



Figure 1. Generalized erythema with yellow pustules particularly on the border of the advancing eruption after the first injection of ustekinumab.

Ustekinumab has recently been reported to be an effective treatment in pustular psoriasis [4]. The paradoxical event of the induction of pustular psoriasis by anti-TNF treatment has raised questions about the possible mechanism of action. It was proposed that a triggering event in predisposed individuals may cause a possible disruption in a sensitive cytokine balance, following TNF α inhibition. That may result in up-regulation of plasmacytoid dendritic cells (PDCs) and the subsequent production of interferon- α . PDCs respond to viral and microbial infections by producing IFN α . Increased IFN α expression in the developing psoriatic lesions of subjects receiving anti-TNF therapy has also been reported [5].

Ustekinumab neutralizes the common p40 subunit of IL-12 and IL-23 of PDCs and thus it blocks the binding of undifferentiated T-cells, suspends the clonal proliferation of Th1 and Th17 cells and downregulates inflammatory cytokines such as IFN α and IFN γ , IL-17 and IL-22 [6]. A possible mechanism of ustekinumab-induced pustular psoriasis may be related to up-regulation of a cytokine-vicious circle in genetically predisposed patients. Further investigation might further illuminate the pathogenesis of such a paradoxical manifestation. ■

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Mesalazine-induced photosensitivity

Drugs are known to cause a variety of cutaneous eruptions, including photosensitivity [1]. We report a case of severe photosensitivity resulting in aggravation of existent eczema, due to mesalazine. A marked improvement was noted after subsequent withdrawal of the drug.

A 56-year-old man with lifelong atopic eczema, which was well controlled using topical therapy, presented with severe photo-aggravation of his eczema and immediate photosensitivity, whilst working on a new assignment in the Middle East (*figure 1*). He received topical emollients, corticosteroids and short courses of systemic steroids, however, the photosensitivity worsened to the degree that he required photo-protective body suits and gloves and his employer was obliged to install ultraviolet screens on his office windows and car. He had a long history of ulcerative colitis



Figure 1. Photo-exposed eczematous eruption secondary to mesalazine.

diagnosed five years prior to his presentation to the dermatology team, which had been quiescent for the last five years on maintenance therapy with mesalazine (Pentasa). Porphyrin and auto-antibody screens were both negative and there was no family history of any photosensitivity. His general practitioner sent him directly for photo investigation in a tertiary referral centre, which showed a low erythema threshold to UVA (1.4 J/cm^2 for both 330 and 350 nm). Provocation testing with both UVA and solar simulators produced a marked eczematous scaling and erythema on the second day of provocation (Custom built arm exposure containing UVA source and 1 KW xenon arc for the solar simulated radiation). An initial diagnosis of photo-aggravated eczema was made. However, the history revealed that he suffered from immediate symptoms of burning and photosensitivity occurring within 30 minutes following exposure to sunlight, with subsequent worsening of his eczema, only in photo-distributed sites. This occurred mainly in the Middle-East, where the sun exposure was much more significant and intense than in the UK. He recalled that these symptoms had occurred within months of starting mesalazine. He was not on any other medication. Drug-induced photosensitivity was suspected and mesalazine was stopped under the guidance of his gastroenterologist. Within ten days of discontinuation his photosensitivity completely disappeared and he was able to venture outside without gloves and facial sun block for the first time in many years. He made a remarkable recovery following discontinuation of mesalazine and his underlying eczema continues to be well controlled.

Drug-induced photosensitivity appears with a combination of a drug and UVA or UVB radiation, although UVA is more often implicated [2]. Photosensitive-drug reactions are divided into phototoxic and photo-allergic types, the latter having an immunological mechanism similar to allergic contact dermatitis. The majority of photosensitive drug reactions are phototoxic, based on a non-immunological mechanism, and are often dose and sun-exposure dependent [1-3]. Photosensitivity due to drugs may induce or aggravate existing dermatoses, as demonstrated here. Withdrawal of the offending drug often results in cessation of the symptoms, which was clearly demonstrated in our patient. Mesalazine (Pentasa) is an amino salicylate, widely used in inflammatory bowel disease. Only one publication in the literature has reported this phenomenon with the drug [4]. Salicylates however, have been associated with photosensitivity, particularly phototoxicity, and we speculate that mesalazine-induced photosensitivity may also be an idiosyncratic phototoxic reaction [5]. The delay in onset of several months after first exposure suggests that photo-allergic reactions may also be involved. Unfortunately we were unable to test this as the patient declined further photopatch tests to drugs as he did not wish to return to the level of discomfort he had recently recovered from. However, the marked rapid improvement following drug discontinuation in this case would favour phototoxicity as a mechanism. Clinicians should therefore be aware of this potential reaction and suspect drugs if patients present with photosensitivity or sudden-onset photo-aggravation of existing dermatoses. ■

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Clinical improvement of pityriasis rubra pilaris with antibiotic therapy

We report a case of pityriasis rubra pilaris treated successfully with antibiotic therapy. Pityriasis rubra pilaris (PRP) is a rare, chronic, papulosquamous disorder of unknown cause characterized by erythematous scaly plaques, palmoplantar keratoderma, and keratotic follicular papules. The disease can be intensely painful, disabling, and difficult to treat. There are several therapeutic approaches for patients with PRP. Currently, retinoid and anti-metabolite treatments are the most successful therapeutic approaches.

A 40-year-old man presented with large erythematous scaly patches and plaques on his trunk, knee, elbow, and the dorsum of his hand (*figure 1A*). He was diagnosed with PRP when he was 20 years old and had undergone various treatments, including systemic vitamin A, methotrexate, calcipotriol, and ultraviolet therapy. However, these therapies failed to result in clinical improvement, and he showed only slight improvement in his condition before treatment. A routine laboratory investigation showed that the complete blood count, blood chemistry, and urine analysis were all within normal ranges. Three months before he visited our department, he underwent an appendix operation and was treated with antibiotics (Benzathine benzyipenicillin 1.2 million unit t.i.d, Isepamicin sulfate 400 mg q.d, and cefaclor 750 mg t.i.d). After surgery, he found that his skin lesions were much improved. We prescribed the same antibiotics, and the patient showed clinical improvement (*figure 1B*), but after he achieved a significant improvement in his clinical status, he stopped taking antibiotics, thereby aggravating his skin lesions. He then resumed a course of antibiotics and experienced clinical improvement with antibiotic therapy.

Several agents are used in the treatment of PRP, including topical corticosteroids, emollients, retinoids,