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Post-Intubation Laryngeal Edema

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Continuing Education Activity

Post intubation laryngeal edema is common after endotracheal intubation and clinically manifests after extubation. Though most cases of post-intubation laryngeal edema are asymptomatic or mildly symptomatic, it is a significant contributor to the development of post-extubation stridor. Almost half of the patients with post-extubation stridor get reintubated. Reintubation event increases the risk of morbidity and mortality of the patients. It is of paramount importance to identify and manage the patients at high risk for post-intubation (extubation) laryngeal edema and stridor, thereby preventing their progression into worsening respiratory failure requiring a reintubation. This activity highlights the role of the interprofessional team in clinical evaluation and management of post-intubation laryngeal edema.

Objectives:

- Summarise the etiology of post-intubation laryngeal edema.
- Summarize the evaluation of post-intubation laryngeal edema.
- Review the management options available for post-intubation laryngeal edema.

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Introduction

Laryngeal injuries are common after endotracheal intubation, which could manifest as varying degrees of edema, ulceration, granulation, and restricted vocal cord mobility, often resulting in luminal narrowing. Among these conditions, laryngeal edema is a common complication following intubation and usually results from the direct pressure and the inflammatory reaction triggered by the endotracheal tube on surfaces of contact.

Nevertheless, although laryngeal edema is documented to be present in almost half of the extubated patients, the majority are asymptomatic or mildly symptomatic. On the other hand, laryngeal edema represents a common cause for breathing difficulty and/or stridor following extubation, thereby makes a common etiology for extubation failure and the need for reintubation. Thus, because reintubation is associated with augmented morbidity and mortality, the issue of post-intubation laryngeal edema is of paramount importance and needs for careful prevention and proper management.[1]

Post-intubation laryngeal edema (rather than the term 'post-extubation laryngeal edema') might be a more appropriate term to denote laryngeal edema, which has got the potential to cause respiratory difficulty and/or stridor following extubation. The pathologic process, which results in edema, indeed, starts soon after intubation though it becomes clinically evident only after removal of the endotracheal tube. The prompt recognition and management of post-intubation laryngeal edema before extubating a patient is extremely important, given the fact that any reintubation

event could increase the morbidity and mortality of the patients.[2]

In this chapter, we discuss the etiology, epidemiology, clinical evaluation, and management of post-intubation laryngeal edema, highlighting the need for identification in high-risk patients with a prompt institution of preventive and treatment measures. The interprofessional team strategies for improving care coordination and communication and, in turn, outcomes will also be addressed.

Etiology

Once the patient is intubated endotracheally, the pressure exerted by the tube incites an inflammatory reaction at the areas of contact, which could explain the basic mechanism for the development of laryngeal edema. Prolonged endotracheal intubation can result in inflammation, swelling, and ulceration of laryngotracheal areas of contact, especially the vocal cords, and the inflated cuff areas. An early study published by Darmon et al. in 1992 which involved 700 consecutive patients requiring endotracheal intubation and mechanical ventilation, noted the duration of ventilation more than 36 hours, and female gender as the risk factors for post-extubation laryngeal edema.[3]

Another study by François et al. conducted on 761 intensive care unit (ICU) patients found trauma at admission, female gender, short duration of intubation (< 1 week), lesser height to tube diameter ratio, and absence of pre-treatment with methylprednisolone as risk factors for the development of post-extubation laryngeal edema.[4] Moreover, the risk for this clinical condition is expected to be significant in patients undergoing head and neck surgery, those being made head down or prone positioned during neurosurgical procedures, pregnancy, fluid resuscitation, neck and airway injuries, inhalational injuries and burns, history of difficult intubation, and self-extubation.

Severe post-extubation (intubation) laryngeal edema requiring tracheostomy in a patient who had undergone bilateral modified radical neck dissection about seven years before the current urologic intervention has been reported with a probable explanation for increased edema risk being the destruction of lymphatics, or venous engorgement of the neck.[5] Gastro-oesophageal reflux is also believed to be a contributory factor for post-intubation laryngeal edema.[6]

In brief, the risk for post-intubation laryngeal edema can be summarized into three categories intubation factors, post-intubation factors, and patient factors.

Intubation factors:

- History of difficult intubation (prolonged intubation attempt)
- Large tube size (smaller height-to-tube diameter ratio)[4]

Post-intubation factors:

- Prolonged endotracheal intubation
- High cuff pressures
- Agitation while intubated
- High cuff pressures
- Self-extubation and reintubation

The patient, setting, and surgery-related factors

- Type of surgery (e.g., head and neck surgery)
- Prone positioning during neurosurgical procedures

- Pregnancy
- Fluid resuscitation
- Neck and airway injuries
- Inhalational injuries and burns
- Lower Glasgow Coma Scale score[7]
- Non-sedation treatment
- Female gender
- Body mass index (>26.5)[8]
- Gastro-oesophageal reflux

Epidemiology

The determination of the exact prevalence of post-intubation laryngeal edema is challenging because of wide variations in definition, diagnostic criteria, and the methods employed for the detection. This variation is further compounded by differences in the study population, and also due to differences like endotracheal tube material with the latter pertaining especially to the older studies.[3]

Grossly, the incidence of post-intubation (extubation) laryngeal edema varies between 5% to 54 % in different studies, whereas the reported incidence of post-extubation stridor ranges from 1.5% to 26.3%.[9] Of note, almost up to 10.5% of patients with laryngeal edema will fail extubation, and require reintubation. The reported overall incidence of post-extubation failure requiring reintubation varies from 1.8% to 31.4%.[10] The numerical differences for all these data are related to the different methods used and the different definitions adopted.

The literature offers other epidemiological data. Concerning gender differences, for instance, the incidence of edema is higher in females. A prospective French study on 136 intubated patients with post-extubation fibreoptic bronchoscopy evaluation observed laryngeal injury in 73% of patients, and laryngeal edema was the most common injury noted in 54.4% of the patients.[6]

Post extubation stridor was present in 13% of the patients. Laryngeal edema with decreased vocal cod mobility was present in almost two-thirds of the patients with stridor. Only half of the patients with post-extubation stridor got reintubated, and only half of the patients who got reintubated had post-extubation stridor. Previous studies had also noted a significant number of intubated patients having varying degrees of edema and ulceration of larynx.[11][12]

Pathophysiology

An endotracheal tube placed even for a short period causes superficial mucosal damage, whereas chronic placement might cause pressure necrosis of deeper layers involving submucosa, perichondrium, and even the cartilage. The laryngeal, as well as tracheal mucosa, are affected similarly. The sites of contact in the larynx usually involve the medial aspect of arytenoid cartilages, vocal cords, cricoarytenoid joints, posterior part of the glottis, and sub-glottis. While the area at the level of vocal cord commonly forms the area of contact in the larynx, the mucosal area in contact with the inflated cuff is involved in the trachea. If the edema involves only the arytenoids and the false cords, it does not cause luminal obstruction. Whenever the edema, ulceration, granulation, or vocal cord dysfunction causes laryngeal luminal narrowing, the resultant increase in airflow velocity manifests as stridor, which is a sign of clinically significant airway obstruction.

There is an association between laryngeal edema with resultant decreased vocal cord mobility, with the latter predisposing to adducted positioning of the cords, thereby increasing the work of breathing. The risk of aspiration is also high in patients with reduced vocal cord mobility, which increases the risk of respiratory failure. It should also be

noted that the above-mentioned factors (edema, ulceration, granulation, or vocal cord dysfunction) causing luminal narrowing will be present as single or in varying combinations in a given patient. It is assumed that stridor and respiratory distress occur when more than half of the luminal area is obstructed.[13]

Histopathology

A study in cynomolgus monkeys has shown significant polymorphonuclear infiltration at the laryngeal contact areas after experimental intubation. This infiltration was maximum at 24-48 hours and was significantly reduced at 96 hours.[14]

History and Physical

Laryngeal edema is the commonest laryngeal injury detected after intubation, and the majority of endoscopically visualized laryngeal edema after extubation remains asymptomatic or mildly symptomatic. Most patients with post-intubation laryngeal edema complain of mild symptoms like throat ache, difficulty to speak, or swallow. However, severe laryngeal edema makes a common etiology of post-extubation stridor in almost two-thirds of the cases, and nearly half of the patients with stridor get reintubated. Associated vocal cord edema may cause significant restriction of vocal cord movements, which will aggravate the stridor.

Though most cases of post-intubation laryngeal edema develop within 24 hours of tube placement and manifest immediately in the ensuing minutes to hours following extubation, a report of intractable post-extubation laryngeal edema presenting as late as 14 hours to 48 hours after post-surgery extubation is also observed in the literature.

Usually, the laryngeal inflammation and swelling usually resolve in one or two days after extubation. If symptoms persist, the possibility of other injuries sustained during endotracheal tube placement such as hematoma, laceration, avulsion, or due prolonged intubation such as ulceration, granulomas, adhesions, and paralysis needs to be looked into. Many of the reintubations in planned extubations occurs within the first 24 hours after the extubation, and rarely reach up to 72 hours.

Evaluation

The vast majority of post-intubation laryngeal edema (which has been ascertained after extubation) is asymptomatic or mildly symptomatic, as shown in the fibreoptic endoscopic evaluation of 136 patients within six hours after extubation [6]. However, post-intubation laryngeal edema with significantly decreased vocal cord mobility constituted almost two-thirds of patients presenting with post-extubation stridor, and nearly half of them got reintubated in the coming hours. As a consequence, several tests have been proposed for the evaluation of airway patency before extubation. These methods include the cuff leak test (CLT), ultrasonography, and video laryngoscopy.

Cuff Leak Test

The CLT is an important non-invasive test evaluation to assess the risk for laryngeal edema and/or post-extubation stridor in intubated patients. Laryngeal edema is the major contributor for post-extubation stridor, as it also causes diminished mobility of oedematous vocal cords. In particular, the CLT ascertains the space available between the larynx and endotracheal tube, and a reduced or absent cuff leak could be due to a variety of causes, including post-intubation laryngeal injuries which involve laryngeal edema (the most common etiology), copious secretions and laryngotracheal narrowing. On the other hand, the cuff leak could be increased by associated or isolated conditions like tracheomalacia.[15]

The CLT could be assessed qualitatively as well as quantitatively. A qualitative assessment is done by deflating the cuff and auscultating the tracheal area for any audible leak. The quantitative test is done by putting the patient in volume control mode and calculating the difference between the inspiratory tidal volume and the average value of lowest three expiratory tidal volumes obtained over a period of 6 breaths (cuff leak volume). A leak or 110 cc or more than 24% of the delivered tidal volume shows a positive leak test, which is considered normal. A negative leak test

predicts risk for post-extubation failure with varying sensitivity and specificity. Hence a negative CLT test is a poor predictor of post-extubation stridor with sensitivity varying between 15 to 85 % and a specificity of 70 to 99 %. Although the positive predictive values differed between various studies depending on the cuff leak cut off values, the negative predictability was more than 90% in most studies. This variation could be explained by the fact that the cuff leak volume is determined not only by the expiratory leak via peri tubal space but also by the inspiratory leak volume, which depends on the inspiratory flow and the compliance of the lung.

A pooled analysis of nine studies published in 2009 estimated the sensitivity and specificity of cuff leak test as 56% and 92%, respectively.[16] Again, a meta-analysis of 14 observational studies showed that performing a cuff leak test reduced the occurrence of post-extubation stridor (4 versus 7%), decreased the rate of reintubation (2.4 versus 4.2%), though the delay in extubation of the patients was increased by 9%.[17] Thus, the 2017 American Thoracic Society (ATS) and American College of Chest Physicians (ACCP) guidelines on liberation from mechanical ventilation in critically ill adults recommend a cuff leak test to be performed on all high-risk patients for post-extubation laryngeal edema and/or stridor which involves patients who have had traumatic intubation, mechanically ventilated for more than six days, a large endotracheal tube, female sex and the ones reintubated after unplanned extubation. Prophylactic methylprednisolone is advised in such patients with negative leak tests, as described above.[17]

Laryngeal Ultrasonography

It measures the Air Column Width (ACW), which is the width of the acoustic shadow at the level cords before and after cuff deflation in the intubated patients. This approach has been shown to predict the risk of post-extubation stridor. The ACWD is the difference in the air column measurement in the intubated and deflated state. Laryngeal ultrasonography is a simple, rapid, and non-invasive evaluation which could be done at the bedside. Ding et al., in a study on 51 patients (out of whom four developed post-extubation stridor) has shown significantly low ACW (4.5mm versus 6.4 mm; p= 0.01) and ACWD (0.35 mm versus 1.5 mm ;p<0.01) in those who developed post-extubation stridor.[18]

Another study by Sutheresan et al. also showed a similar trend with decreased ACW and ACWD in patients who presented with post-extubation laryngeal edema. Again, an investigation on 72 patients found that the laryngeal ACW ratio (ACW before extubation/ACW after intubation) was less than 0.8 in all the five patients who developed post-extubation stridor.[19] However, Mikaeili et al. could not find any significant difference in ACW or ACWD in patients presented with or without post-extubation stridor.[10] In view of the small sample sizes with an extremely small number of patients presenting with post-extubation stridor in the above studies, a robust conclusion about the utility of laryngeal ultrasound is difficult at this stage till larger controlled clinical studies throw more light in this aspect.

Video Laryngoscopy

Video laryngoscopy or fiber optic endoscopy evaluation is conceptually promising in that they would be able to visualize the peri laryngeal structures and abnormalities. Unlike CLT, video laryngoscopy or fibreoptic evaluation can identify and differentiate between the structural versus functional laryngeal abnormalities (e.g., laryngeal edema versus laryngospasm) guiding appropriate management. However, the utility of the above modalities in the prediction of post-extubation laryngeal edema or stridor is yet to be evaluated.

Treatment / Management

Steroids have been studied in the prophylactic administration to prevent post-extubation stridor by various randomized trials and reviews, with most of them suggesting positive results.[4][20][7][21][22] Various evidence-based investigations also supported the clinical benefit of prophylactic steroids in the prevention of post-extubation stridor in high-risk patients.[23][24][25][26][17] It should be noted that the studies which enrolled unselected patients with single-dose steroids did not show any benefit. Thus many experts use methylprednisolone in the dose of 20 mg intravenous (IV) 4 hourly over 12 hours prior to extubation or a single dose methylprednisolone 40 mg IV at least 4 hours before extubation based on the methodologies of the positive randomized trials.

Evidence for clinical benefits of adrenaline nebulization in the management of post-extubation stridor or laryngeal edema lacks in adults though adrenaline nebulization is used in pediatric populations to relieve upper airway edema in acute severe croup.[27][28] MacDonnell et al. in 1995 published a paper where four adult patients with varying aetiologies for upper airway were treated successfully with l-adrenaline nebulizations in the dose of 1 mg in 5 ml of 0.9 % sodium chloride over 10 minutes duration.[29]

The combined use of intravenous steroids and adrenaline nebulization did not mitigate the progression of airway obstruction secondary to laryngeal edema in a randomized study in neonates and children.[30] A recent study compared IV dexamethasone versus budesonide nebulization and suggested the possibility of budesonide nebulization as a potential substitution for IV dexamethasone in enhancing the cuff leak volume in intubated patients.[31]

Heliox (helium and oxygen mixture) has been shown to decrease the post-extubation stridor scores in pediatric trauma patients.[32] Heliox reduces the airways resistance with decreased work of breathing without changing the clinical outcome. Thus, it may only provide more time before a more definite intervention at airway obstruction is executed. The clinical efficacy of Heliox in adults is yet to be proven.

For practical purposes, all patients at high risk for post-extubation laryngeal edema and/or stridor (as mentioned above in the 2017 ATS/ACCP guidelines) should undergo a bedside CLT, and all negative CLT patients (i.e., the negative value from normal cut off with reduced or absent leak) should receive IV methylprednisolone 40 mg at least 4 hours before extubation. The extubation in such cases needs to be monitored closely. An airway exchanger could be kept up to an hour or so in the larynx (after extubation), anticipating the need to thread the endotracheal tube in case of any unexpected difficulty in reintubation. If the patient continues to be asymptomatic after an hour, the active monitoring could be reduced, and routine plans are carried out appropriately.

Symptomatic patients not getting better with anti-edema measures (IV steroids/adrenaline nebulizations) are monitored up to an hour before reintubation. If the patient is significantly symptomatic in the post-extubation period but slowly getting better clinically within this one hour period, IV steroids and adrenaline nebulization are continued for 24 to 48 hours. Reintubated patients are also continued on IV steroids and adrenaline nebulizations for 24 to 48 hours before reassessing for extubation versus tracheostomy. Head end elevation has been ensured to reduce the venous congestion, which is expected to mitigate the edema.

A multicentre randomized study on the trial of non-invasive ventilation (NIV) in 221 patients with post-extubation respiratory failure observed increased mortality in patients who were assigned to the NIV group likely due to delay in reintubation. The authors concluded that NIV neither reduces reintubation rate or mortality in post-extubation respiratory failure.[33] Based on the above findings(though flaws are noted in the above study), the European Respiratory Society (ERS)/ATS guidelines recommend against using NIV in post-extubation respiratory failure.[34]

Differential Diagnosis

The common differential diagnoses include:

- Post-extubation stridor, laryngospasm
- Angioedema, anaphylaxis
- Foreign body in the airway
- Post-surgical hematomas causing airway compression
- Vocal cord palsies following neck surgeries
- Sleep apnoeas

Post extubation stridor is symptomatic (varying severity) form of laryngotracheal narrowing in an extubated patient. It

is clinically diagnosed by the unique inspiratory crowing sound. About two-thirds of post-extubation stridor is caused by severe post-intubation laryngeal edema, and nearly half of the patients with post-extubation laryngeal edema will get reintubated. Laryngospasm is the exaggerated glottic closure reflex due to the stimulation of the superior laryngeal nerve. It could happen abruptly in patients who are extubated after anesthesia. This occurs in patients who are not adequately conscious of countering the laryngeal reflexes in response to vocal cord irritating events like endotracheal tube removal, pooling of secretions, blood, or foreign body in the airway. Treatment is the removal of inciting cause, positive pressure bag, and masking with a simultaneous jaw thrust maneuver. Larson's maneuver is the application of pressure with fingertips at the laryngospasm notch, which could rapidly reverse the condition.

Angiooedema could occur due to various drugs, including opioids, calcium-channel blockers, fibrinolytic agents, and NSAIDs. It usually responds to steroids and antihistaminics. Anaphylaxis is treated with adrenaline injection. Severe bronchospasm and pulmonary edema can sometimes mimic laryngeal edema. Post-extubation stridor could rarely cause negative pressure pulmonary edema.

Pertinent Studies and Ongoing Trials

CLT and Airway Obstruction in Mechanically Ventilated ICU patients (COMIC) is a pilot randomized controlled trial planning to enroll 100 patients which started recruiting patients from June 2018 to primary endpoints being feasibility outcomes and clinical outcomes. The clinical outcomes include post-extubation stridor, reintubation, mechanical ventilation duration, length of stay in ICU, and ICU and in-hospital mortality. Larger RCTs are being planned after the result of this trial.[35] The result is not published to date.

Prognosis

The vast majority of post-intubation laryngeal edema is asymptomatic and does not need any intervention. The degree of severity of edema and the occurrence of associated injuries varies from patient to patient. The overall incidence of post-extubation laryngeal edema is noted to be between 5 to 54 % in various studies, and only about 10.5 % of the patients with post-extubation laryngeal edema will need reintubation. It should be noted that every reintubation event is known to increase the risk of morbidity as well as mortality. Laryngeal edema with resultant restricted vocal cords mobility was the cause for post-extubation stridor in almost two-third of the cases in a study on 136 patients.[6] The risk for post-extubation stridor is usually assessed with a bedside by performing a CLT in high-risk patients, including the ones who underwent traumatic intubation, who are intubated for more than six days, those having a large endotracheal tube, female gender and who were reintubated after unplanned extubation.[17]

If CLT is negative (less than 110 cc), then the extubation is postponed, and the patient is reassessed for extubation after about 4 hours of administration of a single dose of 40 mg IV methylprednisolone. IV steroids have been known to prevent post-extubation stridor in patients who fail the CLT in the select group of high-risk patients.

If the patient still develops post-extubation stridor, measures including IV steroids, nebulized steroids, and adrenaline nebulization are started, and the patient is reintubated if he/she is not improving after close monitoring up to an hour. If reintubated, IV steroids and nebulizations are continued for 24 to 48 hours, and extubation is considered after a repeat CLT. If no improvement is seen in cuff leak volume after 24 to 48 hours, tracheostomy may have to be considered, especially in view of failed previous extubation. The patient will also require a fibreoptic endoscopic evaluation to assess the larynx and tracheal areas in the meantime.

Complications

The majority of post-intubation laryngeal edema is asymptomatic or minimally symptomatic after extubation & does not require any intervention. Post-extubation stridor represents the symptomatic form of severe laryngeal edema in nearly two-thirds of such patients. Almost 50% of patients with post-extubation stridor will require reintubation. it should also be noted that different patients will have varying laryngeal injuries of different grades in isolation or in combination which determines the clinical manifestation.

About 10.5% of the patients with laryngeal edema will progress to worsening respiratory failure and need reintubation despite all medical measures. Again, another small percentage will need tracheostomy after a reassessment with medical management after 24 to 48 hours. Since reintubation events are known to increase the risk of morbidity and mortality significantly, prompt recognition of high-risk patients for post-intubation stridor/laryngeal edema by performing a bedside CLT in the high-risk patients will help to identify and chalk out a preventive measure by administering IV steroids.

Consultations

Most of the post-surgery extubations happen on the table and if stridor occurs, it needs to be differentiated from laryngospasm, angioedema, and rare possibility of anaphylaxis. Certain surgical interventions and surgery positions also increase the risk of post-intubation laryngeal edema, which might contribute significantly to the development of post-extubation stridor.

After aggressive anti-edema measures, as mentioned above, the patient may have to be reintubated after closely observing up to an hour. The same scenario could happen in ICU where the high-risk patients undergo a bedside CLT and receive prophylactic steroids in case of a negative leak test. The risk of post-extubation stridor is high especially if the patient has been intubated for more than 36 hours, with other risk factors being intubated state for more than 6 days, female gender, large for size endotracheal tubes, traumatic intubation, lack of muscle relaxant used during intubation and reintubation after unplanned extubation.

A fibreoptic evaluation may be required to assess the degree of laryngeal and tracheal abnormalities in patients who do not improve the cuff leak volume after treatment for 24 to 48 hours with steroids. The endoscopic evaluation would provide a direct view of laryngotracheal areas. Elective tracheostomy may have to be considered if the fibreoptic evaluation reveals grossly and persistently oedematous laryngeal area or shows irreversible or slowly reversible injuries significantly obstructing the upper airway.

Deterrence and Patient Education

Any electively extubated patient may need reintubation either due to ventilation failure or extubation failure in up to 10% cases. Conservative medical measures and close monitoring are initially tried in symptomatic patients with extubation failure, before proceeding to reintubation mostly happening within the first 24 hours of extubation. The patient and relatives should be made aware of this fact in all planned extubations. In patients admitted to the ICUs, the risk of post-extubation laryngeal edema prompting reintubation in about 10% of the cases needs to be explained to family in advance. The patient and family also need to be aware that tracheostomy would be indicated in a small number of cases who fail to improve after 24-48 hours of anti-laryngeal edema measures after a reintubation event.

Pearls and Other Issues

Laryngeal injuries are common after endotracheal intubation, and post-intubation laryngeal edema is the major contributor of laryngeal injury and post-extubation stridor. About half of the patients who present with post-extubation stridor will require reintubation. Since every reintubation event is a risk factor for increased morbidity and mortality, it is of utmost importance to identify the at-risk patients for post-intubation laryngeal edema and/or stridor by performing a bedside CLT in all the high-risk patients as described above and initiate prompt prophylactic IV methylprednisolone administration in case of a negative CLT.

Enhancing Healthcare Team Outcomes

Post-intubation laryngeal edema is often asymptomatic or minimally symptomatic but could sometimes present with post-extubation stridor (constituting nearly two-thirds of the stridor cases) in which scenario, the patients might end up on reintubation in about half the cases. A well trained and organized anesthesia or ICU team, including medical and nursing staff, as well as respiratory therapists, could safely handle a difficult to intubate and/or ventilate situations

incase it arises during the course of events. Difficult fibreoptic intubations may need expert support, or otorhinolaryngology consult for emergency open tracheostomy. Close coordination and interaction between various health professional teams to ensure the best outcome in a given patient.

Review Questions

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