Pathophysiology and Treatment of Life-Threatening Angioedema

Stephen J. Hoffman  
*Otterbein University, stephen.hoffman@otterbein.edu*

Follow this and additional works at: [https://digitalcommons.otterbein.edu/stu_msn](https://digitalcommons.otterbein.edu/stu_msn)

Part of the [Medical Pathology Commons](https://digitalcommons.otterbein.edu/stu_msn), and the [Nursing Commons](https://digitalcommons.otterbein.edu/stu_msn)

**Recommended Citation**

[https://digitalcommons.otterbein.edu/stu_msn/131](https://digitalcommons.otterbein.edu/stu_msn/131)

This Project is brought to you for free and open access by the Student Research & Creative Work at Digital Commons @ Otterbein. It has been accepted for inclusion in Nursing Student Class Projects (Formerly MSN) by an authorized administrator of Digital Commons @ Otterbein. For more information, please contact digitalcommons07@otterbein.edu.
**Introduction**

Angioedema is a potentially lethal swelling of the interstitial space from extravasation of intravascular fluid, which can impair organ function, cause hypotension, and threaten life. Angioedema may present in any medical setting with a broad range of severity. Angioedema can occur in emergency departments and intensive care units. According to Barbara, Ronan, Maddox, & Warner (2013), “angioedema is of particular importance to... mangoed mad, and can be due to hereditary deficiency or dysfunction of C1-inhibitor, or drugs including ACE inhibitors. Bradykinin is an inflammatory mediator that causes vasodilation, increased vascular permeability, nonvascular smooth muscle contraction, and oedema formation (Spyridonidou et al., 2010). It should be noted that anti-histamines and corticosteroids are ineffective in the treatment of non-histaminergic angioedema and epinephrine is only marginally effective (Moellman, et al, 2014)."

**Characteristics of Edema**

- Non-pitting
- Non-gravity dependent
- Often asymmetric
- Transient
- Self-limiting

**Location**

- Face
- Glottic structures
- Eyelids
- Tongue
- Extremity
- Inguinal cavity
- Trunk
- Throat

**Surgical Risk Assessment Tools**

- In 2001, Otis, Neubauer, Dubovik, Burnham, Kwiatek, & Deek (2001) described the Airway Risk Assessment Tool (ARAT), which is primarily used to risk stratify patients with a history of airway disease or surgery.

**Pathophysiology Histamine vs. Bradykinin**

- Histaminergic angioedema is IgE mediated, type I hypersensitivity reaction that results in the degranulation of histamine from mast cells, primarily mast cells. Histamine release causes localized oedema and increased vascular permeability resulting in the extravasation of intravascular plasma into the interstitial space causing swelling of the subcutaneous and submucosal tissues (Wool et al., 2013).

**Signs and Symptoms**

- Dyspnea
- Continuous cardiac monitor
- Vomiting
- Lethargy
- Genital involvement
- Hoarseness
- Face

**Estimated Classification in Primary Angioedema Cases Presenting to the Hospital**

- In 2013, Ichinose, Ibrahim, and Ireland (2013) proposed a staging system for angioedema: non-stage 1 facial rash, subcutaneous edema, and lip oedema. In this stage, 2 stage 2 upper airway involvement, and 3 stage 3 severe upper airway involvement. This staging system has been used in clinical practice to stratify patients for the appropriate level of care.

- In 2015, Iatrou, et al. (2015) proposed a staging system for angioedema: stage 1 facial rash, stage 2 upper airway involvement, and stage 3 severe upper airway involvement. This staging system has been used in clinical practice to stratify patients for the appropriate level of care.

**Conclusion**

- Healthcare providers need to be able to promptly recognize the clinical manifestations of angioedema because airway involvement and hypotension can be life-threatening. Airway swelling in the ED is a true emergency because it may rapidly become life threatening. Successful management of angioedema is critical to prevent complications and death. Angioedema is a serious medical emergency throughout the hospital and may rapidly become life threatening. Anesthesiologists, as it may present at any point in the perioperative period and it may rapidly become life threatening if it involves airway compromise (p. 335).

- The majority of angioedemas are non-histaminergic reactions, first line management is usually directed at treating all types of angioedema include anti-histamines (H1 and H2 receptor blockers) to blunt the immune response, and epinephrine is used in the treatment of histaminergic angioedema (Wool et al., 2013). For the treatment of ACE inhibitor activity level may be used in sub-acute setting (Sever et al. 2012). Plasma C1-INH concentrate should be administered if the patient is severely affected (acute facial angioedema that is not responsive to antihistamines) can be used to treat bradykinin-mediated angioedema in the acute setting (Moellman, et al. 2015). ACE inhibitor therapy can cause angioedema at any time, but most reactions occur within 24 hours of a dose change (Chan & Selim, 2015). ACE inhibitor converting enzyme (ACE, identical to Kinase II) is responsible for the breakdown of bradykinin at the renin-angiotensin system (RAAS). When ACE (Kinase II) is inhibited, bradykinin levels can progressively increase and cause angioedema. Discontinue the ACE inhibitor. Fresh frozen plasma administration has been successfully used to treat ACE inhibitor related angioedema presumably because plasma contains additional amounts of HAE (ACE), which are not present in donor plasma (Sever et al. 2012). More research work may need to be completed on the potential benefit of administering plasma C1-INH concentrate, BRX receptor antagonist, or kallikrein receptor blockers to patients with ACE inhibitor induced angioedema (off label use) (Moellman, et al. 2014). However, small studies are emerging with promising results. Brain, Zel.wp, Stari, V., et al. (2015) presented a case study that showed rapid resolution of ACE inhibitor induced, refractory, life-threatening laryngeal oedema after a single subcutaneous injection (R2 antagonist) (median time to injection was 0 minutes (95% confidence interval 0 to 30 minutes)).

**References**