

Case Report

Penicillin induced toxic epidermal necrolysis with secondary impetiginization: a rare case

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ABSTRACT

Drug induced allergic reactions can be categorized into IgE-mediated and non-IgE mediated hypersensitivity reactions. Symptoms of IgE-mediated reactions are angioedema, bronchospasm, anaphylaxis, and urticaria that appears within 72 hours and those which are Non-IgE mediated hypersensitivity reactions include morbilliform eruptions, interstitial nephritis, hemolytic anemia, serum sickness, thrombocytopenia, and erythema multiforme, after 72 hours. TEN is defined as an extensive detachment of full-thickness epidermis most often related to an adverse drug reaction. We report a rare case of penicillin induced toxic epidermal necrolysis with Secondary Impetiginization in a 38-year-old male patient with complaints of rashes all over the body, chest pain and dry tongue since seven days. Based on history and clinical examination patient was diagnosed as of penicillin induced toxic epidermal necrolysis with secondary impetiginization and was successfully treated with antihistamines, parenteral antibiotics and corticosteroids.

Keywords: Hypersensitivity reactions, Toxic epidermal necrolysis, Penicillin

INTRODUCTION

Toxic Epidermal Necrolysis (TEN) is rare, but potentially life-threatening disorder. In 1956, Alan Lyell described an eruption resembling scalding of the skin, which is called toxic epidermal necrolysis.¹ Later it was found that most TEN are drug induced and the most common drugs are antibiotics, anti-convulsants, Non-Steroidal Anti-Inflammatory Drugs (NSAIDs), Allopurinol, nevirapine, pyrazolones and barbiturates.² The difference between SJS and TEN is based on the type of lesions and by their extent of skin detachment. Skin detachment from the body is between 3% and 10% in SJS; 11-30% in case of SJS/TEN overlap, and over 15-30% in TEN.³ We report a case of Toxic Epidermal Necrolysis (TEN) due to Penicillin involving 70% of the body surface area, including nasal mucosa, conjunctiva and genital involvement following an IgE mediated skin hypersensitivity to an extended spectrum Penicillin.

CASE REPORT

A 38-year-old male with a history of hypertension and coronary heart disease presented to the emergency department of our hospital with a complaint of rashes all over the body, chest pain and dry tongue since seven days. He was actually alright seven days back when he was administered with injection penicillin by a general practitioner for leg wounds. Within one-week rashes appeared all over the body that initially appeared on the hands and spread to the neck, chest, back, abdomen, upper and lower extremities, within 24 h. On examination, he was disoriented; the rashes were typically edematous and purpuric in nature spreading all over the body. There was involvement in mucosal areas in the form of erythema of the conjunctiva, nasal mucosa, buccal mucosa, and genitalia. Peeling of the skin seen on the back, trunk, face, genitalia, axilla, groin and both upper and lower limbs with moist erosions. Systemic

examination did not reveal any other significant findings. Considering the history, clinical examination, and laboratory findings, the patient was diagnosed as a case of penicillin induced TEN with secondary impetiginization. The patient was treated with parenteral antibiotics, steroids, antihistamines, emollients and nutritional supplements. On causality assessment using Naranjo's causality algorithm, the association was probable.



Figure 1: Toxic epidermal necrolysis affecting the back with peeling of the skin.

DISCUSSION

Toxic Epidermal Necrolysis (TEN) is also called as Lyell syndrome.¹ It is a rapid, extensively spread resembling scalding. Drugs are the most common cause accounting for about 65%-80% of the cases.² Toxic epidermal necrolysis incidence is increased in patients with systemic lupus erythematosus, HIV, brain tumors, use of steroids, graft versus host disease, radiotherapy, bacteria, viruses, fungi, immunization, and idiopathic.³ Although the pathogenesis of TEN is incompletely understood, it has long been suspected to result from drug induced hypersensitive reaction causing keratinocyte cell death result in sloughing of the epidermis attracting inflammatory cells at the affected areas predominately CD8+ cytotoxic T-lymphocytes.⁵ In early lesions, keratinocyte death occurs through apoptosis,⁶ a programmed cell death and disassembly characterized by DNA fragmentation, nuclear condensation, and membrane blebbing. In advanced lesions, necrolysis are noticeable due to detachment of epidermis from the underlying dermis. The onset of symptoms following initial drug administration often ranges from a few hours to three weeks, but rapid reappearance of symptoms after re-challenge within 48 hours or less is observed in an acquired immune response.⁷ Once the diagnosis is made; quick attempting withdrawal of the causing drug should be made thus decreasing mortality. Therapeutic management is by parenteral administration of glucocorticoids, cyclosporine-A, N-acetylcysteine, pentoxifylline, cyclophosphamide, Thalidomide, and

anti-TNF-alpha antibodies for TEN,⁸ though none has conclusively been shown to be beneficial.

CONCLUSION

Though Toxic epidermal necrolysis is rare, proper history should be taken before prescribing drugs like antibiotics, allopurinol, NSAIDs and pyrazoles. Pharmacovigilance should be a part of patient care in order to reduce the occurrence of adverse effects and also encourage practitioners in reporting to gather more regarding adverse drug reactions.

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