# Treatment of Iatrogenic Subglottic Stenosis by Therapeutic Fibreoptic Bronchoscopy

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#### **Abstract**

Endotracheal intubation is a common procedure in intensive care unit. It still qualifies as an invasive procedure. Prolonged endotracheal intubation or intubation with cuffed endotracheal tube in a newborn or young infant is a risk factor for the development of subglottic stenosis. It usually presents with stridor in childhood. The most likely mechanism of subglottic stenosis is an injury of tracheal mucosa, with secondary scar healing that consequently leads to the development of some degree of subglottic stenosis. Depending on the degree of subalottic stenosis, patients may be asymptomatic for a long time or symptoms can occur within several weeks. The incidence of stenosis is very low if intubation lasts less than a week. Sometimes the patient may develop severe subglottic stenosis after short-term endotracheal intubation. Intubation with cuffed endotracheal tube in newborn results in pressure necrosis and sloughing followed by subglottic stenosis. This article presents a case of a patient (Baby A) who had intubation with cuffed tube which landed to airway problem and stenosis and was managed successfully by taking help from an expert pediatric pulmonologist of a neighbouring country.

Key-words: latrogenic subglottic stenosis, Fibreoptic bronchoscopy.

## **Case Report**

A 7 months old boy (Baby A), was born at 32 wks of gestation and diagnosed as a case of preterm (32 weeks), low birth weight (LBW) baby (1.6 Kg) with suspected neonatal sepsis. Up to 14 days of age he was relatively stable and received supportive management at a private hospital in Sylhet. His echocardiography showed secundum atrial septal defect (ASD) and patent ductus arteriosus (PDA).

At 15 days of age, he suddenly had aspiration following feeding increased amount of milk (25ml). He developed severe respiratory distress and endotracheal intubation was done after 12 hours with cuffed endotracheal tube and placed on a mechanical ventilator. He was evacuated to CMH Dhaka with artificial ventilation by bag mask and breathing circuit on

26 August 2017 (day 16). The patient was on ventilatory support for 2 days, then auto extubated and cuffed tube was identified. The patient was quite stable for the next three days. He developed first episodes of stridor and respiratory distress on day 22 and managed with mobilization and chest physiotherapy for one hour. On day 24 he developed 2nd episode of stridor and respiratory distress and managed with nebulization and chest physiotherapy and it took a long time to stabilize him. At 31 days of age, he developed progressive stridor and respiratory distress followed by apnea at one stage with bradycardia when trial of giving orogastric (OG) tube for feeding was attempted. The patient was managed with cardiopulmonary resuscitation and placed on CPAP (continuous positive airway pressure) machine. The patient was irritable and eager to feed and he was sedated with low dose intravenous Midazolam. Chest x-ray (CXR) of the patient showed collapse of the left lungs. Vigorous chest physiotherapy was given for one hour and chest x-ray was repeated. This time collapse of the whole right lung was noticed with the clearance of left lung. The patient's condition improved after 12 hours and CPAP was discontinued. Next morning CXR showed collapse of left lungs again (Fig-2). Considering repeated intermittent collapse and consolidation of alternate lung, a polyp-like lesion intermittently blocking the alternate bronchus was suspected.

CT scan of the chest revealed complete collapse and consolidation of the left lung and an inflammatory pulmonary lesion in right lungs. No polypoid mass was seen after repeated review. Rigid laryngoscopy was done under low anaesthesia and findings were normal airway and laryngeal inlet but swollen and edematous arytenoids. The procedure was postponed as the condition of baby deteriorated. All available pediatric pulmonologists, otolaryngologists in Dhaka city were consulted. Laryngomalacia, gastroesophageal reflux, tracheal stenosis, polyp of airway were considered as differential diagnosis. But the confirmatory diagnosis was not possible as fibreoptic bronchoscopy was not available in Bangladesh. Pulmonologist of the neighbouring country was consulted. Finally, fiberoptic

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bronchoscopy was done by an Indian pediatric pulmonologist who came to Bangladesh with his portable fibreoptic bronchoscopy machine and removed some tissue from trachea during the procedure of bronchoscopy. Findings of bronchoscopy were pharyngomalacia, laryngomalacia, vocal cord nodules, sub-glottic stenosis, tracheomalacia etc.



Fig-1: Baby A

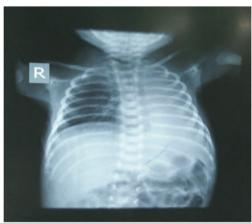


Fig-2: Left sided collapse consolidation on chest x-ray (morning, 31st day).



Fig-3: Right sided collapse consolidation on chest x ray (afternoon 31st day).

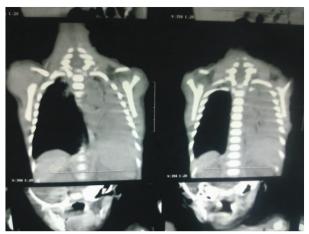


Fig-4: Left sided collapse consolidation on CT scan of the chest.

After bronchoscopy, the condition of the patient improved dramatically and in one week time, he was discharged to mothers care. His follow up showed completely normal lungs and he is one year old now.

## **Discussion**

Subglottic stenosis is a congenital or acquired narrowing of the sub-glottic airway<sup>1</sup>. Although it is relatively rare, it is the third most common congenital airway problem (after laryngomalacia and vocal cord paralysis)<sup>2,3</sup>. Subglottic stenosis can present as a life-threatening airway emergency and can affect both children and adults<sup>4</sup>. Several authors have reported subglottic stenosis in post-intubation cases since the '70s, some other even when the intubation was only for a short period of time<sup>5,6</sup>. It has been demonstrated that the trauma during intubation. situation of hypoxia or hypo-perfusion, cuffed endotracheal tubes and the length of the intubation period are all risk factors for subglottic stenosis. The cause suggested by many authors is a tracheal localized ischemic necrosis and an excessive granulation tissue formation with process of scar contracture<sup>7-10</sup>. Endotracheal tube always lies in the posterior larynx in which the structures vulnerable to pressure are the mucous membrane and mucoperichondrium covering the medial surfaces of the arytenoid cartilages and their vocal processes, the cricoarytenoid joints and the adjoining part of the cricoid cartilage below, and the posterior glottic and interarytenoid region. Although the supraglottic tissues at the level of the false vocal folds and above may become oedematous, they seldom sustain serious damage. Tracheal injuries have become less significant since the introduction of high-volume, low-pressure tracheal cuffs, even though overinflation of the cuff causing injury to the mucosa and ciliary loss is recognized as a risk. The microcirculation in the mucosa and mucoperichondrium is interrupted when pressure from the ETT exceeds capillary pressure. The resulting ischemic necrosis leads to oedema, hyperemia, ulceration.



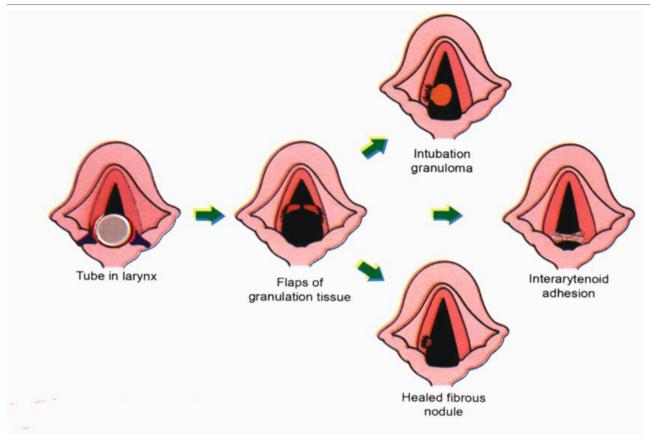


Fig-5: Consequences of intubation.

In neonatal intensive care, it is traditionally recommended to use the uncuffed endotracheal tubes because in children younger than 8 years the narrowest point of the airway is at the level of the circumferential cricoid cartilage. Furthermore, to intubate with the cuffed tracheal tube it is necessary to utilize the tube of smaller diameter and that creates a large increase in airway resistance during mechanical ventilation<sup>11</sup>. The experience of Black et al in 2953 pediatric patients intubated by uncuffed tubes in intensive care unit reports that none of the patients had clinical symptoms of acquired subglottic stenosis<sup>12</sup>. Other Authors showed that the use of cuffed endotracheal tubes in pediatric patients is not associated with an increased risk of subglottic stenosis and moreover avoids the unwanted aspirations of gastric contents 13-15. This reported case shows that the cuffed endotracheal tube can create the conditions favourable for the damage of tracheal mucosa. Moreover, auto-extubation in inflated stage aggravated the situation. The damage sometimes can be caused by the manoeuvres of intubation but more likely is due to the movement of the tube within the trachea particularly during the weaning of mechanical ventilation period, when the child makes the movements and probably also the endotracheal tube has some movements which cause damage of tracheal mucosa and gradually subglottic stenosis may appear.

It is very important to take an accurate past medical history in children who have specific respiratory problems such as moderate respiratory difficulty, noisy breathing, frequent cough or abnormal results in the spirometry with a poor response to the therapies. When there is a history of endotracheal intubation, even for a short period of time (therefore to be considered not only during an intensive care admission but also if the child underwent intubation during general anaesthesia) it is recommended to perform a bronchoscopy in order to exclude subglottic stenosis.

## Conclusion

Any patient who is undergoing or has undergone long-term intubation should be aware of both the acute and long-term complications that may be encountered and should know their management. Any patient with unexplained stridor even after short-term intubation should be investigated for subglottic stenosis or other complications. Scar tissue formation may take time and may present clinical features weeks or months after intubation. Any change in voice should be addressed carefully. Proper selection of size and materials of the tube and use of cuff can reduce complications like subglottic stenosis in preterm low birth weight infants in neonatal intensive care units.

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