Original article

Postextubation stridor in paediatric cardiac surgery patients

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Abstract

Background: Paediatric patients undergoing cardiac surgery are likely to be prone to developing postextubation stridor (PES) due to their airway anatomy and several factors related to surgery, **Aim:** To examine the incidence and risk factors for PES in paediatric patients undergoing cardiac surgery. **Methods:** The study was prospectively conducted in the paediatric cardiac postsurgical ICU (PICU) at a tertiary referral hospital from November 2010 to January 2012. All paediatric patients presenting with immediate stridor or its developing within 24 hours after extubation were included. Only those patients who were ventilated for at least 6 hours after surgery, but not more than 7 days and deemed fit for elective extubation were considered. **Results:** Of the 1328 patients admitted to the PICU, 29 patients (2.18%) met the criteria for PES. Of these, 22 (75.6%) were < 1 year old. Ten patients (34%) did not respond to conservative approach or Noninvasive Ventilation (NIV) and had to be reintubated. Six patients were reintubated within an hour, three in < 6 hours and one after 12 hours of extubation. **Conclusion:** PES is common in paediatric postcardiac surgical patients. Infants are more prone to develop PES. Majority of them can be successfully managed with conservative measures and noninvasive ventilation. The onset of PES varies from immediately after extubation to 3 hours after extubation. Up to one-third of the patients with PES may require reintubation and is common in the first hour after extubation. Patients who develop PES need close observation in the first few hours after extubation.

Keywords: Postextubation stridor, paediatric cardiac surgery.

Introduction

The word stridor is derived from the Latin word 'stridulus' which means creaking, whistling or grating. Stridor or a mechanically produced noise during normal breathing is a frequently encountered clinical symptom in paediatric and neonatal age group. This is caused by airflow limitation through narrowed or partially obstructed respiratory tract. Stridor, in itself is not a diagnosis but merely a symptom of underlying pathology. Stridor can be defined as a variably pitched respiratory sound caused by tissue vibration through an area of decreased calibre. The characteristic features are: airway obstruction at the level of the nasal cavities produce inspiratory low pitched sound called stertor or snoring, supraglottic lesions tend to produce high pitched sounds, subglottic lesions tend to produce biphasic high pitched stridorous sounds and lower respiratory tract lesions produce expiratory wheezing. Most stridulous sounds are musical lasting > 200 ms. These are usually inspiratory

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and harmonious. In vigorous inspiratory efforts, especially in air hunger, stridor is inspiratory and high-pitched.[1]

Stridor increases work of breathing and can result in hypoxia and hypercapnia. These may adversely affect recovery from cardiac surgery causing tachycardia, arrhythmias, myocardial depression, pulmonary hypertension and cardiac failure. Severe hypoxia may result in bradycardia and cardiac arrest. The following survey was conducted to evaluate the incidence of stridor in postoperative paediatric cardiac surgery patients.

Methodology

The study was conducted at the paediatric cardiac intensive care postsurgical unit at Amrita Institute of Medical Sciences and Research Centre (AIMS), a multispecialty teaching and tertiary referral hospital. On approval from the hospital ethics committee, the study was conducted over a period of November 2010 to January 2012. It was a prospective, comparative and observational clinical study.

All paediatric patients presenting with stridor immediately or developing within 24 hours after extubation, ventilated for at least six hours after surgery but not more than seven days and deemed fit for elective extubation were included in the study. Patients with anatomical defects in the upper airways, pre-existing upper airway disease and those that self-extubated were excluded. Patients with incomplete surgical repair, *e.g.*, residual shunt or stenosis evident on postoperative echocardiography were also excluded as this group needed a longer period of ventilation.

During the study period, two paediatric cardiac surgeons performed the operations. Anaesthesia and cardiopulmonary bypass (CPB) were managed by three senior consultant cardiac anaesthesiologists dedicated to cardiac anaesthesia and intensive care. The endotracheal tubes (ETT) used were Smiths Portex uncuffed tracheal tubes with Murphy eye. The ETT size was calculated according to age [formula used for calculating the size of ETT: Internal diameter of the ETT = Age in years/4 + 4 mm]. An ETT tube size of 3.5 mm was usually used for infants < 4 months and 3.5-4 mm was used for older infants. After induction of anaesthesia, the trachea was intubated. The position of the ETT was assessed by direct laryngoscopy showing the line marker at the level of vocal cords and also identified by auscultation and capnography. If the air leak was too high, the ETT was changed to a larger size. Conversely, if there was any resistance while passing the tube into the trachea, a smaller tube was placed.

Data obtained from the operating room (OR) was maintained by the duty respiratory therapist by referring to the OR perfusion chart and anaesthesiologist's OR records. This included the size, type, fixation level of ETT, aortic cross clamp time and the leak percentage measured after connecting to the ventilator and by auscultating the neck.

Postoperative management in the PICU was primarily directed by anaesthesiologists who stayed in the hospital round the clock. However, paediatric cardiologists and cardiac surgeons participated actively in the postoperative care. All patients received sedation, analgesia, muscle relaxation and nursing care as per the standard ICU protocol. The patients were ventilated with different groups of ventilators such as Siemens 300, Maquet Servo*i* or Datex-Ohmeda Engstrom Care station. The mode of ventilation was mostly pressure regulated volume control (PRVC) and weaning took place in pressure support (PSV) and volume support (VS) mode. Ventilator setting changes were done by respiratory therapists as per gas analysis and correction. Trouble shooting and weaning from mechanical ventilation were performed by the respiratory therapist as per the protocol initiated in the ICU for weaning and extubation.

The following criteria were required before weaning:

1. Clinical improvement of the underlying disease as suggested by good spontaneous respiratory efforts, lack of retractions, resolution of

disease, GCS ≥ 11 , capillary refill time < 3 s, age appropriate blood pressure (above 2 SD appropriate for age in absence of inotropes with exception of dopamine 5 µg/kg/min), respiratory rate normal for age (< 60 bpm for infants < 2 months of age, < 50 bpm for 2-12 months, < 40 bpm for children 1-5 years and < 30 bpm for children > 5 years) and SpO_2 more than 94%.

2. pH ≥ 7.35 (acceptable blood gas values)
3. Minimal ventilator support: Fraction of inspired oxygen (FiO_2) ≤ 50%, positive end-expiratory pressure (PEEP) ≤ 5 cm H_2O, positive inspiratory pressure (PIP) ≤ 20 cm H_2O.

All patients were extubated in PICU (not in the OR) after rewarming, gradual arousal from general anaesthesia, establishment of stable haemodynamics and haemostasis. A combined decision for extubation was taken by the attending anaesthesiologist and duty respiratory therapist based on the clinical evaluation and blood gas status. The procedure was conducted by the respiratory therapist and the staff nurse. Patients were observed prospectively for postextubation complications by the respiratory therapist. Observations on adverse events after extubation such as severe desaturation, absence of cry, drowsiness, sternal/chest retractions, prolonged inspiration and expiration, laryngospasm and the requirement of aerosolised medications were noted down.

A protocol was implemented for the management of PES.

Diluted epinephrine (0.5 ml of 1:10,000 solutions) was administered immediately, by nebulisation for its acute anti-inflammatory effect. The action of epinephrine, being transient was also associated with potential risk of rebound oedema. Repeated use of this drug was thus limited.

Treatment with steroids provides a sustained effect due to their anti-inflammatory action. Intravenous dexamethasone was found to be effective in pre-extubation and postextubation states, thus decreasing the risk of postextubation stridor. Inhaled steroids with a similar mechanism of action as systemic steroids should be more advantageous due to direct delivery at the site of action, lesser dose and with fewer side effects. Thus, aerosolised budesonide (2 ml of 0.025%) was found to be effective in reducing the oedema.

Medical treatment consisted of respiratory therapy with postural drainage including prone, quarter/semiprone, airway clearance, bronchial hygiene therapy, antibiotics in the presence of infection and close monitoring.

Different techniques of Noninvasive Ventilation (NIV) were used. Fitting commercial masks on the infant's face without leak was very difficult. Volume of these masks was too large, limiting the effect of NIV. A nasopharyngeal tube with a shorter length (approximately 10 cm), inserted up to 4-5 cm into the nostrils was used to deliver NIV. This was well tolerated and avoided translaryngeal intubation.

No specific comparison was made in this study concerning the ventilators. The most efficient ventilator with better triggering and maximum leak tolerance was adopted. The initial settings on NIV were based on the initial clinical presentation. It was usually commenced using spontaneous time mode with expiratory positive airway pressure (EPAP) of 5 cm H_2O and inspiratory positive airway pressure (IPAP) of 8 cm H_2O. If continuous positive airway pressure (CPAP) alone was required, it was commenced using 4 cm H_2O. FiO_2 setting was adjusted as per the oxygen saturation level and blood gas values. Blood gases were analysed after one hour of continuous NIV to evaluate its effectiveness. Despite this, if the child worsened clinically, elective intubation was done with a smaller tube compared to the previous one. Adequate tracheal seal was ensured.

Patients with respiratory distress were reintubated, provided they met at least one of the following criteria:

1. Clinical deterioration: Respiratory distress, poor respiratory efforts, progressive stridor.
2. A decrease in oxygen saturation to less than 85% despite the use of supplemental oxygen.
3. Worsening blood gas analysis: pH of less than 7.35 with an increase in partial pressure of carbon dioxide of > 15 mm Hg from baseline, PaO_2 of < 50 mm Hg with supplemental oxygen.
4. Hypotension with a systemic blood pressure of < 2 SD for > 30 min despite adequate volume challenge, a diastolic blood pressure drop of > 20 mm Hg.

Scoring of stridor was done according to guidelines by Reber *et al*[2]

1 - Normal breathing sounds detected by auscultation over the trachea.
2 - Stridor over the trachea detected by stethoscope.
3 - Stridor detected without auscultation (audible).
4 - No airway sound detectable over the trachea.

All infants received standard routine care according to our internal protocols, with no changes due to introduction of studied interventions.

Results

During the study period, a total of 1328 patients underwent surgery and were admitted to the PICU. A total of 29 (2.18%) patients met the criteria for PES. Twenty two patients were < 1 year old, four between 1-5 years, while three were above 5 years of age. Among the 29 patients, 20 were male and 9 were female.

Eleven patients (37.9%) who had stridor had undergone corrective surgery for tetralogy of Fallot (TOF), while seven (24%) had undergone ventricular septal defect (VSD) closure *(Figure 1)*. Patients who underwent simple procedures such as atrial septal defect (ASD) with shorter cross clamp time and cardiopulmonary bypass time had fewer postextubation complications compared to the group having more complex surgery or prolonged pump times. Twelve patients who developed stridor had received ventilation for < 24 hours before extubation, while the rest had more than 24 hours of ventilation.

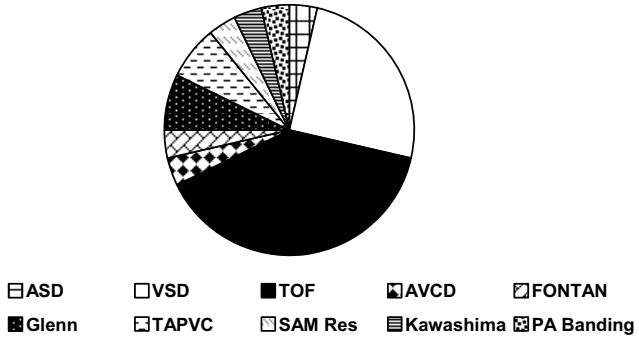

Figure 1: Diagram showing type of paediatric cardiac surgeries undergone by the patients.

Six patients in the study group were dysmorphic, with syndromic features and abnormal facies. None of the patients were malnourished. Stridor could be heard without auscultation (Reber stridor intensity score of 3) in all, except for one, where it was heard with auscultation (Reber stridor intensity score of 2).

Clinical features of stridor included increased resistance during inspiration and expiration, reduced inspiratory and expiratory airflow, reduced tidal volumes and increased chest wall distortion. The onset of stridor varied from immediately after extubation to 12 hours after extubation. Resolution of stridor took a few minutes in some, while it persisted for maximum 9 days in a patient. An otolaryngologist was later consulted for bronchoscopy and the need for tracheostomy.

As shown in (*Figure 2*) 12 out of 29 patients with stridor could be successfully managed with conservative therapy. Conservative management included dexamethasone, which was started electively to prevent stridor immediately after extubation. All these patient groups received aerosolised medications such as salbutamol, adrenaline, budesonide, ipratropium bromide, normal saline and some mucolytics such as N-acetylcysteine.

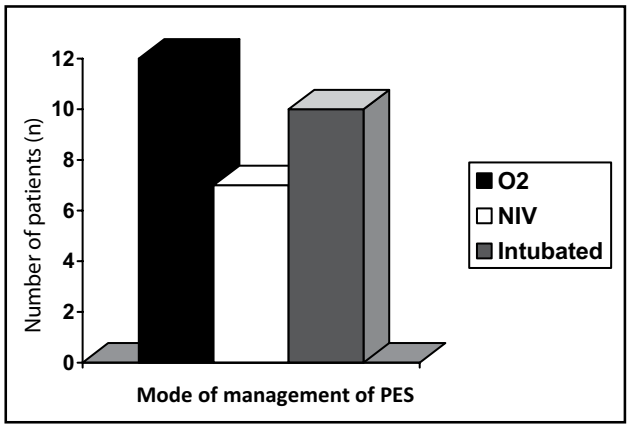

Figure 2: Number of patients requiring different modes of management of stridor

Seven patients who did not improve could be managed with noninvasive ventilation. Ten patients (34%) had severe stridor, not responding to conservative approach or NIV and had to be reintubated. Six patients got reintubated within an hour, three in less than six hours, and one after 12 hours of extubation *(Figure 3)*.

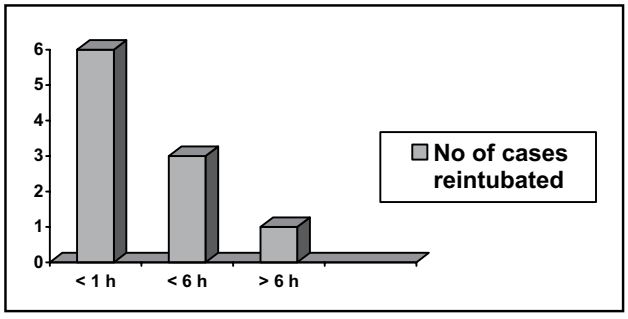

Figure 3: Number of patients with postextubation stridor requiring reintubation.

Discussion

This observational study was initiated to assess the problem of stridor in the paediatric cardiac surgical ICU. The incidence of stridor in our study population was 2.18 %. Koka *et al* reported an incidence of 1% for postextubation croup in paediatric patients.[3] Although there are a number of adult studies reporting incidence of postextubation stridor (PES), studies reporting incidence in paediatric patients are very scarce. The incidence of PES reported in literature varies from 3.5 to 30.2%.[2-8]

Major causes for PES are problems with endotracheal intubation, vocal cord paralysis, congenital laryngeal anomalies such as laryngomalacia, subglottic stenosis, epiglottitis, foreign body aspiration, retropharyngeal abscess, subglottic haemangioma and bacterial infections.

Endotracheal tube and reintubation related factors include incorrect tube size, cuff with high pressure, number of attempts at endotracheal intubation and trauma during the attempt. Care related factors include multiple/traumatic intubations, improper fixation of ETT allowing excessive movement, inadequate analgesia and sedation, patients who try to speak/cry with the tube, too aggressive airway clearing techniques and nasogastric tube. Unplanned extubation may either be self-extubation by the patient or accidental removal by the medical personnel.[9]

Bilateral vocal cord paralysis is a well-documented complication of cardiac/thoracic surgery that transpires due to direct injury occurring during intubation or trauma during thoracic surgery. Most often, the injury is seen to occur on the left side because of the longer course of the left recurrent laryngeal nerve. In bilateral vocal cord paralysis, stridor will be biphasic with severe distress, whereas, in unilateral cord paralysis, the cry will be weak and feeble without any distress. Injury to the recurrent laryngeal nerve may be due to central venous catheterisation, unnatural position of head and neck during surgery and ICU stay, direct injury from traumatic endotracheal tube (ETT) insertion, inappropriate size or faulty insertion of the nasogastric tube, sternotomy/sternal retraction pulling laterally on both subclavian arteries, direct manipulation of heart or hypothermic injury with ice collecting in the pleural cavity. Vocal cord paresis associated with emotional stress increases the severity of stridor.[4]

Angioneurotic oedema may result in acute swelling of the upper airway with resultant stridor and dyspnoea. Patients with bacterial infections are prone to have stridor (bacterial tracheitis). Compression of

the trachea may result from external compression due to vascular anomalies such as double aortic arch, anomalous innominate artery, anomalous left carotid artery, anomalous left pulmonary artery, aberrant subclavian artery and is often aggravated by feeding.[5] Laryngotracheal stenosis (congenital/acquired) is narrowing of the glottis, subglottis and trachea and happens most often in patients intubated for longer time or due to external compression.

Subglottic stenosis is said to be present when the tracheal diameter is < 4 mm in full term infant and < 3 mm in a premature infant. The stenosis is usually most significant, 2 - 3 cm below the vocal cords. Generally, the risk of developing subglottic stenosis increases proportionately through the first week of intubation. Associated symptoms are tachypnoea, hoarseness, shallow breathing and sternal or supraclavicular retraction. Subglottic obstruction may have a barking cough as a symptom and can mimic glottic obstruction. The stridor can worsen while crying or in an excited state, often in supine position where the supraglottic structures may collapse inward. Low birth weight infants are more prone to get stridor. Immediate stridor with choking is strongly suggestive of foreign body.

Other clinical findings include prolonged inspiration due to laryngeal obstruction or a prolonged expiratory phase due to tracheal obstruction. Unilateral decreased air entry may be because of foreign body in the bronchus. Drooling of mouth and leaning over are the other signs.[10] Other major risk factors are pre-existing tracheal irritation, neurological impairment and congenital anomalies.

The onset of symptoms varied from immediately after extubation to a maximum of three hours after extubation. Maximum intensity of signs occurred within four hours and became markedly diminished or nil after 24 hours. The need for reintubation was highest in the first hour after extubation. This observation warrants close bedside monitoring of patients with PES for the first few hours until symptomatic improvement is noted.

75.6% of the patients (22 out of 29) who developed stridor were less than a year old. This indicates a higher incidence of stridor in infants. This is in contrast to a very low incidence of stridor among infants in the study done by Koka *et al* who reported a high incidence in the 1-4 years age group.[3]

Eleven patients (37.9 %) with stridor had undergone TOF repair, while seven (24 %) underwent VSD closure. Many of the patients with TOF, associated with Down's syndrome were difficult to sedate adequately in the ICU. This may partly be a reason for higher incidence of stridor.

Noninvasive positive pressure ventilation can help prevent reintubation due to respiratory insufficiency in general.[3,5] Evidence for benefit of noninvasive positive pressure ventilation in laryngeal oedema, however, is lacking. In this study, 41% of the patients with PES could be managed successfully with conservative management, while 24% patients were managed successfully with NIV without the need for reintubation. NIV, particularly continuous positive airway pressure (CPAP) can be effectively used to relieve respiratory distress due to stridor in children. CPAP relieves the obstruction either by dilating the pharynx, splinting the airways open or by increasing functional residual capacity. The level of CPAP sufficient to relieve the airway obstruction depends on the severity of the obstruction and patient's condition.

More than one-third of the patients with PES required reintubation. This highlights the burden of PES in the paediatric ICU. It is well known that reintubation increases the morbidity and mortality in the ICU.[11,12] Airway obstruction as a reason for reintubation has lower mortality rates as compared to other causes.

This study has several limitations. The sample size is small and larger studies are required to further evaluate correlation between gender, age, type of surgery and the incidence of PES.

Conclusions

Postextubation stridor is not a rare complication in paediatric patients undergoing cardiac surgery.

Infants are more prone to develop postextubation stridor as compared to older children. Majority of the patients with PES can be successfully managed with conservative measures and noninvasive ventilation. The onset of PES varies from immediately after extubation to three hours after extubation. However, reintubation is commonest in the first hour after extubation. The patients need close observation in the first few hours after extubation, especially if they have PES.

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