

Post-Intubation Tracheal Stenosis in Paediatric Patients after Cardiac Surgery

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Children who have undergone successful correction of an underlying congenital heart disease are at increased risk of morbidity and mortality in the post-operative period due to respiratory failure, desaturation, and pneumonia, leading to prolonged ventilation. Prolonged tracheal intubation often leads to tracheal stenosis, which may require surgical correction. The airway of an infant is more vulnerable to occlusion than that of an older child. Both trans-laryngeal endotracheal intubation and tracheostomy may result in frequent and severe complications.¹ These complications are classified into immediate, early and late.² Complications can occur at various stages of endotracheal intubation: during intubation, after intubation, after placement, during extubation and after extubation. We present a report of 2 children who developed tracheal stenosis after prolonged intubation.

Case Report 1

A 2-year-old male child, weighing 10 kg, underwent surgical patch closure of a large perimembranous ventricular septal defect with severe pulmonary artery hypertension (PAH), (90/40 mm Hg). The child's trachea was nasally intubated with 5 mm internal diameter (I.D), non-cuffed, endotracheal tube (ETT). At peak inflation pressure of >20cm H₂O, small air leak was present around the tube. After uneventful surgery, the child was shifted to intensive care unit (ICU) and was extubated on post-operative day (POD)-1. On POD-3 the child was shifted to the ward, where on POD-4, the child had a cardio-respiratory arrest detected by the surgical

resident on duty. The child was immediately intubated orally with non-cuffed ETT size 5.0 mm, I.D, revived and brought back to the ICU. The child later had convulsions for which a computed tomographic (CT) scan was done, which revealed hypoxic brain damage. The child developed pneumonia 2 days after re-intubation and remained intubated for 20 days. Repeat CT scan of head showed marked recovery. With no more incidence of convulsions and resolving of pneumonic patches after appropriate antibiotic therapy, the child was extubated. Immediately after extubation the child developed stridor (both inspiratory and expiratory) with an increase in respiratory rate (55-65 breaths per minute). An ear, nose and throat (ENT) surgeon evaluated the child and performed rigid bronchoscopy under sedation (ketamine and atropine). The child was diagnosed to have a subglottic stenosis of the trachea. The true vocal cords were mobile with a stenosis at the subglottic level with a lumen size of approximately 2 mm. The smallest sized rigid bronchoscope (3mm) could not be negotiated through the stenotic lumen. The ENT surgeon advised splitting of the tracheal ring and the cricoid cartilage, with 4-5 days of elective ventilation after the procedure.

The child was transferred to the operation theatre (OT) on the same day. He was pre-treated with atropine and oxygenated with 100% oxygen via facemask while electrocardiogram leads and pulse oximeter probe were being placed. Anaesthesia was induced with halothane. As it was possible to ventilate the child via bag and mask, rocuronium, (0.6 mg/kg) and fentanyl infusion (1µg/kg/min) were administered. Fiberoptic laryngoscopy was performed and the earlier findings were confirmed. The child was ventilated with 100% oxygen. A horizontal incision was made over the cricoid cartilage. The cricoid cartilage and upper 2 tracheal rings were identified and split. A 5.0 mm, non-cuffed, tracheostomy tube was placed into the distal trachea by the surgeon and the lungs were ventilated through it. At this time, we tried to negotiate a 4.0 mm sized endotracheal tube but our attempts failed. With the help of rigid bronchoscope we were able to negotiate a 4.5 mm sized tube past the stenosis, while the tracheostomy tube was withdrawn. The ETT was fixed after confirming equal bilateral chest expansion and air entry. Post surgery the child was

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ventilated for 5 days. Post extubation there was no stridor and later the child was discharged.

Case Report 2

A 1-month-old male child weighing 3.5 kg was diagnosed to have complete atrio-ventricular septal defect (AVSD), moderate mitral regurgitation, and moderate to severe PAH. The child's trachea was intubated with 4.0 mm ETT, orally. An AVSD repair was performed using standard CPB techniques. Intraoperatively, surgeon placed a catheter into the main pulmonary artery. After weaning off CPB, the child had systemic pulmonary artery pressures (PAP) while on inotropic support of dobutamine (5 µg/kg/min) and sodium nitroprusside (0.1 µg/kg/min). Post repair the child was shifted to the ICU and was ventilated with oxygen and nitric oxide (20 parts/million, SV 300 NO, Siemens, Elema AB, Solna, Sweden). The child was kept sedated with fentanyl infusion (2 µg/kg/min). As the PAP started to decrease, weaning from mechanical ventilation was initiated. Fentanyl infusion, and nitric oxide were tapered, but the child developed episodes of PAH crisis during ETT suctioning and during one such episode developed bradycardia and hypotension. Hence, the child was paralysed and sedated and mechanical ventilation was continued. The child was gradually weaned off nitric oxide over a period of 14 days and extubated at 18 days after the initial surgery. After extubation the child had difficulty in breathing. The arterial blood gas analysis showed gradually increasing arterial carbon dioxide (PaCO₂) tension with a rising PAP. Hence, the child was re-intubated with a smaller sized ETT (3.5 mm), as 4.0 mm ETT could not be negotiated beyond the vocal cords. Later, a tracheostomy was performed. A fiberoptic bronchoscopy was performed which showed bilateral cord mobility with no tracheomalacia. A CT scan of the upper airway revealed tracheal narrowing just superior to the tracheostomy site. Rigid bronchoscopy showed suprastomal granulations. The child was treated with intravenous hydrocortisone (2 mg/kg, 4 hourly) and electively ventilated. A repeat rigid bronchoscopy showed complete resolution of granulation tissue with no narrowing of tracheal lumen. The child was successfully weaned from the ventilator after 2 days. Hydrocortisone was tapered and stopped on day 5, post extubation.

Discussion

Endotracheal intubation (EI) may result in significant injury to the larynx and trachea.

Subglottic stenosis is the most dangerous consequence of this injury in small children. The potential risk factors for post-intubation subglottic stenosis include, the underlying disease requiring EI, the age and body weight, the duration and number of EIs, absence of sedation and the occurrence of infections, hypotensive or hypoxic events during the period of EI and traumatic EI.^{1,2} Sedation is essential in intubated paediatric patients as straining against an ETT may result in repeated trauma to the mucosal and submucosal layer of the trachea leading to mucosal oedema, granulation tissue formation with fibrosis at later stages of healing. Tracheal stenosis is a well-known complication³ that results from prolonged intubation.¹

Zagaloc et al⁴ in their study found that:

- a) Chronic inflammation and fibrosis were responsible for narrowing of the trachea in tracheal stenosis patients.
- b) Metaplastic ossification of cartilage rings occurs only after complete obliteration of the tracheal lumen
- c) Loss of cilia and presence of metaplastic bone tissue are indications of a poor prognosis for tracheal stenosis.

Subglottic tracheal stenosis in childhood can be of 'hard' and 'soft' types occurring typically as primary lesions and stenosis caused by trauma (following intubation or faulty tracheostomy) as the most common secondary lesions. The subepithelial layers play a leading role in the origin of tracheal stenosis.⁵ Severity of stenosis depends upon the degree of involvement of the tracheal wall.³ Involvement of the entire thickness of the tracheal wall results in more severe stenosis. Patients with smaller airways, especially infants and children, have a higher incidence of complications. They are more susceptible to upper airway obstruction secondary to glottic oedema and subglottic stenosis. Whited⁶ reported that intubation for 7 days or less had a low incidence of sequelae, whereas 10 days or more resulted in a high incidence of sequelae. Cuffed tube usage for prolonged intubation and artificial ventilation substantially increases the rate of tracheal and laryngeal injury. Cuff pressure above 25 to 35 mm Hg further adds to the risk by compressing tracheal

capillaries, which predispose to ischaemic mucosal damage.⁷ Selection of an endotracheal tube of the correct size is mandatory. Too large a tube can cause a higher incidence of postoperative sore throat, laryngeal damage and tracheal stenosis.

Depending on the severity of the degeneration and subsequent fibrosis, tracheal stenosis may become symptomatic immediately after extubation, or may present at a later stage with difficulty in breathing. Occasionally, the problem may escape attention for years. Clinically, tracheal stenosis presents as dyspnoea not explained by cardiopulmonary disease in a patient who has received mechanical ventilation. In the subglottic area and particularly the trachea, the lesion is circumferential and located at the level of cuff. Stridor is common after the lumen narrows to less than 5 mm. The exact site of such stenosis can be ascertained by fiberoptic bronchoscopy or contrast enhanced CT scan of the neck.⁸

The treatment strategy for laryngo-tracheal stenosis in children has for a long time been conservative with tracheostomy or bougienation with the hope of a spontaneous resolution of stenosis with the growth of child. Repeated dilatations are done using rigid bronchoscopes (a set of paediatric 3.5-6 mm and adult 7-9 mm), with or without tracheal stenting. Common problems with stents include displacement and obstruction with secretions or granulation tissue. Less commonly the stent may perforate the airway wall, sometimes into the accompanying blood vessels.⁹

If this is not corrective, then a laser resection of the stenosed portion of trachea is performed.¹⁰

Tracheal dilatation, stenting and laser resection are only palliative therapies.¹⁰ The results of these palliative options have proved to be rather disappointing. Treatment like laryngo-tracheal reconstruction with rib cartilage, crico-tracheal resection and laryngofissures are good alternatives for severe stenosis with proliferative scar tissue formation.¹¹ During the splitting of cricoid and tracheal rings, maintaining sufficient airflow to the distal airways remains a challenging task. The use of high frequency jet ventilation (HFJV) is the method of first choice, especially in lesions of upper part of trachea.¹²

Medical management has proved to be successful in select cases. A trial of intravenous/inhaled corticosteroids is indicated when post-intubation tracheal stenosis is diagnosed early. This may influence the inflammatory process favourably and preclude the need for reconstructive surgery. Braidy et al¹³ reported that 200 µg beclomethasone aerosol every 6 hours improved their patient's symptoms. We have used intravenous hydrocortisone in our patient who developed suprastomal granulation. Post treatment bronchoscopy revealed complete resolution of the granulomatous tissue. Treatment strategy also depends upon the site of stenosis. If the stenosis is subglottic, immediate relief can be obtained by a tracheostomy, while awaiting definitive treatment. However, if the site of stenosis is lower down, treatment becomes an emergency. Such treatment may be palliative or definitive. Prevention of post intubation subglottic stenosis is possible by better management of the EI and of the child with a tracheal tube in situ. Sedation of intubated children; skilled EI with appropriate size tube is very important.

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