Cukurova Medical Journal
ÇUKUROVA ÜNİVERSİTESİ TIP FAKÜLTESİ

Cukurova Med J 2022;47(4):1768-1770
DOI: 10.17826/cumj.1170774

EDİTÖRE MEKTUP / LETTER TO THE EDİTOR

Adalimumab induced severe paradoxical psoriasis in a patient with ankylosing spondylitis

Ankilozan spondilit tanılı hastada adalimumabın tetiklediği paradoksal psoriasis

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Cukurova Medical Journal 2022;47(4):1768-1770

To the Editor,

Anti-tumor necrosis factor (Anti-TNF)'s have been used frequently in rheumatology and dermatology. These drugs may cause psoriasiform lesions paradoxically. In this report, ankylosing spondylitis patient who developed severe paradoxical psoriasis while being treated with adalimumab was discussed.

A 23-year-old male presented with erythematous scaly plaques and pustules on the trunk and extremities that had been present for 6 months. The patient had been treated with adalimumab for 1 year for ankylosing spondylitis. There were multiple small erythematous and scaly plaques and pustules on the front and back of the trunk, thick scaled, macerated plaques, erosions and pustules on the palms and soles(Figure1-2-3). Adalimumab was stopped. No triggering factors were detected. In the histopathological examination; confluent parakeratosis, local granular layer loss, irregular acanthosis, neutrophilic spongiosis, munro microabses and spongioform pustules, neutrophilic infiltrates in the epidermis, superficial mild perivascular lymphocytes and plasma cells were observed in the dermis(Figure 4-5-6). The patient was diagnosed with paradoxical psoriasis, methotrexate (15mg/week) treatment was started. Lesions significantly regressed in 1 month follow-up(Figure 7-8-9).

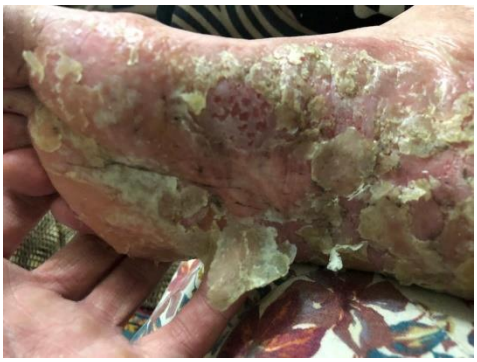

Figure 1. Erythematous macerated plaques and pustules on the sole of the foot.

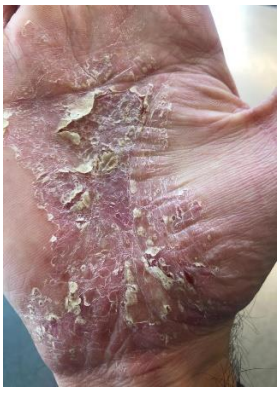

Figure 2. Erythematous and scaly plaque on the palm.

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Geliş tarihi/Received: 05.09.2022 Kabul tarihi/Accepted: 01.11.2022

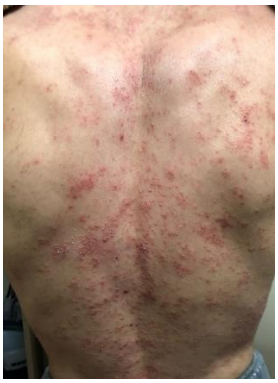

Figure 3. Severe pustular eruption on an erythematous area on the back of the trunk

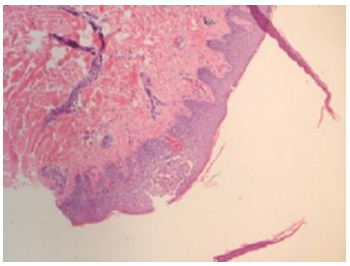

Figure 4. Parakeratosis, acanthosis, tortuous and dilated blood vessels, and the spongiform pustule of Kogoj (Haematoxylin and eosin x100).

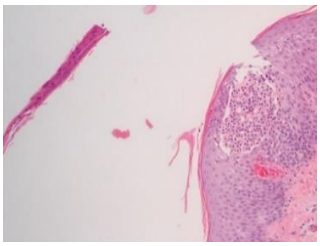

Figure 5. Neutrophils in the epidermis, Kogoj pustule, spongiosis, parakeratosis and granular layer loss (Haematoxylin and eosin x200).

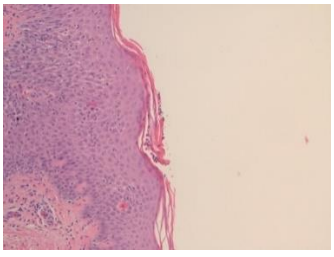

Figure 6. Neutrophils in the epidermis, Munroe abscess, spongiosis, parakeratosis and granular layer loss (Haematoxylin and eosin x200).

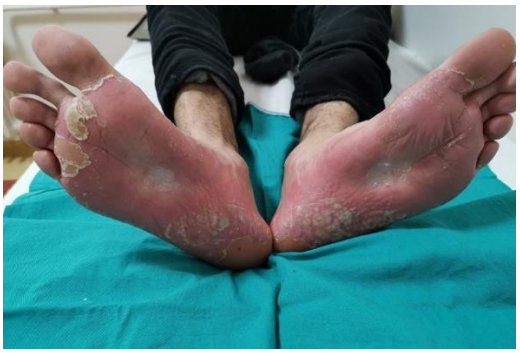

Figure 7. Erythema, desquamation and pustules on the sole of the foot (1 month after the termination of adalimumab).

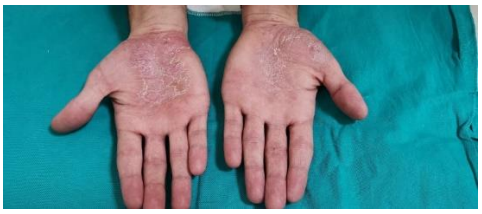

Figure 8. Erythematous desquamation of the palms (1 month after the termination of adalimumab).

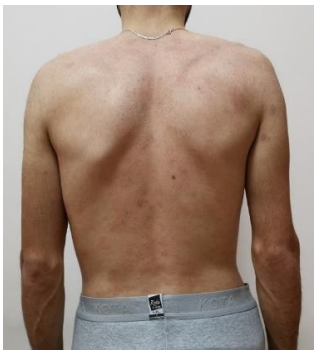

Figure 9. Superficial desquamation on the back of the trunk (1 month after the termination of adalimumab).

The mechanism of paradoxical psoriasis can be summarized as blockade of TNF-alpha. Because of this blockade, innate immune response activation exaggerates and type I interferon (IFN) increases. Dentritic cells which activated in paradoxical psoriasis secrete IFN. IFN stimulates the secretion of TNF-alpha and IL-23. TNF-alpha suppresses the release of IFN with negative feedback[1,2]. Blockade of TNF increases the production of type-I IFN. Paradoxical psoriasis is an expression of an ongoing

type I IFN-mediated innate immunity independently of the T cell, but the relationship between the increase in the amount of IFN and keratinocyte hyperproliferation has not been clearly understood[3]. Factors that induce paradoxical psoriasis remain unclear. Due to concurrent bacterial superinfections have been identified in a significant number of patients, infections can induce the development of paradoxical psoriasis[4]. We could not show any trigger factors, including infections.

Histopathological appearance of the disease mostly overlaps with early psoriasis lesions. Severe dermal innate immune system cell infiltration (dentritic cell, neutrophil, eosinophil, mast cell, macrophages) and increase of mediators managing the innate immune response are observed[1,5,6].

Treatment recommendations are based on case series experience. In the mild disease topical corticosteroids and phototherapy-acitretin combination are appropriate, anti-TNF therapy does not need to be discontinued in the first step. In more severe patients, it is recommended to discontinue anti-TNF therapy and switch to another class of biological agent. Dapsone can be considered in pustular form. Considering the patient's comorbidities, methotrexate or cyclosporine may be added to the treatment. There are also examples of cases which treated with ustekinumab in the literatüre, but it should be kept in mind that ustekinumab treatment may paradoxically cause psoriasis[7-10].

Anti-TNF drugs which used in the treatment of psoriasis can paradoxically cause psoriasiform lesions. Paradoxical psoriasis usually progresses mildly, severe forms are observed less frequently as in our case. In this report, we discuss a case of severe paradoxical psoriasis secondary to adalimumab treatment.

Yazar Katkıları: Çalışma konsepti/Tasarımı: EZ; Veri toplama: EZ, GSK; Veri analizi ve yorumlama: EZ, PK; Yazı taslağı: EZ; İçeriğin eleştirel incelenmesi: EZ, GSK; Son onay ve sorumluluk: EZ, GSK, PK; Teknik ve malzeme desteği: EZ; Süpervizyon: EZ, GSK; Fon sağlama (mevcut ise): yok.
Etik Onay: Bu çalışma için yerel veya ulusal yönergelere uygun olarak etik onay gerekli değildir. Tıbbi vakalarının ayrıntılarının ve beraberindeki görüntülerin yayınlanması için hastadan yazılı bilgilendirilmiş onam alındı.
Hakem Değerlendirmesi: Editoryal değerlendirme.
Çıkar Çatışması: Yazarlar, bu makalenin yazarlığı ve / veya yayınlanmasıyla ilgili çıkar çatışması ilan etmemişlerdir.
Finansal Destek: The authors received no financial support for the research and/or authorship of this article.
Author Contributions: Concept/Design : EZ; Data acquisition: EZ, GSK; Data analysis and interpretation: EZ, PK; Drafting manuscript: EZ; Critical revision of manuscript: EZ, GSK; Final approval and accountability: EZ, GSK, PK; Technical or material support: EZ; Supervision: EZ, GSK; Securing funding (if available): n/a.
Ethical Approval: Ethical approval is not required for this study in accordance with local or national guidelines. Written informed consent was obtained from the patient for publication of the details of their medical case and any accompanying images.
Peer-review: Editorial review.
Conflict of Interest: The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.
Financial Disclosure: Yazarlar, bu makalenin araştırması ve / veya yazarlığı için herhangi bir mali destek almamışlardır.

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