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Evaluation of the Growth of Preterms Born in 2001–2002 During Adolescence

2001–2002 Yılında Doğan Prematüre Bebeklerin Adölesan Dönemde Büyümelerinin Değerlendirilmesi

Kübra Baskın[1,2], Sultan Kavuncuoğlu[2]

[1]Department of Pediatric Immunology and Allergy, Gazi University, Faculty of Medicine, Ankara, Türkiye
[2]Department of Pediatrics, University of Health Sciences Türkiye, Kanuni Sultan Süleyman Training and Research Hospital, İstanbul, Türkiye

ABSTRACT

Objective: In this study, we aimed to evaluate growth characteristics of preterms followed up in a neonatal intensive care unit (NICU) during adolescence.

Methods: The preterms born in 2001–2002 who were followed up in the NICU of our hospital were examined in 2016. Data from the prenatal, natal, and postnatal periods, as well as at the 6–7 years, were obtained from a database with follow–up assessments during adolescence. Measures of growth (height, weight, body mass index), the percentile curves for Turkish children, and the target height formula were used in the assessment. Factors affecting growth, such as maternal problems and neonatal morbidities, were questioned.

Results: Thirty–seven preterm babies (19 females and 18 males) were included in the study. The mean gestational age was 31.5 (28–34) weeks, and the birth weight was 1298 (820–1870) grams. Six newborns (17.6%), and three adolescents (8.1%) were below 3rd percentile for height. All cases had height percentiles within the normal range at 6–7 years of age. Twenty–six adolescents (70.3%) achieved the target height. Eleven premature (29.7%) during the neonatal period, one child (2.7%) during the school period, and one adolescent (3%) were below 3rd percentile for weight. During the neonatal period, one case for height and two cases for weight were above 90th percentiles. While, during school period, 6 cases for height, 2 cases for weight were above 90th percentiles; 1 case for height, 6 cases for weight were above 90th percentiles during adolescence. The frequency of obesity increased from 2.7% to 13.5% during adolescence. 90% of individuals below 3rd percentile of birth weight reached normal weight percentiles during adolescence. Prenatal, natal, and postnatal risk factors, as well as duration of breastfeeding, were not found to be effective in achieving the target height during school–age and adolescent periods.

ÖZ

Amaç: Çalışmamızda yenidoğan yoğun bakım ünitemizde (YYBÜ) izlenen prematürelerin adölesan dönemde büyüme özelliklerini değerlendirmeyi amaçladık.

Yöntemler: 2001–2002 yıllarında doğan ve hastanemiz YYBÜ takip edilen prematüreler 2016 yılında değerlendirildi. Veritabanından hastalara ait prenatal, natal, postnatal, 6–7 yaş ve adolesan dönem takip verileri elde edildi. Değerlendirmede büyüme ölçütleri (boy, kilo, vücut kitle indeksi), Türk çocuklarının büyüme eğrileri ve hedef boy formülü kullanıldı. Büyümeye etki eden faktörler (maternal sorunlar, neonatal dönem morbiditeleri vb.) sorgulandı.

Bulgular: Çalışmaya 37 preterm (19 kız, 18 erkek) dahil edildi. Ortalama gebelik yaşı 31,5 (28–34) hafta, doğum ağırlığı 1298 (820–1870) gramdı. Altı yenidoğan (%17,6) ve 3 adolesan (%8,1) boyca 3 persentil altındaydı. Tüm olgular 6–7 yaşta boyca normal persentillerdeydi. Yirmi altı adolesan (%70,3) hedef boya ulaşmıştı. Yenidoğan döneminde 11 prematüre (%29,7), okul döneminde bir çocuk (%2,7) ve bir adolesan (%3) ağırlıkça 3 persentil altındaydı. Yenidoğan döneminde, bir olgunun boyda ve 2 olgunun ağırlıkta 90 persentil üzerinde olduğu görüldü. Okul döneminde 6 olgu boyda, 2 olgu ağırlıkta 90 persentil üzerinde iken; adolesan dönemde 1 olgu boyda, 6 olgu ağırlıkta 90 persentil üzerindeydi. Obezite sıklığı adolesan dönemde %2,7'den %13,5'e yükselmişti. Doğum ağırlığı 3 persentil altındaki olguların %90'ı adolesan dönemde normal ağırlık persentillerine ulaşmıştı. Okul çağı ve adolesan dönemde hedef boya ulaşmada prenatal, natal ve postnatal risk faktörleri ile anne sütü alma süresinin etkili olmadığı saptandı.

Sonuç: Çalışmamızda, okul döneminde tüm olguların boyca normal persentillerde olmasına rağmen ergenlikte olguların %8,1'nin 3 persentil altında olduğunu saptadık. Ayrıca ağırlıkça 3 persentil altında

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Address for Correspondence/Yazışma Adresi: Kübra Baskın, Department of Pediatric Immunology and Allergy, Gazi University Faculty of Medicine, Ankara, Türkiye
E-mail / E-posta: akbaskin@hotmail.com
ORCID ID: orcid.org/0000–0002–4294–7492

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ABSTRACT

Conclusion: In this study, we found that all cases had normal percentiles for height in the school period, but on the other hand, 8.1% cases were <3rd percentiles during adolescence. Also, the frequency of cases that were <3rd percentiles for weight was similar during the school period and adolescence. These results reflected that, while catch–up growth could be achieved during the school years, it was not achievable during adolescence. It demonstrates that the growth of individuals born preterm is a dynamic phenomenon.

Keywords: Adolescent, growth, preterm birth, risk factors

ÖZ

olan olguların sıklığı okul dönemi ve ergenlik döneminde benzerdi. Bu sonuçlar, büyüme yakalamasının okul yıllarında sağlanabilmekle birlikte, ergenlik döneminde sağlanamadığını yansıtmaktadır. Bu durum prematüre doğan bireylerin büyümelerinin dinamik bir süreç olduğunu göstermektedir.

Anahtar Sözcükler: Adolesan, büyüme, preterm doğum, risk faktörleri

INTRODUCTION

In parallel with advances in perinatal and neonatal care worldwide, the survival of premature babies has increased, and more are being discharged from the hospital. The follow–up of high–risk preterm infants has highlighted several key questions concerning growth patterns and developmental milestones. Prematurity is associated with a range of short– and long–term complications, including impaired somatic growth, neurodevelopmental delays, and increased risk of chronic health conditions. These outcomes may persist into adolescence and adulthood, underscoring the importance of longitudinal monitoring. A holistic examination of the long–term consequences of premature birth is of great importance for prognosis.

Preterm infants are prone to early postnatal growth restriction; nevertheless, most subsequently achieve somatic catch–up growth. Increases in weight and length typically begin within the first months of life and often normalize by two years of age (1). Some postnatal comorbidities, such as bronchopulmonary dysplasia (BPD), retinopathy of prematurity (ROP), and neurodevelopmental impairment, are also reported to affect catch–up growth (2).

Numerous studies have examined long–term monitoring in very–low–birth–weight premature infants (3-5). Although studies on somatic growth and neurodevelopmental evaluation are available in infancy, early childhood, and school–age, data on adolescent follow–up are limited (6-8).

For this reason, we aimed to evaluate the somatic growth of preterm infants born in 2001–2002 who were followed up in our neonatal intensive care unit (NICU) during adolescence, with a specific focus on their anthropometric outcomes.

MATERIALS AND METHODS

Premature infants born in 2001–2002 at 28–34 weeks of gestational age (GA) according to the Ballard Score and followed up in the NICU of Ministry of Health, Bakırköy Obstetrics and Pediatrics Training and Research Hospital were included in the study. Individuals who are deceased, have metabolic disease, and have anomalies are excluded. Growth curves by Kurtoğlu et al. (9) were used to evaluate somatic growth during the neonatal period. Cases whose birth weight were <3rd percentile (rather than the more commonly used <10th percentile threshold) were recorded as small for GA (SGA) and divided into symmetrical or asymmetrical SGA categories. This stricter definition was chosen to increase specificity, ensuring that infants classified as SGA truly represented those at highest risk of growth failure and adverse outcomes, and to enable a more accurate comparison with cases presenting growth retardation during childhood and adolescence. Cases at the 3rd–90th percentile were considered as appropriate for GA (AGA), and cases >90th percentile were considered as large for GA (LGA). Also, we categorised cases according to GA [extremely preterm (less than 28 weeks), very preterm (28 to less than 32 weeks), moderate to late preterm (32 to 37 weeks)] and birthweight (<1000 g: extremely low birthweight, 1000 to 1499 g: very low birthweight, 1500 to 2500 g: low birthweight).

The following prenatal risk factors were assessed: premature rupture of membranes, chorioamnionitis, maternal diabetes mellitus, hypertensive disorders of pregnancy, presence of chronic disease, and intrauterine growth retardation. Among natal risk factors, the following were investigated: history of fetal distress, placenta previa, and placental detachment. The following postnatal risk factors were investigated and recorded: duration of stay in the NICU, mechanical ventilation (administration and duration), surfactant therapy, sepsis, hypoglycemia, hyperbilirubinemia, respiratory distress syndrome (RDS), BPD, intraventricular hemorrhage, posthemorrhagic hydrocephalus, and ROP. Additionally, breastfeeding duration was ascertained and recorded.

The somatic growth of these children was previously evaluated at school age (6–7 years). Data from the prenatal, natal, and postnatal periods and at 6–7 years of age were obtained from the database. Values >90th percentile during the neonatal, childhood, and adolescent periods were used as a criteria for advanced growth (10).

A single physician (KB) assessed the cohort at 14–15 years of age, following signed parental and child consent. The systemic examination of the patients was performed. Puberty status was assessed according to Tanner staging (11,12). Ages at menarche and thelarche were recorded based on parental recall.

The standing height and weight of the cases and parents were measured by the same physician (using a Sinbo weighing instrument and a Harpenden stadiometer) at least twice, and the averages were used for analysis. Values of the cases were placed in the percentile curves prepared for the Turkish children by Neyzi et al. (13). The target length based on the mother's and father's heights was also calculated and checked after the percentile chart was marked. When calculating the target height, the difference between men and women, according to each society's standards, was considered. For Turkish society, this difference was 13 cm (13,14).

Target height for a girl was calculated as $\frac{(\text{Father's height} - 13\text{ cm}) + (\text{mother's height})}{2}$

Target height for a boy was calculated as $\frac{(\text{Mother's height} + 13\text{ cm}) + (\text{father's height})}{2}$

Body mass index (BMI) was calculated as weight (kg)/height (m^2). Cases were identified according to the World Health Organization BMI classification (15). BMI percentiles were assessed using reference values for Turkish children (16).

This study was approved by the Ethics Committee of Ministry of Health İstanbul Kanuni Sultan Süleyman Training and Research Hospital (approval number: 15929, date: 30.09.2015). Patients and their parents provided the written informed consent.

Statistical Analysis

The findings were analyzed statistically using the Statistical Program in Social Sciences version 24.0 (2016). When evaluating the study data, McNemar test, Fisher exact test, Mann–Whitney U test, and Kruskal–Wallis test were used in addition to descriptive statistical methods (mean, median, standard deviation, frequency). Categorical variables were presented as numbers and percentages. Continuous variables were presented as mean ± standard deviation or median (minimum-maximum), depending on the distribution. The results were evaluated using a 95% confidence interval, with significance set at $p < 0.05$. Missing data were taken into account during analyses.

RESULTS

In this study, 37 adolescents born preterm (19 females, 18 males) were evaluated. Most cases (n = 20, 54%) were SGA; 40.5% (n = 15) were AGA; and most cases (n = 30, 80%) had birth weights of 1000–1500 grams. While the most common prenatal risk factors were intrauterine growth retardation (IUGR) and maternal hypertension, the most common postnatal risk factor was hyperbilirubinemia. Demographic characteristics and prenatal, natal, and postnatal risk factors of the cases are summarized in Table 1.

Growth in the cases during the newborn and school periods (6–7 years) was compared with adolescent growth (Table 2). During adolescence, the mean age of cases was 14.6 ± 0.78 years, the mean height was 162.3 ± 7.1 cm, the mean weight was 56.1 ± 13.8 kg, and the mean BMI was 21 ± 4.04 kg/m^2. Values for height, weight, BMI, and pubarche onset by gender are presented in Table 3. Age at pubarch onset was 12.06 ± 1.2 years, and at menarche onset was 12.2 ± 1.08 years. Only one case was treated for precocious puberty; in the other cases, the onset of pubertal growth and development was prompt.

While 6 cases (17.6%) were found to be <3rd percentiles for height in the newborn period, there were no cases <3rd percentiles for height in the school period. Three adolescents (8.1%) were <3rd percentiles for height (Table 2). Twenty–six cases (70.3%) reached target height by adolescence (Table 4). Of the eleven cases (6 males, 5 females) that did not reach target height, 4 were AGA, 3 were symmetrical SGA, and 4 were asymmetric SGA in the neonatal period (Table 4). Eleven premature (29.7%) during the neonatal period, one child (2.7%) during the school period, and one adolescent (3%) were <3rd percentile for weight.

Table 1. Demographic, perinatal and neonatal characteristics of cases.

	n	%
Gender		
Female	19	51.4
Male	18	48.6
Intrauterine growth		
Symmetric SGA*	8	21.6
Asymmetric SGA*	12	32.4
AGA*	15	40.5
LGA*	2	5.4
GA (week)		
≤28	4	10.8
29–32	20	54
≥33	13	35.1
Birth weight (gram)		
<1000 g	3	8.1
1000–1500 g	30	81
>1500 g	4	10.8
Prenatal risk factors		
IUGR	10	32.3
Hypertensive disorders of pregnancy	10	32.3
Maternal smoking	8	25.8
Mother's age >35 (year)	6	19.4
PROM	4	12.9
Presence of chronic disease	3	9.7
Plesantal dysfunction	2	6.5
Natal risk factors		
Fetal distress	8	100.0
Placenta detachment	4	50.0
Postnatal risk factors		
Hyperbilirubinemia	25	71.4
RDS	13	37.1
Surfactant therapy	13	37.1
Mechanical ventilation	13	37.1
Sepsis	11	31.4
ROP	6	17.1
Apnea	6	17.1
Intracranial hemorrhage	5	14.3
Hypoglycemia	2	5.7
PDA	2	5.7
Hydrocephalus	1	2.9
BPD	1	2.9

*AGA: Appropriate for gestational age, LGA: Large for gestational age, SGA: Small for gestational age, BPD: Bronchopulmonary dysplasia, IUGR: Intrauterine growth retardation, PDA: Patent ductus arteriosus, PROM: Premature rupture of membranes, RDS: Respiratory distress syndrome, ROP: Retinopathy of prematurity.

During the neonatal period, one case for height and two cases for weight were >90th percentiles. While during school period, 6 cases for height and 2 cases for weight were >90th percentiles; 1 case for height and 6 cases for weight were >90th percentiles during adolescence (Table 2). The Frequency of LGA was 5.4% during the neonatal period. Obesity prevalence, when evaluated according to BMI percentiles, was 2.7% during the school period and increased to 13.5% during adolescence.

To evaluate growth trajectories, height and weight percentiles at birth, at age 6–7 years, and during adolescence were examined. No statistically significant difference was observed between height percentiles at birth and in adolescence. Two of the six patients whose birth height was <3rd percentile (33.3%) were <3rd percentile for height currently, and the remainder (n = 4, 66.7%) reached the target height (Table 5). While all individuals with a birth weight <3rd percentiles reached normal weight percentiles at age 6–7 (p = 0.021), 90.9% reached normal weight percentiles during puberty (p = 0.002). Intrauterine growth characteristics (SGA, AGA, LGA), parental heights, and BMI did not significantly affect this group's attainment of the target height (Table 5).

Eight of 11 patients whose birth weight was <3rd percentile, and 18 of remaning 26 patients whose birth weight was >3rd percentile could reach target height. Birth weight was not found to have a statistically significant effect on target height. (p = 1.00)

When the relationship between birth height percentiles and prenatal, natal, and postnatal risk factors were examined, the incidence of hypoglycemia was higher in patients whose height <3rd percentile (p = 0.030). No statistically significant differences were observed between other risk factors and height percentiles at birth (Table 6). There was also no statistically significant association between birth weight percentiles and the aforementioned risk factors (Table 7).

Height and weight percentiles during school age and adolescence were examined according to the intrauterine growth status of the patients (AGA, LGA, SGA), and no statistically significant differences were found. Also, no statistically significant association was found between prenatal, natal, and postnatal risk factors and height and weight percentiles during school age and adolescence.

Breastfeeding duration ranged from 15 days to 2.5 years among the cases. There was no statistically significant relationship between breastfeeding duration and patients' growth (length and weight percentiles) during school–age and adolescence.

DISCUSSION

This study examined the somatic growth features of high–risk preterm infants, most of whom weighed <1500 grams, at approximately 14 years of age. Our results suggested that the growth trajectories of children born preterm differed across follow–up periods. Growth of the cases during the newborn and school periods (6–7 years) was compared with adolescent growth.

While 6 cases (17.6%) for height, 11 cases (29.7%) for weight were found to be <3rd percentiles in the newborn period, all cases caught up in length gain during school age. Only one child was found to be <3rd percentiles for weight. Three adolescents (8.1%) for height, 1 adolescent (3%) for weight were found to be <3rd percentiles. Twenty–six cases (70.3%) could reach target height by adolescence.

Different views exist in the literature regarding catch–up growth. Catch–up growth occurs during the first three years of life in cases without severe neurological impairment, cardiac disease, or genetic disease or syndrome (17). Niklasson et al. (18) showed that normal growth could be achieved at the age of 2 years with optimal postnatal care, even in cases with IUGR, in the absence of perinatal complications and severe congenital anomalies.

Unlike our study, in a study that evaluated neurodevelopmental and physical growth of very low birth weight preterms at an average of 87 months after birth, the rate of cases <3rd percentile was reported as 6% in height and 7% in weight (7). A study of 4423 late preterm children evaluated from birth to 14 years reported that preterm children had lower height and weight than full–term children across all age groups (19). In the Danish birth cohort in which children born between 1996 and 2003 were followed until age 18, GA was associated with height in infancy, but the difference in height between preterm and term children decreased during childhood. It has been reported that many individuals born preterm remain shorter than those born at term during adolescence. It has also been reported that BMI, which was relatively lower in preterm infants, became equal to that of term infants during childhood, with no significant difference by adolescence (20). We found the BMI during adolescence to be similar to normal values according to the WHO classification.

Table 2. Weight and height measurements of the patients at birth, 6–7 years of age and adolescence.

	Birth height		Birth weight		Height (6–7 years)		Weight (6–7 years)		Height (adolescent)		Weight (adolescent)	
	n	%	n	%	n	%	n	%	n	%	n	%
<3rd p	6	17.6	11	29.7	0	0.0	1	3.0	3	8.1	1	2.7
3–10 p	7	20.6	9	24.3	0	0.0	3	9.1	2	5.4	2	5.4
11–25 p	10	29.4	5	13.5	3	9.1	9	27.3	8	21.6	7	18.9
26–50 p	7	20.6	6	16.2	10	30.3	8	24.2	13	35.1	8	21.6
51–75 p	3	8.8	4	10.8	7	21.2	5	15.2	9	24.3	9	24.3
76–90 p	0	0.0	0	0.0	7	21.2	5	15.2	1	2.7	4	10.8
91–97 p	0	0.0	2	5.4	5	15.2	0	0.0	1	2.7	5	13.5
>97th p	1	2.9	0	0.0	1	3.0	2	6.1	0	0.0	1	2.7

p: Percentile.

In the EPICure study, which evaluated extremely premature infants, the authors reported that some catch–up growth in height and weight occurred between 2.5 and 6 years of age, but growth parameters at age 6 remained below population norms. In this study, individuals born preterm were lighter and shorter at 19 years of age than the control group (21).

In a study that examined growth characteristics of very low–weight premature babies at an average postnatal age of 36 months, it was found that gender and AGA or SGA status had no effect on growth. Mechanical ventilation, chronic disease, advanced intracranial hemorrhage, ROP, and lack of breastfeeding were identified as risk factors for growth retardation (6). Akar et al. (8) emphasized in their study evaluating somatic growth in late preterm preschoolers that being AGA or SGA was not a statistically significant predictor of reaching the target height. By contrast, in a Brazilian birth cohort study, preterm children with SGA and low birth weight were shorter and thinner than term children at 5 years (22). Similar to the previous studies (6,8), in our study intrauterine growth characteristics and the aforementioned postnatal risk factors were not associated with growth at school age or during adolescence.

Prenatal risk factors that affect growth include maternal hypertension, chronic disease, gestational diabetes, maternal smoking, and placental dysfunction (23,24). However, our study revealed that prenatal and natal risk factors were not associated with growth at birth, during school age, and during adolescence.

Breastfeeding is also important for growth. In a study, the growth of SGA babies fed breast milk was faster than that of babies fed formula (25). In our study, no statistically significant relationship was observed between breastfeeding duration and patients' growth, measured by height and weight percentiles, during school age and adolescence.

Our study revealed that during the neonatal and school–age periods 2 cases, during adolescence 6 cases for weight were >90th percentiles. Two cases who were >90th percentiles for weight at school age were asymmetrical SGA at birth. Their birth weights were 1100 grams and 1600 grams. While one case had a history of RDS, BPD, PDA, and mechanical ventilation, the other case had only hyperbilirubinemia. Of the six cases who were >90th percentiles for weight during adolescence, 5 were SGA (asymmetric: 3, symmetric: 2) and, one was AGA at birth. Birth weights ranged from 1000 to 1600 grams. In most cases, sepsis, apnea, and hyperbilirubinemia were present as postnatal risk factors. The frequency of obesity increased from 2.7% to 13.5% during adolescence. There was no significant association of intrauterine growth status or prenatal, natal, and postnatal risk factors with weight and height in adolescence.

Few studies have examined the potential link between prematurity and the risk of childhood obesity and have yielded different results. Unlike our study, a study examining extremely preterm infants reported that females with high gains in height/weight or weight during the 2 years after NICU discharge had greater odds of obesity at 10 years, but not at 15 years (26). Another study revealed that premature infants had a greater likelihood of childhood obesity at ages 8 to 11, and no significant difference in childhood obesity risk was found between SGA and AGA preterm infants (27).

Table 3. Height, weight, BMI and pubarche onset distrubition according to gender during adolescence.

	Boys	Girls
Height (cm, median)	166.6	159
(min–max)	(144.5–179.7)	(148–172)
Weight (kg, median)	54.4	51.8
(min–max)	(32.8–93.5)	(41–82.1)
BMI (kg/m^2, median)	19.9	20
(min–max)	(15.7–33.6)	(16,3–27.9)
Pubarche onset (year, median)	13	11.5
(min–max)	(11–14)	(9–14)

BMI: Body mass index, Min: Minimum, Max: Maximum.

Table 4. Characteristics of patients according to reaching target height status in adolescence.

	Reaching target height	
	Yes	No
	n = 26	n = 11
Gender		
Female	15	5
Male	11	6
Intrauterine growth		
Symmetric SGA	6	3
Asymmetric SGA	9	4
AGA	10	4
LGA	1	–

AGA: Appropriate for gestational age, LGA: Large for gestational age, SGA: Small for gestational age.

Table 5. Distribution of clinical features according to height percentiles at birth.

Birth height	<3rd p		3–97th p		p[a]
	n	%	n	%	
Intrauterine growth					
Symmetric SGA	4	66.7	4	14.8	0.052
Asymmetric SGA	2	33.3	10	37.0	
AGA	0	0.0	11	40.7	
LGA	0	0.0	2	7.4	
Reaching target height					
Yes	4	66.7	19	70.4	1.000
No	2	33.3	8	29.6	
Mother's height (mean ± 2 SD) (cm)	160.0 ± 6.2		159.4 ± 5.7		0.800
Father's height (mean ± 2 SD) (cm)	173.7 ± 8.5		170.2 ± 7.2		0.313
BMI (mean ± 2 SD) (kg/m^2)	19.5 ± 3.0		21.7 ± 4.4		0.259

[a]Fisher exact test. Due to the distribution of data, cases were analyzed as <3rd percentile and 3–97th percentile.
p: Percentile, BMI: Body mass index, SD: Standard deviation, SGA: Small for gestational age, AGA: Appropriate for gestational age, LGA: Large for gestational age.

In a French cohort that prospectively evaluated very preterm infants, the obesity rate in adolescence was 13.9% (10.5–18.3%), which is similar to that was observed in our study. This study also revealed that the change in length during the neonatal hospital stay of preterm infants was negatively associated with risk of overweight or obesity at 5 and 15 years, and that the change in BMI between discharge and 2 years was positively associated that risk (28). Unfortunately, we did not have data on patients' growth in length during their NICU stay. Another study evaluating four birth cohorts during adolescence revealed that BMI was similar between preterm and term peers, while very preterm individuals had an increased risk of overweight (29).

Growth in height accelerates during puberty, and the peak of this acceleration occurs in early puberty (Tanner stage 2) in girls and in mid–puberty (Tanner stage 4) in boys. Although the acceleration in height growth occurs earlier in girls, it ends earlier than it does in boys (30). The literature is inconsistent regarding the effect of preterm birth on timing of puberty and pubertal growth patterns. While one study assessing 129 preterms compared to terms found no differences in pubertal growth spurt (31), in another study the onset of puberty (assessed according to Tanner staging) was later for preterm girls than term controls (32). In the ESTER Preterm Birth Study, timing of pubertal growth, age at menarche or voice break were found to be similar in preterm and term individuals (33). When we evaluated our cases with respect to pubertal development, we observed that only 1 case was treated for precocious puberty, whereas the remaining cases had normal pubertal onset and development.

CONCLUSION

Although our study has certain limitations, we believe it is valuable as it includes long-term data from our country. Key limitations of

Table 6. Distribution of prenatal, natal and postnatal risk factors according to height percentiles at birth.

Birth height	<3rd p		3–97th p		p[a]
	n	%	n	%	
Prenatal risk factors					
Hypertensive disorders of pregnancy	3	60.0	6	27.3	0.295
IUGR	2	40.0	6	27.3	0.616
Mother's age >35 (year)	1	20.0	4	18.2	1.000
PROM	1	20.0	3	13.6	1.000
Presence of chronic disease	1	20.0	2	9.1	1.000
Plesantal dysfunction	1	20.0	1	4.5	0.342
Smoking/substance use	0	0.0	7	31.8	0.283
Natal risk factors					
Fetal distress	1	100.0	6	100.0	*
Placenta detachment	0	0.0	4	66.7	0.429
Postnatal risk factors					
Hyperbilirubinemia	4	66.7	18	69.2	1.000
Surfactant therapy	2	33.3	10	38.5	1.000
Hypoglycemia	2	33.3	0	0.0	**0.030**
Sepsis	2	33.3	8	30.8	1.000
Mechanical ventilation	2	33.3	10	38.5	1.000
RDS	1	16.7	11	42.3	0.370
ROP	1	16.7	5	19.2	1.000
Hydrocephalus	0	0.0	1	3.8	1.000
BPD	0	0.0	1	3.8	1.000
Intracranial hemorrhage	0	0.0	5	19.2	0.555
PDA	0	0.0	2	7.7	1.000
Apnea	0	0.0	5	19.2	0.555

[a]Fisher exact test, *p-value could not be calculated due to data distribution. BPD: Bronchopulmonary dysplasia, IUGR: Intrauterine growth retardation, PDA: Patent ductus arteriosus, PROM: Premature rupture of membranes, RDS: Respiratory distress syndrome, ROP: Retinopathy of prematurity.

Tablo 7. Distribution of prenatal, natal and postnatal risk factors according to weight percentiles at birth.

Birth weight	<3rd p		3–97th p		p[a]
	n	%	n	%	
Prenatal risk factors					
IUGR	4	40.0	6	28.6	0.685
Hypertensive disorders of pregnancy	4	40.0	6	28.6	0.685
Mother's age >35 (year)	3	30.0	3	14.3	0.284
Presence of chronic disease	2	20.0	1	4.8	0.237
PROM	2	20.0	2	9.5	0.577
Plesantal dysfunction	1	10.0	1	4.8	1.000
Smoking/substance use	1	10.0	7	33.3	0.222
Natal risk factors					
Fetal distress	2	100.0	6	100.0	*
Placenta detachment	1	50.0	3	50.0	1.000
Postnatal risk factors					
Hyperbilirubinemia	9	90.0	16	64.0	0.218
Sepsis	4	40.0	7	28.0	0.689
Apnea	3	30.0	3	12.0	0.322
Surfactant therapy	3	30.0	10	40.0	0.709
ROP	2	20.0	4	16.0	1.000
RDS	2	20.0	11	44.0	0.259
Mechanical ventilation	2	20.0	11	44.0	0.259
Hypoglycemia	1	10.0	1	4.0	1.000
PDA	1	10.0	1	4.0	1.000
Intracranial hemorrhage	1	10.0	4	16.0	1.000
Hydrocephalus	0	0.0	1	4.0	1.000
BPD	0	0.0	1	4.0	1.000

[a]Fisher exact test, *p-value could not be calculated due to data distribution. BPD: Bronchopulmonary dysplasia, IUGR: Intrauterine growth retardation, PDA: Patent ductus arteriosus, PROM: Premature rupture of membranes, RDS: Respiratory distress syndrome, ROP: Retinopathy of prematurity.

our study include the participation of high-risk preterm infants requiring intensive care and the relatively small sample size, which restrict the generalizability of the findings. Multicenter studies with larger sample sizes will contribute to the literature. A further limitation of our study is the definition of SGA restricted to neonates with birth weight below the 3rd percentile. While this approach identifies the most severely growth-restricted infants, it excludes those between the 3rd and 10th percentiles who are commonly included in the conventional definition. As a result, our findings may not be fully generalizable to the broader SGA population and should be interpreted with caution when compared to studies employing the standard <10th percentile threshold. Moreover, assessing the age of thelarche based solely on parental recall constitutes a limitation in the evaluation of pubertal development.

Our findings indicate that somatic growth in preterm infants is a dynamic, non–linear process with distinct patterns across developmental stages. Although catch–up growth in height and weight was largely achieved by school age, a subset of adolescents exhibited growth failure or excessive weight gain during puberty.

These observations underscore the clinical importance of long–term, structured follow–up for preterm infants that extends beyond early childhood. Regular anthropometric monitoring into adolescence and beyond is essential to detect deviations from expected growth trajectories. Such follow–up enables timely nutritional, endocrinological, and psychosocial interventions, which may improve long–term health outcomes and quality of life.

Ethics

Ethics Committee Approval: This study was approved by the Ethics Committee of Ministry of Health İstanbul Kanuni Sultan Süleyman Training and Research Hospital (approval number: 15929, date: 30.09.2015).

Informed Consent: Patients and their parents provided the written informed consent.

Footnotes

Authorship Contributions

Surgical and Medical Practices: K.B., S.K., Concept: K.B., S.K., Design: K.B., S.K., Data Collection or Processing: K.B., S.K., Analysis or Interpretation: K.B., S.K., Literature Search: K.B., S.K., Writing: K.B., S.K.

Conflict of Interest: No conflict of interest was declared by the authors.

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REFERENCES

1. Euser AM, de Wit CC, Finken MJ, Rijken M, Wit JM. Growth of preterm born children. Horm Res. 2008; 70: 319-28.
2. Raaijmakers A, Allegaert K. Catch-up growth in former preterm neonates: no time to waste. Nutrients. 2016; 8: 817.
3. Hack M, Fanaroff AA. Outcomes of children of extremely low birthweight and gestational age in the 1990s. Semin Neonatol. 2000; 5: 89-106.
4. Hack M, Klein NK, Taylor HG. Long-term developmental outcomes of low-birth-weight infants. Future Child. 1995; 5: 176-96.
5. Enhancing the outcomes of low-birth-weight, premature infants. A multisite, randomized trial. The Infant Health and Development Program. JAMA. 1990; 263: 3035-42.
6. Yeşinel S, Aldemir EY, Kavuncuoğlu S, Yeşinel S, Yıldız H. Evaluation of growth in very low birth weight preterm babies. Turk Pediatri Ars. 2014; 49: 289-98.
7. Koç Ö, Kavuncuoğlu S, Ramoğlu MG, Aldemir E, Aktalay A, Eras Z. School performance and neurodevelopment of very low birth weight preterm infants: First report from Turkey. J Child Neurol. 2016; 31: 170-6.
8. Akar S, Kavuncuoğlu S, Akın MA, Aldemir E, Demirhan A. Somatic growth features of moderate-late and ID early preterm infants at early childhood. J Behcet Uz Child Hosp. 2021; 11: 226-32.
9. Kurtoğlu S, Hatipoğlu N, Mazıcıoğlu MM, Akın MA, Çoban D, Gökoğlu S, et al. Body weight, length and head circumference at birth in a cohort of Turkish newborns. J Clin Res Pediatr Endocrinol. 2012; 4: 132-9.
10. Rogol AD. The child with tall stature and/or abnormally rapid growth. UpToDate [Internet]. Waltham (MA): UpToDate Inc.; [cited 2026 Mar 13]. Available from: https://www.uptodate.com/contents/the-child-with-tall-stature-and-or-abnormally-rapid-growth
11. Marshall WA, Tanner JM. Variations in the pattern of pubertal changes in boys. Arch Dis Child. 1970; 45: 13-23.
12. Marshall WA, Tanner JM. Variations in pattern of pubertal changes in girls. Arch Dis Child. 1969; 44: 291-303.
13. Neyzi O, Bundak R, Gökçay G, Günöz H, Furman A, Darendeliler F, et al. Reference values for weight, height, head circumference, and body mass index in Turkish children. J Clin Res Pediatr Endocrinol. 2015; 7: 280-293.
14. Cinaz P, Darendeliler F. Normal büyüme. In: Cinaz P, Darendeliler F, editors. Temel Çocuk Endokrinolojisi. 1st ed. İstanbul: Nobel Tıp Kitabevi; 2013. p. 35. Available from: https://www.nobelkitabevi.com/temel-cocuk-endokrinolojisi.
15. World Health Organization. BMI-for-age (5-19 years) [Internet]. Geneva: WHO; [cited 2026 Mar 13]. Available from: https://www.who.int/tools/growth-reference-data-for-5to19-years/indicators/bmi-for-age
16. Neyzi O, Günöz H, Furman A, Bundak R, Gökçay G, Darendeliler F, et al. Türk çocuklarında vücut ağırlığı, boy uzunluğu, baş çevresi ve vücut kitle indeksi referans değerleri. Çocuk Sağlığı ve Hastalıkları Dergisi. 2008; 51: 1-14.
17. Allen MC. Outcome and follow-up of high-risk infants. In: Taeusch HW, Ballard RA, editors. Avery's diseases of the newborn. 7th ed. Philadelphia: WB Saunders; 1998. p. 413-463. Available from: https://www.sciencedirect.com/book/9780721678244/averys-diseases-of-the-newborn
18. Niklasson A, Engstrom E, Hard AL, Wikland KA, Hellstrom A. Growth in very preterm children: A longitudinal study. Pediatr Res. 2003; 54: 899-905.
19. Yoshida-Montezuma Y, Kirkwood D, Sivapathasundaram B, Keown-Stoneman CDG, de Souza RJ, To T, et al. Late preterm birth and growth trajectories during childhood: A linked retrospective cohort study. BMC Pediatr. 2023; 23: 450.
20. Vinther JL, Ekstrøm CT, Sørensen TIA, Cederkvist L, Lawlor DA, Andersen AN. Gestational age and trajectories of body mass index and height from birth through adolescence in the Danish National Birth Cohort. Sci Rep. 2023; 13: 3298.
21. Jańczewska I, Wierzba J, Jańczewska A, Szczurek-Gierczak M, Domżalska-Popadiuk I. Prematurity and low birth weight and their impact on childhood growth patterns and the risk of long-term cardiovascular sequelae. Children (Basel). 2023; 10: 1599.

22. Rocha AS, Ribeiro-Silva RC, Silva JFM, Pinto EJ, Silva NJ, Paixao ES, et al. Postnatal growth in small vulnerable newborns: A longitudinal study of 2 million Brazilians using routine register-based linked data. Am J Clin Nutr. 2024; 119: 444-55.
23. Mittendorf R, Herschel M, Williams MA, Hibbard JU, Moawad AH, Lee KS. Reducing the frequency of low birth weight in the United States. Obstet Gynecol. 1994; 83: 1056-9.
24. Lin X, Zhou L, Si S, Cheng H, Alifu X, Qiu Y, et al. Association of the comorbidity of gestational diabetes mellitus and hypertension disorders of pregnancy with birth outcomes. Front Endocrinol (Lausanne). 2024; 15: 1468820.
25. Lucas A, Fewtrell MS, Davies PS, Bishop NJ, Clough H, Cole TJ. Breastfeeding and catch-up growth in infants born small for gestational age. Acta Paediatr. 1997; 86: 564-9.
26. O'Shea TM, Register HM, Yi JX, Jensen ET, Joseph RM, Kuban KCK, et al. Growth during infancy after extremely preterm birth: Associations with later neurodevelopmental and health outcomes. J Pediatr. 2023; 252: 40-7.e5.
27. Ou-Yang MC, Sun Y, Liebowitz M, Chen CC, Fang ML, Dai W, et al. Accelerated weight gain, prematurity, and the risk of childhood obesity: A meta-analysis and systematic review. PLoS One. 2020; 15: e0232238.
28. Simon L, Hadchouel A, Arnaud C, Frondas-Chauty A, Marret S, Flamant C, et al. Growth trajectory during the first 1000 days and later overweight in very preterm infants. Arch Dis Child Fetal Neonatal Ed. 2023; 108: 149-55.
29. Vinther JL, Cadman T, Avraam D, Ekstrøm CT, Sørensen TIA, Elhakeem A, et al. Gestational age at birth and body size from infancy through adolescence: An individual participant data meta-analysis on 253,810 singletons in 16 birth cohort studies. PLoS Med. 2023; 20: 1004036.
30. Wood CL, Lane LC, Cheetham T. Puberty: Normal physiology (brief overview). Best Pract Res Clin Endocrinol Metab. 2019; 33: 101265.
31. Persson I, Ahlsson F, Ewald U, Tuvemo T, Qingyuan M, von Rosen D, et al. Influence of perinatal factors on the onset of puberty in boys and girls: Implications for interpretation of link with risk of long-term diseases. Am J Epidemiol. 1999; 150: 747-55.
32. Hui LL, Leung GM, Lam TH, Schooling CM. Premature birth and age at onset of puberty. Epidemiology. 2012; 23: 415-22.
33. Suikkanen J, Nurhonen M, Cole TJ, Paalanne M, Matinolli HM, Tikanmäki M, et al. Preterm birth and subsequent timing of pubertal growth, menarche, and voice break. Pediatr Res. 2022; 92: 199-205.