

DRUG INDUCED HYPERSENSITIVITY- A CASE STUDY**S. Sindhu, S. Vedha Pal Jeyamani*, V. Chellapriya, V. Keethana and S. P. V. S. S.****Anuroopa**

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ABSTRACT

Hypersensitivity reactions against nonsteroidal anti-inflammatory drugs (NSAIDs) like diclofenac can elicit noticeable allergic reactions including systemic anaphylaxis and dermatological lesions. We present a case on diclofenac induced hypersensitivity erythematous Oedematous plaque and urticarial weal positive. No allergic history and no other drug intake were reported by the patient. The goal of treatment is to reduce the Severity of the allergic reaction and to minimize the further complication. Patients and physicians must be aware of the risk of cutaneous sensitisation induced by diclofenac, a drug which is extensively used as self-medication. Discontinuation is

the best therapeutic approach and the patients should be counselled about avoidance of OTC medications. The pharmacist, along with the prescriber has a duty to ensure that patients are aware of the risk of side effects. With their detailed knowledge of medicine, pharmacists have the ability to relate unexpected symptoms experienced by patients to possible adverse effects of their drug therapy.

KEYWORDS: Hypersensitivity, Diclofenac, Rashes, Case report, non-steroidal anti-inflammatory drugs (NSAIDs).

INTRODUCTION

Diclofenac is one of the most popular drugs world-wide belonging to the family of nonsteroidal antiinflammatory drugs (NSAIDs). Adverse drug reactions (ADRs), which occur in about every fifth patient, are well-known side effects mainly caused by the NSAIDs' acidic properties.^[1] Hypersensitivity to NSAIDs can be classified according to the time of onset and

clinical manifestations into acute and delayed categories. Acute reactions start immediately to several hours after drug administration and include.

(1) Respiratory reactions: Observed in patients with Aspirin Exacerbated Respiratory Disease (AERD), also called aspirin triad, Samter's disease, or aspirin intolerant asthma. These individuals experience a chronic disease characterized by chronic rhinosinusitis, severe persistent and steroid-dependent asthma, with or without nasal polyposis. Acute asthma exacerbations occur when they receive aspirin or classic NSAIDs. These asthma attacks are severe and may be life-threatening. Various genetic polymorphisms have been associated with this condition.

(2) Cross reacting urticaria and angioedema: Exacerbations of urticaria and/or angioedema induced by COX-1 inhibitors are observed in up to one third of patients with chronic urticaria, more often with drugs of the heteroaryl group (naproxen, diclofenac and ibuprofen). Various genetic polymorphisms, including genes coding for HLA antigens, LTC₄ synthase, 5-lipoxygenase, and the high affinity receptor for IgE have been observed in these patients.

(3) Urticaria, angioedema and anaphylaxis induced by multiple NSAIDs: In patients who do not suffer other morbid conditions NSAIDs can precipitate acute urticaria, angioedema or systemic reactions. This variant of hypersensitivity is more prevalent in atopic individuals and facial angioedema is the most frequent clinical manifestation. It has been associated with A444-C allele of LTC₄ synthase.

(4) Urticaria, angioedema and anaphylaxis induced by a single NSAID: More frequently triggered by pyrazolones, but also reported for aspirin, paracetamol, ibuprofen, diclofenac and naproxen.

These reactions constitute about 30% of adverse reactions to NSAIDs and are observed with increased frequency in patients with previous history of atopic disease, food or drug allergy. The clinical manifestations include urticaria, angioedema, laryngeal edema, anaphylaxis, generalized pruritus, rhinitis or bronchospasm. Delayed reactions begin after 24 hours of NSAID exposure, can be induced by a single or multiple cross-reacting NSAIDs and are clinically expressed either as organ specific or as multisystemic diseases.^[2]

Examples of organ specific diseases are

(1) Skin: Maculopapular exanthemas, fixed drug eruptions, Bullous reactions (erythema multiforme, Stevens-Johnson syndrome, toxic epidermal necrolysis), acute generalized exanthematous pustulosis, contact and photocontact dermatitis.

(2) Lung: Pneumonitis.

(3) Central nervous system: Aseptic meningitis.

Herewith we are reporting a case of diclofenac induced hypersensitivity necessitating hospitalization.

CASE REPORT

A 29-year-old female was admitted to the hospital with chief complaints of rashes all over the body along with erythematous Oedematous plaque and urticarial weal positive from day before admission. The Patient has no history of similar complaint in past. No history of any allergic disorder or allergy to medication. Patient had no family history of drug allergy. Patient was not on any other medication.

On physical examination and systemic examination, the patient was confirmed with diclofenac drug hypersensitivity and had no relevant previous history with diclofenac hypersensitivity reaction on her past. Apart from platelet count all other lab value and vitals were normal. No sun exposure was allowed during the testing and other treatments were paused. The drug induced hypersensitivity was confirmed by patch test.

The patient was a normal lifestyle oriented person with normal bowel bladder, sleep. She takes mixed diet and had a habit of drinking caffeine more than 3 cups per day, the patient is an illiterate and belongs to low socio-economic category. The patient was prescribed with Anti-Histamines and topical applications. The goal of treatment is to reduce the Severity of the allergic reaction and to minimize the further complication. The patient was counselled accordingly with aspects to diseases, drug and lifestyle modification to control and emphasized regarding the importance of therapy and to improve the quality of life of the patient.

DISCUSSION

Hypersensitivity is a state of altered reactivity in which the body reacts with an exaggerated immune response to a foreign agent, which have anaphylaxis and allergy are the usual forms as response. The patient has a typical erythematous demetons plaque all over the body. On

lab investigation, the platelet counts were increased and rest of all other lab parameters were found normal. Diclofenac is one of the most popular drugs world-wide belonging to the family of nonsteroidal antiinflammatory drugs (NSAIDs) as explained by Andrea *et al.*^[3] Diclofenac causes either Hypersensitivity rashes or urticarial angioedema which are the common and highly documented.^[5] Patients should be alarmed about their hypersensitivity and drug induced allergy so that the drug can be prevented for future use. Discontinuation of the drug is the best therapeutic approach and patient counselling regarding the condition was given for better patient care.

CONCLUSION

Diclofenac induced hypersensitivity is more common among Indian population due to lack of awareness and irrational drug usage along with higher usage of OTC medications. Hence it's essential for a pharmacist to create awareness and to improve the quality of life of the patients. And Diclofenac should be prescribed with caution along with allergy check test or patch test when it's used for the first time. Avoidance of the medication in cases of a previously reported proven allergy to Diclofenac is of great importance.

COMPLIANCE WITH ETHICAL STANDARDS

Written informed consent was obtained from the patient for publication of the case study, inclusion of the accompanying images. Copies of written consent may be requested for review from the corresponding author.

CONFLICT OF INTREST

The authors declare no conflicts of intrest concerning the content of this case report.

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