions	Alopecia	Latency months	Personal history psoriasis	Family history psoriasis	Manage-ment	Stop anti-TNFα	Outcome after management	Histopath of the scalp
6	Perman *et al*,. 2012 [10]	F	7	JIA	ADA	NM	scalp (pustular and plaque-type) ears and heels	sever-al-cen-timeter, boggy, erythem-atous, erodied, hemor-rhagically crusted alopecic plaque on the crown.	9	no	no	topical treat-ment and systemic ATB, CyA 8 months, switch to ABA, MTX at time to submission (for primary disease)	yes (switch)	scarring alo-pecia	suggestive of early psoriasis. PAS (-).
7	Perman *et al*,. 2012 [10]	M	11	CD	IFX	NM	scalp > trunk, genitals		26	no	no	topical treatment and sys-temic ATB	no	clearance	NM
8	Perman *et al*,. 2012 [10]	M	16	UC	IFX	NM	scalp > face, trunk, buttocks, extremities, palmoplantar	erythema-tous, scaly, eroded and crusted alopecic plaques	5	no	no	topical treament, switch to ADA (for primary disease)	yes (switch)	clearance	NM
9	Perman *et al*,. 2012 [10]	F	14	CD	IFX	NM	scalp, face		2	no	yes	topical treatment, stop IFX, MTX (for primary disease)	yes	neraly clearance	NM
10	Perman *et al*,. 2012 [10]	F	18	CD	IFX	NM	scalp, postauri-cular		7	no	no	topical treatment, stop IFX	yes	no improve-ment	NM

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11	Osório *et al.*, 2012 [5]	F	26	CD	IFX	no	scalp, axillae, umbilicus	scalp psoriasifom eruption of an erythematous and scaly nature, with hyperkeratotik and exudative lesions, inducing non-scarring alopecia	3	no	no	topical treatment, switch to ADA, MTX	yes (switch)	flare-ups	regular acanthosis with hyperkeratosis and a variable number of neutrophils in the epidermis, as well as mixted or predominantly lymphocytic infiltrate in the dermis.
					ADA							topical treatment, switch to IFX	yes (switch)	flare-ups	
					IFX							topical treatment and MTX	no	flare-ups	
12	Osório *et al.*, 2012 [5]	F	31	CD	IFX	AZA	scalp, axillae, genitals		9	no	yes	topical trea-ment,stop IFX, MTX (2 months)	yes	clearance	
13	Osório *et al.*, 2012 [5]	F	23	CD	ADA	AZA	scalp, axillae		46	no	no	topical treatment	no	neraly clearance	
14	Osório *et al.*, 2012 [5]	M	30	CD	ADA	AZA	scalp, axillae, genitals		11	no	no	topical treatment, CTC (1 month) and stop ADA,MTX (3 months)	yes	clearance	
15	Osório *et al.*, 2012 [5]	F	25	CD	ADA	no	scalp, axillae, inframammary folds, genitals		2	no	no	topical treatment and MTX, CTC (2 weeks) and MTX, CyA	yes	mild improve-ment	

	Reference	Sex	Age	Disease	Anti-TNFα	Concomitant IT	Psoriasiform eruptions	Alopecia	Latency months	Personal history psoriasis	Family history psoriasis	Manage-ment	Stop anti-TNFα	Outcome after management	Histopath of the scalp
16	Doyle *et al.*, 2011 [1]	F	21	CD	IFX	NM	trunk, extremities. Later: scalp	large scaly alopecic plaques on the scalp	2	NM	NM	intralesional triamcinolona and topical CTC.	no	clearance (but the plaques on the body occasionally flared and eventually evolved into pustular psoriasiform lesions)	psoriasiform epidermal hyperplasia, confluent parakeratosis containing neutrophils, similar seen in psoriasis. Increased number of catagen/telogen and miniaturized hair follicles, with peribulbar lymphocytic inflammation, similar to alopecia areata. Also, peribulbar infiltrate contained eosinophils and numerous plasma cells. PAS (-). Steiner(-)
17	Doyle *et al.*, 2011 [1]	F	27	CD	IFX	NM	papulo-pustular eruption on the scalp, palms, extremities, axillae and groin.	scalp alopecia	NM	NM	NM	topical treatment and UVB, stop IFX,certolizumab pegol	yes	hair regrowth	2 scalp biopsies showed increased catagen/telogen hair follicles and hair miniaturization. The remaining hairs showed peribulbar inflamation including lymphocytes and numerous plasma cells. The superficial portion showed pustular psoriasis-like changes and increased number of eosinophils. PAS (-). Steiner (-)

	Reference	Sex	Age	Disease	Anti-TNFα	Concomitant IT	Psoriasiform eruptions	Alopecia	Latency months	Personal history psoriasis	Family history psoriasis	Manage-ment	Stop anti-TNFα	Outcome after management	Histopath of the scalp
18	Doyle *et al.*, 2011 [1]	F	39	CD	ADA	NM	scalp	large scaly psoria-siform plaques on the scalp with significant alopecia	NM	NM	NM	topical treatment	no	dramatic im-provement	increased catagen/telogen hair follicles and miniaturization of hairs. Lymph-oplasmacytic inflammation of the residual fibrous stelae. The superficial aspect showed mild psoriasiform hyperplasia, minimal vacuolar interfase changes and a superficial predominantly lymphoid infiltrate with occasional eosinophils and plasma cells. PAS (-). Steiner (-).
19	Andrisani *et al.*, 2013 [17]	F	26	CD	IFX	no	scalp	diffuse er-ythemao-tous scaly plaque on the scalp with severe hair loss and intense itching	2	no	no	stop IFX, topics and oral CTC, UST	yes (switch)	clearance	2 biopsies with characteristics of psoriasis without any evidence of scarring alopecia: lymphoid perifol-licular infiltrate with follicular atrophy and a parakeratosis and superficial dermic perivascular infiltrates.
20	Toda-Brito *et al.*, 2015 [2]	F	24	CD	ADA	no	scalp, limbs, palms and soles	patches on the scalp with exudative discharge and pro-gressive alopecia	7	no	no	stop ADA, topical treatment	yes	complete hair regrowth	psoriasiform epi-dermal changes in association with alopecia areata-like changes

	Reference	Sex	Age	Disease	Anti-TNFα	Concomitant IT	Psoriasiform eruptions	Alopecia	Latency months	Personal history psoriasis	Family history psoriasis	Manage-ment	Stop anti-TNFα	Outcome after management	Histopath of the scalp
21	Ribeiro LB *et al*,2015 [12]	M	28	CD	IFX	no	scalp, armpits, naval, perianal	2 alopecia plaques in parietal region. The oldest lesions showed aspects of AA and the most recent presented erythe-ma and desqua-mation	36	no	no	topics and intrale-sional CTC, coal tar shampoo	no	complete hair regrowth	extensive paraker-atosis, epidermal hyperplasia, tortuous capil-laries and mononucle-ar perifollicular inflammatory infiltrate, and intense miniatur-ization
22	Ribeiro LB *et al*.,2015 [12]	F	14	CD	IFX	no	scalp, trunk, armpits, pubic region, breasts, plantar region, elbows, knees	scaly pso-riasiform plaques with alopecia on the scalp with tortuous vessels	4	no	no	topical CTC, coal tar shampoo	no	hair regrowth	hyperkeratosis pronounced miniaturization with only 50% of hair terminals in anagen and mononuclear infiltrate, discrete perivascular and multifocal intrafollicular
23	Udkoff J *et al* [18]	F	23	CD	IFX	AZA	scalp	diffuse alopecia and ery-thematous plaque with scaling that pro-gressed and devel-opment of pruritic le-sions that covered the scalp	8	NM	NM	Stop IFX, oral mi-nocycline, topical CTC, coal tar shampoo with keto-conazole and salicyl-ic acid, UST	yes	improvement	chronic folliculitis and perifolliculitis with dermal scar-ring and naked hair shafts in the dermal stroma. Histologically consistent with folliculitis decal-vans but clinically suggestive of alopecia and scalp psoriasis second-ary to IFX

	Reference	Sex	Age	Disease	Anti-TNFα	Concomitant IT	Psoriasiform eruptions	Alopecia	Latency months	Personal history psoriasis	Family history psoriasis	Manage-ment	Stop anti-TNFα	Outcome after management	Histopath of the scalp
24	Our patient	M	50	PS	ADA	no	scalp, arms	scaly psoriasiform plaques with alopecia on the scalp	10	yes	no	stop ADA, topical CTC, CyA	yes	improvement	epidermal changes included hyperplasia, parakeratosis and neutrophils. The dermal inflammatory infiltrate contained lymphocytes, eosinophils and plasma cells. In addition, we observed increased in catagen /telogen and miniaturized hairs and peribulbar lymphocytic inflammation. PAS (-).

ATB = antibiotic; ABA = abatacept; ADA = adalimumab; RA = rheumatoid arthritis; AS = ankylosing spondylitis; AZA= azathioprine; CD = Crohn's disease;

CTC = corticosteroids; CyA = ciclosporine; IFX = infliximab; IR = immunosuppressive therapy; JIA = juvenile idiopathic arthritis; MTX = methotrexate;

NM = not mentioned; PS: psoriasis; UC = ulcerative colitis; UST= ustekinumab