Case report

Anaesthesia and laryngomalacia - A case report

Swati Verma*, Madhusudan Upadya

Email: swatzee2011@yahoo.com

Abstract

Laryngomalacia is one of the commonest causes of congenital stridor in neonates. The treatment depends on the severity of the stridor. We present a case of 12 day old neonate with a history of stridor who was diagnosed to have laryngomalacia on clinical examination and confirmed by flexible fibreoptic bronchoscopy. The neonate underwent excision of excessive aryepiglottic fold under general endotracheal anaesthesia with controlled ventilation as the definitive surgery.

Keywords: Excision, flexible fibreoptic bronchoscopy, general anaesthesia, laryngomalacia.

Introduction

Laryngomalacia, also known as floppy larynx syndrome is the weakness of the laryngeal structures such as epiglottis and aryepiglottic folds which causes the airway to collapse during respiration. Laryngomalacia is a congenital defect caused by the immature and incomplete development of the muscles and nerve supply to the larynx. It presents in the first few days of life or up to two months and worsens at the age of six months. The condition usually improves as the child grows beyond six months of age. Most infants are symptom free by 2 years of age although the stridor can be present for up to four years.

Case Report

A 12 day old neonate from South India presented with history of stridor which was high pitched and inspiratory crowing noises. Noises increased when the baby was lying supine, during crying or agitation. The baby's cry was normal and feeding intolerance was not noted.

On physical examination, the infant was active, there was no tachypnoea, vital signs were normal and oxygen saturation was 100%. Auscultation above the sternal notch demonstrated inspiratory noises. Rest of the systemic examination was normal. Neck computerised tomography (CT) scan was done to rule out other causes of stridor in a neonate such as subglottic stenosis, laryngotracheal cleft, foreign bodies or vallecular cyst. Oropharyngeal topography was also done to delineate any abnormality in the path of the passage of endotracheal intubation or fibreoptic bronchoscope. There was no anatomical obstruction of the airway on CT scan in this baby.

This neonate weighing 2.45 kg was posted for diagnostic flexible fibreoptic bronchoscopy under general anaesthesia to rule out other causes of stridor, grade the severity of disease, vocal cord lesions and look for other inflammatory conditions. The neonate was permitted to be fed breast milk 4 hours and glucose water 2 hours prior to the procedure. After written informed consent, the baby was shifted to operation theatre. Monitoring of electrocardiogram, oxygen saturation using pulse oximetry, end tidal carbon dioxide with a capnometer

Swati Verma, MD
Senior Resident, Department of Anaesthesiology, Kasturba Medical College, Mangalore, Manipal University, India

Madhusudan Upadya, MD
Professor and Head of Anaesthesiology, Kasturba Medical College, Mangalore, Manipal University, India

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and noninvasive blood pressure were commenced. The infant was induced with sevoflurane in 100% oxygen, a 24 G intravenous peripheral venous access was secured in the right upper arm and fluid infusion started. Further sedation was achieved with titrated intravenous bolus doses of 0.1 mg midazolam and anaesthesia maintained with intermittent intravenous boluses of 1 mg propofol. Child received supplemental oxygen *via* bronchoscope port to maintain oxygen saturation above 90%. The supraglottis was visualised during spontaneous respiration and major areas of collapse were noted. Respiratory rate, blood pressure, heart rate were continuously monitored throughout the procedure and saturation never dropped to below 98%.

A 2.6 mm diameter bronchoscope was chosen for evaluation of the airway. While advancing the bronchoscope, the entire larynx and vocal cords were evaluated and laryngomalacia score was assessed without spraying lignocaine. Application of lignocaine for topical anaesthesia during flexible fibreoptic bronchoscopy reduces the tone of upper airway muscles causing upper airway obstruction. Floppiness of the larynx thus produced alters the assessment of the grade and severity of disease. This increase in laryngeal collapse increases the difficulty of the lower airway examination and might result in missed diagnosis, incorrect assessment of lower airway and result in more frequent surgical intervention than necessary. At the end of the procedure, anaesthetics were cut off, the child woken up and shifted to postoperative ICU.

In order to quantify the degree of laryngomalacia, separate scores for the function of arytenoids and epiglottis as described by Sivan Y *et al* were used *(Table 1)*.[1] The laryngomalacia score consisted of the sum of arytenoid score and epiglottis score. A total score of ≥ 4 is considered consistent with laryngomalacia and significant. Our patient had a total score of 4.

Since the laryngomalacia in the infant was significant, he underwent excision of excessive aryepiglottic fold mucosa after three days under general anaesthesia. Baseline monitoring included 3-lead electrocardiogram, pulse oximeter, end tidal capnometer and noninvasive blood pressure. The neonate was induced with sevoflurane in 100% oxygen followed by muscle relaxation with atracurium after confirming ability to ventilate using face mask. The child was intubated with 3 mm I.D uncuffed RAE tube and fixed at preformed curvature after confirming bilateral equal air entry. After the completion of procedure, neuromuscular blockade was reversed with neostigmine and glycopyrrolate and waited till he started breathing spontaneously. Neonate was shifted to neonatal intensive care unit with endotracheal tube *in situ* for 48 h for the subsidence of oedema. Intravenous dexamethasone was given to control oedema. The child was extubated after 48 h in operation theatre under direct vision with fibreoptic bronchoscope. Gradually, child improved, was tolerating breast feeds well and discharged after 4 days. On subsequent follow ups till 8 months, child was active with good weight gain and no complains of stridor were noted.

Table 1: Laryngomalacia Scoring System[1]

Arytenoid score (AS)	
0 points	No discernible collapse into glottis with inspiration
1 point	Subtle collapse of arytenoids into glottis
2 points	Collapse of arytenoids into glottis, 25 to 50% of vocal cords obscured
3 points	Collapse of arytenoids into glottis, about 75% of vocal cords obscured
4 points	Collapse of arytenoids into glottis, 100% of vocal cords obscured
Epiglottis score (ES)	
0 points	Normal epiglottis, no folding during inspiration
1 point	Slight length-wise folding of epiglottis
2 points	Moderate fold of epiglottis without contact between lateral edges
3 points	Intermittent contact of lateral edges of epiglottis
4 points	Continuous contact and even overlap of lateral edges of epiglottis

Discussion

Laryngomalacia is the most common cause of congenital stridor in newborns, affecting 45-75% of all infants with stridor.[2,3] The high pitched noise of inspiratory stridor occurs when supraglottic structures collapse into the airway during the inspiratory phase of respiration. Laryngomalacia has a disease spectrum that can be divided into

mild, moderate and severe type.[4] Children with mild disease usually present with on and off inspiratory stridor, those with moderate disease have stridor with feeding-related symptoms which improves with acid suppression treatment and children with severe disease require surgical interventions such as supraglottoplasty. Other medical comorbidities associated with laryngomalacia are gastroesophageal reflux disease, neurological disease, additional airway lesions, congenital heart disease and the presence of a syndrome or genetic disorder.[5]

Stridor with respiratory compromise and feeding difficulties with failure to thrive, severe airway obstruction with significant retractions, pectus excavatum, cor pulmonale, pulmonary hypertension and hypoxia are absolute indications for surgery. Relative indications are aspiration with recurrent pneumonia, weight loss without true failure to thrive and a difficult to feed child who has not responded to acid suppression therapy.

In our case, the neonate presented to the hospital with symptoms of positional stridor and typical presentation of laryngomalacia. It was confirmed by flexible fibreoptic bronchoscopy under general anaesthesia in operation theatre. Larynx and vocal cords were examined and laryngomalacia score was assessed without spraying or infusing lignocaine as it is known that applying lidocaine solution to the upper airway structures for topical anesthesia during flexible fibreoptic bronchoscopy results in worsening stridor and clinical signs of upper airway obstruction.[6] Fibreoptic laryngoscopy findings were prolapsing epiglottis, large floppy arytenoids prolapsing into the glottis during inspiration, short aryepiglottic folds and normal subglottic region. Laryngomalacia score was determined to be 4 which was significant enough to be surgically corrected. Three days later, excision of excessive aryepiglottic fold mucosa was done under general anesthesia. The child improved well after surgery and had no symptoms thereafter till 8 months of regular follow up.

Conclusion

Laryngomalacia is one of the most common causes of stridor in neonates. General anaesthesia has been found to be more useful and accurate than awake fibrescopy to evaluate the severity of the disease and rule out other causes of neonatal stridor so that definitive surgical treatment can be instituted. Administration of anaesthesia to a neonate is in itself a challenge which is compounded in these cases by the presence of laryngomalacia. Careful attention to detail is required for a successful outcome.

References

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