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Diclofenac Induced Toxic Epidermal Necrolysis: A Case Report

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Abstract

Toxic epidermal necrosis (TEN) is a potentially fatal cutaneous condition that is typically brought on by medication. One of the most frequent classes of medications linked to drug-induced TEN are non-steroidal anti-inflammatory medicines (NSAIDS)(1,2). Diclofenac is a recently developed NSAID that preferentially inhibits cyclooxygenase-2. It is frequently employed in the treatment of musculoskeletal conditions. In this report, a 80-year-old woman who received an IM diclofenac injection for bodily symptoms is described as having TEN caused by the drug.

1. Introduction

Non-steroidal anti-inflammatory medicines (NSAIDS) are one of the classes of medications that are frequently used in healthcare management because they are so effective at treating pain and inflammation. Diclofenac, a relatively novel NSAID, has potent anti-inflammatory properties comparable to aceclofenac and preferentially inhibits cyclooxygenase-2[(3)]. An oral non-steroidal anti-inflammatory medicine called diclofenac is frequently recommended to treat pain and inflammation in rheumatologic illnesses. Diclofenac primary mechanism of action is the suppression of prostaglandin production. The enzyme cyclooxygenase, which is necessary for the generation of prostaglandins, is severely inhibited by diclofenac.Diclofenac is generally well tolerated, with most side effects being small, treatable, and primarily GI-related. Vertigo, tremor, paraesthesia, and vertigo are among additional negative effects. Here, we provide a case of the clinically novel, previously unreported diclofenac induced [(4)] TEN in the Indian population.

Here we are reporting a case of 80 year old female with TEN

2. Case Report

An 80-year-old female patient was seen by a general practitioner with the main complaints of a body rash, skin peeling on both upper and lower limbs, the trunk, and the back. The woman mentioned receiving a diclofenac[(5)] injection from a nearby doctor because she was experiencing body aches. She also had a rash that started on her hands' fingers and then expanded to her trunk and both upper limbs. The rash extended over her face, including the oral mucosa, the area around both of her eyes, both lower limbs, and eventually the entire torso. The rashes on her face, including the areas around both eyes, upper limbs , and lower limbs, torso, and both backs , began to peel the following day. There was no prior history of burning micturition, diarrhoea, hemorrhagic urination, photophobia, or burn injuries. The patient had no prior history of comparable problems. She has been taking levothyroxine 25 mcg once daily for her

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hypothyroidism for 12 years, and she has been taking amlodipine 5 mg once daily for her hypertension for 10 years. Six months prior, the patient had received the second Covishield treatment against Covid-19. Back then, she didn't have any hypersensitive reactions.

A general examination found no pallor, icterus, cyanosis, clubbing, or lymphadenopathy, as well as normal blood pressure, temperature, and pulse and respiration rates. During the systemic assessment, no noteworthy findings were discovered. The results of the local/dermatological testing were as follows:

• Oral aphthae; • Lips showing superficial erosions with crusting; • Crusting on bilateral perioral area; • Bilateral conjunctivae showed erythema; • Presence of erosions on vaginal mucosa; • Scalp was red; • Presence of generalised purpuric macules on face, inside mouth, on both upper limbs, the entire trunk, and on both lower limbs Haemoglobin was found to be 8 g/dl during a complete blood count, but all other values were within normal ranges. Both serum urea and serum creatinine levels rose. 50 mg/dl of serum urea and 1.5 mg/dl of serum creatinine.

SCORTEN resulted in a score of 3.

• Blood urea > 28 mg/dl, > 30% epidermal detachment, and > 40 years of age

Based on the narrative and clinical examination, TEN[6] owing to diclofenac ingestion was diagnosed.

Intravenous fluids, multivitamins, corticosteroids, and oral antifungals were administered to the patient. On the lesions, liquid paraffin was administered topically. For five days, the lesions received wound care. On the lips, regular saline soaks were utilised. To stop the lesions from becoming infected again, antibiotic prophylaxis was administered.

The patient spent seven days in the hospital.

According to the WHO-UMC causality assessment[7], it was "likely";

• Positive De-challenge test since the skin lesions stopped spreading after the medication was withdrawn; • Positive temporal correlation between drug consumption and the formation of skin lesions; • No alternative pathophysiological explanation for the skin lesions;

3. Discussion

TEN is an uncommon, severe, acute, and sometimes fatal cutaneous condition characterised by widespread erythema, necrosis blistering, and mucous membrane and epidermal separation. Over 80% of TEN cases have a medication-related cause.[8]

Uncertainties exist regarding the pathophysiology of TEN at its core. It is thought to be cytotoxic T cell mediated drug hypersensitivity that is human leukocyte antigen dependent. At least 200 medications have been linked to SJS/TEN, according to reports. A drug or drug metabolite may act as a hapten or pro-hapten, may directly interact with the HLA protein, may change the specificity of HLA, or may bind to the T-cell receptor directly, which can ultimately result in T-cell activation. Cytotoxic CD-8+ cells that have been primed destroy autologous targets after activation.In addition to the substantial link between SJS/TEN and specific HLA alleles, genetic diversity in drug metabolising enzymes and rug transporters, which is crucial for the pharmacokinetics of medicines, also appears to be involved in the pathophysiology of SJS/TEN. Malignancies, vaccinations, and viral or bacterial infections can all interact intricately with the immune system and raise the risk of SJS/TEN[9].

Septicaemia is the most typical acute consequence.

8 In the long run, SJS/TEN may have an impact on numerous internal organ systems, including the pulmonary, gastrointestinal/hepatic, oral, otorhinolaryngologic, gynecologic, genitourinary, and renal systems, in addition to the eyes and skin. [10]

Ten can affect people of any age, although TEN caused by NSAIDs has a higher mean age than SJS (57.7 3.6 vs. 4.8 2.6 years). Female sex tends to be more susceptible to TEN in general. Proprionic acid derivatives, acetaminophen, acetic acid derivatives, and salicylic acid are said to be the most often used NSAIDs in SCARs. For TEN caused by NSAIDs, the reported mean time to symptom onset is 3.8

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days. When the TEN symptoms first appeared in a previous case report using aceclofenac

4. Conclusion

The severe, potentially fatal mucocutaneous adverse drug reactions Stevens-Johnson syndrome and toxic epidermal necrolysis have a high morbidity and fatality rate. Early diagnosis of the disorder, stopping any suspicious drugs, supportive therapy, starting a particular therapy, managing complications, and preventing further episodes are all critical components of management.

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Conflict of Interest: The authors declare that was no conflict of interest

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