International Journal Dental and Medical Sciences Research
Volume 7, Issue 1, Jan - Feb 2025 pp 320-324 www.ijdmsrjournal.com ISSN: 2582-6018

Complete Airway Obstruction Immediately After Extubation

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Date of Submission: 15-02-2025 Date of Acceptance: 25-02-2025

ABSTRACT: We report a case of upper airway obstruction occurring immediately after extubation after palatine tonsil surgery. The obstruction was caused by severe, progressive supraglotticoedema, which totally obscured the laryngeal inlet. No swelling had been observed during initial laryngoscopy and intubation. Immediate re-intubation of the patient's trachea was difficult but necessary to secure the airway. Further evaluation revealed extensive soft tissue swelling, maximum at the level of the hyoid and extending downwards into the trachea. The cause of such severe oedema in this case is not certain, but may be due to instrumentation carried out at the end of operation. We review the appropriate literature, describe the postoperative management and suggest precautions in similar patients.

Keywords : Intubation Tracheal; Complications, Laryngeal Oedema; Airway, Obstruction

I. INTRODUCTION:

Post extubation airway obstruction, though relatively uncommon after prolonged tracheal intubation, [for example in intensive care units,] is seldom of sufficient severity to require immediate re-intubation.[1,2,3] In the immediate postoperative period, airway obstruction necessitating reintubation is similarly rare and was found to have an incidence of 0.17% and 0.19% respectively in two large series which examined 10 060 and 13 593 postanaesthesia care unit admissions.[4,5]

Laryngeal edema is a common cause of airway obstruction after extubation in intensive care patients and is thought to arise from direct mechanical trauma to the larynx by the endotracheal tube.[6,7] The severity of airway obstruction due to laryngeal edema varies. In more severe cases, the edema can lead to acute respiratory compromise necessitating emergency reintubation.[8–13]

Airway obstruction after surgery has also been reported secondary to several physical factors, such as compressive effects of haematomata,[14] bilateral recurrent laryngeal nerve palsies,[15] massive tongue swelling,[16] paradoxical vocal cord motion[17] and obstruction by a mucous cast.[18] Hereditary angioedema[19] and anaphylaxis[20] may also cause acute postoperative upper airway obstruction.

We report a case of airway obstruction, occurring immediately after extubation. While the aetiology is uncertain, direct trauma to the larynx during removal of palatine tonsils could not be ruled out. We discuss the management of the acute event and strategies to avoid similar event.

II. CASE REPORT:

A 22-yr-old, 95-kg man presented for elective surgery for removal of palatine tonsil. He had no previous medical history of diabetes, hypertension but he was a smoker consuming 10 cigarette per day. Physical examination was normal and visual assessment of his airway did not suggest that he would pose any difficulties on intubation.

Premedication consisted of glycopyrolate 0.02mg and diazepam 10mg orally, 2 h before operation. Anaesthesia was induced with propofoland atracurium. Laryngoscopy revealed a grade 1 view (Cormack and Lehane) and the patient's trachea was intubated using a 8.5 mm internal diameter cuffed nasal tracheal tube. A saline-soaked throat pack was inserted and anaesthesia was maintained with isoflurane and nitrous oxide in oxygen, with subsequent doses of atracurium 10 mg as required to facilitate mechanical ventilation. Ceftriaxone 1.0 g and dexamethasone 8 mg were also administered during surgery.

With the patient in a supine head-up position, he underwent uneventful palatine tonsil removal. Duration of surgery was 1.5 h and the patient remained hemodynamically stable throughout.

On completion of surgery, after antagonism of neuromuscular block, the patient was breathing spontaneously generating good tidal volume. The throat pack was removed and the oropharynx was suctioned under direct vision (with gentle laryngoscopy) and observed to be dry, with no evidence of oedema. Extubation was performed

under deeper plane of anaesthesia to avoid coughing or straining on the tracheal tube. Immediately after extubation, the patient was noted to be making good respiratory effort but appeared to have an obstructed airway. Worryingly there was no audible stridor, which suggested total or near-total obstruction. Attempts to relieve the obstruction by airway manoeuvres and insertion of a Guedel airway were unsuccessful. Oxygen saturation was maintained initially as he had been breathing 100% oxygen before extubation. But when it started to decrease, in the face of persistent total airway obstruction, the decision to re-intubate the patient's trachea was taken.

Succinylcholine 100 mg was given and laryngoscopy revealed the laryngeal opening to be completely obscured by supraglottic swelling and tissue oedema. In view of the urgency of the situation, an oral tracheal tube of size 6.5mm internal diameter was advanced into the centre of the swelling where the laryngeal opening was assumed to be situated. Fortunately, the tube passed through the oedematous tissue to enter the trachea, and the patient's lungs proved easy to ventilate. Further sedation was given and he was admitted to the intensive care unit for continuing sedation and mechanical ventilation, pending resolution of the swelling. Dexamethasone 6 mg i.v. every 6 h was prescribed.

As expected, considerable swelling developed at the sites of operation over the course of the following 48hr. Fibreoptic laryngoscopy was performed on the second postoperative day to assess the degree of supraglottic swelling. At this time, there was no leak of air when the cuff of the tracheal tube was deflated during ventilation. Gross swelling/oedema of the laryngeal inlet was still present, particularly around the false cords. However, when the fibreoptic laryngoscope was advanced gently through this area, an excellent view of the true cords was obtained. They were also noted to be mildly oedematous. Sedation and ventilation were continued for another 24 h, at which time fibreoptic laryngoscopy was repeated. On this occasion, the oedema was found to have increased, with the true cords appearing much more swollen.

On day 4 after operation, there was still no audible leak around the tracheal tube and biopsy of the swelling was considered. By the following day, a considerable leak had developed around the tube when the cuff was deflated, and a decision to extubate under deep inhalation anaesthesia was taken. After gaseous induction of anaesthesia with sevoflurane in 100% oxygen, an airway exchange catheter, connected to an oxygen source, was introduced into the patient's trachea via the tracheal tube. As there was no evidence of respiratory obstruction, the tracheal tube was removed leaving the oxygen catheter in situ. Anaesthesia was discontinued and the oxygen catheter was removed with the patient awake. His oxygen saturation remained 99% on air and he was discharged from the intensive therapy unit later that day. No further airway complications occurred and he made an otherwise uneventful recovery.

A range of laboratory tests to identify an allergic reaction (IgE and DLC) were negative. Skin testing to PVC, povidine iodine and latex was also negative.

III. DISCUSSION:

Early recognition of laryngeal edema is essential since these patients have the highest risk of evolving to respiratory distress and extubation failure. Even before extubation, signs indicative of laryngeal edema may be present.

Airway oedema is to be expected in surgical procedures similar to that described in this case, and postoperative airway compromise is not uncommon. Obstruction of the airway is seldom complete, however, and generally takes some hours to develop, allowing time for intervention before life-threatening obstruction supervenes. The search for a test that adequately identifies patients at risk for extubation failure is ongoing. Recently, the cuff leak test has gained interest. The test is non-invasive, relatively easy to perform and is thought to give an indication of the patency of the upper airway. When the ventilated patient is allowed to exhale with a deflated cuff, expired air normally escapes from the otherwise closed circuit. The volume of leaked air can be measured by spirometry functions of the ventilator.

Miller and Cole made the first attempts to make the cuff leak test quantitative, by measuring the amount of air leak and correlating the cuff leak volume to the likelihood of developing laryngeal edema and PES. They calculated the cut-off value with the highest sensitivity and specificity.[21] Almost none of the patients with cuff leak volume >110 ml developed PES: the specificity of this cut-off value was 99% and the negative predictive value for absence of PES was 98%. If a leak <110 ml was present, only two-thirds of patients developed PES - making the sensitivity 67%.[21]

Although the exact cause of the oedema in this patient remains unclear, many factors have been identified as potential causes of swelling in maxillofacial surgery. Patient positioning, fixation of tracheal tubes, gauze throats packs and direct surgical trauma may contribute to airway oedema.

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Intraoperative use of steroids cannot be relied upon to prevent the occurrence of oedema.[1,22]

Zulian and colleagues[23] produced a checklist for extubation of oral and maxillofacial surgical patients, and recommended that the extent of surgically induced oedema be evaluated before extubation of the trachea. However, they do not suggest how this should be achieved. In our case mouth opening may be limited due to removal of palatine tonsil and trauma to palatine tonsil lead to the limited attempt of direct laryngoscopy.

Oedema at the level of the glottis may thus not be revealed, even if laryngoscopy is attempted. The flexible fibreoptic laryngoscope could be used in such circumstances to see the airway down to the level of the glottis, introduced either nasally or orally, without undue stress on the surgical correction. We recommend that in cases where occult oedema may be present, particularly if no air leak is heard after deflation of the tracheal tube cuff, the fibreoptic laryngoscope should be used before extubation.

Medical therapeutic strategies include systemic administration of steroids and nebulization of epinephrine. Corticosteroids downregulate the inflammatory response by inhibiting the recruitment and action of inflammatory cells.[24] Together with a decrease in capillary vessel dilatation and permeability, this inhibition reduces edema.[25] The most effective dose has not yet been determined. We suggest a dose of 0.5 mg/kg prednisolone intravenously per day. The effectiveness of glucocorticoids in post-extubation laryngeal edema has not been confirmed in randomized controlled trials. In our experience, however, the potential benefit outweighs the risk of adverse events. Moreover, most adverse events are pharmacological effects of corticosteroids that are likely to disappear after the treatment period.[26]

Furthermore, epinephrine nebulization is another potentially effective therapy. Epinephrine acts through local stimulation of α-adrenergic receptors on vascular smooth muscle cells, thereby causing vasoconstriction and decreased blood flow, which diminishes edema formation. Randomized controlled trials that prove efficacy of epinephrine in post-extubation laryngeal edema in adults are again lacking. Likewise, there is no consensus about the potentially effective dosage of epinephrine nebulization. A dose of 1 mg epinephrine in 5 ml normal saline has proved successful in some cases of upper airway obstruction in adults.[27] Rebound edema is known to occur and close observation is essential.[28] Side effects can occur, especially in patients with coronary artery disease.[28]

Maintaining the airway, adequate oxygenation and relieving distress associated with obstruction are primary treatment goals. Several treatment modalities, including reintubation, are available and will be discussed below will be discussed(Figure 2).

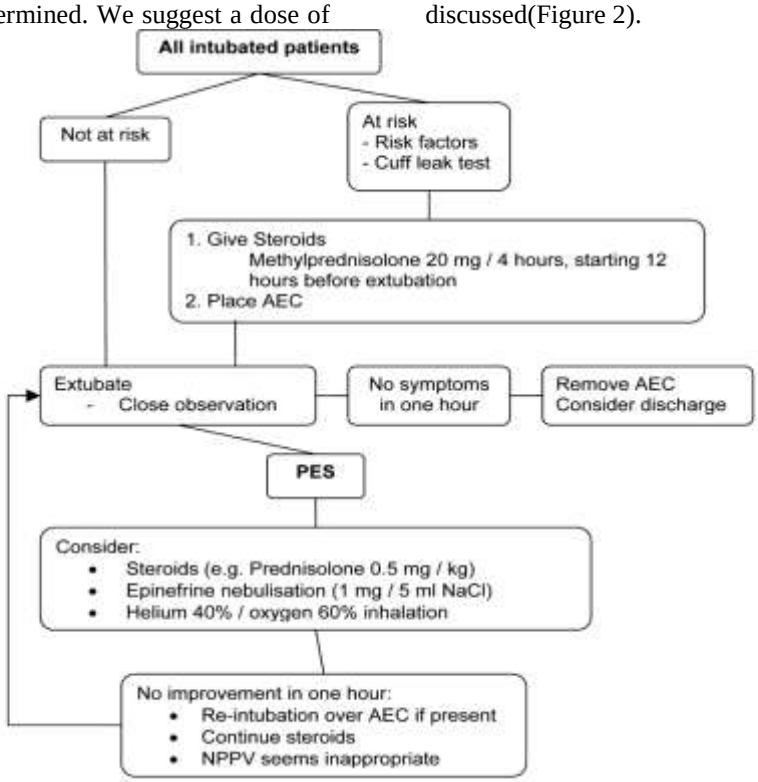

FIGURE 2: POST-EXTUBATION LARYNGEAL EDEMA THERAPY FLOW CHART

IV. CONCLUSION :

Laryngeal edema most often presents as inspiratory stridor and may be associated with respiratory failure due to airway obstruction. Subsequent reintubation leads to increased costs, morbidity and mortality. Female gender, a relatively small tracheal diameter or a large tube size and a long duration of intubation have been identified as risk factors in different studies. A positive cuff leak test with leak volume <110 ml increases the risk for development of PES and subsequent reintubation significantly. Multiple-dose regimens of corticosteroids starting at least 12 hours before extubation can prevent the development of laryngeal edema in patients with these risk factors. In our case highlights a high index of suspicion and the use of a fibreoptic laryngoscope before extubation in such cases may prove efficacy.

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