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Acquired angioedema

Angioedema adquirido

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Acquired angioedema is a rare disorder that is characterized by deficiency of C1 inhibitor, a regulatory protein which predominantly controls formation of bradykinin from high molecular weight kininogen. Its deficiency leads to excessive bradykinin generation predisposing patients to develop recurrent attacks of bradykinin-mediated angioedema. Attacks are triggered by trauma or psychological stressors and often involve the upper airway and larynx.[1,2] The accompanying image demonstrates prominent angioedema of the lips during an acute attack that occurred intraoperatively (Fig. 1).

Unlike hereditary angioedema, acquired angioedema manifests in the 4th decade of life. Patients typically have no family history of angioedema and often present with dysphagia, throat tightness, dyspnea, or stridor. Presence of stridor portends impending complete airway obstruction and necessitates early intubation. Anesthesiologists should be aware of this condition as airway management often warrants a cautious approach to avoid a "cannot ventilate cannot intubate" situation. Angioedema of the lips can impede access to the oral cavity while an edematous tongue often acts as a physical barrier by occupying the entire oropharynx. Consequently, placement of airway devices, fiberoptic intubation and laryngoscopy are exceedingly challenging. In addition, positive pressure ventilation via a supraglottic device or mask is

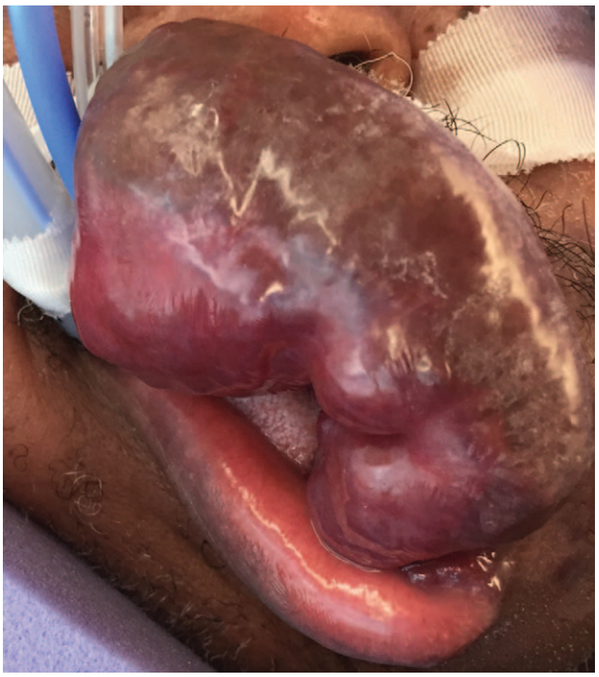

Figure 1. Prominent angioedema of the lips during an acute attack that occurred intraoperatively.
Source: Authors.

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ineffective in the presence of edematous vocal cords. It is prudent to prepare for an emergent surgical airway before intubation as laryngoscopy attempts can rapidly worsen laryngeal edema hastening loss of airway patency. Extubation is undertaken after resolution of airway edema.[2,3]

Unlike histaminergic angioedema that is commonly seen in immunoglobulin E (IgE)-mediated allergic reactions, bradykinin-induced angioedema does not resolve with administration of epinephrine, steroids, and antihistamines. Acute attacks are treated by restoring levels of C1 esterase inhibitor with plasma-derived concentrate, recombinant C1 inhibitor or fresh frozen plasma. Alternatively, icatibant, a bradykinin receptor antagonist or ecallantide, a plasma kallikrein inhibitor can be used to terminate acute attacks.[1,2]

Ethical responsibilities

Protection of people and animals. No experiments on people or animals were done.

Confidentiality of the data. All protocols at our institute were followed and patient or hospital identifiers have been removed from all images.

Right to privacy and informed consent. As patient and hospital identifiers have been removed, no informed consent was solicited for this production.

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Conflicts of interest

The authors have no conflicts of interest to disclose.

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