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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, MI, 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 wide 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."



("Angioedema - Pharmacy and Drugs," n.d.)



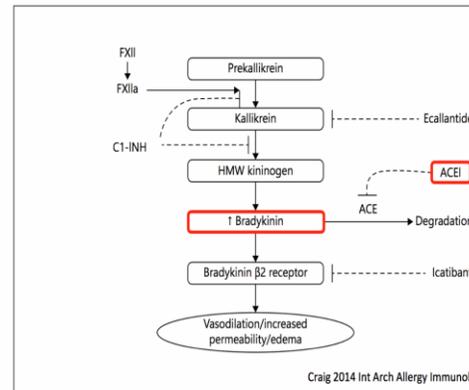
("Learn It," n.d.)

Underlying Patho

The following article describes the process of the Pathophysiology concept behind Ace-inhibitor Angioedema (Campo, Fernandez, Canto, & Mayorga, 2013) "ACEI-AAG is due to excessive accumulation of bradykinin. The reason that only a small fraction of patients treated with ACE inhibitors develop ACEI-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 ACEI-AAG." While many may wonder what Bradykinin role is according to ("Angioedema," n.d.) "Plasma globulins called kininogens release bradykinin and cause vascular permeability and Inhibition of ACE hinders the degradation of bradykinin"

Significance of Patho

The pathophysiologic 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 treating a mast cell reaction as a chronic condition which could compromise the patients airway resulting in death or a tracheostomy.



(Src="https://i2.wp.com/Emcrit.org/Wp-Content/Uploads/Gravatar/Farkas-Josh-Wc.jpg?w=80, Pulmonary, & Vermont, 2015)

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.

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 medication. The advanced practice nurse must know not only what type of Angioedema the patient is suffering from but how to properly treat the specific ailment the patient is experiencing. Also we must be able to bring a calm compassionate manner to the bedside of these patients who are fearful, anxious, and fighting for their airway in some cases. The hard choice must be made to take control of the airway if needed in some cases whether that be intubation or tracheostomy placement but this is the choice that lies heavy upon the advanced practice nurse.

Conclusion

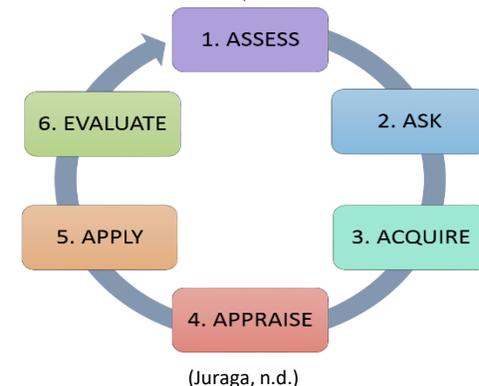
The potential of adverse effects from ACE-inhibitors should be heavily taken into consideration when reviewing the patient record and when prescribing this medication. The dangers of not being prepared to deal with this potential life threatening event could result in catastrophic consequences for the patient. The CRNA or Advanced practice nurse must be ready to identify the different causative factors and treat them in accordance with the proper treatment protocol. While ACE-inhibitors are a wonderful and useful drug that can treat multiple disease processes the risk must always way heavy on the minds of those treating patients taking these class of medications. The goal should be not only to spread awareness but knowledge that expressly deals with the identification and treatment of angioedema.

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(Juraga, n.d.)