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Research Article

Angiotensin-Converting Enzyme (ACE) Induced Angioedema: A Case Report

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ABSTRACT
A thirty five-year-old woman developed angioedema during her treatment with the drug perindopril, an angiotensin-converting enzyme (ACE) inhibitor for hypertension. The woman received perindopril at a dose of 10 mg once daily for hypertension. After six hours of administration, she developed swollen lips and lower part of the face, and difficulty upon swallowing. Investigations revealed the high blood pressure with an increase in pulse and respiratory rate. The pharynx was oedematous. She was diagnosed with perindopril-induced angioedema. Perindopril was discontinued and the patient received intravenously hydrocortisone stat and oxygen 4L/min and followed up with cetirizine 10 mg od and prednisolone 10 mg tds for 5 days. The patient had fully recovered after five days. A Naranjo assessment score of 7 was obtained, indicating a probable relationship between the patient's symptoms and her use of drug perindopril.

Keywords: ACE inhibitors, angiotensin-converting enzyme, perindopril, ACE inhibitor-induced angioedema, West Indies, Trinidad and Tobago

INTRODUCTION

Angiotensin-converting enzyme (ACE) inhibitors are the most common prescribed medications either alone or in combination with other drugs for the treatment of hypertension, congestive cardiac failure, myocardial infarction, renal failure, and diabetic nephropathy. [1] The widespread use of ACE inhibitors is associated with various adverse effects including angioedema. [2] ACE inhibitors are the leading cause of drug induced angioedema.ACE inhibitor-related angioedema is a class effect that can affect 0.1% to 0.5% of patients taking the drug. [3] It is rarely documented in Trinidad and Tobago. Perindopril is a commonly prescribed ACE inhibitor which is considered to be generally safe and well tolerated. We report a case of angioedema following the use of perindopril.

Case Report: This is the case of a 35-year old woman of the African ethnic group, presented to her physician with an elevated blood pressure and a past history of polycystic ovarian syndrome. She was placed on drug perindopril 10mg once daily. Six hours post administration of the first dose of perindopril, patient re-presented to the medical emergency unit of the hospital with swollen lips and lower face associated with difficulty upon swallowing (Fig. 1). The pharynx was also oedematous. On examination, her blood pressure was 155/95 mmHg. The pulse rate was 98 beats per minute and respiratory rate was 24 breaths per minute respectively. There was no swelling on any other part of her body. She had neither skin rashes nor pruritus. The patient has never had any previous episodes of swelling. The patient had no history of allergies to any foods or drugs, and there was no significant family history; thus, a clinical assessment of perindopril-induced angioedema was made. The perindopril was discontinued and she was treated with intravenously hydrocortisone 200 mg stat and oxygen 4L/min and followed up with tablets cetirizine 10mg od and prednisolone 10mg tds for 5 days. The patient was reviewed after 5 days at outpatient unit. She was fully recovered and there was no symptoms of swelling. The Naranjo assessment score of 7 indicated a probable relationship between the patient's symptoms and her use of drug perindopril. The patient was subsequently placed on a combination of valsartan and hydrochlorothiazide (160/12.5 mg) to treat her hypertension.

DISCUSSION

Angioedema is swelling of the deep dermis and subcutaneous tissues. It is caused by exposure to drug, venom, dietary, or extracted allergens. It is characterized

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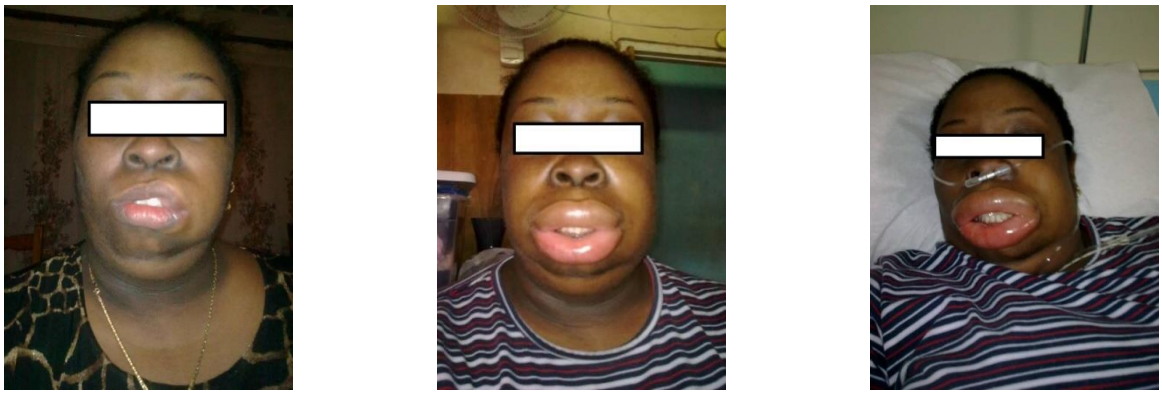

Fig. 1: Photographic documentation of angioedema of the lower face and lips

by locally diffuse and painful soft tissue swelling that may be asymmetric, especially on the eyelids, lips, face and tongue but also on the back of the hands or feet and on genitalia. [4] Hereditary angioedema is caused by a deficiency or dysfunction of C1 inhibitor, a protein that regulates the classical complement activation pathway. [4] The causes of Non-hereditary angioedema are variable and include acquired C1 esterase inhibitor deficiency, idiopathic or due to an allergic reaction to food, inhalants or immune complex diseases. [4] The mechanism of ACE inhibitors induced angioedema is thought to be related to its effect on the kallikrein-kinin system. Kallikrein is a protease that converts high-molecular-weight kininogens into kinins, primarily bradykinin. [5] ACE inhibitors inhibits the breakdown of kinins and thereby increasing availability of bradykinin in circulatory plasma. These kinins have multiple effects and are probably mediators of various reactions including urticarial, flushing, pruritic reactions and angioedema. [3]

ACE inhibitors-induced angioedema can be potentially a life-threatening adverse effect that can occur at any stage of treatment, even after prolonged exposure for up to several years. The incidence of ACE inhibitors-induced angioedema is presently underestimated and the actual incidence can be far higher because of poorly recognised presentation due to its late onset, concomitant use of other drugs, and long term treatment. Additionally, a spontaneous reporting bias can contribute to an actual higher incidence of this phenomenon. [6] Further, the incidence of ACE inhibitors-induced angioedema is reported 3 to 5-foldshigher among black peopleof African or American origin. [7] In the present case, this can partially account for occurrence of angioedema since the patient was of African descent.

The use of ACE inhibitors is of significant importance in Trinidad and Tobago since ACE inhibitors are available to public on prescription in all public sector hospitals and also in community pharmacies as a part of chronic disease assistance programme (CDAP). Hence it is assumed that a large number of patients would be using ACE inhibitors for their cardiovascular and renal impairments. Moreover, approximately 40% of Trinidad population is of African ethnic origin. This population would be at higher risk of developing angioedema due to higher incidence rate among blacks.

This case is presented for the purposes of documentation since a larger part of the Trinidad population is of African ethnic origin and a careful supervision is required to monitor this rare but potential adverse effect. It is suggested that the ACE inhibitors should be initially started under direct medical supervision and patient should also be informed about the possibility of ACE-inhibitors induced angioedema.

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