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Comprehending Angioedema: ACE-Inhibitor Reaction
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Introduction/Case
In anesthesia one of the specialty areas would be the airway and angioedema affects the airway even sometimes to the point of compromising the airway to the point of emergency interventions. According to (“Angioedema - Immunology; Allergic Disorders,” n.d.) “Angioedema is edema of the deep dermis and subcutaneous tissue” due to increased vascular permeability include the following mast-cell, bradykinin and complement mediators”. While ACE-inhibitors are well known for use to combat hypertension they are also according to (“Angiotensin-converting enzyme (ACE) inhibitors,” n.d.) used for “Coronary artery disease, Heart failure, Diabetes, Kidney disease, Mj, Scleroderma and Migraines”. Which makes there treatment field more widely used than many truly understand. This makes the risk of ACE-inhibitor induced angioedema even greater with such a large range of patients. While many causes of angioedema are known According to (“ACE-inhibitor-induced Angioedema” UpToDate, n.d.) “Angiotensin-converting enzyme (ACE) inhibitors are the leading cause of drug-induced angioedema in the United States because they are so widely prescribed”.

Sings and Symptoms
The specific Signs and symptoms according to (“Angioedema - Immunology; Allergic Disorders,” n.d.) “edema is often asymmetric and mildly painful. It often involves the face, lips, and/or tongue and may also occur on the back of hands or feet or on the genitals. Edema of the upper airways may cause respiratory distress and stridor; the stridor may be mistaken for asthma. The airways may be completely obstructed. Edema of the intestines may cause nausea, vomiting, colicky abdominal pain, and/or diarrhea.”

Underlying Patho
The following article describes the process of the Pathophysiology concept behind ACE-inhibitor Angioedema (Campo, Fernandez, Canto, & Mayorga, 2013) “ACE-I AAG is due to excessive accumulation of bradykinin. The reason that only a small fraction of patients treated with ACE inhibitors develop ACE-I AAG may relate to genetic variations in bradykinin metabolism. During treatment with ACE inhibitors, various alternative enzymatic pathways for metabolism of bradykinin become critical (e.g. plasma aminopeptidase P). Individuals with lower activity of these alternative clearance pathways may be at increased risk of bradykinin accumulation and ACE-I AAG.” While many may wonder what Bradykinins role is according to (“Angioedema,” n.d.) “Plasma globulins called kinogens release bradykinin and cause vascular permeability and inhibition of ACE hinders the degradation of bradykinin”.

Significance of Patho
The pathophysiology concepts are significant in many ways such a treatment pathway and understanding risks involved. While many treatment options are different they all share a common goal which is to stop the progression of airway compromise. The key is choosing the correct treatment plan in order to stop the process that is occurring whether treating for Mast cell reaction, Bradykinin and complement, Acute, chronic, hereditary or acquired angioedema. The chosen treatment pathway can have great success or end with the death of a patient if the wrong treatment guidelines are chosen. While this can be exemplified by the example of mast cell reaction as a chronic condition which could compromise the patients airway resulting in death or a tracheostomy.

Implications for Nursing
While a nurse in advanced practice in any setting must be vigilant of this lurking threat to the most important aspect of life we call the airway the CRNA must be extra vigilant of the medications there patient is taking whether the ACE-inhibitor is new or a lifelong process the risk must always weigh heavy on the minds of those treating patients taking this class of medications. The goal should be not only to spread awareness but knowledge that expressly deals with the identification and treatment of angioedema.

References

Additional Sources
Kraschnewski, J., J., & Tobias, J. D. (2016). The contact activation and regulatory processes the risk must always weigh heavy on the minds of those treating patients taking this class of medications. The goal should be not only to spread awareness but knowledge that expressly deals with the identification and treatment of angioedema.

Treatment
According to (“Angioedema - Immunology; Allergic Disorders,” n.d.) “For bradykinin-mediated angioedema, epinephrine, corticosteroids, and antihistamines have not been shown to be effective. Angioedema due to ACE inhibitor use usually resolves about 24 to 48 h after stopping the drug. If symptoms are severe, progressing, or refractory, treatments used for hereditary or acquired angioedema can be tried. They include fresh frozen plasma, C1 inhibitor concentrate, and possibly ecallantide (which inhibits plasma kallikrein, required for the generation of bradykinin) and icatibant (which blocks bradykinin).” Also according to (“Angioedema - Immunology; Allergic Disorders,” n.d.) “Securing an airway is the highest priority obviously when the patient can no longer keep their own airway secure.”

[Image 124x145 to 240x234]

[Learn It, n.d.]