Case Report:
Abatacept-Induced Psoriasis in a Rheumatoid Arthritis Patient

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Abstract

Objective: To report a case of drug-induced psoriasis in a girl with rheumatoid arthritis.

Case Report: A 12-year old, Saudi patient with rheumatoid arthritis on abatacept presented to the Dermatology Clinic of Aseer Central Hospital, Abha City, Saudi Arabia, complaining of itchy skin rash all over the body since 3 months. On examination, there were scaly well-defined plaque and patch, mainly involving the trunk and the scalp. The diagnosis for this case was “abatacept-induced psoriasis”. Abatacept was discontinued and the dose of methotrexate was increased in addition to prednisolone. Clobetasol propionate cream was administered for the skin lesion. Two months later, the psoriatic lesions resolved.

Conclusions: Abatacept-induced psoriasis is rare. Its exact mechanism is not yet clearly resolved. Management is by discontinuation of abatacept and the application of clobetasol propionate cream. Further research is still needed so as to achieve a better understanding of the mechanism of drug-induced psoriasis.


Introduction

RHEUMATOID arthritis is an autoimmune disease of unknown etiology, characterized by chronic and debilitating symmetrical polyarthritis, which results in joint damage and deformities [1].

Psoriasis is a chronic inflammatory immune-mediated skin disease. It is characterized by hyperproliferation and aberrant differentiation of keratinocytes [2,3]. Psoriatic arthritis is a chronic inflammatory disease affecting about 6-10% of patients with cutaneous psoriasis, with significantly higher prevalence in patients with extensive skin involvement [4].

The pathogenesis of rheumatoid arthritis is not clearly understood. The combination of rheumatoid arthritis and psoriasis is very rare. It is believed that there may be shared autoimmune or genetic factors [1].

Abatacept is a new agent, approved as a first-line treatment for rheumatoid arthritis [5]. It prevents the activation of T-cells by binding to the ligands CD80/CD86 on the surface of APCs, thus competing for them with CD28 expressed by T-cells. As an important indirect effect within the inflammatory cascade, the production of cytokines and autoantibodies is inhibited [6,7].

Only few cases of psoriasis during treatment with abatacept have been reported [8,9]. Therefore, we aim to report this case of drug-induced psoriasis in a 12-year old Saudi girl with rheumatoid arthritis.

Case Report

On March 2015, a 12-year old, Saudi girl presented to the Dermatology Clinic of Aseer Central Hospital, Abha City, Saudi Arabia, complaining of itchy skin rash all over the body since 3 months.

One year earlier, she was admitted to the Pediatrics Hospital with history of joint pain and swelling that started initially at ankle joint bilaterally, then knee joint, elbow, wrist and proximal interphalangeal joint, associated with morning stiffness, restricted movement and disrupted her daily activities. She proved to have rheumatoid arthritis and received methotrexate (12.5mg once/week), prednisolone, (5mg po od) folic acid (1mg po od), naproxen (250mg/day), and vitamin D3 (800 IU po od). However, due to the lack of sufficient response she was started on abatacept (125mg Sc weekly). Three months later, the patient developed this itchy skin rash all over the body including the scalp.
There was a positive family history of rheumatoid arthritis, but not psoriasis. On examination, there were scaly well-defined patch, mainly involving the scalp Fig. (1), psoriatic plaque with slivery scales involving the trunk, with sharp demarcation Fig. (2).

Laboratory investigations revealed elevated erythrocyte sedimentation rate (85mm/hour), leukocytic count of $4.6 \times 10^9/L$, positive rheumatoid factor and high C-reactive protein level.

A skin biopsy was taken for histopathological examination, which showed hyperplasia of epidermis with squared off rete ridges, elongation of dermal papillae, dilated superficial blood vessels, hypogranulosis and parakeratosis plus remnants of neutrophils. A collection of neutrophils was seen within stratum spinosum Fig. (3).

The diagnosis for this case was “abatacept-induced psoriasis”. Topical steroids were administered and abatacept was discontinued and the dose of methotrexate was increased to 15mg/weekly in addition to prednisolone (10mg od).

Clobetasol propionate (Dermovate) cream was administered for the skin lesion. After two weeks the patient showed mild skin improvement. The plan was to follow the patient till resolving of the rash. Two months later, the psoriatic lesions resolved completely.

Discussion

Psoriasis is a cutaneous disorder characterized by epidermal hyper-proliferation and inflammation. Pro-inflammatory cytokines, such as IL-17, TNF-α and IL-23, have been recently suggested to play a crucial role in the pathogenesis of psoriasis [10-11].

Our reported case of psoriasis was caused by abatacept which was administered for the treatment of rheumatoid arthritis. This is a rare complication of attributed to treatment by abatacept. Konsta et al., [7] reported that 17 of 3,277 rheumatoid arthritis patients (0.5%) who received abatacept developed psoriasis as an adverse event.

Few cases of psoriasis during treatment with abatacept have been reported [7,8,12]. In a meta-analysis of 13 relevant trials, four of 1332 rheumatoid arthritis patients who received abatacept as monotherapy as well as 13 of 1945 patients treated with abatacept plus disease-modifying anti-rheumatic drugs developed psoriasis as an adverse event [7].

For management of our case, abatacept was stopped, and as a substitute, methotrexate and prednisolone were increased, while dermovate...
cream was administered for treatment of psoriatic skin lesions, which were resolved within two months.

In an open multi-center trial, Svartholm et al., [13] reported that rapid clinical healing of the psoriasis infiltration in 75% of the patients with two applications of clobetasol propionate per week. They stated that this regimen could keep the disease in remission throughout a 4-month observation period.

Our case represents an onset of psoriatic lesions in the absence of family history or other triggering factors, three months after initiating abatacept for the treatment of rheumatoid arthritis. Though the underlying mechanism for this adverse event remains unclear, the possibility of blocking a specific biological activity might explain this pathological condition [12].

The precise mechanisms for this adverse event are still under discussion. Notably, Bouguermouh et al., [14] reported that B7 co-stimulatory molecules-mediated CD28 signaling in CD4+ naïve T cells downregulates the induction of Th17, which suggested the possibility that blocking CD28 increases the differentiation of Th17.

It has been suggested that the interference with CTLA-4 signals in regulatory T-cells might result in the impaired suppressive functions of those cells and in the exacerbation of Th17 immunity [8].

Cai et al., [15] noted that the role of T-cells in psoriasis pathophysiology is now well recognized. van Kuijk et al., [16] reported that activated T-cells are abundant in the inflamed joints of both psoriatic and rheumatoid arthritis, showing a similar profile of pro-inflammatory cytokine expression.

Tanita et al., [17] noted that the expression of nuclear pSTAT3 in epidermal keratinocytes was prominent, suggesting Th17 differentiation in the dermal papule and suggests one of the possible mechanisms of the psoriasiform drug eruption caused by abatacept.

In conclusion, the abatacept-induced psoriasis is rare. Its exact mechanism is not yet clearly resolved. Management is by discontinuation of abatacept and the application of dermovate cream. Further research is still needed so as to achieve a better understanding of the mechanism of drug-induced psoriasis.

References


