



Aceclofenac-Induced Bilateral Eyelid Angioedema

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Abstract

Among the most often observed drug hypersensitivity reactions in clinical practise are those brought on by nonsteroidal anti-inflammatory medication (NSAIDs). These reactions are characterised by angioedema, urticaria or both. Mainstay of treatment is cessation of NSAID intake. We describe a rare case of bilateral localised eyelid angioedema caused by aceclofenac.

Keywords: Angioedema; Hypersensitivity, Urticaria, Aceclofenac.

Introduction

Angioedema and urticaria triggered by hypersensitivity to nonsteroidal anti-inflammatory medications (NSAIDs) are becoming more common^{1,9}. Atopic dermatitis, female sex, youth, a history of chronic urticaria, and the use of NSAIDs for the alleviation of acute pain can all be predisposing factors.⁷ An improved understanding of the pathogenesis of adverse reactions to NSAIDs has resulted from the description of two distinct arachidonic acid cyclo-oxygenases (COX), designated COX-1 and COX-2¹. There are three main clinical phenotypes: Nonsteroidal anti-inflammatory drug-induced urticaria/angioedema (NIUA), NSAID-exacerbated cutaneous illness, and NSAID-induced urticaria and angioedema. For effective therapy, the kind of cutaneous reaction must be classified.²

We herein present a 28-year-old female with aceclofenac induced bilateral angioedema.

Case report

A 28-year-old female presented with abrasion on forehead due to fall from stairs associated with swelling of the left eye (Figure 1). The patient had taken aceclofenac for pain relief. Swelling was noticed after 6 hours of intake of aceclofenac.

Initially the swelling was attributed to the trauma and the treatment was continued. Following day patient presented with typical picture of angioedema bilaterally, not associated with itching (Figure 2). On examination, bilateral diffuse oedema was observed in both eyelids of both eyes, associated with reddish discoloration of the skin. Patient had a blood pressure of 110/70 and pulse rate of 92/min. The NSAID was suspected as the causative agent for the same and discontinued. There was no symptom or evidence suggesting laryngeal oedema. Patient was treated with bilastine 20 mg after dinner. Angioedema resolved within 10 hours. Patient was given reassurance

and advised for follow up for recurrence or any other complaint.



Figure 1 Unilateral angioedema involving left upper eyelid



Figure 2 Bilateral angioedema involving both upper and lower eyelid

Discussion

Aceclofenac a phenylacetic acid derivative has been demonstrated to be an effective agent in the management of trauma pain. Aceclofenac is well tolerated, with most side effects being treatable, and primarily GI-related.^{3,4} NSAIDs are a common over-the-counter remedy to relieve inflammation. However Prevalence rates of urticaria and angioedema with NSAIDs ranging from (0.1-0.3%) has been steadily increasing, which are partially attributed to the size of the exposed (at risk) population^{1,2,9}. Widely accepted theory for NSAID induced urticaria states that COX 1 inhibition causes a shunting of arachidonic acid metabolism towards the 5-lipoxygenase pathway, which leads to an increase in the synthesis and release of cysteinyl leukotrienes.^{1,4} Indomethacin, naproxen, and diclofenac, NSAIDs that primarily inhibit COX-1 enzymes, have greater rates of hypersensitivity reactions than weak COX-1 inhibitors and selective COX-2 inhibitors, which are frequently better tolerated with a lower likelihood of hypersensitivity

reactions.¹⁰ Patients showing hypersensitivity to NSAIDs can be also cross reactive to high dose paracetamol. The timing of symptoms, specifics of the symptoms felt, a correlation between symptoms and consumption of the offending drug(s), concurrent prescriptions, and personal history of atopy must all be included in a thorough history. Delayed NSAID hypersensitivity reactions are rare, and the time from NSAID treatment to the onset of symptoms ranged from 2 days to 6 weeks. Physical examination is crucial, especially for NSAID hypersensitivity reactions involving the skin. A correct categorization, accurate prognostic assessment, and suitable care are made possible by pattern identification of skin lesions, evaluation of mucosal involvement, and assessment of the extent of skin involvement.¹⁰ Since skin prick tests with NSAIDs have not been successful and no accurate in vitro testing are available, the diagnosis of NSAID sensitivity is typically made by history. Oral test administration is the sole reliable diagnostic procedure.^{5,6,8} For effective therapy, the kind of cutaneous reaction must be classified as it is safe to utilise chemically unrelated COX-1 inhibitors in patients with isolated NSAID-induced responses.² For treatment of angioedema strict NSAID avoidance is mainstay of the therapy. Evidence of airway obstruction, if present necessitates immediate airway management. Concern or evidence for Anaphylaxis necessitates administration of adrenaline, corticosteroid and H1 & H2 antagonist. Hereditary angioedema if suspected may necessitate use of icatibant. Patient must be observed for worsening of angioedema.¹¹

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