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Research Article

Paradoxical Psoriasis: The Flip Side of TNF-α Inhibitors

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Abstract

Background : Tumor necrosis factor-alpha inhibitors (anti-TNF-α) have revolutionized the management of chronic autoimmune diseases, including inflammatory bowel disease, rheumatoid arthritis, psoriasis, and psoriatic arthritis. Despite their efficacy, these agents have paradoxically been associated with the induction or exacerbation of psoriatic lesions, a phenomenon increasingly reported in the literature.

Objective : This study aimed to describe the epidemiological and clinical characteristics of paradoxical psoriasis induced by anti-TNF-α therapy, analyze the therapeutic approaches employed, and assess patient outcomes.

Methods : We conducted a retrospective analysis of 10 cases of paradoxical psoriasis reported to the National Center of Pharmacovigilance, Chalbi Belkahia (Tunis, Tunisia), between June 2018 and June 2023. Cases were selected from a database connected with VigiBase® and analyzed using the French method of

imputability. Only cases where anti-TNF-α agents were strongly suspected of inducing or exacerbating psoriasis were included.

Results : The study included 10 patients (7 women, 3 men) with a mean age of 42.8 years. Eight patients had no prior history of psoriasis before starting anti-TNF-α therapy. Psoriatic lesions appeared after an average of 25.6 months (range: 2 months-6 years) following treatment initiation. Clinical presentations included plaque psoriasis (4 patients) and pustular psoriasis (4 patients), while guttate and inverse forms were less frequent. The most frequently affected underlying conditions were ankylosing spondylitis (n=3), rheumatoid arthritis (n=3), psoriatic arthritis (n=2), and Crohn's disease (n=2). Management strategies varied: anti-TNF-α therapy was discontinued in 8 patients, leading to remission in 7. Four patients were switched to another anti-TNF-α agent, and none experienced recurrence.

Conclusion : Paradoxical psoriasis represents a significant adverse effect of anti-TNF-α therapy, with variable clinical presentations and management outcomes. Although switching to a different anti-TNF-α agent may be a viable option, careful monitoring and individualized treatment strategies are essential for optimal outcomes. Further studies are needed to clarify underlying mechanisms and identify potential risk factors for this paradoxical reaction.

Keywords: psoriasis; paradoxical reaction; TNF-α inhibitor; chronic autoimmune diseases; pharmacovigilance

INTRODUCTION :

The management of various chronic autoimmune diseases, including inflammatory bowel disease and rheumatoid arthritis, has been revolutionized by tumor necrosis factor-alpha (TNF-α) inhibitors [1]. This group of drugs includes infliximab, etanercept, adalimumab, and more recently certolizumab and golimumab. Commonly reported adverse effects include autoimmune disorders, severe infections, and neoplasms [2]. Recently, these agents have also gained approval for treating psoriasis and psoriatic arthritis [3]. However, an increasing number of published case reports suggest that TNF-α inhibitors may paradoxically induce new-onset psoriasis

or worsen pre-existing psoriatic lesions in individuals undergoing anti-TNF-α therapy [4]. Notably, these adverse effects have been associated with the pharmacological class rather than any specific drug, as evidenced by cases involving different anti-TNF-α agents [5]. Paradoxical psoriasis occurs in approximately 5% of patients receiving TNF-α inhibitors [6, 7].

This study aimed to describe the epidemiological and clinical characteristics of cases of paradoxical psoriasis associated with TNF-α inhibitors reported to the National Pharmacovigilance Center in Tunisia, and to explore management options and assess patient outcomes.

Generating such data is essential to enhance drug safety monitoring, foster early recognition of adverse effects, and guide therapeutic decision-making in daily practice. In Tunisia, where paradoxical psoriasis remains poorly documented, these findings can reinforce pharmacovigilance systems and evidence-based prescribing. At the international level, they add to the growing body of real-world data informing global clinical pharmacy practice. Ultimately, our study conveys a key message to policymakers and clinical pharmacists : paradoxical adverse reactions should be systematically integrated into treatment guidelines, patient counseling, and pharmacovigilance policies to ensure safer and more rational use of TNF-α inhibitors.

METHODS:

This retrospective study analyzed cases notified to the National Pharmacovigilance Center, Chalbi Belkahia (Tunis, Tunisia), between June 2018 and June 2023. We first identified cases in which anti-TNF-α agents were suspected of either inducing de novo psoriasis or exacerbating preexisting psoriasis, using our database, which is directly connected with VigiBase®. Cases were included if they contained sufficient clinical, therapeutic, and follow-up information to support analysis. Exclusion criteria were incomplete reports, alternative etiologies for psoriatic lesions (infections, concomitant medications), or uncertain exposure timelines.

For causality assessment, we applied the French imputability method [8], which evaluates each drug individually. This method assigns:

- Chronological score (C0–C3): evaluates the temporal relationship between drug exposure and reaction onset, the evolution after withdrawal or continuation, and the response to re-exposure.
- Semiological score (S0–-S3): considers clinical features and biological findings suggestive of drug involvement.
- Bibliographic score (B0–B3): reflects the level of documentation of the adverse reaction in the scientific literature.

The intrinsic imputability score (I = C + S) was calculated for each case, and then combined with the bibliographic score to determine the final causality assessment.

Over the 6-year study period, 10 cases of anti-TNF-α–induced or –exacerbated psoriasis were identified among 18,750 cases reported to our pharmacovigilance department, corresponding to a frequency of 0.05% (Figure 1).

Statistical Analysis

Data were analyzed using descriptive statistics. Categorical variables are presented as frequencies and percentages, while continuous variables are expressed as means with ranges (or medians with interquartile ranges, if applicable). Given the limited sample size, no inferential statistical tests were applied.

RESULTS:

Our study included ten patients: seven women and three men (Table 1). The mean age was 42.8 years (range: 13–66 years). Comorbidities included diabetes in three patients, hypertension and dyslipidemia in one patient, and cutaneous leishmaniasis in another. There was no family history of psoriasis except in one case (case 5). Three patients already had mild, stable psoriatic eruptions that spread after the administration of anti-TNF-α agents; two were being treated for psoriatic arthritis and the third for Crohn’s disease, with a personal history of inverse psoriasis well controlled by corticosteroids. The remaining seven patients had no known personal history of psoriasis before initiation of anti-TNF-α treatment. Three patients were treated for ankylosing spondylitis (AS), three for rheumatoid arthritis (RA), and one for Crohn’s disease (CD). The duration of underlying inflammatory diseases ranged from one year to 25 years.

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As the first anti-TNF-α therapy, four patients received adalimumab, three received certolizumab, two received etanercept and one received infliximab. Anti-TNF-α drugs were associated with other immunosuppressive medications in six cases: corticosteroids (n=3), azathioprine (n=2) and methotrexate (n=1). The time of onset of paradoxical psoriasis ranged from two months to six years after starting anti-TNF-α treatment, with a mean onset time of 25.6 months after drug initiation. The time of onset after the last administration was specified in four cases, ranging from two to 20 days.

Clinical presentations varied among patients. Plaque psoriasis (Figure 2) and pustular psoriasis (Figure 3) were each reported in four cases. One patient developed guttate lesions (Figure 4), and another presented with exacerbation of inverse psoriasis. Psoriatic eruption mainly affected the scalp, back, hands, thighs, legs and less frequently the face, chest, axillary folds, elbows and buttocks. No nail involvement was observed. Three cases were confirmed histologically, showing lymphocytic infiltrates in the epidermis, epithelial hyperplasia with acanthosis and hyperkeratosis with parakeratosis.

Management varied among patients. The same anti-TNF-α agent was continued in two cases. One patient achieved significant improvement within one month, with complete resolution during the one-year-follo-up (case 8). The other experienced psoriasis flares controlled by systemic corticosteroids (case 2). Anti-TNF-α therapy was discontinued in eight cases (cases 1, 3, 4, 5, 6, 7, 9 and 10). All but one achieved complete remission of skin lesions after drug withdrawal ; in one case, lesions persisted for over two years despite cessation (case 1). Among the seven cases with favorable outcome, remission ocuured within a period of one to several months, achieved with topical corticosteroids in six patients and methotrexate in one case (case 4).

Alternative strategies also varied. In three cases, the incriminated anti-TNF-α agent was switched to another anti-TNF-α with no recurrence. For example, one patient improved after stopping adalimumab and was remained psoriasis-free on certolizumab (case 4). Another improved after etanercept withdrawal and tolerated infliximab without relapse (case 5). In a third case, lesions resolved after stopping

certolizumab and infliximab was introduced without recurrence (case 6). Adalimumab was successfully reintroduced in one patient (case 3). The remaining four patients received alternative immunosuppressive therapies (cases 1, 7, 9 and 10) with no further relapse of psoriatic eruptions.

DISCUSSION:

Paradoxical psoriasis following anti-TNF-α therapy has been well described in the literature. A large prospective observational study demonstrated a higher incidence of new-onset psoriasis in patients receiving anti-TNF-α therapy compared to those on traditional disease-modifying antirheumatic drugs (DMARDs) [9], with prior reports estimating 2%–5% of patients developing this adverse effect [10]. The incidence varies by underlying disease, ranging from 1.04–2.31 per 1,000 person-years in RA, 0.5–1% in AS, to 3.7% in inflammatory bowel disease (IBD) [11–13].

Paradoxical psoriasis affects both sexes without a clear age effect, although higher risks have been noted in females, younger patients, smokers and IBD patients [3, 13, 14]. In our case series, the female predominance (F/M=2.3) reflects this trend, likely influenced by the high proportion of female patients included. Most reported cases, including ours, lack a personal or family history of psoriasis [10, 15], though some studies suggest family history may increase risk [16]. In our series, patients primarily received anti-TNF-α therapy for inflammatory rheumatic arthritis (three ankylosing spondylitis, two rheumatoid arthritis, two psoriatic arthritis), and IBD (two Crohn's disease). Paradoxical psoriasis may occur in all diseases treated with TNF-α inhibitors, though prevalence appears higher in RA [15]. Lesion onset is variable, ranging from days to years after treatment initiation. In our cases, most lesions appeared within the first year; however, patients with a prior history of psoriasis may develop lesions earlier, as reported in the literature [10, 15]. In addition, skin lesions tend to appear sooner in IBD patients than in RA patients, with an average onset of three months versus nine months [17]. Interestingly, in our Crohn's disease patients, lesion onset timing resembled that in RA rather than typical IBD patterns.

Regarding drug-specific risk, infliximab is the most widely used, but paradoxical psoriasis is frequently reported with adalimumab [3, 17]. Literature reports mixed findings for etanercept and golimumab, with few dermatological events for the latter

[4, 9, 18, 19]. Recent studies suggest that certolizumab is the least associated with the development of paradoxical reactions [20]. In our series, one case was successfully managed by switching from adalimumab to certolizumab, highlighting its potential as an effective alternative [21].

Clinical presentation of paradoxical psoriasis is heterogeneous. Pustular palmoplantar psoriasis predominates (>50%), followed by plaque (14.7%) and guttate forms (10.9%) [2, 15, **22**]. Commonly affected areas include palms, soles, scalp and trunk, with occasional nail involvement [4]. Patients with prior psoriasis often develop lesions of a different morphology [23], whereas in our series, three such patients exhibited the same pattern. Paradoxical psoriasis differs from classic psoriasis by more frequent flexural and palmoplantar involvement and distinct histopathological patterns combining psoriatic, eczematiform, and lichenoid features [24]. Laboratory studies report elevated interferon-alpha (IFN-α) and IL-17A–expressing T cells, correlating with disease severity [16].

Management depends on skin severity and underlying disease control. Mild cases may continue anti-TNF therapy with topical/systemic psoriasis treatments. Worsening lesions or unstable primary disease may require switching TNF- α agents or moving to a different biologic class [16, 22, 24, 25]. In our series, eight patients discontinued therapy, resulting in lesion improvement, while two continued treatment ; one experienced flare, and the other improved. Literature indicates that rechallenge with the same class often results in recurrence, although some patients tolerate switches to another TNF-α inhibitor without relapse [15, 22, 23, 26]

The pathogenesis of paradoxical psoriasis likely involves complex immunologic mechanisms [17]. TNF-α inhibition disrupts cytokine balance, leading to overproduction of INFα by plasmacytoid dendritic cells, triggering innate immune responses independent of T cells [11, 16, 24]. This mechanism is supported by our observations of rapid lesion onset and the resolution of psoriasis after discontinuation of therapy, suggesting a direct link to TNF-α blockade. Genetic susceptibility also contributes, with SNPs in TNF-α, TNFR1B, TNFAIP3, IL23R, FBXL19, CTLA4, SLC12A8, and TAP1 implicated. Notably, the rare C allele of TNF-α rs1799964 may increase risk, particularly in IBD patients receiving adalimumab [16].

Strengths and limitations

This case series provides valuable real-world data on anti-TNF-α–induced paradoxical psoriasis in Tunisian patients, a region with limited published evidence. Cases were systematically collected through the National Pharmacovigilance Center, and the series captures a diverse clinical presentations and therapeutic responses, offering practical guidance for clinicians.

Limitations include the small sample size, retrospective design, and heterogeneity of underlying diseases and treatment regimens. Long-term follow-up was limited, and some clinical details may be incomplete. Comparisons with other populations relied on previously published studies, which may affect generalizability. Additionally, the lack of mechanistic or genetic analyses prevents a deeper understanding of pathogenesis, and the absence of a control group restricts the ability to quantify relative risk. Despite these constraints, the study provides meaningful insights into the clinical spectrum, management, and outcomes of paradoxical psoriasis in a local context.

Conclusion :

Paradoxical psoriasis is an uncommon but important adverse reaction to anti-TNF-α therapy, with variable clinical presentations and patient responses. Clinicians should monitor patients closely, recognize early lesions, and report cases to pharmacovigilance systems. Management should be tailored : mild cases may be treated symptomatically while continuing therapy, whereas severe or persistent lesions may require switching to another anti-TNF-α agent or a different biologic class. Awareness, timely recognition, reporting, and individualized treatment are key to optimizing patient safety and therapeutic outcomes.

List of abbreviations:

AS: ankylosing spondylitis

AGEP: acute generalized exanthematous pustulosis

CD: Crohn’s disease

DMARD: disease-modifying antirheumatic drug

IBD: inflammatory bowel disease

IFN-α: interferon-alpha

RA: rheumatoid arthritis

TNF-α: tumor necrosis factor-alpha

Author Contributions:

YSM and MBB: Writing-original draft; FMZ: Writing-review and editing; ID: Investigation; IA: Supervision; SEA: Validation, YSM: visualisation. All authors have read and agreed to the published version of the manuscript.

Availability of Data and Material : Data are available from the corresponding author on request.

Ethics Committee Approval and Consent to participate:

This study was conducted using anonymized pharmacovigilance data reported to the National Pharmacovigilance Center (CNPV), Tunisia. According to national regulations, retrospective analyses of anonymized pharmacovigilance reports do not require additional approval from an ethics committee. Therefore, separate ethical approval and informed consent from patients were not applicable.

Conflicts of Interest :

All authors declare no conflicts of interest.

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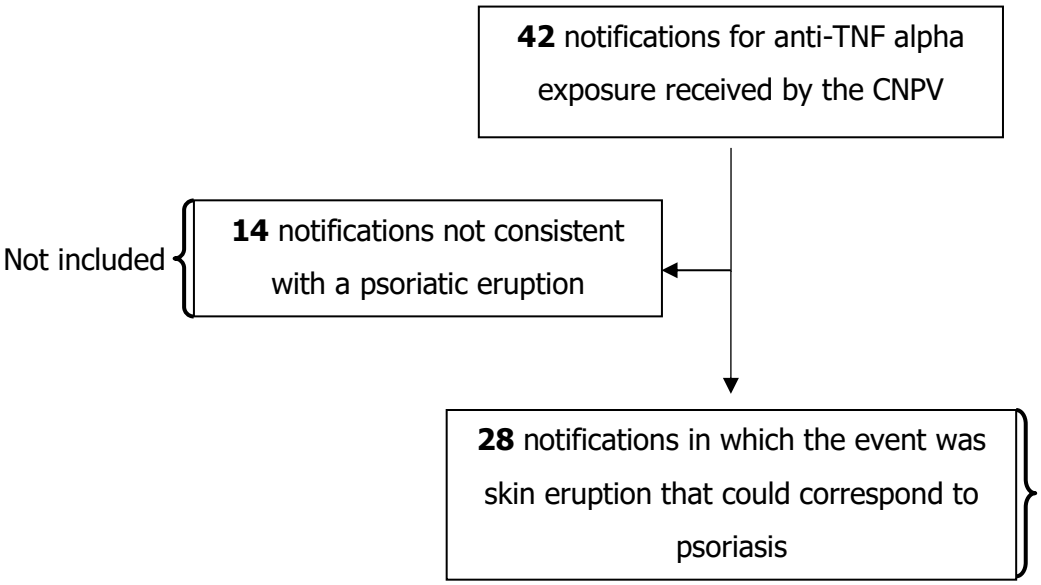
Table 1 : Main characteristics of our ten patients

Case number	Gender	Age	Inflammatory disease	History of psoriasis	Type of anti TNF-α	Delay of onset	Spreading of lesions	Type of psoriasis	Concomitant treatment	Management	Outcome after management	Alternative
1	F	49	RA	No	etanercept	2 years	legs, back	Pustular	None	Discontinuation	Persistance (> 2 years)	MTX
2	F	62	PA	personal	adalimumab	6 years	Buttocks lower limbs hands	Pustular	MTX	Continuation	Fluctuation of lesions	adalimumab
3	F	39	AS	No	adalimumab	10 months	scalp	Plaque	None	Discontinuation	Negative rechallenge	adalimumab
4	F	30	PA	personal	adalimumab	3 years		Plaque	MTX	Discontinuation	Regression No recurrence	certolizumab
5	M	40	AS	familial	etanercept	few months	scalp elbows lower limbs	Guettae	Azathioprine	Discontinuation	Regression No recurrence	infliximab
6	F	56	RA	No	certolizumab	3 months	Upper limbs thighs neckline	Plaque	None	Discontinuation	Regression No recurrence	infliximab
7	F	32	CD	Personal	infliximab	6 years	folds	Inverse	None	Discontinuation	Regression No recurrence	NS
8	M	47	AS	No	certolizumab	8 months	Palmoplantar Lower limbs	pustular	None	Continuation	Stabilisation of lesions	certolizumab
9	M	13	CD	No	adalimumab	2 months	trunk	pustular	None	Discontinuation	Regression No recurrence	NS
10	F	60	RA	No	certolizumab	2 years	back	Plaque	None	Discontinuation	Regression No recurrence	NS

F : female ; RA : rheumatoid arthritis ; MTX : methotrexate ; PA : psoriatic arthritis ; AS : ankylosing spondylitis ; M : male ; CD : Crohn's disease ; NS : not specified



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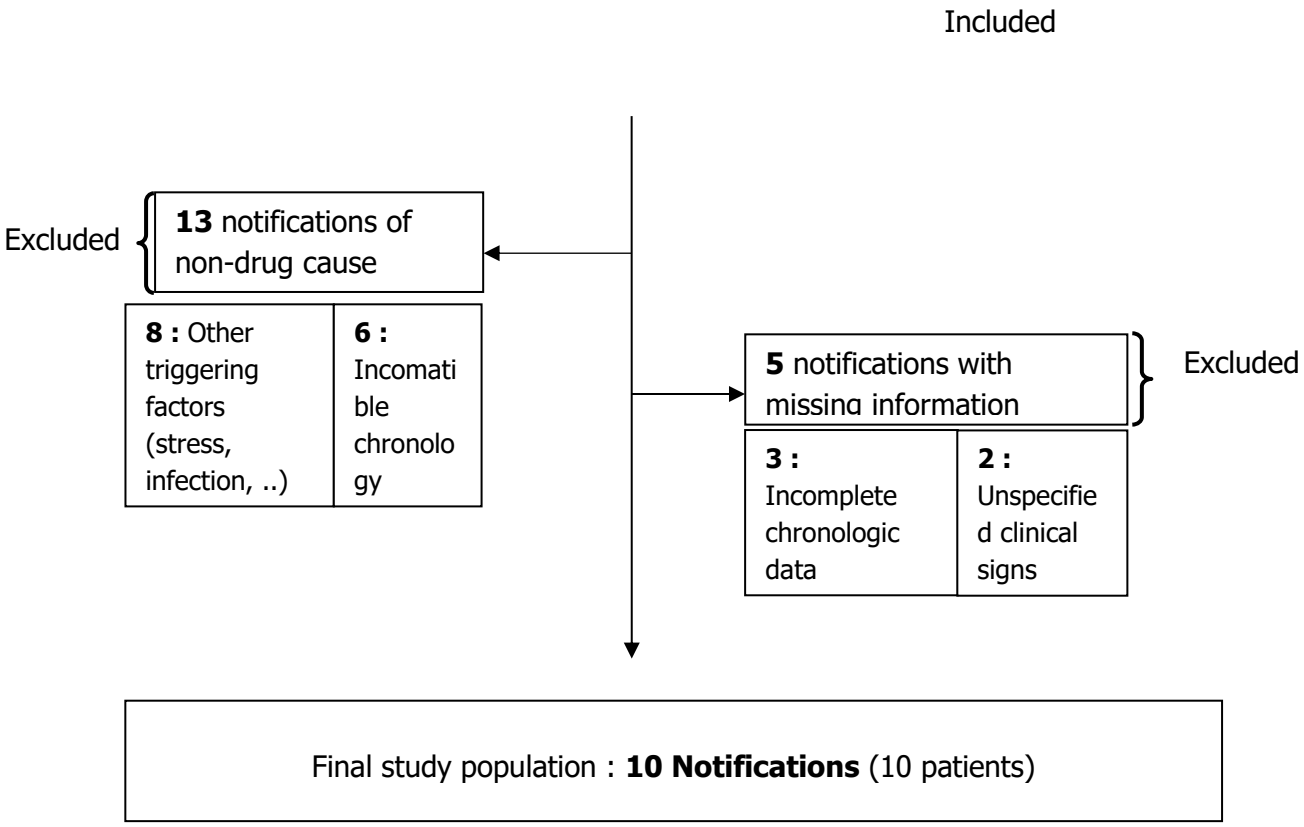

Figure 1 : Patient selection flowchart

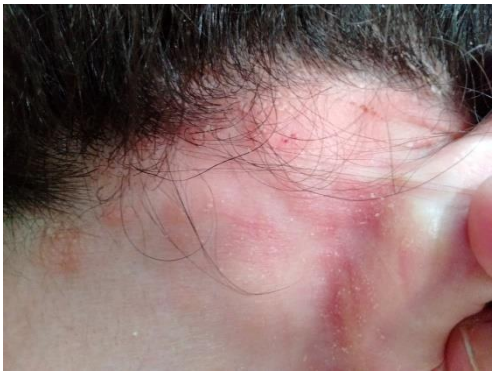

Figure 2 : Plaque psoriasis eruption on the forehead of patient 3

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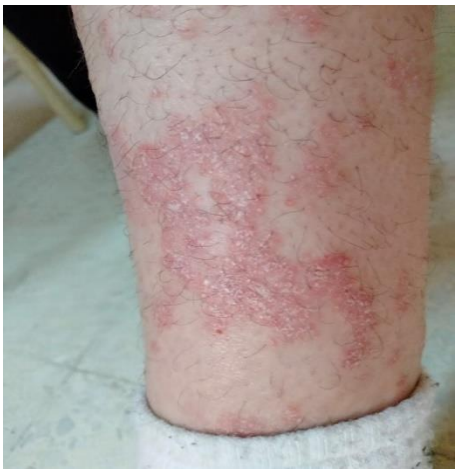

Figure 3 : Pustular psoriasis eruption on the right leg of patient 8

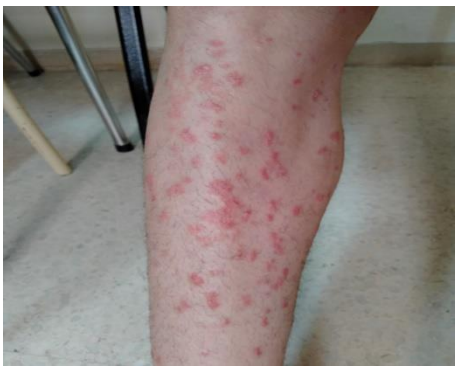

Figure 4 : Guettae psoriasis eruption on the right leg of patient 5