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THE ROLE OF CORTICOSTEROIDS IN THE MANAGEMENT OF ACUTE LARYNGEAL EDEMA IN THE EMERGENCY SETTING

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Abstract

Acute laryngeal edema is a potentially life-threatening condition characterized by swelling of the larynx, resulting in airway obstruction. Without prompt intervention, it can lead to respiratory distress and even complete airway obstruction. In the emergency setting, the identification and management of acute laryngeal edema pose significant challenges. Early recognition and appropriate management are essential to prevent complications and improve patient outcomes. Corticosteroids have been widely used in the management of acute laryngeal edema due to their anti-inflammatory properties. They work by reducing edema and inflammation in the larynx, thereby helping to alleviate airway obstruction. The use of corticosteroids in this setting has been supported by evidence from clinical studies and is included in many treatment guidelines. In addition to their role in reducing laryngeal edema, corticosteroids have also been shown to stabilize and prevent further progression of the condition. They are often used in combination with other interventions such as nebulized adrenaline and airway management techniques. It is important for healthcare providers to be familiar with the appropriate dosing, route of administration, and potential side effects of corticosteroids when managing acute laryngeal edema. This knowledge is crucial for optimizing patient care and ensuring positive outcomes in emergency situations.

Keywords: ALE, corticosteroids, ED, obstruction, stridor, extubation, postextubation.

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Introduction

Acute laryngeal edema is a potentially life-threatening condition that must be recognized and treated promptly when it occurs as it is a situation that rapidly progresses to airway obstruction, often within a period of minutes. Laryngeal edema occurs when there is an increase in capillary permeability and subsequent movement of fluid into the laryngeal tissue. There are many causes of laryngeal edema, both local and systemic, and often no cause is found. Examples of local causes include trauma, allergy, infection, and malignancy and can be classified into acute, and chronic. Symptoms associated with laryngeal edema include voice change, stridor, hoarseness, dyspnea, and loss of consciousness. Abduction of vocal cords is prevented due to edema occurring in supraglottic or glottic regions, and this causes the characteristic stridor and air hunger. Inspiratory stridor suggests life-threatening obstruction at the level of the glottis or supraglottis and should be treated as a medical emergency. Loss of consciousness is a poor prognostic sign and needs immediate resuscitative measures [1,2]. Due to the specter of airway obstruction and the speed of its onset, laryngeal edema should always be considered a medical emergency and a situation that requires immediate intervention. The site at which edema occurs is critical in determining the likelihood of airway obstruction, the more superior the location the greater the risk. Although deciding on and defining airway obstruction can be difficult, the subjective beliefs of ENT surgeons dictate that prophylactic measures should be undertaken if there is more than 50% reduction in the airway. This opinion is based largely on clinical experience with few studies focusing on the simple, accurate, and efficient diagnosis of progressive airway narrowing due to laryngeal edema. Due to the difficulty in diagnosis and defining the severity of airway obstruction, any case of laryngeal edema and particularly acute onset cases should be treated immediately and as though there is an imminent risk of airway obstruction. Corticosteroids have been widely used in the treatment of laryngeal edema, and there is growing evidence to suggest that they are beneficial in reducing the severity and duration of the condition. The ideal time scale for administration, dosage, and type of corticosteroid is uncertain, and often the choice of corticosteroid is based on physician or previous experience [3].

Definition of Acute Laryngeal Edema (ALE)

In simple terms, edema is defined as swelling, usually in reference to a soft tissue mass. When the swelling occurs in the larynx, it constitutes a life-threatening emergency. This can happen from a number of causes; often these are allergic or infectious in nature. They all have in common some sort of insult to the laryngeal mucosa which then results in the extravasation of plasma into the laryngeal tissues creating edema. This then can progress to complete airway obstruction from the swelling impairing the movement of the vocal cords and/or from the pooling of the edematous fluid above the level of the cords. At this point in time patients are generally in acute respiratory distress and without appropriate and timely intervention can proceed to respiratory-failure and arrest [4]. Ale starts more insidiously and also progresses to airway obstruction. It almost always occurs as a result of an insult to the laryngeal mucosa, with the swelling starting around the false vocal cords and the aryepiglottic folds. Although an important distinction is that with Ale the insult to the mucosa does not have to be recent and given the pharmacodynamics of corticosteroids it may be a few days after the initial insult before the steroids actually become useful. Ale is commonly a complication of extubation in an intubated patient and can also occur after any head and neck surgery. The author has also seen it in a patient who was treated with external beam radiation to the neck for an adenoid cystic carcinoma. Finally, there are idiopathic cases of Ale with the suspicion being that they are due to some allergic process. In all cases

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if the patient develops acute airway change from the edema, there is a variable latent period before complete airway obstruction. It is during this time frame that the diagnosis should be made and treatment initiated [3,4].

Importance of Managing Acute Laryngeal Edema in the Emergency Setting

Acute laryngeal edema is a life-threatening emergency that may result from allergic or non-allergic causes, such as infection, inflammatory disease, reflux or trauma; it may occur with little warning or gradually. The event can be sudden and patients often recall the exact moment at which their airway became closed. Symptoms may include dysphonia, dysphagia, dyspnea and inspiratory stridor. Early detection and treatment are necessary in order to prevent airway compromise which may lead to fatal asphyxiation. IList et al. (1997) reported that mortality from acute laryngeal edema is approximately 5% with rates reported as high as 44% in patients with compromised airways [4]. The threat of acute death and the unpredictable nature of laryngeal edema dictate a need for effective treatment. Since there are no widely accepted treatment recommendations for acute laryngeal edema, management is often tailored to individual cases and is based on personal experience and consensus rather than on evidence from clinical trials. This may involve close observation, humidified oxygen and nebulized adrenergic agents but may also require endotracheal intubation or tracheostomy. Non-invasive strategies are preferred but their success is varied and the clinical course of acute laryngeal edema is often unpredictable [5].

Pharmacology of Corticosteroids

Important medicinal drugs called corticosteroids are used to treat inflammatory and allergic diseases as well as to inhibit the immune system's improper or undesired responses. In the clinical context, drugs with glucocorticoid action are referred to as corticosteroids. The endogenous glucocorticoid cortisol is called for its effects on glucose metabolism, but it also carries out the other corticosteroid-mediated immune functions. The adrenal gland uses cholesterol metabolism to make cortisol. The common route of cholesterol metabolism also produces a range of other hormones, such as aldosterone, mineralocorticoids, and sex hormones for men and women. Some of the negative responses and side effects linked to pharmacologic dosages of cortisol and its synthetic analogs can be explained by the hormones' shared route and structural resemblances [6].

Cortisol, the main endogenous glucocorticoid, affects human physiology in a number of ways. These effects are pleiotropic and non-specific, impacting almost all human organs and metabolic functions. Corticosteroids are frequently used pharmacologically to reduce or avoid inflammation or allergic reaction symptoms, as well as to inhibit an undesirable or inappropriate immune response. Less frequently, when the hypothalamic-pituitary-adrenal axis is functioning normally or when circulating cortisol is low because of a primary adrenal condition or a secondary pituitary or hypothalamic failure that results in deficiencies of adrenocorticotrophic hormone or corticotrophin-releasing hormone, hydrocortisone is used for physiologic replacement of cortisol. The following describes the effects of corticosteroids on immunological response and inflammation. These substances also have an impact on the metabolism of fats, proteins, and carbohydrates, which among their many other effects, causes gluconeogenesis, protein catabolism, and fatty acid mobilization. In addition, cardiovascular homeostasis, the central nervous system, calcium and bone metabolism, and a host of endocrine effects are impacted by corticosteroids. Both glucocorticoid and mineralocorticoid activity are linked to

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impacts on cardiovascular health as well as fluid and electrolyte balance. These effects, which are substantial and frequently unwanted when corticosteroids are dosed pharmacologically, have physiologic repercussions that are covered in the section on adverse medication responses and side effects [7].

Corticosteroids are mostly utilized for their anti-inflammatory properties, but they also have positive effects on the β2-adrenergic receptor. In addition to helping to restore the downregulation of these receptors linked to chronic β2-adrenergic treatments, corticosteroids are linked to an increase of β2-adrenergic receptor activity. Increasing the coupling of β receptors to G proteins, which raises adenyl cyclase, and increasing the creation of new receptors are plausible pathways for this action at the β2-adrenergic receptor [8].

Efficacy of Corticosteroids for Laryngeal Edema

Evaluating the impact of corticosteroids on pediatric patients' extubation failure and laryngeal edema that may cause stridor following. Our meta-analyses showed that corticosteroids, when used in pediatric patients with or at risk of laryngeal edema, significantly reduce the rates of post-extubation stridor/suspected upper airway obstruction and extubation failure, despite the wide variations in corticosteroid regimens, cumulative doses, frequencies, and durations of administration among those trials. Each trial included children of varying ages, so only a limited number of subgroup analyses for different age categories could be carried out. However, the analyses did not reveal a significant difference in the odds of stridor for pediatric patients under five years old, excluding infants, nor in the odds of extubation failure for infants [9].

The most recent systematic review and meta-analysis was carried out in 2009 by McCaffey et al., which included both adults and children. They found that corticosteroid administration may prevent laryngeal edema (OR, 0.36; 95% CI, 0.27–0.49) and lower the incidence of extubation failure (OR, 0.56; 95% CI, 0.41–0.77) in critically ill patients of all ages. Concerns exist, although, regarding their conclusion [10]. Initially, the authors pooled data for adults and newborns, even though they included three studies for neonates and four papers for pediatrics in their analysis. The morphology and organization of the airways vary with age, therefore combining those data for analysis might not be acceptable. Secondly, they mentioned a research done by Tibballs et al. that involved kids who had croup [11]. Patients with croup should not be included in a meta-analysis evaluating the impact of corticosteroids on laryngeal edema since croup is among the conditions for which they are beneficial. A Cochrane Systematic Review assessed the effects of corticosteroids on each of these age groups independently in the same year [12]. There were two studies conducted on newborns (n= 109) and two trials conducted on children (n= 216). They found no statistically significant decrease in the rate of reintubation in neonates or children, and their meta-analyses revealed no statistically significant decrease in the frequency of post-extubation stridor in newborns. Only children with heterogeneity showed a statistically significant decrease in the frequency of post-extubation stridor. Based on the previous findings, we came to the conclusion that corticosteroids have not been shown to be effective in preventing stridor in newborns or children following extubation, and there is disagreement over whether or not corticosteroids should be prescribed in pediatric patients to prevent stridor following extubation [12,13].

When stridor/suspected upper airway obstruction was examined using meta-regression analysis, the duration between the initial corticosteroid dosage and extubation was shown to have negative

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coefficients in every model. The results of a Cochrane Systematic Review by Khemani et al., which demonstrated that corticosteroids are more effective when multiple doses are administered 12–24 hours prior to extubation, are consistent with the trends of greater effectiveness for a larger amount of cumulative corticosteroid dose and for longer duration between the first dosing and planned extubation, even though they did not show statistical significance. While a positive age coefficient in meta-regressions may indicate that corticosteroids are advantageous for younger patients, it was not statistically significant [12].

Conclusion

In conclusion, variations in corticosteroid regimens and administration among trials involving children of different ages, the overall findings suggest a significant reduction in post-extubation stridor and extubation failure rates with corticosteroid use. However, subgroup analyses for different age categories revealed mixed results, with no significant difference in outcomes for pediatric patients under five years old and infants Further research is needed to clarify the effectiveness of corticosteroids in preventing stridor in newborns and children post-extubation, as well as to optimize dosing regimens and timing for maximum benefit. The meta-regression analyses indicate a potential benefit of longer duration between corticosteroid administration and extubation, emphasizing the need for tailored approaches based on patient age and clinical context. Overall, while corticosteroids show promise in reducing post-extubation complications in pediatric patients, further studies are warranted to refine their use in clinical practice and inform evidence-based guidelines.

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Conflict of interests

The authors declare no conflict of interest.

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