Angiotensin converting enzyme related angioedema

Andrea Sims
Otterbein University, andrea.sims@otterbein.edu

Follow this and additional works at: http://digitalcommons.otterbein.edu/stu_msn
Part of the Medical Pathology Commons, and the Nursing Commons

Recommended Citation
Angiotensin converting enzyme related angioedema
Andrea Sims BSN, RN, CEN
Otterbein University, Westerville, Ohio

Introduction
A 54-year-old female patient arrives to the emergency department (ED) via squad complaining that 2 days ago her lips were puffy, her face and eyes were swelling. She denied any recent travel, new medications or inciting foods. She denied any history of atopy or asthma. She was known to take Levothyroxine but was not on any other medications. She denied hospital admission during the past year. She was 5'10" and weighed 185 lbs. She had hypertension for which she takes Lisinopril and hydrochlorothiazide. According to her medical history, she has a history of chronic urticaria. She was started on a prednisone taper and discharged home.

Pathophysiological process
Angiotensin converting enzyme (ACE) is activated by the negative feedback loop of the renin-angiotensin-aldosterone system (RAAS). Both kidney and lung systems are involved in RAAS regulation. ACE has two main substrates: angiotensin I and kallikrein-kinin system. Angiotensin I is converted to angiotensin II (a potent vasoconstrictor) by ACE in the lungs. Angiotensin II causes vasoconstriction and aldosterone hypersecretion through aldosterone production and constricts blood vessels. Bronchial tone is increased with the production of bradykinin. ACE inhibitors interfere with the RAAS by blocking the conversion of angiotensin I to angiotensin II by inhibiting the ACE enzyme (Winters, M., Rosenbaum, S., et al., 2010). The effect of lower blood pressure with ACE inhibitors is also thought to be the pathophysiology involved in ACE angioedema. Histamine release may be involved in the pathophysiology of angioedema but common treatment regimens such as antihistamines, histamine blockers, steroids, and adrenaline do not markedly improve swelling, therefore, it is unlikely that histamine plays a significant role in this form of angioedema (Rasmussen, Mey, & Busse, 2011).

Signs and symptoms
Angioedema (AE) is characterized by non-pitting edema of the dermis and subcutaneous tissues. The most common sites of involvement are the lips, face, tongue, and mucous membranes. Angioedema is not associated with wheals or urticaria. It is different from urticaria in that it is typically self-limiting and often resolves within 72 hours. Facial swelling, dysphagia, and dyspnea are some of the symptoms that may require primary or advanced airway control (Appendix A).

Acute swelling can occur in the extremities, genitalia, and throat. Airway compromise is rare but drooling, stridor and throat. Observation of the patient for 12 to 24 hours in the ED or an admission to the hospital (Rasmussen, Mey, & Busse, 2011) will depend on the severity of the edema and potential for airway compromise. The practitioner should take the patient’s history and review their medications. It is prudent that this diagnosis is recognized, the patient is given educational material, and the patient is monitored for future reactions.

Implications for nursing care
The most important implication for nursing care is to recognize the life threatening airway compromise that can occur with this type of reaction. Observation of the patient for 12 to 24 hours in the ED or an admission to the hospital (Rasmussen, Mey, & Busse, 2011) will depend on the severity of the edema and potential for airway compromise. The practitioner should take the patient’s history and review their medications. It is prudent that this diagnosis is recognized, the patient is given educational material, and the patient is monitored for future reactions.

References
Andrea Sims BSN, RN, CEN
Otterbein University, Westerville, Ohio

Winters, M., Rosenbaum, S., et al. (2010). The incidence of angioedema, related to angiotensin-converting enzyme inhibition, is 0.1% to 1% of patients. A retrospective analysis by Tai et al. states that new therapies are being evaluated with some success in fresh frozen plasma which contains ACE inhibitors. An additional treatment option that has been used with some success is inotropic drugs which increases the cardiac output (Rasmussen, Mey, & Busse, 2011).