**Hypermobile laryngeal granulomas: A potential cause of false negative cuff leak test**

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Sir,

Airway obstruction and post extubation stridor (PES), particularly when unanticipated, can bring forth a life-threatening situation. Intubation granulomas, even though benign and the majority being managed conservatively, can be a cause of airway obstruction and fatal respiratory distress following extubation. Although the cuff leak test (CLT) has been mentioned in the literature as a reliable predictor of PES, its clinical utility in recent years is now being questioned due to its low sensitivity and low positive predictive values. We report a case of an 8-year-old male child who developed PES in spite of more than recommended cuffed leaked volume. Thus, we believe that a single CLT should not be presumed to be a sole predictor of PES.

An 8-year-old male patient (20 kg of body mass, 130 cm in height) admitted in neurointensive care unit (NICU) with tracheal intubation (atraumatic) was performed with a low pressure, high volume cuff leak test (5.5 mm internal diameter) and subsequently mechanically ventilated. The patient was managed conservatively. On the ventilator, the cuff pressure was maintained between 20–25 cm H₂O. On the 5th day of NICU admission, we planned a tracheal extubation after successful weaning over a period of 48 hours. As a routine protocol prior to extubation, we performed both a quantitative and qualitative CLT. An audible leak was clearly noticed with the stethoscope while the cuff leaked volume was 56 ml when the inspired tidal volume was 200 mL on assist control mode (the quantitative CLT was done based on the method proposed by Miller and Cole) [1]. An arterial blood gas (ABG) analysis was also carried out and was found to be within normal limits. However, immediately following extubation, a loud and clear stridor was audible and was associated with suprasternal retraction and paradoxical respiration. Chest auscultation showed diminished air entry bilaterally. A thorough oropharyngeal suctioning was performed again followed by nebulisation with racemic epinephrine and budesonide. However, the respiratory distress continued and gradually the oxygen saturation started declining (SpO₂ 90–91%). As such, a trial of positive pressure ventilation was given after sedating the child with propofol. On ventilating, mild resistance was noticed. We then performed video laryngoscopy [Image 1] and visualised bilateral hypermobile vocal process granulomas (VPG) intermittently obstructing the glottis on inspiration. Immediately, we intubated the trachea with a smaller size endotracheal tube (4.5 mm ID). The child was subsequently tracheotomised and managed conservatively on steroid treatment and antibiotics. The later course of the NICU stay was uneventful and the child was shifted to ward on the 14th day.

Post-intubation laryngeal granulomas (vocal process granulomas) which are typically associated with traumatic intubation, prolonged intubation, the use of oversized ETT, excessive cuff pressure, a hyperextended or hyperflexed neck are further compounded by movement of the patient, coughing, swallowing, infection and laryngopharyngeal reflux [2]. Predisposing factors include adult females, obesity with short neck, pre-existing airway abnormalities and fragility of the laryngotracheal mucosa [3]. Presumably, in our case, the main causes were the duration of intubation (5 days) along with excessive patient movement, coughing and bucking on the ETT whenever we intended to withdraw sedation in order to evaluate the GCS. This incited laryngeal inflammation which then ulcerated and transformed into a sessile and later on bilateral pedunculated granulomas.

In the above-described case, the pedunculated granulomas were hypermobile (visualised on a video laryngoscopy) which intermittently obstructed glottis transversely during inspiration, resulting in airflow obstruction. Since the dynamic obstruction was intermittent in nature, it resulted in falsely negative CLT. As such, the unanticipated post-extubation stridor required reintubation after failed initial trials of nebulisation and positive pressure ventilation.

Cuff leak tests were introduced in an attempt to predict PES and reduce the incidence of extubation failure. Although a substantial amount of the available literature confirms its usefulness [1, 4–6], we believe that this test should not be completely presumed to be conclusive. In our case, the cuff leaked volume was 28% (56 mL) of the inspired tidal volume (200 mL), a percentage much higher than recommended for a negative CLT (10% as proposed by Sandhu et al. [7]). Our report is further strengthened by certain studies which have questioned the clinical utility and reliability of CLT. Shin et al. [8] reported that the CLT does not reliably identify those patients who will require reintubation in a trauma population. Similarly, Ochoa et al. [9], in a systematic review and meta-analysis, highlighted that the presence of a detectable leak has a low predictive value and does not exclude PES or the need for re-intubation, even though they also pointed to the concern that the absence of a leak should alert the clinician to a higher risk of PES. Again,
a recent study by Patel et al. [10] demonstrated that the CLT or a combination of the CLT with laryngeal parameters failed to accurately predict PES. Moreover, the probability that the CLT can increase the risk of oropharyngeal and subglottic secretions into the airway during cuff deflation and mechanical ventilation does exist and cannot be denied.

Extubation of intensive care unit (ICU) patients is potentially a high risk procedure. There is no single test that can reliably predict post-extubation stridor. We believe that a combination of tests should be performed (as suggested by Patel et al. [10]) in addition to a fibreoptic laryngoscopy prior to extubation so as to exclude any new onset of anatomical defects similar to our case. This, in turn, can somewhat reduce the incidence of post extubation upper airway obstruction.

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References:

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Silicone stents save lives without surgery in postintubation subglottic stenosis
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To the Editor,

The estimated prevalence of postintubation subglottic tracheal stenosis has been stated as 4.9 cases per million per year [1]. In most cases the ischemic injury occurs at the level of the cuff, in the subglottic region [2]. Life-threatening complications may occur, including stridor, hypercapnia, hypoxia, reintubation and a delay in weaning of intensive care unit patients. Most cases typically present weeks to months after intubation [3].

There are several treatment options for benign tracheal stenosis. Early low-dose systemic corticosteroids have been shown to be effective in postintubation tracheal stenosis management [4]. Tracheoplasty may be performed with tracheal anastomosis. Patients may require a permanent or transient tracheostomy. Surgery usually includes complex tracheal stenosis, subglottic involvement or associated tracheomalacia. Even following laryngotracheal resection, restenosis may occur. Tracheal dilation and stenting is another alternative modality of treatment for patients who are not surgical candidates.

A 56-year-old male patient with a diagnosis of coronary artery disease, developed an acute myocardial infarction. The patient had chronic obstructive pulmonary disease and diabetes mellitus type 2. He was admitted to the intensive care unit. The patient was intubated due to acute hypoxemic respiratory failure. He required prolonged mechanical ventilation during 21 consecutive days. Following the 10th day of his extubation, progressive stridor and dispnea occurred. A fiberoptic bronchoscopy was performed on the patient with a provisional diagnosis of subglottic stenosis (Fig. 1A). Following mechanical dilation and the use of an argon laser, the placement of a Vergnon silicone stent was performed using rigid bronchoscopy. Subsequently, the proper position of the stent was confirmed by a fiberoptic bronchoscope (Fig. 1B).