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Vocal fold paralysis following surgical ductal closure in extremely low birth weight infants: A case series of feeding and respiratory complications

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Abstract

Surgical closure of a Patent Ductus Arteriosus (PDA) continues to be frequent among Extremely Low Birth Weight (ELBW) infants, despite improvements in the medical management of PDA's and rising questions about its pathophysiologic role. Among other possible complications of this surgical intervention, left vocal fold paralysis (LVFP) has been reported. Only more recently, however, neonatologists are realizing the frequency and impact of this complication on chronic respiratory and feeding difficulties in the ELBW population. In this case series, we describe the clinical course of three sets of multiple births, for which at least one infant underwent surgical closure of his PDA and subsequently developed feeding and/or respiratory difficulties due to LVFP, and compare them to their respective siblings who did not sustain this complication.

Keywords

Patent Ductus Arteriosus; Vocal cord paralysis; Infant, Premature; Infant, Low Birth Weight; Feeding

Introduction

Surgical closure of the patent ductus arteriosus (PDA) has been considered a relatively safe treatment option for infants failing medical management.[1] More recently, however, the benefits of ligation vs. medical closure have been questioned, especially when considering extremely low birth weight infants. (ELBW, birth weight <1000 grams) Multiple complications have been reported with surgical closure of the PDA, including pneumothorax, chylothorax, infection, increased risk of bronchopulmonary dysplasia and neurodevelopmental impairment, and left vocal fold paralysis. (LVFP) [2,3]

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Paralysis or paresis of the left vocal fold, due to injury of the left recurrent laryngeal nerve during surgical closure of the PDA, has been reported in infants who had postoperative stridor, hoarseness, or inability to wean from mechanical ventilation. [4,5] We present a case series of ELBW infants who had vocal fold paralysis after surgical closure of their PDA and presented primarily with feeding difficulties, with or without respiratory issues. Our intent is to demonstrate that LVFP following PDA ligation in ELBW infants not only causes acute airway and feeding problems, but can impact feeding and respiratory status long after initial hospital discharge.

Patients and Methods

This was a retrospective chart review of all ELBW infants hospitalized in the neonatal intensive care unit at Duke University Medical Center between January 2004 and December 2006, who underwent surgical closure of their PDA. Demographic data, clinical course with respect to respiratory and feeding specifics during initial hospitalization and after discharge, were recorded. All cases of paralyzed left vocal fold were confirmed by direct laryngoscopy performed by an attending pediatric otolaryngologist. Reactive airway disease was determined by need for bronchodilator aerosol treatments at any time up until their 2 year developmental follow-up visit. Included, and whose clinical course is described in more detail, is a case series of three sets of multiple births, each of which had at least one sibling develop left vocal fold paralysis following surgical closure of their PDA.

Results

Family “1” was a set of triplets born at 26 weeks gestation.

Triplet A and Triplet B’s PDA closed spontaneously and by indomethacin, respectively. Triplet C required PDA surgery on day of life 9 secondary to hypotension and an increased need for respiratory support, after an attempt at medical closure was unsuccessful.

Triplet A’s clinical course was the most benign overall. He had no significant respiratory complications during or after initial hospitalization, and did not have any difficulties with the transition to oral feeds. While Triplet B remained on oxygen the longest, he had no further respiratory complications after NICU discharge. His feeds were initially thickened when starting to bottle feed due to poor coordination, but he subsequently tolerated non-thickened feeds without difficulty.

Triplet C, who underwent surgical ligation of his PDA, remained intubated for a total of 29 days, also requiring another 26 days of CPAP and 13 days of supplemental oxygen via nasal cannula. His course was complicated by aspiration pneumonia a few days after the initiation of oral feeding. Persistence of a weak cry and feeding difficulties, including bradycardia and desaturations with oral feeding, prompted flexible laryngoscopy which demonstrated left vocal fold paralysis on day of life 99.

All three infants were discharged home on full oral feeds of hydrolysate formula without added thickeners. They were all sent home being treated with anti-reflux medications and triplets B and C were equipped with a home apnea monitor. Triplet C’s ENT follow-up exams at 6 months and 14 months demonstrated persistence of left vocal fold dysfunction, but improving movement. His weak cry slowly improved over the first year of life and he had no further episodes of aspiration pneumonia or swallowing difficulties, though he was the only sibling to develop reactive airway disease requiring aerosol treatments during his first year. None of the infants were readmitted to the hospital for respiratory, feeding or other concerns, following their initial NICU hospitalization.

Family"2" were quadruplets born at 25 weeks gestation. Unfortunately, one of the infants ("A") was unable to be resuscitated at birth.

Infant C and D's PDA closed with indomethacin, whereas infant B required surgical closure on day of life 23. Infant B, indeed, had the most complicated NICU course, including a grade III–IV intraventricular hemorrhage, fungal and gram positive sepsis, and surgical necrotizing enterocolitis.

Infants C and D were weaned off oxygen prior to initial hospital discharge and had no ongoing respiratory problems. Infant B was intubated for 28 total days and received oxygen via CPAP, oxyhood or nasal cannula throughout his hospitalization. He required supplemental oxygen, diuretics, and aerosol treatments up until and after discharge.

All 3 infants were discharged home on complete oral feeds of transitional or preterm milk-based formula, without added thickeners. Infants B and D were discharged home on anti-reflux medications and only infant B equipped with a home apnea monitor.

As an outpatient, Infant B developed failure to thrive with worsened symptoms of gastro-esophageal reflux and an increased need for respiratory support. A modified barium swallow study as an outpatient showed evidence of aspiration of thin liquids. He, therefore, underwent surgery for a Nissen fundoplication and gastrostomy tube placement, and poor left vocal fold movement was noted during intubation for surgery. He was readmitted to the hospital on 3 separate occasions for respiratory and feeding problems and continued to be dependant on tube feedings for nutrition at 2 years of age. The family chose to have no ENT follow-up, though his hoarse voice continued through his 2 year developmental follow-up visit.

Family "3" was a set of triplets born at 24 weeks gestation.

All three infants required surgical closure of a PDA, after receiving indomethacin, secondary to feeding intolerance, hypotension, and decreased renal perfusion. Ages at surgery were 24 (A), 23 (B) and 20 (C) days of life.

Triplet A was intubated for 28 days, required 25 days of CPAP and 12 days of supplemental oxygen via nasal cannula. At 97 days old, a weak cry and poor progression to oral feeds was noted by our speech therapist, prompting an ENT evaluation that revealed left vocal fold paralysis. A modified barium swallow study showed aspiration of thin liquids and she was eventually discharged home on nasogastric tube feedings of maternal breast milk, anti-reflux medications, and home monitor. However, because of poor growth, increased symptoms of gastro-esophageal reflux, and frequent monitor alarms, she was re-admitted for elective gastrostomy tube placement soon after discharge. At 18-month follow-up, she was taking no feeds by mouth and had acquired an oral aversion. In addition to her persistent symptoms of gastro-esophageal reflux, she also required inhaled steroids and bronchodilator aerosol treatments for reactive airway disease.

Triplet B remained intubated for 43 days and subsequently required another 10 days of CPAP and continued supplemental oxygen via nasal cannula through discharge. ENT consult was obtained when his cry remained weak and feeding problems persisted, with frequent oxygen desaturations during swallowing. Flexible laryngoscopy revealed left vocal fold paralysis at day of life 75. A modified barium swallow study was obtained due to an inability to progress with oral feeds, which confirmed aspiration of thin and thick liquids. Though he was initially discharged home on nasogastric tube feeds of fortified maternal breast milk, anti-reflux medications, supplemental oxygen, and home monitor, he subsequently had to be readmitted four months later for elective gastrostomy tube placement, secondary to poor weight gain and oxygen desaturations with feeds. At 18-month follow-up visit, he continued to require partial

gastrostomy tube feeds. He developed reactive airway disease shortly after initial NICU discharge as well, necessitating inhaled steroid and bronchodilator treatments.

Triplet C was also observed to have oxygen desaturations when transitioning to oral feeds and an ENT consult was obtained. Flexible laryngoscopy revealed good vocal fold function bilaterally without any evidence of paralysis. In comparison to his siblings, Triplet C successfully learned to feed by mouth and was discharged home on full oral feeds of maternal breast milk fortified with formula powder. He was electively readmitted once for an inguinal hernia repair, but otherwise had no subsequent hospital admissions or specialist referrals. He, unlike his siblings, did not develop reactive airway disease.

Table 1 describes the infants included in this case series.

Discussion

Despite current discussion among neonatologists as to the clinical importance of a persistent PDA in extremely preterm infants, medical and surgical treatment has remained the standard of care.[6] Surgery is the method of closure in approximately 25% of extremely low birth weight infants, as reported by the National Institute of Child Health and Human Development Neonatal Research Network. [7,8]

Multiple publications have highlighted the merits of surgical closure of a symptomatic PDA and its overall short and long-term safety and efficacy. However, studies have also demonstrated that paralysis of the left vocal fold remains a complication of this type of surgery. Clipping of the duct is the preferred method at our institution, as it is in many other neonatal units, because of the overall decreased surgical dissection time and reduced risk of damage to the great vessels, but this method has also been reported to bear a higher incidence of nerve damage and subsequent LVFP compared to ligation. [9] The two surgical techniques have not been compared in a randomized controlled trial.

Pereira et al. studied the clinical course of neonates with vocal cord paralysis after PDA ligation. They reported an increased duration of mechanical ventilation, stridor, and feeding difficulties requiring tube feedings in patients with LVFP versus those who did not develop this complication post-operatively. Upon follow-up at 9 months of life, all patients had satisfactory compensation by the right vocal cord and none of the patients had sustained feeding difficulties, though many continued to demonstrate a weak cry. Their results showed an 11 % prevalence of LVFP in patients undergoing PDA ligation.

We have found the prevalence of vocal fold paralysis and related complications to be considerably higher amongst ELBW infants undergoing surgical PDA closure. Our 3-year chart review showed 22 of 55 (40%) of surviving ELBW infants who underwent surgical ductal closure resulted in left vocal fold paralysis. We also observed that a persistent inability to protect their airway during swallowing often negatively impacted their feeding abilities long-term, with 14 of the 22 (64%) of these infants requiring gastrostomy tube placement. Furthermore, chronic microaspiration may contribute to the development of reactive airway disease, as 19 of the 22 (86%) required the need for chronic aerosolized treatments.

Initial diagnostic visualization of the vocal folds with the flexible fiberoptic endoscope will reveal limitation in abduction of the true vocal fold from the midline with deep inspiration. The vocal fold may be resting in the midline position, facilitating right-sided vocal fold compensation with a misleading strong cry and absence of aspiration. The vocal fold may be resting in the paramedian position, in which case the right true vocal fold compensation may be incomplete, resulting in a hoarse cry with or without aspiration. If the vocal fold rests in the

lateral position, then the probability of compensation for vocalization or for feeding purposes is very low. S1. (Figure/Video 1) demonstrates LVFP by fiberoptic exam.

Most recoveries of function will occur by two-years from the date of injury. Recovery results in cases in which the paralysis was due to traction during the surgery rather than ligation of the recurrent laryngeal nerve. A short time to recovery suggests simple, brief disruption of axonal flow. A longer time to recovery suggests that the axons were disrupted by shear forces but the overall nerve structure remained intact, and the time to recovery was the time required for the axons to re-grow.

In addition to visualization of the vocal cords by an otolaryngologist, most of our infants with LVFP undergo a modified barium swallow study. This videofluoroscopic study is performed after a clinical suspicion of aspiration or laryngeal penetration is raised; however, some infants present with “silent” aspiration and have no clinical signs suggestive of their inability to protect their airway during swallowing. Thickening of formula or breast milk with crushed baby cereal or other commercial thickening product can often prevent penetration into the laryngeal vestibule and aspiration.

In this case series, we presented 3 families of multiples of ELBW infants with at least one sibling developing LVFP following surgical PDA closure. We propose that by using the siblings as a comparison, while not necessarily an exact match in terms of acquired morbidities, we were able to compare outcomes of this complication by holding genetic backgrounds, social structure and follow-up constant. Our aim was to demonstrate that infants with vocal fold paralysis may present with feeding difficulties and require further medical and surgical interventions due to this complication. We would like to highlight this significant long term morbidity which should be taken into consideration when evaluating infants for surgical vs. medical closure of their PDA.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Patient characteristics

	Birth Weight (g)	Method of PDA Closure	Paralyzed Vocal fold?	Documented Aspiration?	NICU LOS (days)	Days on O2 or d/c home	RAD?	Required G-tube?
Family "1" (GA 26wks)								
Infant A	970	Spontaneous	No	No	120	41	No	No
Infant B	890	Indo treatment	No	No	109	90	No	No
Infant C	895	Surgery	Yes	No	116	68	Yes	No
Family "2" (GA 25wks)								
Infant A (died)								
Infant B	810	Surgery	Yes	Yes	213	d/c	Yes	Yes
Infant C	810	Indo treatment	No	No	117	54	No	No
Infant D	570	Indo prophylax	No	No	120	87	No	No
Family "3" (GA 24wks)								
Infant A	650	Surgery	Yes	Yes	199	65	Yes	Yes
Infant B	710	Surgery	Yes	Yes	127	d/c	Yes	Yes
Infant C	750	Surgery	No	No	106	70	No	No