

Postextubation stridor in the ICU

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Abstract

Endotracheal intubation is one of the commonest procedures performed in the intensive care unit. Once the requirement of the tube is no longer present and the patient has recovered the ability to maintain airway, clear secretions and has adequate gas exchange, extubation is done. Occasionally, the patients can develop post-extubation stridor. This CME article describes the various causes of postextubation stridor, its clinical features, diagnosis and management.

Keywords: Epinephrine, postextubation stridor, risk factors, steroid

Introduction

Endotracheal intubation is a commonly performed procedure in the intensive care unit. It is mainly used to ensure a secure airway and to facilitate mechanical ventilation. When the need has passed and the patient recovers his ability to breathe and maintain airway, the trachea is extubated. This is usually an uneventful transition back to the natural airway of the patient. Occasionally, the patient can develop stridor and breathing difficulty after extubation. This article details the causes, prevention and management of postextubation stridor (PES) in the intensive care unit.

Definition and incidence

Postextubation stridor is defined as presence of an inspiratory noise after extubation. The reported incidence of postextubation stridor varies between 1.5 to 26%.¹

Clinical features

The most evident clinical feature is an inspiratory noise. When this causes significant obstruction to movement of air through the glottis, the patient will manifest with indrawing of intercostal muscles, suprasternal retractions, restlessness and the

accessory muscles of respiration could be active. This could lead to hypoxia and hypercarbia with associated tachycardia, hypertension, arrhythmias and occasionally cardiac failure. Breathing strongly against a closed or partially closed glottis can lead to negative pressure pulmonary oedema. This happens in patients who have good respiratory efforts and is common in young, healthy people with good muscle power but have laryngeal obstruction.

Causes

Stridor after extubation may occur acutely due to several reasons: Trauma during intubation resulting in glottic oedema, residual effects of sedatives and neuromuscular blockers, airway obstruction due to inadequate pharyngeal tone, pharyngeal or neck haematoma, nerve palsy, hypocalcaemia after thyroidectomy and rarely even a foreign body. Laryngeal oedema is the commonest cause in the ICU. Stridor can occur after a few days or months after extubation and this is usually due to granulation tissue at the glottis or laryngotracheal stenosis but this is beyond the purview of this article.

Risk factors for development of postextubation stridor²⁻⁵

Postextubation stridor occurs mainly due to laryngeal oedema. The causes can be classified into tube factors, airway factors, patient factors and disease factors.

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Tube factors: These are when a large endotracheal tube has been used for the airway (> 45% of the diameter of the trachea), prolonged intubation (> 5 days), higher cuff pressure (> 25–30 cm H₂O), small airway as in children, infants and in the female gender.

Airway factors: These include injury to airway due to trauma or during intubation, airway surgery, repeated attempts at intubation as in difficult airway, reintubation (because of multiple attempts), self-extubation where the cuffed endotracheal tube is pulled out by the patient himself with the cuff still inflated or there has been excessive movement of the endotracheal tube within the glottis due to agitation and under-sedation.

Patient and disease factors: These include old age, overweight (BMI > 26.5 kg/m²), high SAPS II score, high APACHE score and in patients with medical reasons for admission to ICU, where the severity of illness is higher.

Prevention

Careful assessment of the patient's eligibility for extubation is vital. It must be ensured that the patient has the ability to maintain his airway. The level of consciousness should be adequate as can be defined by a GCS > 8. He should be able to cough and clear the airway of secretions. This is indicated by a peak expiratory flow rate of > 60 L/min and a vital capacity at least three times the tidal volume. He should not require suctioning more than once every two to three hours. If any of these factors are present, extubation can fail.

Postextubation stridor due to laryngeal oedema in the ICU can be prevented to a large extent by using appropriate sized tube; 7 mm ID for women, 8 mm ID for men, keeping them intubated for a minimum duration of intubation, inflating the cuff to a pressure not exceeding 25 cm H₂O and any other modifiable risk factor.

Diagnosis

The diagnosis and differentiation of stridor due to functional factors as against structural problems, either a videolaryngoscope or a fiberoptic bronchoscope is required to visualise the vocal cords.

Ultrasonography (USG): USG has been advocated recently where the air column width (ACW) at the level of vocal cords before and after deflation of the cuff is measured. The difference (ACWD) between the two is calculated. Patients who developed a stridor had a significantly lower ACWD, (0.35 vs 1.5 mm; P < 0.01) and lower ACW during cuff deflation (4.5 vs 6.4 mm; P = 0.01) compared to patients who did not.⁶

Cuff leak test: A cuff leak test can be performed to clinically check whether there is glottic oedema. The ventilator is set to assist-control (*i.e.*, VCV mode). The throat is cleared of all airway secretions. The inspiratory and expiratory tidal volumes are recorded and then the cuff is deflated. The expiratory tidal volumes are now recorded over the next six breathing cycles. It is not appropriate to accept only the first leak volume since it may lead to error. Record those six expiratory volumes and *average the three lowest values*. The cuff leak is the difference between the inspiratory tidal volume and the average expiratory tidal volume.

Miller and Cole in their study on cuff leak volumes found that the volume with the best predictive value for PES was 110 ml. If leak < 110 ml, 67% developed stridor after extubation. Of the patients with >110 ml, practically nobody developed stridor. Only 6 patients out of the 100 developed stridor. The mean leak was actually around 350 ml.⁷

Among the ten studies listed, the cut-off values for a cuff leak have ranged from 88 ml to 140 ml (10% of inspired tidal volume) as the leak cut-off. The general observation was that the absence of a leak does not preclude the possibility of a successful extubation. In the presence of a cuff leak, one may be reasonably confident that post-extubation stridor will not occur.⁸

Procedure for extubation after prolonged intubation in the ICU

Perform cuff leak test for all patients before considering extubation. Measure the cuff leak volume (CLV) to see if it is less than or more than 100 ml (in an adult of average size). If the CLV is > 110 ml, there is no risk of postextubation stridor. If

< 110 ml, and if there are additional risk factors such as too large a tube, prolonged intubation, previously failed extubation, and high cuff pressures, administer steroids about 4–5 hours prior to extubation.

Steroids: Methyl prednisolone 20 mg IV every four hours, dexamethasone 0.1 mg/kg IV starting at least 4 hours prior to extubation can be used. It is preferable to start steroid the day prior so that multiple doses are given prior to extubation. The last dose is timed to be given just prior to extubation. Removal of the tube may be done over a tube exchanger if there is a fear of losing the airway after the procedure.

Observe closely after extubation for an hour. If there are no symptoms, the airway exchange catheter can be removed. If the patient develops stridor, the following options may be considered. Continue Methyl prednisolone 20 mg every 4–6 hours and nebulise Budesonide 1 mg.

Epinephrine: If stridor is severe and is due to laryngeal oedema, epinephrine can be nebulised to produce mucosal vasoconstriction and reduction in the oedema. If the patient is < 4 years, 2.5 mg epinephrine diluted with 4–5 ml normal saline can be given. In patients > 4 years, 5 mg dose is sufficient with no more dilution.

If racemic epinephrine is available, 0.5 ml of 2.25% solution diluted in 2.5 ml normal saline can be used. Excessive doses of epinephrine may cause chest pain, tachycardia, hypertension, myocardial ischaemia, cardiac arrhythmia. If epinephrine is being administered more frequently than 2 hours, close cardiorespiratory monitoring is indicated. It is also important to watch for a rebound oedema once the effect of the nebulised epinephrine wears off. If stridor is severe and unresponsive, reintubation may be necessary.

Noninvasive ventilation (NIV): NIV for postextubation stridor and consequent respiratory failure has been associated with increased mortality, probably due to the increased delay to intubation. Therefore, the use of NIV for extubation failure due to postextubation stridor is not recommended.

Heliox: Inhalation of heliox (helium-oxygen mixture) decreases airway resistance and work of breathing. There is no evidence on the efficacy of heliox administration in adults but reduction in respiratory distress score by 38% is reported in children.⁹ The use of heliox is not associated with any change in outcome. It decreases work of breathing and buys time to establish a definitive solution for the upper airway obstruction. This may be particularly useful in difficult to intubate patients.

Conclusions

Postextubation stridor is common in the ICUs. Prevention is the best method and cuff leak volume check is advocated for all patients before extubation. Steroids can be helpful and three doses can be given in advance of extubation. If stridor develops, nebulised adrenaline may help. NIV and Heliox may buy time but may also delay a much required intubation. If all else fails, reintubation and tracheostomy may become necessary.

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