

CASE REPORT

Postoperative supraglottic edema following palatoplasty

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ABSTRACT

Insidious and progressive airway obstruction may occur following palatoplasty and establishment of a patent airway or reintubation may be extremely difficult. We present a case of unanticipated, severe postoperative supraglottic edema after repair of cleft palate in a 17-month-old child.

Key words: Anesthesia; Postoperative Complications; Airway Obstruction; Edema; Laryngeal Edema

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INTRODUCTION

Significant airway problems are frequently encountered after repair of a cleft palate in patients with craniofacial abnormalities.¹ The anesthetic management of cleft palate repair with an otherwise normal pediatric airway is rarely complicated. We report a child who underwent cleft palate repair and developed supraglottic edema with life-threatening upper airway obstruction shortly after extubation.

CASE REPORT

A 17-month-old, 12 kg boy was scheduled for repair of the cleft palate. History was unremarkable except for recurrent upper respiratory tract infection (URTI). Last URTI was four weeks back. Physical examination revealed nasal crusting, cleft of the palate and an otherwise normal airway. The child received oral triclofos 500 mg, 2h before surgery.

In the operating room, standard monitoring was established. Anesthesia was induced with halothane 0.6%-2% in nitrous oxide 50%, and oxygen via face mask. An intravenous access was secured. Patient received glycopyrrolate 0.04 mg and fentanyl 20 µg. Tracheal intubation with oral uncuffed RAE tracheal tube (size 4.5) was facilitated by vecuronium 1.2 mg. There was an acceptable air leak at 20 cmH₂O airway pressure. Anesthesia was maintained with isoflurane 0.6% in nitrous oxide 66% and oxygen. A roll was placed under the shoulders to extend the neck

and tip the head down. A Dingman mouth gag was used and an oropharyngeal pack was inserted by the surgeon. A Veau Wardill-Kilner palatoplasty was performed. The intraoperative course was uneventful. Total blood loss was estimated at 70 ml, and 570 ml of Ringer's lactate solution was infused during the 2h operation. Paracetamol suppository 250 mg and additional fentanyl 10 µg provided analgesia. At the end of surgery, the throat pack was removed. A tongue stitch was placed. Laryngoscopy was performed for oropharyngeal suction and to check for surgical bleeding. Residual neuromuscular block was antagonized with glycopyrrolate 0.12 mg and neostigmine 0.6 mg. When the child was fully awake, spontaneous ventilation was adequate, and protective airway reflexes had returned, the trachea was extubated. Following extubation, the child was agitated and restless with nasal flaring and suprasternal and intercostal retractions. 100% oxygen was administered via face mask. Saturation decreased to 80%. He was placed in the lateral position. The tongue was pulled forward by the tongue suture. Hydrocortisone 50 mg and dexamethasone 1 mg was administered. Adrenaline (1ml of 1: 10 000 diluted in 4ml saline) was nebulized. SpO₂ increased to 90%. Labored breathing persisted with rib retractions suggestive of upper airway obstruction. At about 15 min post-extubation a gentle laryngoscopy was performed again to rule out any blood clot, secretions or gauze piece obstructing the airway. The epiglottis was found to be bulbous and edematous.

Approximately 30 min post-extubation, the child developed

supraglottic edema following palatoplasty

tachypnea, with conducted sounds on auscultation, and a decrease in SpO₂. Immediate tracheal intubation became imperative. Inj. propofol 10 mg was administered. After confirming ability to ventilate the lungs with 100% oxygen by facemask, suxamethonium 6 mg was administered. Laryngoscopy with external laryngeal manipulation revealed a Cormack grade 4 view. The epiglottis could not be identified and the area of false vocal cords was grossly edematous. The tracheal tube was placed blindly on a depression in the edematous soft tissue. After three unsuccessful attempts a 4.5 size uncuffed tracheal tube was placed in the trachea. Lung fields were clear following tracheal suctioning and SpO₂ increased to 100% with 100% oxygen.

The child was transferred to the Intensive Care Unit. The lungs were mechanically ventilated overnight. Neuromuscular blocking agents were discontinued. Sedation was provided with i.v. midazolam and morphine. The child received ceftriaxone and hydrocortisone along with other supportive treatment. The following day (day 2), the tracheal tube was dislodged when the patient moved vigorously. The child was reintubated with size 4.5 non-cuffed portex tracheal tube as he was unable to maintain an adequate airway. At laryngoscopy the supraglottic structures could be identified but were still edematous. The child maintained normal vital parameters with spontaneous respiration on continuous positive airway pressure (CPAP) of 5 mmHg. On day 3, he developed fever that responded to cefoperazone and sulbactam. On day 6, the child was afebrile, conscious, sedated, hemodynamically stable and there was no supraglottic edema. The trachea was successfully extubated and he was transferred to the ward on day 7 and was subsequently discharged home on day 10.

DISCUSSION

The overall risk of airway obstruction after a cleft palate repair is 5.7% and often occurs within the first 2h postoperatively.² It is most likely to occur in children with associated cranio-facial abnormalities. A history of snoring, apnea during feeds and a protracted feeding time is suggestive of chronic airway obstruction, none of which were present in the present child.

Significant airway compromise following palatoplasty may be due to tongue swelling, laryngeal edema, pharyngeal

secretions, blood clot, retained throat pack, prolonged action of anesthetic agents, or a combination of factors. Several case reports describe the insidious onset of postoperative macroglossia minutes or hours after extubation.³⁻⁶ Pressure associated with an excessively opened Dingman gag for a prolonged period can induce ischaemia, resulting in necrosis of the tongue muscle, venous stasis and lymphedema, leading to tongue swelling.^{7,8} This rapid onset suggests ischemia of the tongue with reperfusion hyperemia and capillary leakage as important mechanisms. The use of mouth packs and oral airways were thought to be contributory.^{3,6} Palatoplasty by itself reduces the air passage.⁹ Hyperextension of the head or Trendelenberg positioning may also lead to impaired arterial inflow and decreased venous drainage of the tongue.^{1,5,7} Prolonged duration of surgery correlates with increased incidence of airway obstruction.²

In the case described, the exact etiology of supraglottic edema is unclear. A possible explanation is that inadvertent compression of the epiglottis occurred from the tongue blade of the Dingman retractor or from improper or overzealous pharyngeal packing, which likely resulted in arterial and venous compromise. The supraglottic injury was further exacerbated by an extreme head on neck extension which compounds venous stasis and lymphedema by gravitational fluid shifts.

Development of postoperative supraglottic edema following palatoplasty may lead to complete airway obstruction, necessitating immediate re-intubation which may be extremely difficult, as seen in the present case. It is suggested that the entire oropharyngeal cavity including the tongue and surrounding soft tissue be examined thoroughly at the completion of cleft palate repair before the tracheal tube is removed. If any signs of lingual or supraglottic edema exist, it may be prudent to retain the tracheal tube and observe the patient for progression of edema. Prolonged intubation may be required in such cases to ensure a patent airway.

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