CASE REPORT

Neonatal Post-Intubation Subglottic Stenosis

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Summary

Acquired subglottic stenosis is a complication of neonatal endotracheal intubation. Although it is rare, it contributes significantly to the morbidity and physical well being of post extubated neonates. A 20-day old neonate, ventilated for meconium aspiration syndrome and persistent fetal circulation, presented with marked stridor and respiratory embarrassment. A stenosed subglottic area was found on rigid bronchoscopy. Anterior cricoid split was performed to relieve the obstruction. He is asymptomatic post operatively.

Key Words: Acquired subglottic stenosis, Neonatal, Post-intubation

Introduction

Subglottic stenosis is the narrowing of the subglottic airway or lumen. It is situated in the region of the cricoid cartilage. Cricoid cartilage, being a complete, non-pliable and non-expandable ring, is also the narrowest area of the entire human airway.

Subglottic stenosis can be divided into congenital or acquired. Congenital subglottic stenosis is secondary to maldevelopment of the cricoid cartilage in utero. The commonest cause of acquired subglottic stenosis is endotracheal intubation. Other causes include external trauma, high level tracheotomy, infection, inflammation, chemical or thermal burn, tumour and dystrophic cartilage.

Case Report

We report herein a 20-day old child who presented with marked stridor and chest recession, not altered with positional change. He was born 8 days post date, via emergency low segment caesarean section for fetal distress and meconium stained liquor with a birth weight of 4.6 kg. Postnatally, he was ventilated for meconium aspiration syndrome for six days. Size 3.5 mm endotracheal tube was inserted with ease. During ventilation, the endotracheal tube was reinserted twice due to tube blocked and failed extubation. In addition, he had persistent fetal circulation which required high pressure ventilation which later successfully reverted. He was also treated for presumed sepsis and packed cells were transfused. Five days post extubation he was discharged. He had noisy breathing since discharge from the hospital. Despite this, he was able to feed adequately. His parents were worried when the noisy breathing persisted and got worse.

He was given intravenous dexamethasone and budesonide nebulization. Flexible nasopharyngeal laryngoscopy was carried out and no gross laryngeal pathology was noted.

However, the respiratory distress progressively worsened and carbon dioxide retention occurred. Echocardiogram showed a normal heart. Rigid

This article was accepted: 7 October 2003
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laryngoscopy was carried out. The supraglottis and glottis were discovered to be essentially normal. Following this, rigid bronchoscopy was performed and the subglottis was found to be circumferentially stenosed resulting in a lumen that was only 25 - 30% patent (Grade II - III). A size 3.5 mm (inner diameter) rigid bronchoscope could not be passed beyond the stenosis.

He was intubated and ventilated with a size 2.5 mm endotracheal tube. A day later the child was able to breathe spontaneously with the endotracheal tube in situ.

The following day an anterior cricoid split (anterior laryngotracheal decompression) was carried out under general anaesthesia. A transverse skin incision was made over the cricoid cartilage. The cricoid ring, the thyroid lamina and the first two tracheal rings were exposed. A midline vertical split was created from the midpoint of the thyroid cartilage until the second tracheal ring, thereby exposing the airway lumen. The cricoid cartilage was circumferentially thickened. Stay sutures were placed through the cut edges of the trachea (Fig. I). A size 3.5 mm endotracheal tube was inserted. Skin sutures were loosely placed to approximate the wound and a corrugated drain was inserted to prevent emphysema, pneumomediastinum or pneumothorax. The tube was left in place as a stent for seven days.

Twenty four hours before extubation, the child was given intravenous dexamethasone 1 mg / kg / 24 hour in three divided doses and this was continued for further five days thereafter. He was successfully extubated after being ventilated for eight days in total. He was weaned off oxygen over the next three days. The child tolerated extubation well but developed prominent emphysema over the neck and upper chest which resolved spontaneously later.

Discussion

Whilst the incidence of congenital subglottic stenosis is unknown, the incidence of acquired subglottic stenosis is estimated to be 2% in the neonatal intensive care settings[1]. The incidence is even lower if meticulous care is taken to minimize trauma to the airway in intubated neonates.

Possible mechanism of subglottic stenosis in this child is trauma of the subglottic mucosa. The placement of endotracheal tube causes oedema and hyperemia of the mucosa. It then progresses to pressure necrosis of the mucosa and exposure of the perichondrium of the cricoid cartilage. Scarring of the subglottic area is the end result of the infected perichondrium.

In endotracheal intubation, the size of the tube used in relation to the larynx, the amount of air leak, temporal

Table I: Subglottic Stenosis Grading System

Grade	Severity of Obstruction
I	obstruction of 0 – 50% of the lumen
II	obstruction of 51 – 70% of the lumen
III	obstruction of 71 – 99% of the lumen
IV	100% obstruction or no detectable lumen

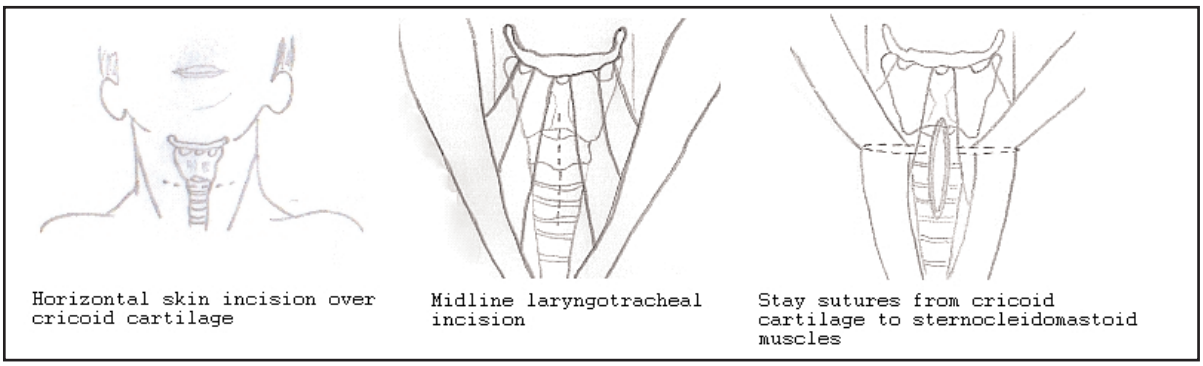

Fig. 1: Surgical Diagram

duration of the intubation, motion or friction of the tube and the number of intubation attempts, adversely contribute to subglottic stenosis. Septicaemia, local bacterial infection, hypoxia, anaemia, poor nutrition and poor perfusion contribute significantly to the development of subglottic stenosis.

The clinical presentation in this child was the same as described in the literature. Neonates with subglottic stenosis can present with obstructive breathing, which manifests as typically biphasic (both inspiratory and expiratory) stridor, laboured breathing and respiratory distress. The severity of the clinical manifestation correlates well with the amount of obstruction to the lumen of the airway. The severity of the obstruction can be graded I – IV (Table I)[2].

Flexible nasopharyngeal laryngoscopy with topical anaesthesia, can be used to assess pathology in the nose, nasopharynx, oropharynx, hypopharynx, larynx, arytenoids, epiglottis and vocal cords. Rigid bronchoscopy is best in assessment of the pathology in the glottis and subglottis regions.

The mode of treatment will depend on the severity of clinical presentation and response of the treatment. As a first line of treatment, intravenous dexamethasone and budesonide nebulization were given to this child. Other anti-inflammatory and vasoactive medications such as oral steroids and inhaled adrenaline can also be tried. If the obstructive symptoms persists, especially when they are well (i.e. free of lung pathology, sepsis) and at rest, surgical intervention needs to be considered. As a general rule, patients with Grade I and II stenosis will do well without surgical operation, whereas Grade III and IV cases will require surgical intervention. However, the prime consideration and determination has to be the clinical manifestation.

Various methods have been described to correct subglottic stenosis based on the severity, the location and the pathology involved. Mild cases can be managed expediently with a wait and watch policy. Most of these cases resolve spontaneously with growth. Endoscopic techniques have evolved primarily to treat moderate soft tissue narrowing. Repeated dilatation and carbon dioxide laser have been used extensively to ameliorate soft tissue stenosis[3]. Endoscopic management is not favoured when there is significant damage or abnormality of the cricoid cartilage as in Grade III and IV stenosis. In Grade III and IV stenosis, open surgical techniques such as laryngoplasty (Evan Todd) and single stage laryngotracheal reconstructive have been described. Tracheostomy has been performed in the past to provide relief to the obstruction while a more definite procedure was contemplated.

In this child, we opted for anterior cricoid split. Anterior cricoid split evolved in an attempt to avoid tracheostomy as tracheostomy in infants and neonates carries a significant rate of morbidity and mortality[3]. This procedure was described by Cotton and Sied in 1980. They developed this technique to relieve subglottic stenosis that was the result of small cricoid ring or in cases of soft tissue subglottic stenosis with normal cricoid cartilage. They proposed that the patient should have adequate pulmonary reserve (less than 30% oxygen requirement), weigh more than 1500 gm and should be free of active infections and should not suffer from congestive heart failure. Studies conducted in the past have shown that 77 – 85% of the children undergoing this procedure can be extubated successfully without a need for tracheostomy[6].

We have demonstrated that the anterior cricoid split is a viable surgical option in neonatal post-intubation subglottic stenosis.

References

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