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Journal of Dermatology and Dermatitis

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Research Article

Paradoxical Psoriasis during IL-17 blockage: two memorable patients

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Received date: July 19, 2021; **Accepted date:** July 23, 2021; **Published date:** July 29, 2021

Citation: A M G Brunasso, C Massone. (2021) Paradoxical Psoriasis during IL-17 blockage: two memorable patients. *J. Dermatology and Dermatitis*. 6(2); Doi: 10.31579/2578-8949/080

Abstract:

47 year-old man who suffered for plaque psoriasis since 2013 was previously treated with topicals, UVB photherapy, acitretin, methotrexate and adalimumab (Imraldi®) with scarce response. Brodalumab (IL-17 receptor chain A blocking antibody) at 210 mg sc day 0, week-1, week-2 and every 2 weeks was initiated (baseline PASI of 16) with fast improvement of psoriasis (PASI-90 at week 4) and new onset of erythema, pustules and pain in the palmoplantar area after each subcutaneous Brodalumab administration and progressive improvement after 5-8 days.

Keywords: old man; uvb photherapy; corticosteroids; biopsy

Case 1

47 year-old man who suffered for plaque psoriasis since 2013 was previously treated with topicals, UVB photherapy, acitretin, methotrexate and adalimumab (Imraldi®) with scarce response. Brodalumab (IL-17 receptor chain A blocking antibody) at 210 mg sc day 0, week-1, week-2 and every 2 weeks was initiated (baseline PASI of 16) with fast improvement of psoriasis (PASI-90 at week 4) and new onset of erythema, pustules and pain in the palmoplantar area after each subcutaneous Brodalumab administration and progressive improvement after 5-8 days (Fig. 1a, 1b). Diagnosis of palmoplantar pustulosis (PPP) was made, blood laboratory diagnostics were in the normal range. The patient denied consensus for a skin biopsy. Due to the discomfort associated to the onset of PPP after each Brodalumab injection a switch to Guselkumab (IL-23 neutralizing antibody, 100 mg sc at week 0, week-4 and every 8 weeks) was decided, with complete improvement of both PPP and plaque psoriasis after 14 weeks.

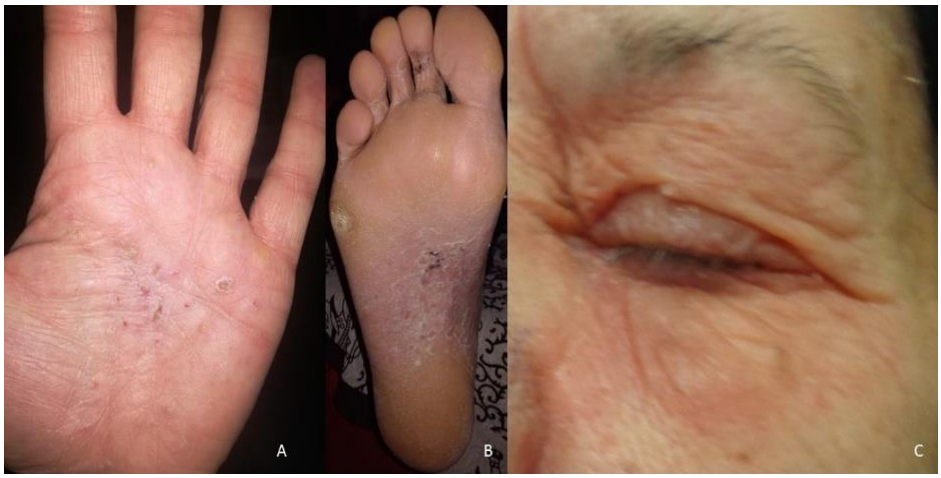

Figure 1A: *pustules and scaling with crusts on the left palmar region.*

Figure 1B: *keratotic plaque with crusting, erythema and some pustules on the right plantar region*

Figure 1C: *well defined erythematous, scaling plaque on periocular area*

Case 2

79-year-old female affected by plaque psoriasis since 1970, previously treated with topicals, UVB phototherapy, methotrexate with scarce response-initiated adalimumab (Humira®) 4 years before and withdrew therapy after 2 months because of the new-onset of Dermatomyositis (DM), prednisone 0.5 mg/kg per o.s was given for 2 years and during tapering psoriasis returned. The patient was reluctant to biological therapy and only after 2 years she accepted to initiate Secukinumab 300 mg sc at weeks, 0, 1, 2, 3 ,4 and every month thereafter for a diffuse plaque psoriasis (PASI: 19.2). Complete response with PASI-100 was observed at week-12, but she developed eye-lid lesions never seen before, denying pruritus, use of eye-drops or cosmetics and no other therapies were conducted. (Fig 1c) A skin biopsy was not performed because of dissent of the patient. Topical corticosteroids and thereafter pimecrolimus cream were required to control the eye-lid psoriasiform-eczema.

A paradoxical adverse event (PAE) is defined as the unexpected new onset or worsening of a condition during biologic therapy, when that particular condition otherwise responds well to the biologic agent involved [1]. New development of plaque or pustular psoriasis and psoriasis onset in anatomical sites never compromised before are the most common reported PAE [1,2]. Herein, we report two different psoriasis-PAE during anti-IL-17 therapy: PPP and eye-lid psoriasiform-eczema; interestingly in the second case a prior PAE was reported during adalimumab (DM) [3,4]. TNF-α blocking agents may aggravate/trigger the onset of DM as demonstrated in a systematic literature review, in contrast to the proposed action as potential steroid-sparing agents in DM and polymyositis [3,4]. Psoriasis-PAE appears in 2-5% of anti-TNF-α treated patients and is driven by an ongoing type-I IFN innate immune response in the absence of T cell driven inflammation in contrast to chronic plaque psoriasis [1,2-5]. It has been postulated that psoriasis-PAE are seen exclusively during TNF-α blockade, but few cases have been reported during anti- IL-17 therapies, even if the underlying mechanism still needs to be elucidated [6-8]. The low-frequency of presentation (less than 5%) indicates that other factors such as genetic predisposition or trigger factors influence the abrupt onset [1,5]. Noteworthy, epidermal trauma (Koebner phenomenon) which induces antimicrobial peptides (AMP)-expression by keratinocytes and attracts plasmacytoid dendritic cells (pDCs) into the skin, enhances the production of type-I IFN and the new onset of psoriasis [4]. We hypothesize that chronic trauma (Koebner phenomenon) in the palms and soles might trigger such condition as in our first patient [4]. The new onset of eye-lid psoriasiform-eczema in our second case, affecting an area usually spared by plaque psoriasis (involved only erythrodermic-psoriasis) has been previously reported in other three patients [8]. In both cases the rapid-onset of psoriasis-PAE suggests also a genetic predisposition (mean 2.5 weeks). We highlight that in our first case, anti-IL-23 therapy resulted in complete resolution of both the classical and paradoxical psoriasis.

Conflicts of interests: The authors have no conflict of interest.

The content of this paper has not been previously presented

We did not receive funding sources for this project

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DOI: 10.31579/2578-8949/080