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**Angiotensin converting enzyme related angioedema**

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**Introduction**

A 54 year old female patient arrives to the emergency department (ED) via squad complaining that 2 days ago her lips and face were swollen. She was diagnosed with hives and told to take an antihistamine. Today she woke up with her tongue protruding out of her mouth, lips, face and eyes were swollen. She does not present with pruritus or urticaria. Squad personnel established an IV, gave her loratadine and a prednisone 50 mg PO which was not tolerated and she presented with non-tender swelling without any change in her complexion. She is allergic to penicillins and is drug class specific, not dose specific, with angioedema from ACE inhibitors. From a history and physical exam can help the practitioner make the correct diagnosis.

**Pathophysiological process**

Angiotensin converting enzyme (ACE) is activated by the negative feedback loop of the renin-angiotensin-aldosterone system (RAAS). It is stimulated in the kidneys by deceased circulating blood pressure, serum sodium concentrations, or seeing low renal perfusion; in hypertensive patients this could be from renal artery stenosis. Renal stimulation create formation of angiotensin I is then converted to angiotensin II (a potent vasoconstrictor) by ACE in the lungs. Angiotensin II causes vasoconstriction and aldosterone secretion, which stimulates aldosterone and water retention in the tubules of the kidneys that contributes to the increase in blood pressure (Appendix A). Another function of ACE is that it breaks down the endothelins (B2 receptors, leading to a change in vascular integrity and subsequent edema).

The adaption or lack of degradation of bradykinin by ACE allows edema to occur. Eventually angiotensin II levels return to near normal which more common pathways are used for activation and hypotension control. While non-pitting edema can be seen, ACE inhibitors interfere with the RAAS by blocking the conversion of bradykinin to kinogen. Kinogen is converted to kininogen in the liver, which is then converted to active bradykinin by ACE inhibitors 

**Implications for nursing care**

The most important implication for nursing care is to recognize the life threatening airway compromise that can occur with ACE inhibitors. Current Medicinal Chemistry, 19 (2013) 499-528, Bruse, P., & Rudnick, M. (2012). Non-histaminergic angioedema: Focus on bradykinin mediated angioedema. Clinical & Experimental Allergy, 43, 385-394. 10.1111/j.1365-2222.2012.04219.x. Studies have shown that many ACE inhibitors are being evaluated with kallikrein inhibitors and bradykinin receptor antagonists but are not approved yet in the United States for ACE inhibitors. An additional treatment option that has been used is the use of fresh frozen plasma which contains ACE which breaks down bradykinin but also kininase, a bradykinin substrate (Lewis, 2013).

**References**


