Case Report

Deflazacort-induced Acneiform Eruptions

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A 38-year-old male suffering from a low backache since 3 months was diagnosed as a case of L4-L5 disc prolapse after magnetic resonance imaging examination. He was treated with tolperisone, aceclofenac, and paracetamol in these drugs deflazacort added later. From the 2nd day of an addition of deflazacort in the therapy, sharply marginated, infiltrative, and erythematous skin eruptions with discrete itching sensations were seen. It was diagnosed as deflazacort-induced acneiform eruption and treated with doxycycline for 2 months which led to the disappearance of acneiform eruptions.

**Keywords:** Acneiform eruptions, deflazacort, doxycycline

**Abstract**

INTRODUCTION

Acne is a dermatological disease of pilosebaceous follicle. Depending on etiological factors, it may be of hormone dependent acne, drug-induced acne, or mechanical acne. Drug-induced acne is the occurrence of acne-like eruptions arising after drug intake. Specific features associated with drug-induced acne include a monomorphic pattern, lesion location away from seborrheic area, and history of recent drug intake. A number of drugs are either directly involved or are associated with drug-induced eruptions including corticosteroids, neurological, and immunomodulating drugs.[1] We present and discuss a case of deflazacort, a derivative of prednisolone, induced acneiform eruptions.

CASE REPORT

A 38-year-old male of weight 60 kg was suffering from a low backache for 3 months. He used topical diclofenac sodium gel intermittently to get relief from a low backache. However, during the last 15 days, the intensity of pain increased hence he was prescribed tablet aceclofenac 100 mg with paracetamol 325 mg twice daily and pantoprazole 40 mg once daily for 5 days and advised bed rest. When he reported after 1 week, the intensity of pain was further increased as he avoided bed rest and was involved in heavy physical work. Due to the severity of pain, he was not able to move his entire right leg and even slight movement was responsible for severe pain. On examination, right lower limb radiculopathy was observed with loss of power in the right toe. On magnetic resonance imaging (MRI) examination diffuse posterior protrusion with large postero-central extrusion with an inferior migration of L4-L5 disc was causing severe compression of the thecal sac, bilateral recess was observed. Focal postero-central protrusion of L5-S1 disc compressing thecal sac, bilateral budding nerve root, and causing mild canal narrowing was observed. After MRI examination, L4-L5 disc prolapse was diagnosed. Relief from pain was not seen.

He was prescribed tablet deflazacort 36 mg twice daily for 5 days and tablet tolperisone 450 mg once daily for 5 days in addition to tablet aceclofenac (100 mg) and paracetamol (325 mg) twice daily for 5 days, tablet pantoprazole 40 mg once daily for 5 days.

The patient showed improvement in the painful condition due to the addition of deflazacort but from the 2nd day of the addition of deflazacort in the therapy, sharply marginated, infiltrative, erythematous skin eruptions with discrete itching sensations was seen. Comedones were absent. These eruptions distributed over the neck, chest, upper abdomen, back, and both the arms but not involved face [Figures 1-3].

Within 5 days of deflazacort therapy, the patient showed dramatic improvement with the treatment, intensity of pain reduced, the movement started, and power in the right toe. On

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The dose of deflazacort was tapered and withdrawn over another 15 days. The 30 days course of drug therapy involving various drugs such as 20 days deflazacort, 10 days tolperisone, and 30 days aceclofenac, paracetamol, and pantoprazole was responsible for recovery of the patient from the painful situation.

During the entire 20 days course of deflazacort therapy number and size of acneiform eruption increased. No effect of tolperisone withdrawal was observed in the pattern and intensity of acneiform eruptions. The therapy with aceclofenac, paracetamol, and pantoprazole was not shown any positive or negative effect on pattern and progress of acneiform eruptions. However, when the deflazacort was withdrawn, there was no further increase in number and size of eruption seen. Based on the relation between duration of deflazacort therapy and eruptions, diagnosis of deflazacort-induced acneiform eruption was made.

After the withdrawal of deflazacort drug, tablet doxycycline 100 mg twice daily for 1 month was prescribed for the management of acneiform eruptions. Adapalene 0.1% gel and benzoyl peroxide 5% gel to apply over the eruptions in the night for 1 month was also advised. During 1 month of therapy with doxycycline and adapalene and benzoyl peroxide, lesions were partly controlled. The same treatment for acne control was advised for another 1 month which led to the disappearance of acneiform eruptions.

**Discussion**

In this case, eruption was diagnosed as drug-induced (deflazacort induced) acneiform eruptions. Acneiform eruptions were visible from the 2nd day of deflazacort therapy. Progression of acneiform eruptions was stopped after the completion of 20 days deflazacort course indicating the strong role of deflazacort behind the eruptions.

The role of diclofenac was eliminated as the causative agent for acneiform eruption as it was withdrawn much before the onset of eruptions. Tolperisone was also not suspected as causative agents since its withdrawal did not affect the course and pattern of acneiform eruptions. Aceclofenac and pantoprazole were also ruled out as factors behind acneiform eruptions because these drugs were started 5 days earlier and no eruptions were found, moreover, drug was continued for 10 days more after progression of acneiform eruptions was stopped. Aceclofenac and pantoprazole did not show any change in the behavior of the acneiform eruptions.

The development of monomorphic follicular inflammatory papulopustules with an absence of comedones often presenting acutely, distributed on the site not commonly affected by acne is typical of acneiform drug eruptions. Acneiform eruptions can be induced by many drugs. Among the several drugs which are associated with acneiform eruptions, corticosteroids constitute a
Steroid-induced acne may be characterized by monomorphic inflammatory papules and pustules. Glucocorticoids increase toll-like receptor 2 expressions in human keratinocytes which are stimulated by Propionibacterium acnes or proinflammatory cytokines and leads to acne.

P. acnes secretes many enzymes such as proteases, hyaluronidases, and neumaminidases which are involved in epithelium permeabilization and inflammatory infiltration. It also produces chemotactic factors and proinflammatory cytokine leading to the appearance of the clinical picture. In the acute inflammatory lesions, polymorphonuclear cells are seen while in chronic cases mononuclear cells, mainly T-cells of the CD4 phenotype are involved.

Treatment of steroid-induced acne involves withdrawal or decrease in dose of the steroid, sensitive antimicrobial agent and benzoyl peroxide. Doxycycline was used to control the bacteria involved in the deflazacort-induced acneform eruption because among the antimicrobial agents, tetracyclines mainly doxycycline and minocycline are most commonly used drugs for the management of acne due to their better efficacy and low level of bacterial resistance. Since adapalene and benzoyl peroxide are most effective topical agents for the management of acne, we used both the drugs. Adapalene modulate keratinization and inflammatory process. Benzoyl peroxide markedly reduces P. acnes by a direct toxic effect on the bacteria. It also has anti-inflammatory property.

**Conclusion**

Deflazacort was considered as safer molecule however it is not free from inducing drug-induced acneform eruptions. While prescribing deflazacort previous drug history including drug eruptions should be taken.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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**Conflicts of interest**

There are no conflicts of interest.

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