Journal of Neonatal Biology
ISSN: 2167-0897

Kiran, et al., J Neonatal Biol 2015, 4:2
DOI: 10.4172/2167-0987.1000173

Case Report Open Access

Laryngomalacia in Neonates: A Review and the Surgical Management or Severe Cases

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Received date: Feb 09, 2015; **Accepted date:** Apr 10, 2015; **Published date:** Apr 17, 2015

Abstract

Congenital stridor is one of the rare presentations of respiratory distress at birth. The commonest cause of congenital stridor is laryngomalacia, which accounts for 60% of the causes, seldom requiring any intervention. The current report describes a case of neonatal stridor which needed evaluation and intervention at the earliest.

Keywords: Stridor; Laryngomalacia; Congenital; Intervention

Introduction

Laryngomalacia is the most common cause of stridor in neonates. Holinger found laryngomalacia in 60% of children with congenital laryngeal anomalies who presented with stridor [1]. Most children with laryngomalacia stridor resolve within 18 to 24 months of birth and require no specific treatment [2]. However, neonates with severe laryngomalacia may develop marked upper airway obstruction resulting in cor pulmonale, life-threatening apneic episodes, feeding difficulties, obstructive sleep apnea, and failure to thrive [3]. While tracheostomy has been used in complex neonates in whom laryngomalacia is associated with other anomalies or medical problems, surgical resection of supraglottic tissues supraglottoplasty has been the favored approach. We report a case of laryngomalacia in a neonate managed with early evaluation and intervention with supraglottoplasty

Case Report

A female baby delivered by elective caesarean section to primigravida at 38 weeks of gestation was referred on day 8 of life with distress and stridor since birth. There was feeding difficulty and one episode of aspiration following feed. At admission baby had tachypnea with suprasternal recessions and inspiratory stridor in supine position. Weight at admission was 2.38 Kg as to the birth weight of 2.75 Kg. Systemic examination was normal. Baby was nursed in prone position. A CT scan demonstrated dilated pharyngeal and laryngeal airway up to false vocal cords with a normal mediastinum. Flexible fiber optic laryngoscopy was performed by ENT surgeon which demonstrated laryngomalacia with curved epiglottis falling forward and excess aryepiglottic fold. Supraglottoplasty was performed with excision of excess aryepiglottic mucosa. Following the procedure baby was put on O_2 T piece with ET tube insitu for 3 days and then extubated. As no stridor or respiratory distress was noted, nasogastric feeds reinitiated. Baby was discharged on breast feeds and antireflux measures. Adequate weight gain was documented at subsequent visits.

Discussion

The differential diagnosis for neonatal stridor is vast and encompasses lesions throughout the airway tract. In a case series evaluating cause of stridor in babies it was found that Laryngomalacia (19.4%) was the commonest congenital cause [4]. Other diagnoses that should be entertained include: craniofacial anomalies, choanal atresia, encephalocele, turbinate hypertrophy, vocal cord paralysis (unilateral & bilateral), congenital or acquired subglottic stenosis, laryngeal webs, cysts or clefts, papillomatosis, GER (gastroesophageal reflux), tracheomalacia, vascular ring, mediastinal masses and foreign bodies [5].

It is especially important to know the differential diagnosis for stridor in the neonate because 17.5% (12-27%) of patients with stridor and respiratory symptoms have two or more unrelated airway lesions. Gonzales reported that 27% of patients with laryngomalacia had synchronous airway lesions and half of these were below the vocal folds [6]. GER has been documented in up to 80% of patients with laryngomalacia. Evaluation and treatment of this should be considered in symptomatic patients. Failure of surgical correction has been attributed to uncontrolled reflux.

The stridor in laryngomalacia is caused by obstruction at the level of the supraglottis when epiglottic and arytenoid tissues prolapse into the rima glottidis with inspiration. Not much is known about the exact cause. There are two schools of thought. Those favoring a structural etiology stipulate that the anatomic findings of a tubular epiglottis, shortened A-E folds and large arytenoids in these patients cause a narrowing of the supraglottic and the resultant higher airway pressures forces collapse of these tissues into the glottic opening. This theory is supported by histologic exams which demonstrate normal cartilage in autopsy specimens. Others believe that a neurogenic etiology is responsible for this disorder. Neuromuscular immaturity, hypofunction or incoordination is believed to cause flaccidity of the supraglottic structures. Supporters of this theory point to studies which show an association of other neurologic disorders including central apnea, hypothermia and GER in some patients. In addition, experimental evidence has demonstrated medial prolapse of the larynx in denervated larynges in animals and in addition there are many reported cases of acquired laryngomalacia following closed head injury.

Although usually a benign disorder, complications do occur. Feeding difficulties, obstructive sleep apnea, bradycardias, cor pulmonale, failure to thrive and pectus excavatum (due to persistent retractions) can all be seen in severe cases. The natural history in most patients is for spontaneous recovery, however because of frank airway obstruction or other complications 10-15% of babies require surgical intervention to improve their airway4. Tracheostomy has been the standard treatment for upper airway obstruction for over a century. With the development of laryngoscopy and endoscopic techniques more site specific treatments could be developed. In the early twentieth century a snare was used to amputate a portion of the epiglottis. Following this, hypomandibulopexy airway utilized in an attempt to improve the airway by suspending the hyoepiglottic ligament to the mandible. Contemporary surgeons began looking for alternative treatments for this entity. In general these methods addressed the three anatomic abnormalities exhibited by these patients: the floppy epiglottis, the redundant arytenoid tissue and the shortened aryepiglottic folds [7]. Various forms of aryepiglottic fold excision, laser division and microsurgical trimming was performed. Currently popular is the CO_2 laser supraglottoplasty technique in which the aryepiglottic folds are divided and the redundant arytenoid tissue is vaporized. In 6% of neonates undergoing supraglotoplasty, revision surgery is needed to remove the redundant mucosa when stridor persists beyond surgery.

To conclude laryngomalacia is indeed the most common cause of congenital stridor, which has a benign course in about 80-98% of cases, resolving spontaneously with no long term sequel. The mainstay is going to be observation and parental reassurance. Supraglottoplasty and surgical correction can be very effective for complicated cases and have drastically reduced the need for tracheotomy in these patients. Lastly, esophageal reflux disease, upper respiratory infections and any associated illnesses, such as heart, lung or neurological disease, should be aggressively treated as well in order to maximize the good outcomes in these children.

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