Case Report **IJCR (2020) 4:144**

International Journal of Case Reports (ISSN:2572-8776)

Visceral Angioedema caused by Angiotensin Receptor Blocker

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ABSTRACT

In literature we see clear evidence of Angiotensin Converting Enzyme inhibitors (ACEI's) causing visceral angioedema, but further evidence is required to establish the causality for Angiotensin Receptor Blockers (ARB's). With the widespread usage of these anti-hypertensives and the potential serious adverse effect in the form of angioedema; clinicians should consider medication induced as part of their differentials.

Keywords: Visceral Angioedema, Angiotensin Receptor Blocker

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How to cite this article:
Arun Minupuri, Roshni Patel, Jesus Salas Noain. Visceral Angioedema caused by Angiotensin Receptor Blocker. Inter-national Journal of Case Reports, 2020 4:144

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Website: https://escipub.com/

Introduction:

The incidence of angiotensin-converting enzyme inhibitors (ACEI's) induced angioedema is about 0.1 to 0.7 percent, while angiotensin receptor blockers (ARB's) have an incidence of 0.1 to 0.3 percent [1]. Additionally, in both classes of drugs, African Americans tend to have a higher incidence [2]. Both of these drugs are used to treat hypertension and CHF and the common side effects include cough, headache, dizziness, weakness, and angioedema [3].

Current literature provides a clear relationship between ACEIs and visceral angioedema, but not with ARBs [3]. In our case, we present a relatively rare presentation of visceral neck angioedema associated with the use of ARB.

Case Presentation:

A 61-year-old African-American female presented to our institution and described her symptoms as "throat closing up and lightheadedness". Denied any fevers/chills, cough, wheezing, chest pain, abdominal pain, or sick contacts. Her medical history was significant for hypertension, asthma, chronic obstructive pulmonary disease and obstructive sleep apnea. Smoking history positive for 15 pack years.

In the emergency department, patient presented with tachypnea, tachycardia and stridor. She was afebrile, labs revealed a white count of 11.8, and a lactate of 2.8. Initial management consisted of multiple rounds of nebulizer treatments and intravenous steroids. With no significant improvement, a trial of intramuscular epinephrine was given; which led to improvement in her symptoms.

Due to the patient's stridor, contrast-enhanced computed tomography (CT) of the neck was done; which showed inflammation/edema of the nasopharyngeal soft tissues and epiglottic folds. Her losartan was discontinued, as a concern for ARB-induced visceral edema. She continued to receive steroids and nebulizer treatments. C1 esterase and C4 complement levels turned out to be negative.

Repeat imaging of the neck next day showed improvement of the mucosal inflammation/edema.

Discussion:

When a patient presents with laryngeal edema, the differentials include, tonsillitis, peritonsillar abscess, idiopathic and hereditary anaphylaxis, foreign body, and medication side-effects. Workup includes CBC, erythrocyte sedimentation rate, C-reactive protein, C1-esterase, complement levels, and neck imaging. In our case with other differentials being ruled out, the most likely etiology was ARB induced visceral edema.

ARB's work by preventing angiotensin II to bind to angiotensin II AT1 receptors, a pathway that does not directly interfere with the kinin pathway [4]. Hence it would be rational to conclude that ARB's do not cause bradykinin related vasodilation and subsequent angioedema; but emerging evidence point towards the contrary as there have been increased levels of bradykinin in patients who are on ARB's [3, 5]. One proposed mechanism state that increased AT 1 receptor blockade by ARB leads to a feedback increase in angiotensin II levels, and this increase is enough to suppress the activity of ACE levels leading to increase in bradykinin levels [3].

ARB's related visceral angioedema is considered a rare occurrence, but the widespread usage of these drugs and the potential life-threatening side effect, should alert clinicians to consider medication induced as part of their differentials.

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