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Review

Nutrition of preterm infants with bronchopulmonary dysplasia after hospital discharge – Part II

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Abstract

Preterm infants with bronchopulmonary dysplasia often present with severe growth failure at discharge from the neonatal intensive care unit. Catch-up growth accelerates after hospital discharge, nevertheless, feeding problems may need a specialized approach. Following the revision of the scientific literature on the most relevant aspects on nutrition of patients with bronchopulmonary dysplasia after hospital discharge in Part I, in this article the Authors present and discuss important issues such as catch up growth, swallow dysfunction, gastroesophageal reflux, and how to improve feeding competences.

Keywords

Bronchopulmonary dysplasia, catch up growth, gastroesophageal reflux, swallow dysfunction, very low birth weight preterm infant.

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Introduction

Lung growth and maturation continue after hospital discharge and bronchopulmonary dysplasia (BPD) patient's nutritional requirements and feeding problems will need a specialized multidisciplinary approach during follow-up.

Following the revision of the scientific literature on the most relevant aspects on nutrition of BPD patients after hospital discharge in Part I, in this article we are discussing such issues as:

- Common problems (catch up growth, swallow dysfunction, gastroesophageal reflux).
- How to improve feeding competences?

Catch-up growth

Preterm infants accrue significant nutrient deficits during hospitalization, and at the time of discharge most very low birth weight preterm infants have moderate to severe growth failure [1]. Infants with significant morbidities and infants with extremely low birth weight have more severe growth failure since they regain birth weight at a later age, and they gain weight more slowly. Catch-up growth accelerates after hospital discharge.

The follow-up and treatment of BPD patients after hospital discharge is better performed in a specialized multidisciplinary setting, both because the child is most often an extreme preterm, and because he or she may need respiratory support and respiratory medications. Tracheostomy and ventilatory support may be needed in some patients, and a significant number will need oxygen at home for a variable period, along with bronchodilator therapy, inhaled steroids, fluid management, nutritional support, respiratory physiotherapy, infection preventive measures including vaccination against influenza and pneumococcus, and prophylaxis of respiratory syncytial virus infection with monoclonal antibody Palivizumab [2].

BPD and long term oxygen use have not been consistently found as predictors of poor growth [3]. This is in contrast to the observation of BPD as a contributing factor in poor growth of the hospitalized very low birth weight preterm infant prior to initial discharge [4, 5].

Different studies on BPD patients showed delayed catch-up growth for weight, height and head circumference at different moments of evaluation [6-11]. Two studies published during the eighties showed that by two years of age 20-35% of BPD patients presented a height that was below -4SD (standard deviation), 66% presented a weight below -4SD, and 28% presented a head circumference below -4SD [9, 10]. A different study, in 1995, including 406 patients, revealed that BPD patients aged between eight and ten years presented lower Z scores than no BPD patients, -0.4 (± 1.3) for height, -0.5 for weight (± 1.3), and -1.4 (± 1.3) for head circumference [6]. However, after controlling for possible confounders (using analysis of covariance), no significant differences were demonstrated between the two groups.The authors concluded that significant differences were noted between children with and without BPD for weight and head circumference but not height, and when possible confounders were taken into account, the differences were no longer appreciated. Thus, the previously reported poor growth in children with BPD may have been related to other factors and not necessarily to BPD.

Even if the majority of the studies show an inconsistent catch-up growth among BPD patients, the prevalence and degree of malnutrition are difficult to evaluate in this population of patients [11]. From one study to another, the results are not consistent, highlighting the difficulty in finding the best criteria for the assessment of malnutrition for these children [11]. Some studies use centiles for the expression of height, weight and head circumference, other studies use Z scores of height, weight and head circumference, or even Z score of weight/height. The disparity among studies also results from their antiquity, the definitions used for BPD, and the fact that the populations of preterm were different [11]. Poor growth may be the result of a complex interaction of a number of factors, including inadequate nutrition, morbidities affecting energy requirements, endocrine abnormalities, central nervous system insults, medications that may affect protein and energy metabolism, and others. While inadequate nutrition itself may impact brain maturation and growth during a vulnerable period, it may also more broadly affect health by compromising other organs maturation, impairing immune function, and diminishing reserves for recovery from chronic or intercurrent illness or surgery.

More recent published literature refers that, in preterm neonates, catch-up and pulmonary alveolar growth occur during the first two years of life; 10% to 25% of preterm infants with BPD are under-nourished after two years of age, and 30% to 60% of them also suffer from persistent airway obstruction, hyperinflation and bronchial hyperreactivity [12]. Nutritional status at the age of two years in children who had BPD in infancy influences nutritional and pulmonary outcomes in childhood [13].

Growth retardation, delayed bone mass accretion and delayed catch-up growth in infants who develop BPD have been reported in some studies [14-17].

The etiology of this delayed growth performance is multifactorial including the limited nutrient intake because of restricted fluid intake, feeding intolerance and/or extended parenteral nutrition, extremely prematurity, and the interference with growth processes by exogenous steroids, when prescribed to enhance pulmonary function [18, 19].

Dexamethasone, commonly used in extreme premature infants as a therapy to promote earlier weaning from the ventilator, has the negative effects of a potent steroid on growth and mineral and bone metabolism [20]. Ward et al. verified that dexamethasone was associated with abrupt growth restriction without recovery by term age [19]. Although the infants included in the study were born appropriate for gestational age (birth weight: 782 ± 185 g, gestational age: 25 ± 1 wk), length fell to < 5% percentile during dexamethasone treatment with only 1/17 infants demonstrating significant catch-up (> 5% percentile) by term age. Weight fell to < 5% percentile in 13/17 infants during dexamethasone and only 2/13 infants crossed above the 5% percentile by term age [21].

Postnatal steroids induce abnormalities in bone metabolism by interfering with one or more aspects of the growth hormone-insulin-like growth factor (GH-IGF-1) axis [19]. Bone cell activity is suppressed during steroid therapy, as indicated by reduced circulating osteocalcin (a bone-formation marker) and N-telopeptide (a bone resorption marker), although both markers rose by 10 days after the completion of dexamethasone therapy [19]. Even tapered dosing regimens of dexamethasone are associated with restriction in weight, length and head circumference growth and abnormalities in biochemical markers of bone turnover [18, 20]. The steroid-induced abnormalities observed in extreme premature babies were reproduced in the early weaned piglet model, thus proving that the restrictions in growth and bone are a result of the steroid drug and not only a result of the lung disease or extreme prematurity [21-23].

Preterm infants, especially those with BPD, have a multitude of feeding problems and it is reasonable that the restricted growth could result from inadequate nutrient delivery rather than a direct effect of the steroid drug. Two prospective descriptive studies demonstrated that nutrient intake of dexamethasone-treated infants was not different either during or after dexamethasone or when compared to no treated infants matched for size and gestation [18, 19]. Thus, the catabolic effects of dexamethasone on protein metabolism and its interference with the GH-IGF-1 axis are the more likely explanations for the immediate influence of the drug on normal development [24, 25]. Administration of GH with or without IGF-1 only partially attenuated the steroid-induced abnormalities in growth and bone metabolism [21]. The role of nutrition in attenuating the negative effects of steroid drugs on growth processes during drug administration or as rehabilitation after the completion of drug treatment needs to be investigated.

Swallow dysfunction

Infants with BPD can experience significant feeding difficulty, possibly secondary to tachypnea interfering with coordination of sucking. This is especially problematic as these infants often have increased metabolic demands and caloric requirements imposed by chronic hypoxia [26]. Indeed, feeding difficulties, decreased nutrient intake, decreased fluid tolerance secondary to BPD, and increased metabolic needs in infants with BPD often result in long-term reduced rates of growth [9, 27]. Palatal grooves caused by prolonged intubation may also make it difficult to achieve a proper seal between the tongue and palate [28].

Successful feeding in infants with BPD is further compromised by acute oxygen desaturation during feedings [29, 30]. Craig et al. have noted that the breathing patterns during feeding in infants with BPD did not demonstrate the striking regularity seen in control term infants [31]

Gewolb and co-workers analyzed the rhythmic differences during feeding in infants with BPD and post menstrual age matched control group without BPD, from initiation of bottle feeding until discharge, with simultaneous digital recordings of pharyngeal and nipple (teat) pressure [32]. Unlike the control group, there was no significant correlation between post menstrual age and stability of suckle rhythm, aggregation of suckles or swallows into runs, or length of suckle runs. Comparing those infants > 35 weeks' post menstrual age, the group with BPD had significantly decreased stability of suckle rhythm, decreased aggregation into suckle runs, and decreased length of suckle runs. Percentage of swallows in runs was also decreased in the cohort with BPD, as was length of swallow run. Thus, in infants with BPD, anticipated maturational patterns of suckle and swallow rhythms that normally occur in preterm infants did not occur. Delay in attainment of stable suckle and swallow rhythms

in preterm infants, especially after 35 weeks' post menstrual age, may predict subsequent feeding and neurological problems [32].

Infants with BPD do not follow predicted maturational patterns of suck-swallow rhythmic integration [33]. Individual rhythms of suck, swallow, and respiration are disrupted in preterm infants with BPD. Integration of respiration into suck-swallow efforts is critical for establishing coordinated suckle feeding. One study quantitatively assessed the coordination of respiration and swallow in infants with and without BPD [34]. Participants were studied at postmenstrual age of 32 to 40 weeks and postnatal age of 2 to 12 weeks using digital recordings of pharyngeal pressure, nasal thermistor flow, and thoraco-abdominal plethysmography. Apnoeic swallows were significantly increased after 35 weeks in infants with BPD compared with non-BDP infants, as were swallow-breath phase relationships involving apnoea. The BPD cohort also had significantly higher swallow-breath coefficients of variation and breath-breath coefficients of variation than non-BPD infants, indicating less rhythmic coordination of swallowing and respiration during feeding. Results emphasize the need for frequent rests and closer monitoring when feeding infants with respiratory compromise. Quantitative assessment of the underlying rhythms involved in feeding may be predictive of longer-term feeding and neurological problems [34].

Cervical accelerometer with digital signal processing can identify signals that are consistently associated with swallowing during feeding of infants. It is shown that these signals, called initial discrete sounds, become more uniform with advancing postmenstrual age in healthy preterm infants. In a study by Reynolds and co-workers there was no significant correlation between variance index and postmenstrual age for the BPD cohort [35]. The variance index of infants with BPD was significantly different from that of infants without BPD. While the variance index for the healthy control group decreased with advancing postmenstrual age, the variance index of the BPD group increased with advancing postmenstrual age.

Oral feeding has been reported to compromise breathing among preterm infants with BPD during hospitalization or shortly after discharge. However, limited information is available concerning whether preterm infants with BPD remain vulnerable to feeding and growth insufficiency after a longer term of follow-up. The study performed by Li-Ying and co-workers examined the effect of severity of BPD on pulse oxygen saturation (SpO_2) during feeding and growth in very low birth weight preterm infants during infancy, at 2, 4, and 6 months of corrected age [36]. Infants with severe BPD showed significantly lower mean levels of SpO_2 during feeding at 2-6 months corrected age. Those with severe BPD further exhibited higher rates of growth delay (weight < 10^{th} percentile) throughout the study period. Among VLBW infants, severe BPD had an adverse relation with subsequent weight measures after adjustment for medical and demographic confounding variables.

Gastroesophageal reflux

Premature infants have an increased risk of developing gastroesophageal reflux. Having an infant or child with gastroesophageal reflux can be extremely overwhelming for parents and families. Reflux may be painful, causing infants to cry constantly, refuse to eat, spit up frequently, and sleep poorly. The daily routine of giving medication, dealing with the constant vomiting and special feedings, and the frequent doctor visits can be exhausting. If gastroesophageal reflux is left untreated, long-term complications such as feeding disorders, inadequate weight gain, narrowing of the oesophagus, and damage to the tissue in the oesophagus (called Barrett's syndrome) can develop. For non-complicated reflux, no intervention is required for most infants. Effective parental reassurance and education regarding regurgitation and lifestyle changes are usually sufficient to manage infant reflux. Sandifer syndrome, apnea and apparent life-threatening events are the extraesophageal manifestations of gastroesophageal reflux in infants. Pharmacotherapeutic agents used to treat gastroesophageal reflux encompass antisecretory agents, antacids, surface barrier agents and prokinetics. Currently, North American Society for Pediatric Gasroenterology, Hepatology and Nutrition (NASPGHAN) and European Society for Paediatric Gastroenterology, Hepatology and Nutrition (ESPGHAN) practice guidelines concluded that there is insufficient evidence to justify the routine use of prokinetic agents. Omeprazole may be used for short-term treatment of gastroesophageal reflux with erosive esophagitis in infants aged from 1 to 12 months. Although Nissen fundoplication is now well established as a treatment option in selected cases in children, its role in neonates and young infants is unclear and is only reserved for selective infants who did not

respond to medical therapy and have life-threatening complications of gastroesophageal reflux [37, 38].

Infants with BPD have a higher incidence of gastroesophageal reflux [39]. Infants with gastroesophageal reflux may suffer from frequent aspiration, pneumonia, apnoea, and failure to thrive [40]. Pulmonary deterioration, failure to growth, and refusal to eat may herald gastroesophageal reflux in preterm neonates [37]. Prolonged gastric tube use and feeding intolerance increase the risk for gastroesophageal reflux in low birth weight infants with BPD [40].

In preterm infants with BPD, transient lower oesophageal sphincter relaxations are the predominant mechanism underlying gastroesophageal reflux, and oesophageal clearance mechanisms are fully functional, which is similar to that seen in healthy preterm infants [41]. Fundoplication and gastrostomy is effective in facilitating growth and feeding in addition to decreasing oxygen requirements in infants with severe BPD and gastroesophageal reflux [42]. Randal Giuffre et al. analysed the effect of fundoplication and gastrostomy in patients with BPD and gastroesophageal reflux [42]. The postsurgical respiratory response was observed to be a rapid decrease in oxygen requirements and an absence of further aspiration episodes. A mean decrease of 0.14 in fractional inspired oxygen concentration was noted by 30 days postoperatively, and by 180 days the decrease in fractional inspired oxygen concentration was 0.22. All infants were fed by gastrostomy by postoperative day 4, with no evidence of clinical reflux. The nutritional response was noted to be an increase in growth velocity with increasing age (i.e., catch-up growth) and ease of feeding. At both 30 and 180 days postoperatively, the mean growth velocity was more than double the preoperative growth velocity. In addition, ease of postoperative feeding reduced the nursing care requirements and allowed earlier discharge from hospital. Nissen fundoplication is a feasible, effective and safe operation in severe gastroesophageal reflux, and may be used when medical treatment fails [43].

How to improve competences?

Feeding problems depend on the severity of BPD. Safe and successful oral feeding requires appropriate maturation and coordination of sucking, swallowing and respiration. Infants with BPD often have difficulty achieving coordinated suckle feeding, possibly secondary to tachypnea interfering with suck coordination, which limits the use of bottle or breast feeding initially [32, 44, 45]. These children have low sucking pressure and sucking frequency, short sucking burst duration, high respiratory rate, greater decrease in oxygen saturation, long deglutition apnea and low feeding efficiency [44, 46]. Optimal oral feeding should occur when a regular rhythmic relation exists between suck, swallow and respiration; however, infants with BPD do not seem to follow predicted maturational patterns of suck-swallow rhythmic integration [32].

A multidisciplinary approach is essential for therapeutic success. If aspiration is suspected or risk of aspiration is a factor, instrumental assessments of swallowing, such as video fluoroscopic swallowing assessment or fiber-optic endoscopic evaluation of swallowing may be necessary following the clinical evaluation [47].

Sensory feedback is essential. Nonnutritive sucking, for example with a pacifier or a finger, provides benefits to the ability of oral feeding skills. This therapy improve feeding tolerance, accelerates the transition from tube to oral feed, increase weight gain, improve breastfeeding scores and increase gastric motility [44]. Concomitant stimulation of oral-motor skills should occur in all tube-fed patients to prepare them for eventual feeding by mouth when there is no longer a risk of aspiration and swallowing functions have matured [45]. At this stage, gustatory stimuli (with a little of milk or other sugary flavors) may be used for the child to come into contact with the taste, smell and texture [46]. As respiratory status improves bolus feedings may be initiated [45]. After the introduction of solid foods, it is important to progress in consistencies and textures [48]. The delay in the introduction of solid foods can result in food refusal and occasionally the development of food aversions. Infants with anatomical or physiologic abnormalities present a higher risk of having difficulty establishing and maintaining oral feeding due to inability to initiate oral feedings within deadlines appropriate to the age [47]. There are many therapeutic techniques that can be used depending on each clinical case. Strategies such as postural techniques intended to improve safety and efficiency while allowing for oral feeding [47, 49]. Some children show significantly improved oral skills and timing of swallowing with posture and position changes [47, 50]. The environment should be quiet without unnecessary stimulation because these children

are already easily overwhelmed by tactile, visual, auditory and kinesthetic stimuli. Sleep cycles should be respected whenever possible [45]. Other strategies include diet modifications (variations in texture, tastes and temperature of food), changes in feeding routine or changes in presentation of food. Oral sensorimotor intervention is a method whose benefits are still inconclusive but promising that involves techniques that are directed toward improving a child's ability to accept, manipulate and swallow foods successfully. This may include techniques like facial massage, vibration, tapping of oral musculature, stroking the face, the use of a brush or other kinds of stimulation in order to desensitizing and improving function [47, 48]. Oral-motor dysfunction during feeding should be recognized as soon as possible. The parents/ caregivers should be informed about maneuvers to improve neuromuscular coordination during feeding such as thickened feeds [45].

It is essential that the treatment sessions also involve parent and caregiver education in every meeting, as well as offer home programs and suggestions for how to work with children at home on a daily basis [47].

Declaration of interest

The Authors declare that there is no conflict of interest.

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