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ORIGINAL ARTICLE

Do preterm infants with Bronchopulmonary dysplasia have a unique postnatal weight gain pattern?

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ABSTRACT

Objectives: To investigate the weight gain pattern of preterm infants with bronchopulmonary dysplasia (BPD) during the hospital stay using weekly weight assessment methods.

Methods: This single-center, retrospective, cohort study was carried out in Zekai Tahir Burak Maternal Health Education and Research Hospital between 2014 and 2018. One hundred fifty-one preterm infants <32 weeks of gestation and <1500 g of birth weight with BPD were compared to 251 babies without BPD in terms of weekly weight gain, standard deviation score (SDS), and fall in weight SDS till discharge.

Results: Mean body weight was significantly lower in babies with BPD in all weeks except postnatal week (PW) 8. The groups had similar daily weight gain between birth and discharge ($p = .78$). Infants with BPD had lower weight SDS on postnatal day (PD) 14 and 21, and discharge, however similar on PD 28. The fall in SDS between PW 4 and discharge was significantly higher in the BPD group. Infants with BPD had higher fall in weight SDS between birth and discharge ($p = .022$). Discharge weight SDS was associated with gestational age and weight SDS on PW 4 in the whole cohort.

Conclusion: Infants with BPD showed a unique and unsteady pattern of growth compromise during the NICU course, most explicitly in early postnatal life and between PD 28-discharge. Future studies should consider not only the early postnatal life but also the period after four weeks of life till discharge to design an optimal nutrition strategy and decent growth for preterm infants with BPD.

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Chronic lung disease of prematurity; growth; early postnatal life

Introduction

Bronchopulmonary dysplasia (BPD) remains the most common morbidity of extremely preterm infants albeit with ameliorated care and minimal invasive handling implemented from the first breath onward [1]. Severe lung injury was speculated to evolve as a multifactorial process likely triggered by some yet unknown intrauterine mechanisms [1]. Evidence points out the undoubtful causality link between intrauterine growth compromise and BPD [2]. Besides, postnatal suboptimal nutrition was speculated to disrupt alveolarization in extremely preterm babies whose lung development is still at the saccular-alveolar stage [2]. A considerable number of cohort studies exist addressing the unignorable relationship between calorie/protein intake and growth in babies with BPD. Nevertheless, it remains controversial whether it is a real cause-and-effect relationship or just a marker of BPD/critically ill state [3–8]. Several studies sought a plausible association between the development of BPD and postnatal growth restriction [9–11]. First and foremost, the energy requirement of critically ill babies with evolving BPD is undoubtfully higher than the others [4]. However, it seems unlikely to calculate the exact energy and nutrient requirements and plan a tailored nutrition prescription for this high-risk group. Another point that has yet to be clarified is the timing, amount, and type of nutrition that should be provided to reduce the incidence of BPD [3,12]. Last but not

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least, current nutrition guidelines have yet commented on neither a nutrition itinerary nor a specific nutrient that should be supplied in a certain amount to stop or treat BPD.

Along with the evidence of the fundamentality of optimal nutrient provision for proper lung development initiated in fetal life, the ideal postnatal growth pattern of preterm babies remains questionable [13,14]. Currently used terminologies for growth faltering lack accuracy to opt for which growth pattern is abnormal [14]. Moreover, the lack of an ideal growth monitoring tool for preterm infants and the variability of growth compromise terminology make the assessment of the association challenging. A couple of studies suggested a decent mean in-hospital weight gain despite a high percentage of growth restriction based on the current definitions [9,10]. This was striking in the study of Natajaran et al. revealing an extrauterine growth restriction (EUGR) defined as <10% in more than half of the BPD cohort during the hospital stay despite an optimal mean weight gain of 30 g/kg/d [9]. This study validates the lack of reliability of currently used "abnormal growth" terminologies. Moreover, evidence is prompting clinicians to seek new definitions given the controversial benefit of "EUGR" for the prediction of adverse outcomes. Another point is the uncertainty about how to act in the case of EUGR given the possibility of being normal because of the preset growth potential of the baby [11]. On the other hand, evaluation of growth at only term gestational age or discharge seems useless and insufficient given the unsteadiness of growth during the NICU stay. Considering these gaps, we opted to carry out a study in preterm infants to find out the weight gain pattern of preterm infants from birth to discharge.

Methods

We conducted this study in Zekai Tahir Burak Maternal Health Education and Research Hospital. Our retrospective data included preterm infants <32 weeks of gestation and <1500 g of birth weight born between January 2014 and December 2018. Records of all inborn preterm babies who survived beyond 48 h were accessed. Whilst those with BPD constituted the study group, babies without BPD were incorporated into the control group. Exclusion criteria were <23 weeks of gestation, death <48 h of life, and major congenital/chromosomal anomalies. The institutional ethics committee approved the study. Informed written or verbal consent was obtained from the families.

Diagnosis of BPD was based on the classification of Jobe and Bancalari [15].

Maternal characteristics comprised of delivery method, antenatal steroid, preterm premature rupture of membranes (PPROM), chorioamnionitis, preeclampsia, and multiple pregnancies. Prenatal diagnosis of intrauterine growth restriction was gathered from the maternal obstetrics files. Infant characteristics included APGAR scores, the requirement for advanced resuscitation in the delivery room, small for gestational age, surfactant requirement, hemodynamically significant patent ductus arteriosus (hsPDA), intraventricular hemorrhage (IVH), late-onset sepsis (LOS), necrotizing enterocolitis (NEC), length of hospital stay, and mortality.

Postnatal weight was recorded daily by bedside nurses. We calculated the growth velocity *via* a two-point birthweight model. Weekly weight percentiles and standard deviation scores (SDS) based on the 2013 Fenton growth chart were plotted and calculated. The fall in SDS between the two points was calculated weekly. Post-discharge weight data were obtained from the outpatient clinic records. Duration of nothing per oral (NPO), time to introduce EN, availability of mom's milk, day to reach full enteral feeding and birthweight, duration of PN, feeding intolerance, and presence of mortality before/after full EN were all noted from the files.

All extremely preterm infants were initiated on mother's milk within the first 48 h of life unless there was a contraindication. Trophic feeding was initiated at the earliest with mother's milk by 10–20 ml/kg/d in preterm babies <32 weeks of gestation and sustained 3–5 days. In case of the unavailability of mom's milk, the preterm formula was introduced. Donor milk was unavailable in our country. The daily enteral increment was 20–30 ml/kg at the discretion of the neonatologist. Standard fortification of human milk commenced once the baby reached and tolerated 80 ml/kg/d enteral feeding. However, the fortification was held off under certain circumstances including feeding intolerance, necrotizing enterocolitis, or spontaneous intestinal perforation.

Nine different standard PN solutions that were administered based on postnatal age and birth weight were available in our pharmacy. Electrolyte-free PN solution was provided soonest following the insertion of an umbilical venous catheter. Protein and lipid catering was started by 2–2.5 g/kg/d and 0.5–1 g/kg/d in PN with daily advancement up to 3.5 g/kg/g of protein and 3 g/kg/d of lipid in extremely preterm infants.

Weaning from PN was initiated with gradual tapering of lipid infusion once the baby tolerated 80 ml/kg/d of enteral nutrition, continued with tapering of protein and dextrose once reached 100 ml/kg/d enteral amount.

Table 1. Demographical and gestational characteristics of the groups.

	BPD $n = 151$	Control $n = 251$	p
Gestational age, weeks[a]	27 ± 1.41	28.05 ± 1.75	**<.001**
Birth weight, g[a]	985.5 ± 201.6	1061 ± 243	**.038**
Male, *n* %	26 (54.2)	219 (48.5)	.27
C/S, *n* %	121 (81)	210 (84)	.37
SGA, *n* %	7 (4.2)	39 (15.7)	**.017**
5 min APGAR[b]	7 (2–9)	8 (3–9)	**.004**
Antenatal steroid, *n* (%)	100 (66)	180 (72)	.24
PPROM, *n* (%)	43 (29)	46 (18.8)	.068
Clinical chorioamnionitis, *n* (%)	28 (18.8)	24 (9.5)	.053
Preeclampsia, *n* (%)	7 (4.5)	48 (19.5)	**.004**
Multiple pregnancy, *n* (%)	19 (12.5)	58 (23.2)	.059

Data presented as mean ± SD, median (min-max), or count (percentages).
[a]Mean ± SD. [b]Median (min-max).
Significant *p* values are highlighted. C/S: Cesarean section; SGA: Small for gestational age; PPROM: Preterm premature rupture of membranes.

Table 2. Preterm morbidities and nutrition data of the groups.

	BPD $n = 151$	Control $n = 251$	p
Surfactant requirement, *n* (%)	115 (77)	130 (52)	**.001**
Duration of invasive ventilation, days[b]	14 (0–68)	1 (0–46)	**<.001**
hsPDA, *n* (%)	106 (70.8)	104 (41.6)	**<.001**
Late-onset sepsis, *n* %	78 (52)	73 (29)	**.001**
IVH (>Grade 2), *n* %	25 (16.7)	28 (11.3)	**.001**
ROP requiring treatment, *n* (%)	46 (31)	13 (5)	**<.001**
Length of NICU stay, days[b]	96 (60–183)	55 (1–235)	**<.001**
Duration of PN, days[a]	19.35 ± 12.23	14.65 ± 7.04	**.003**
Day to reach full enteral nutrition[b]	17.5 (7–69)	14 (5–102)	**.003**
Length of NPO[b]	2 (0–18)	1 (0–39)	**.048**
Day to catch-up birth weight[b]	14 (5–33)	12 (2–26)	**<.001**
Feeding intolerance, *n* (%)	91 (60)	141 (56)	.36
NEC (>Grade 2), *n* (%)	3 (1.6)	5 (1.7)	.617

Data presented as mean ± SD, median (min-max) or count (percentages).
[a]Mean ± SD. [b]Median (min-max).
Significant *p* values are highlighted. hsPDA: hemodynamically significant patent ductus arteriosus; IVH: Intraventricular hemorrhage; ROP: Retinopathy of prematurity; PN: Parenteral nutrition; NPO: Nothing per oral; NEC: Necrotizing enterocolitis.

The decision for the transition to full EN was made by the attending neonatologist based on the baby's feeding tolerance and weight gain velocity.

Statistics

Mean or median were used to describe continuous variables while discrete variables were characterized as proportions. Kolmogorov–Smirnov or Shapiro-Wilk test was used to assess the normal distribution of the variables. Normally distributed continuous variables were compared *via* mean and standard deviation along with the comparison of non-normal data *via* median (min-max). Categorical data were summarized as counts and percentages. While normally distributed continuous variables were compared *via* Independent Samples t Test, Mann Whitney U test was used to compare continuous non-normally distributed variables. IBM SPSS Statistics 20 program was used. Two-tailed *p*-value was used with a statistical significance of a p-value $< .05$.

Multivariate logistic regression analysis was performed to reveal independent factors for discharge SDS. In this model, all variables that were significantly different between the groups in the univariate analysis were included.

Results

A total of 402 infants were eligible for the study. Of those, 151 had BPD. The remaining 251 constituted the control group. The gestational age and birth weight were significantly lower in the BPD group (27±1.41 and 28.05±1.75 weeks, 985.5±201.6 and 1061±243 g, in the BPD and control groups, respectively) (Table 1).

The incidence of SGA and maternal preeclampsia was lower in the BPD group ($p = .017$ and .004, respectively). Nonetheless, babies with BPD had higher rates of PPROM and clinical chorioamnionitis even if it was insignificant ($p = .053$ and .068, respectively).

Patients with BPD were more likely to have preterm morbidities namely hsPDA, severe IVH, ROP, culture-proven sepsis, and prolonged invasive ventilation ($p < .05$) (Table 2).

The length of PN was longer in the BPD group ($p = .03$). Day to reach full enteral nutrition and catch-

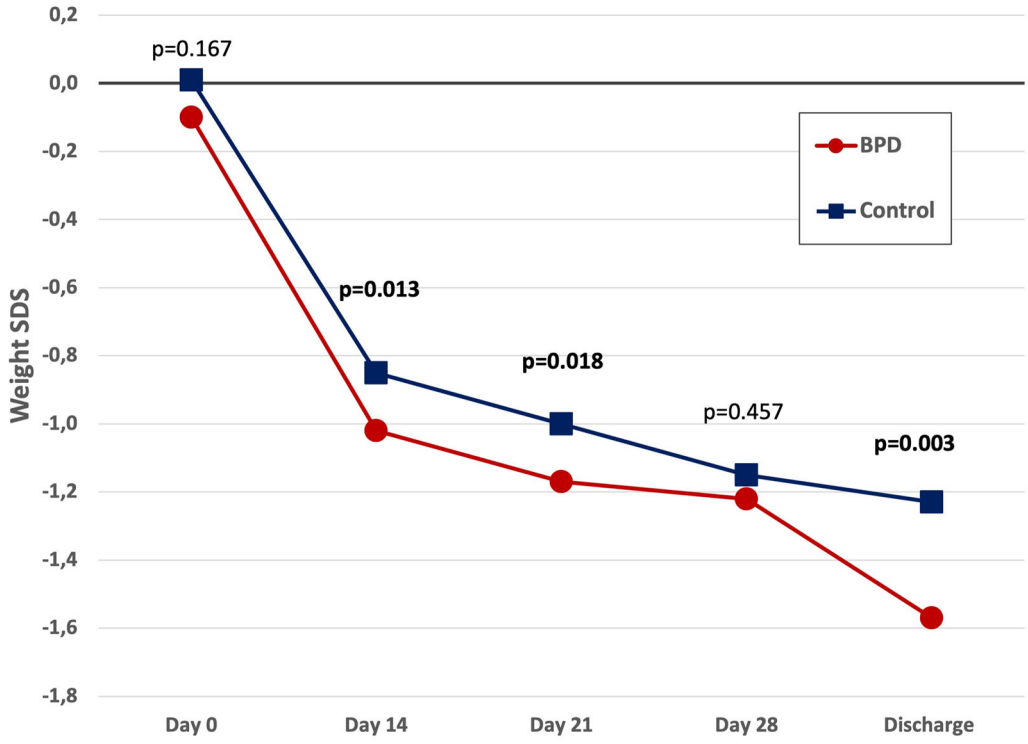


Figure 1. Serial weight SDS of the groups based on the PD.

up birthweight were later in babies with BPD ($p = .003$ and $< .001$, respectively). However, there was no statistically significant difference in terms of feeding intolerance, necrotizing enterocolitis, or spontaneous intestinal perforation ($p > .05$).

Infants with BPD had lower weight SDS on postnatal day (PD) 14–21 and discharge, however similar to PD 28 (Figure 1). The daily weight gain was lower in the BPD group during the first three weeks, statistically similar in PW 4 and 6, but higher in PW 8 (Figure 2). The BPD group had a more fall in weight SDS between birth and discharge ($p = .022$) (Figure 3). When the fall in weight SDS was assessed weekly, the drop between PW 4 and discharge was noted as significantly higher in the BPD group [–0.41 (–2.69 to 1.85) and −0.09 (–2.37 to 1.65) in the BPD and control group, respectively, $p = .012$]. Once we made a subgroup analysis based on the severity of BPD, no statistically significant difference was noted regarding daily weight gain [17.6 (14.1–22.2), and 18 (14.7–22.6) g/kg/d in mild and moderate-severe BPD groups respectively; $p = .68$].

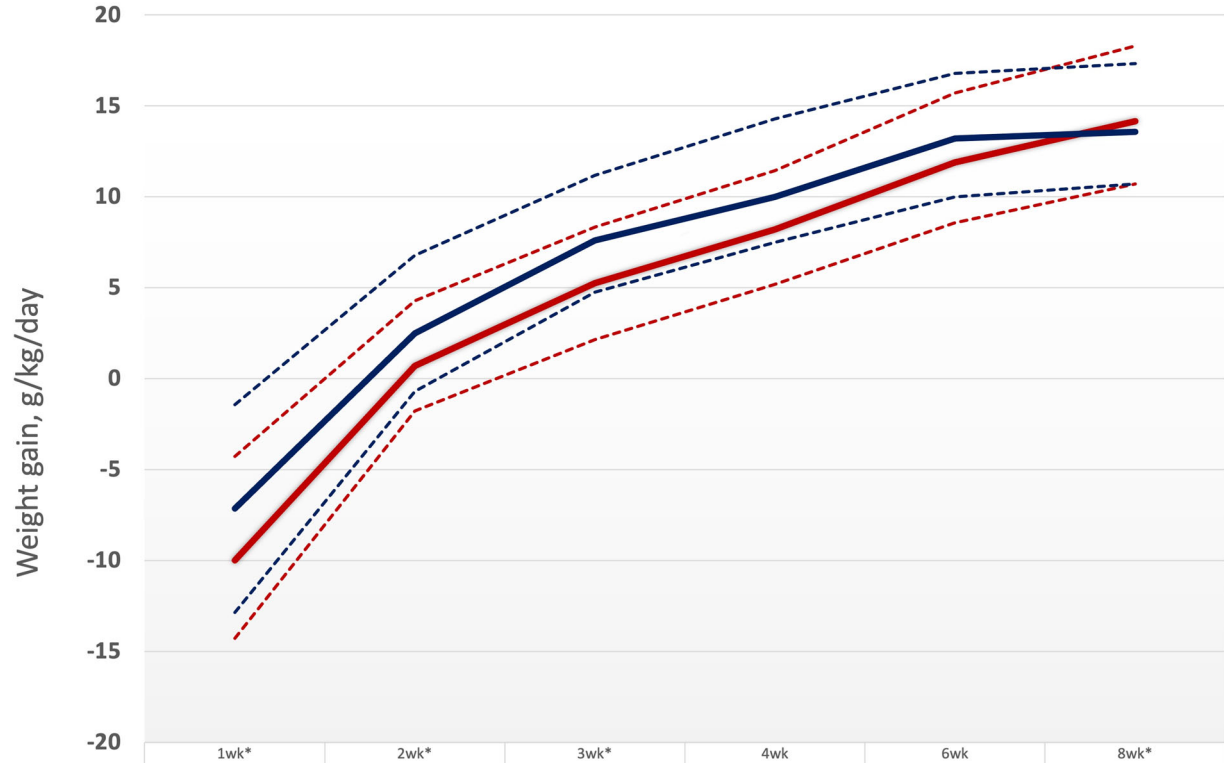
We sub-categorized the infants based on gestational age (23–25, 26–28, and 29–31 weeks) and assessed the weight gain pattern of the subgroups. Our study revealed no statistically significant difference between the infants with BPD and control regarding daily weight gain of the subgroups except for deeper weight loss of 26–28 weeks' infants with BPD compared to the control group in PW 1 ($p = .028$).

Multivariate logistic regression analysis revealed that gestational age (OR: 0.70; 95% CI: 0.55–0.90; $p = .005$), NPO (OR: 1.2; 95% CI: 1.13–1.42; $p < .001$) and lower weight SDS on PW 4 (OR: 20; 95% CI: 10.5–38.3; $p < .001$) were independent risk factors for discharge weight SDS in the whole cohort.

Discussion

This study demonstrates the variability of the weight gain pattern of infants with BPD on weekly weight gain/SDS and fall in SDS basis during the hospital stay. Babies with BPD were found to have a deeper weight loss and restricted early postnatal weight gain, however similar in terms of weight gain following PW 4 and superior weight gain on PW 8. The weekly fall-in SDS was statistically similar except between PW 4 and discharge. Additionally, the fall in SDS between birth and discharge was significantly higher in the BPD group compared to others. Both groups tracked nearly the same trajectory after the initial physiological weight loss.

The coexistence of BPD and growth compromise has been a topic of interest to researchers for decades [9–11,15]. Poor growth following suboptimal nutrition initiated in early postnatal life has been shown many times in babies with BPD [3–7]. However, it remains challenging to draw a concrete conclusion and take new strategies due to the lack of a gold standard



	1wk*	2wk*	3wk*	4wk	6wk	8wk*
BPD 25%	-14,29	-1,79	2,14	5,18	8,57	10,71
BPD 50%	-10,00	0,71	5,24	8,21	11,90	14,15
BPD 75%	-4,29	4,29	8,33	11,43	15,71	18,30
Control 25%	-12,86	-0,71	4,76	7,50	10,00	10,71
Control 50%	-7,14	2,50	7,60	10,00	13,20	13,57
Control 75%	-1,43	6,79	11,19	14,29	16,79	17,32

Figure 2. Daily weight gain of the groups based on the PW. Daily weight gain of the BPD group was significantly lower than the control group on PW 1, 2, and 3 ($p < .05$, .001, and .006, respectively). on the other hand, it was statistically similar on PW 4 and 6 ($p = .09$ and .34, respectively) while the BPD group gained higher weight on PW 8 ($p = .008$).

growth monitoring tool and variability in the growth assessment methods and among different trials done so far. Malikiwi and colleagues reported statistically similar weight SDS during the first 28 days in newborns <28 weeks of gestation with and without BPD [3]. Even though that study provided detailed nutritional data, the small sample size and assessment of the growth at only two points were the limitations.

While a similar decline in weight SDS was noted during the first four weeks of life in babies with BPD and non-BPD, SDS of the BPD group showed a gradual decline after the PW 4 along with recovery in babies without BPD [5]. Despite the similar drop in SDS until PW 4, weight SDS continued to decrease in both groups with a pronounced decline in babies with BPD in our study.

Klevebro and colleagues revealed that babies with 27–30 weeks of gestation and developed BPD experienced a lower growth trajectory on PW 4–5 compared to infants without BPD however, amended growth rates from the PW 7 of life [16]. The mean gestational age of the cohort and the results of this study might be in keeping with ours in terms of weekly weight gain patterns. Along with the lack of growth data on PW 5 and 7, weight gain on PW 8 was statistically higher in the BPD group in our study. Additionally, it was the PW 4 that weight SDS was on dip since birth. Despite the multicenter and large-scale design with a cohort comprising >20 000 infants, the authors couldn't base the results upon a presumptive cause.

A very recent cohort study comprising > 90 000 preterm infants <32 weeks of gestation revealed that babies with BPD had a lower birth weight, and regained weight slowly, but ended up with a higher weight SDS than non-BPD babies at 36 weeks. It seems inappropriate to compare with our study because we assessed the fall in SDS based on discharge values [11]. Nonetheless, a persistent decline of SDS till and even after 36 weeks was resembling our study. The reason for not preferring assessment at 36 weeks corrected is the lack of evidence showing an association between term corrected age and outcomes such as neurodevelopmental benefits. This study emphasizes the necessity of follow-up of growth in preterm babies

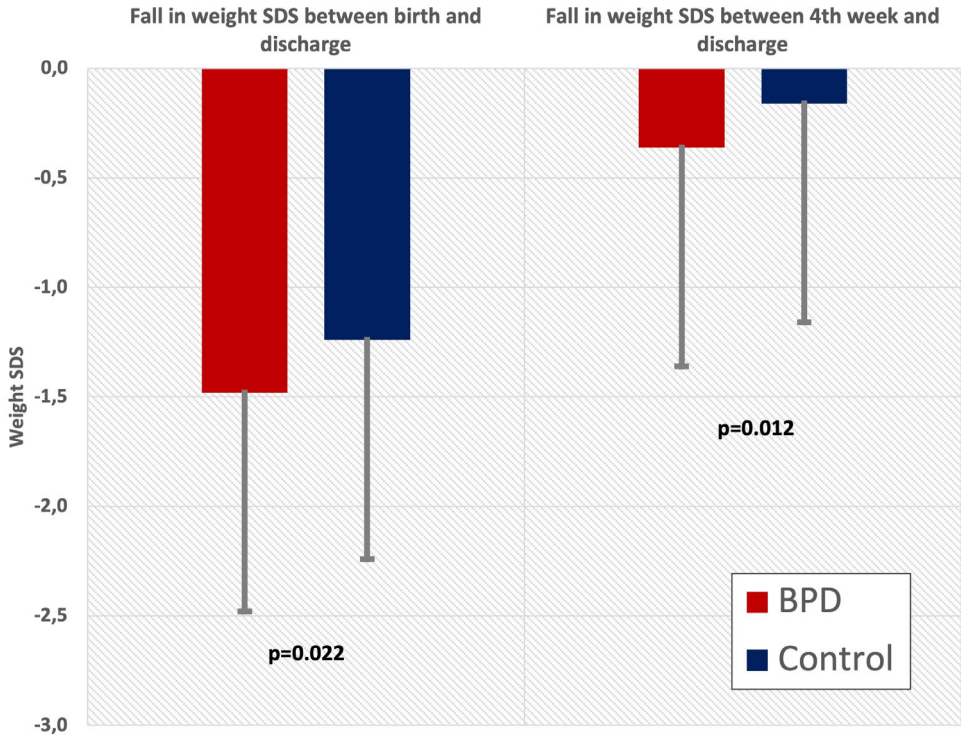


Figure 3. Fall in weight SDS of the groups.

with bearing in mind to track a parallel trajectory instead of enforcing weight gain to recover the birth percentile. As we appreciate from the results, our study groups tracked a similar percentile line following the initial physiological weight-loss period till discharge.

On the other hand, subgroup analysis based on the severity of BPD revealed no significant difference regarding weight gain parameters between mild and moderate-severe BPD groups. Data exists reporting a difference in growth parameters based on the severity of the disease [17,18]. However, the similarity of the weight gain patterns of the subgroups was hypothesized to be due to the low sample size of the BPD group.

Even though a list of advantages was published in favor of standard PN, it might not meet the nutritional requirement of sick preterm babies in evolving BPD process. The tendency of fluid restriction and/or prolonged transition period from PN to EN might account for suboptimal calorie catering with the usage of stock PN solutions. Miller and colleagues categorized the nutrition phases as full PN, transitional PN + EN, and full EN to reveal periods of growth compromise [19,20]. This transitional period was reported to carry a high risk for postnatal growth restriction due to insufficient protein/-calorie intake in that study. We hypothesized that our study group might have been exposed to a longer period of inadequate nutrition given the longer duration of the transitional phase in the BPD group. Eventual prolonged weaning of standard PN might have ended up with a higher fall-in weight SDS presumably due to insufficient protein intake. This transitional phase coincides with PW 2–3 with a lower SDS of the BPD group in our study. So, usage and weaning of standard PN might have ended up with a weight gain compromise in this transitional phase.

However, to our knowledge, a significant fall in weight SDS between PW 4 and discharge was mentioned and emphasized for the first time in the literature. There might be a couple of explanations. Firstly, often standard, and rarely individualized fortification of mother's milk might not have provided optimal calorie and/or protein catering given the considerable percentage of preterm babies on full EN. Indeed, it was emphasized in a recent Cochrane meta-analysis suggesting individualized fortification despite moderate-low certainty evidence [21].

Secondly, we acknowledge that the higher proportion of SGA babies in the control group might be a consequence of selection bias. Thirdly, increased incidence of accompanying morbidities like LOS, ROP, and prolonged invasive ventilation might have hampered optimal nutrition and proper weight gain in the BPD group with lower gestational age and birth weight. The close association of poor postnatal weight gain with ROP was suggested several times by various researchers [22]. Moreover, this period coincides with

corticosteroid treatment in preterm babies with BPD. Indeed, corticosteroids are known to account for protein breakdown along with a couple of trials reporting no unfavorable impact of postnatal steroid usage for prevention or treatment of BPD on growth [21,23]. So, babies might have undergone less protein accretion with steroids in a period of unclear protein intake given the variability of the protein content of breastmilk [24]. So, one should bear in mind the possibility of confounders and eventual misconceptions following the results.

We acknowledge the limitations of our study including the lack of details on the nutrition and calorie data. So, it seems not possible to conclude an association between calorie/protein intake and weight gain. Babies might have received inadequate nutrition likely due to the usage of standard PN and lack of tailored nutrition plans. The nutrition guidelines lack a thorough nutrition strategy for sick preterm and term infants. ESPGHAN Committee on Nutrition recently published a guideline and suggested providing optimized nutrition above presumptive requirements during the recovery phase to compensate for the losses of the catabolic phase and trigger growth [25]. In addition, there is no consensus on the nutrition of evolving and established BPD babies most of whom are critically ill. Our current nutrition practice requires review and amendments, particularly for sick preterm babies.

Another limitation was the assessment of growth only on a weight basis without head circumference and longitudinal growth. Recent studies strongly propose growth monitoring not only with weight gain or SDS but also with head circumference and linear growth [13]. In addition, we preferred not to use EUGR terminology due to weak clinical and prognostic utility. A notable proportion of preterm infants were reported to have EUGR at term equivalent age or discharge [26,27]. Not all growth-faltering cases can be linked to inadequate nutrition, a substantial proportion of these infants might be already growth-restricted at birth [11]. In addition, Greenbury et al. referred mean SDS for birth weight as lower than the reference data including babies born between 1983 and 1993 indicating a higher degree of IUGR [11]. So, a notable portion of these babies might have been mislabeled even though they tracked preset growth potential. Last but not least, one should bear in mind the possibility of non-nutritional growth impairment reasons during follow-up. Besides, weekly growth assessment till discharge with various tools were the strengths of our study. Thus, we noted a significant relationship between the drop in SDS between PW4-discharge and BPD for the first time. This fall in SDS after day 28 could be indicating the lack of optimal nutrition strategies not only for the first weeks of life but also during the full enteral nutrition phase.

In conclusion, our results validate the formerly referred robust association of BPD and growth compromise and the unsteady growth pattern during the NICU course presumably due to the prolonged transitional phase and inadequate nutritional intake. Large-scale trials are warranted to elucidate a nutrition strategy tailored for babies with BPD to improve growth. Moreover, future studies should focus not only early postnatal period but also on following the first 28 days of life to improve the nutrition and eventual growth outcomes in very preterm babies.

Disclosure statement

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