



TOXIC EPIDERMAL NECROLYSIS DUE TO ACECLOFENAC

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ABSTRACT

Toxic epidermal Necrolysis (TEN) are serious disorders commonly termed as idiosyncratic reactions to drug, the most common being antiepileptic(phenytoin, barbiturates, carbamazepine and lamotrigine), sulfonamides, trimethoprim, ampicillin, allopurinol and NSAIDS (especially phenylbutazone and oxicam derivatives). Here we report a case of TEN in a patient who developed the lesion after oral administration of ACECLOFENAC. These drugs have rarely been implicated in this disorder. The suspected drug in this case was aceclofenac. The patient was managed with antibiotics, corticosteroids and parenteral fluids and recovered well.

KEY WORDS: Adverse drug reaction, Toxic Epidermal Necrolysis, Aceclofenac



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INTRODUCTION

Toxic epidermal necrolysis (TEN) is a rare life threatening idiosyncratic mucocutaneous reaction characterized by widespread epidermal necrosis followed by epidermal detachment. Drugs, infection and immunization are the most common causes of TEN. Drug induced TEN is the commonest cause in which antipsychotics, antibiotics, nonsteroidal anti inflammatory drugs(NSAIDS) and allopurinol are the most commonly implicated agents. We report a case of TEN which was induced by aceclofenac, that has been rarely implicated in the causation of TEN.¹

CASE REPORT

A young girl aged 23years was referred to the casualty department with extensive epidermal detachment all over the body and lesions around the axilla, inner thigh, oral and ocular region of after suspected drug intake of aceclofenac leading to the provisional diagnosis

of toxic epidermal necrolysis (TEN). A day prior to the admission, she had presented with complains of fever and generalized body pain for which Tab Aceclofenac was taken twice daily. One was taken in the morning, following evening she developed intense itching all over the body along with severe congestion of eye. After 8-12hrs she developed high grade fever, malaise, sore throat and erythematous maculopapular rash all over the body, face along with intense discharge from eye. She was immediately referred to a tertiary care hospital. She was conscious and febrile with temperature 101°C, heart rate was 102 bpm and blood pressure was 110/70mmHg. Cutaneous examination showed involvement of about 80% of total body surface area, with skin necrosis, tenderness and a positive nikolskys sign. There was ulceration and crusting on axillary and facial region. She also developed dysphagia owing to severe oral ulceration.

Table 1
Common blood investigations

Investigations	Values on admission
Hb	13.2 mg/dl
Platelets	1.06lac
Neutrophils	86
Lymphocytes	11
Eosinophils	03
ESR	10
RBC	4.57
Urine	Colour: pale yellow Pus/RBC/cast: nil
ECG	Within normal limits
Sugar	141mg/dl
Blood urea	15.8mg
Creatinine	0.79
Total bilirubin	0.69mg
HIV, HbsAg	Non reactive
Bleeding time	2 minutes
Clotting time	5 minutes

She also complained of intense itching all over the body, and was started on intravenous fluids, injection hydrocortisone 100mg BD, injection Clarithromycin 500mg IV BD, injection metrogyl 400mg TDS. Wound care was given by applying topical antiseptics on eroded areas along with nonadherent dressings and Chlorhexidine for mouth wash. Following few days of continuous therapy with injection hydrocortisone 100mg, injection pheniramine, injection ranitidine was given with

calamine lotion for topical application. Artificial eye drops, along with ciprofloxacin eye ointment HS was given. The condition of the patient improved. Parenteral therapy was later replaced by oral drugs. The oral lesion was managed by chlorhexidine mouth wash, white petroleum jelly for the lips, syrup mucaine gel for gastric irritation. The patient showed steady improvement with the therapy given and was discharged, after 10 days, without any sequelae.

Toxic epidermal necrolysis following Aceclofenac administration.



Figure 1

Figure2

DISCUSSION

This is most likely case of drug –induced TEN and it was an acute presentation, which occurred after the drug was administered, Systemic Lupus Erythematosus(SLE) and rheumatological disorder were ruled out. Pemphigus was ruled out, as the result of the

Tzanck smear were negative. TEN was diagnosed from the history and typical clinical features and biopsy was not needed. The prodrome was shorter (6-12hrs) than the usual 1-3 days. Rechallenge was not done due to the severity of reaction². The mortality rate varies

from 20% to 45%. Death is due to septicemia or worsening of the attack. Healing of the skin would normally leave little scarring though hypertrophic scars have been reported⁴. Stevens–Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are severe cutaneous adverse reactions (SCARs) to drugs, characterized by extensive detachment of epidermis and erosions of mucous membranes⁵. There is a growing evidence that SJS and TEN are a single disease with common causes and mechanisms (Auquier-Dunant et al., 2002). The principal difference is the extent of skin detachment is limited in SJS and more widespread in TEN⁶. Even though rare (two cases/million population/year), SJS and TEN have a significant impact on public health because of high mortality (20–25%), frequent lasting disability, and reluctance of survivors and their physicians to subsequent use of medications. In 1995, a first case–control study (SCAR-study) assessed the risks

of SJS and TEN related to medications⁷. High relative risks (RRs) were observed for anti-infective sulfonamides (especially cotrimoxazole), carbamazepine, phenytoin, phenobarbital, non-steroidal anti-inflammatory drugs (NSAIDs) of the oxicam type, allopurinol, chlormezanone, aminopenicillins, cephalosporins, quinolones, and cycline antibiotics. These results contributed to several decisions of regulatory agencies, for example, withdrawal of chlormezanone from the market, restricted indications for cotrimoxazole and Phenobarbital.

CONCLUSION

TEN in this patient was probably caused by tablet Aceclofenac; hence, it is further emphasized on the need of test drug administration even for common drugs.

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