Persistent Post-Extubation Stridor in an Intensive Care Unit: A Decision Dilemma

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Stridor is a harsh, wheezing, often high-pitched sound produced by rapid, turbulent flow of air through a narrowed supraglottic region to proximal trachea and can be inspiratory, expiratory, or biphasic.1 The incidence of post-extubation stridor varies from 2 to 42% in pediatric intensive care unit (ICU).2 Factors like traumatic intubation, multiple attempts, prolonged intubation, use of cuffed or inappropriate sized tube, lower age, inadequate analgesia, and sedation are associated with significant risk of post-extubation stridor.2,3 Here, we report a case of persistent post-extubation stridor in a patient with traumatic brain injury, who was medically managed, thus avoiding reintubation. Informed consent for reporting this case was obtained from the child’s parents.

A 2-year-old, 15 kg, male child, presented to the emergency department with a history of fall from the first floor (10–12 feet). He was tracheally intubated with a 3.5-mm uncuffed tube in view of low Glasgow Coma Scale (GCS) of E1V2M5. Noncontrast computed tomography (CT) scan of head revealed right basifrontal contusion with fracture of right frontal bone, which was managed conservatively. Extended Focused Assessment of Sonography in Trauma, CT scan of spine and torso, and X-ray of long bones were normal. On receiving the patient in the ICU, his endotracheal tube (ET) was changed from uncuffed to 3.5 mm cuffed tube. The tube was snugly fitting the trachea and there was no air leak at cuff pressure of 22 mm Hg. The child received midazolam and fentanyl infusion while on mechanical ventilation. During his ICU stay, he had two episodes of tube blockage due to thick secretions for which his ET was changed twice, using same sized tube without encountering any difficulty. Over the next 3 days, the patient developed paroxysmal sympathetic hyperactivity for which tablet bactroban (5 mg thrice daily) and propranolol (5 mg thrice daily) was administered via Ryle’s tube. Patient’s GCS gradually improved to full score and repeat CT head revealed resolving changes. Gradual weaning from the ventilator was initiated. Injection dexamethasone 0.2 mg/kg intravenously was administered about 6 hours prior to planned extubation and after a positive cuff leak test, trachea was extubated on sixth day postinjury. Post-extubation, he developed inspiratory stridor, bilateral wheeze, and respiratory rate of 30 to 35 per minute which aggravated on crying. Vocal cord edema was suspected as a cause of stridor because of history of multiple intubation attempts. As his vitals and arterial blood gas (ABG) parameters remained normal, we did not proceed directly with the tracheal intubation and decided to closely monitor the child. He was put on a high-flow nasal cannula (flow rate 10 L/min; fraction of inspired oxygen 0.4) and saturation remained around 92 to 95%. Patient was started on injection dexamethasone 3 mg twice daily, along with nebulization with salbutamol, budesonide, and adrenaline every 6 hours for 2 days. Within 2 days, wheezing subsided, frequency of stridor became intermittent, being more pronounced only on crying, while chest X-ray, vitals, and ABG were normal. An otolaryngologist consultation was taken, following which flexible fiberoptic laryngoscopy was done which revealed left vocal cord restricted mobility and left arytenoid prolapse. X-ray neck revealed no subglottic stenosis, and no intervention was to be done from their side. Patient was medically managed and nebulization was continued. Dexamethasone was stopped after 2 days; vitals were closely monitored and the child was shifted to the ward after 72 hours of observation (ICU stay of 11 days).

Stridor in the pediatric population has multiple etiologies.1,2 Studies have shown multiple endotracheal intubations

ISSN 2348-0548.
attempts and prolonged intubation to cause laryngeal injury ranging from mild edema to vocal cord immobility.\textsuperscript{1–3} The larynx in infants and young children is prone to intubation injuries because of its funnel shape and relatively thin mucosa. Post-intubation laryngeal injuries in children are relatively common (97%), of which 88% are significant.\textsuperscript{4} Laryngeal erythema, edema, vocal process ulceration, granulation, vocal fold immobility, and subglottic narrowing are seen commonly. The vocal processes of arytenoids, is the site most vulnerable to intubation injury.\textsuperscript{5} The cuff leak test is of limited significance for predicting post-extubation stridor in children.\textsuperscript{3} Dexamethasone with anti-inflammatory action have been shown to prevent post-extubation stridor by 40%.\textsuperscript{2,3,6} Both aerosolized epinephrine and budesonide are equally effective in response to post-extubation stridor.\textsuperscript{7} Despite high incidence of post-extubation stridor, most of the lesions tend to heal spontaneously with no or minimal consequences and with correct medical treatment, only few need reintubation or surgical intervention.\textsuperscript{2–4} Veder et al studied 150 children in pediatric ICU and 28 of them developed stridor following extubation. Out of these, 3 children (10.7%) were reintubated in view of respiratory distress and only one required surgical intervention.\textsuperscript{2} Incidence of reintubation after extubation failure in children range from 16 to 19%\textsuperscript{5} and this increases the chance of subsequent extubation failures and need for tracheostomy. Tracheostomy in small children has its own concerns like tube blockade, impairment of speech development, changes in lifestyle, and anxiety in parents.\textsuperscript{4} As there is a risk of negative pressure pulmonary edema development when child breathes against partially closed glottis; close monitoring of GCS, ABG, and X-ray chest is of paramount importance to take prompt decisions regarding reintubation if condition worsens. Underlying cardiac and pulmonary dysfunction in patients with acute head injury requires a lower threshold for reintubation. As our patient was conscious and neurologically intact with stable vitals and oxygenation, we decided to closely monitor the child and medically manage with oxygen inhalation, nebulization, and intravenous steroid administration. The child improved over time and reintubation could be avoided.

As an intensivist one should be aware of various possible risk factors for post-extubation stridor and initiate timely preventive therapies. Preventive measures include use of correct sized ET, fixation at appropriate length, and regular measurement of cuff pressure when cuffed ET is used. Once stridor happens, the decision to reintubate should be based on the cause of stridor, general condition of the child, vitals, his reserves, underlying neurological, cardiac, and lung conditions. Close monitoring can enable us to avoid reintubation to prevent its adverse consequences.

\textbf{Funding}
None.

\textbf{Conflict of Interest}
None declared.

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