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Maternal Height and Infant Body Mass Index Are Possible Risk Factors for Developmental Dysplasia of the Hip in Female Infants

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Developmental dysplasia of the hip (DDH) is a wide-spectrum disease with a multifactorial etiology and, despite its prevalence, no definitive etiology has yet been established. The aim of this study was to investigate new risk factors for DDH by evaluating newly defined potential risk factors. A total of 71 infants were separated into 2 groups: Group I, 28 female first-born infants diagnosed with DDH and their mothers; and Group II, 43 healthy female first-born infants and their mothers. The maternal height and weight before pregnancy, infant height and weight at birth, and body mass index (BMI) of both mother and infant were determined. Calculations were made of the ratios between these parameters. Of the examined risk factors, only maternal height and the ratio of maternal height to infant BMI (MH/I-BMI) were found to be significant for DDH in infants. In conclusion, the results of this study show that a short maternal height and a low MH/I-BMI increase the risk of DDH. Further studies with a larger series are necessary to confirm these results.

Key words: developmental dysplasia of the hip, maternal, neonatal, risk factors

D evelopmental dysplasia of the hip (DDH) is a common congenital abnormality that affects the developing hip joint of the newborn [1]. The reported incidence of DDH varies from 1.5 to 2.5 per 1,000 live births [2, 3]. DDH represents a spectrum of hip joint disorders, ranging from hip dysplasia to irreducible hip dislocation [4, 5]. The etiology of DDH remains unclarified though several theories have been proposed, including inheritance, mechanical or environmental factors, hormone-induced joint laxity, and primary acetabular dysplasia [6].

A number of deformities are associated with DDH,

including neuromuscular fetal abnormalities such as arthrogryposis, calcaneovalgus, and plagiocephaly. In addition, swaddling of newborns with hips extended and adducted, breech presentation, twin pregnancy, oligohydramnios, and positive familial history have been associated with DDH. A 5 : 1 female dominance has been noted, and ligamentous laxity and maternal relaxin hormone levels have also been implicated [7, 8].

The aim of the present study was to investigate the height, weight, and body mass index (BMI) of both the infant and the mother in addition to the known factors for DDH and thereby identify and assess newly defined

potential risk factors in a group of infants with DDH in comparison to a control group. The hypothesis of this study was that a larger infant born to a mother of small stature could be a predictive factor.

Materials and Methods

Using data in hospital admission databases, we determined that a total of 203 DDH patients were treated at 2 institutions (the workplaces of authors 1 and 3) between January 2009 and January 2014. The IRB approval number of this study was B.10.4.İSM.4.06.68.49/. To eliminate any gender bias, only first-born female infants (age, 2 months – 5 years) and their mothers (age at first delivery, 20–35 years) were included in both the test and control groups. The inclusion criteria also stipulated full-term infants in both groups. Group I consisted of 28 mothers and their infants who had been treated for DDH following a diagnosis based on ultrasonography (Graf Type ≥ 2c) or radiography.

Exclusion criteria were male gender, risk factors for DDH such as oligohydramnios, breech presentation, multiple pregnancy, transverse presentation, known congenital abnormalities of the fetus, a strong family history including parents, siblings, grandparents and first cousins, torticollis, postural or congenital talipes equinovarus, congenital talipes calcaneovalgus, metatarsus adductus and swaddling. In addition, neonates with neuromuscular or syndromic causes of dislocation (meningomyelocele, cerebral palsy), or teratologic dislocation, were excluded as the hip abnormality was secondary rather than primary and therefore not defined as true DDH. Infants in poor overall condition or who had been admitted to the Neonatal Intensive Care Unit were also excluded.

Group II, the control group, consisted of 43 consecutive mothers and healthy infants, who had presented at the pediatric out-patient clinic between July 1, 2014 and August 31, 2014. The infants and mothers were evaluated to ensure that they met the inclusion criteria of full-term, first-born healthy female infant aged between 2 months and 5 years, with no DDH (according to the hip ultrasonography screening data of patients) or any of the other known risk factors mentioned above, and no congenital abnormalities (talipes equinovarus, torticollis, meningomyelocele).

The following data were taken from patient records: maternal height (MH) (cm), maternal weight before pregnancy (MW-BP) (kg), infant height at birth (IH-B) (cm) and infant weight at birth (IW-B) (g). BMI values were calculated for both mother and infant. Calculations were then made of the following proportions: maternal weight/maternal height (MW/MH), infant weight/infant height (IW/IH), infant BMI/maternal BMI (I-BMI/M-BMI), maternal height/infant height (MH/IH), and maternal height/infant BMI (MH/I-BMI), and the results were compared between the 2 groups.

Statistical analyses were calculated using the SPSS 17.0 statistical software (SPSS for Windows, SPSS Inc., Chicago, IL, USA). Continuous variables with normal distribution were compared between groups using the Student's t-test, and those with non-normal distribution using the Mann-Whitney U test. The odds ratios (ORs) for all parameters were calculated using univariate logistic regression analysis, and the Shapiro-Wilk test was used for tests of normality. Power analysis was applied to parameters found to be statistically significant. A value of $p < 0.05$ was considered statistically significant.

Results

The results for both groups are summarized in Table 1. The parameters stated above were analyzed between the 2 groups. The average MH was calculated as 161 (range, 157.25–166.50) cm in Group I and 164 (160–168) cm in Group II, and the difference between the groups was found to be statistically significant ($p = 0.022$). The average MH/I-BMI was calculated as 12.31 (11.37–12.92) in Group I and 12.77 (12.12–13.79) in Group II, and the difference between the groups was found to be statistically significant ($p = 0.038$). No statistically significant differences were determined in any other parameters and the ratios were similar between the 2 groups (all p values > 0.05). The ORs for the risk factors are shown in Table 2. The MH OR was calculated as 0.90 (082–0.99; 95% confidence interval, CI) for Group I, which was statistically significant ($p = 0.027$). The OR for MH/I-BMI was 0.67 (0.46–0.9; 95% CI) for Group I, which was also statistically significant ($p = 0.044$). Power analysis was applied to the statistically significant parameters and the powers of MH

Table 1   Comparison of risk factors between groups

	Group I (n : 28) Mean ± SD Median (25th–75th) percentile	Group II (n : 43) Mean ± SD Median (25th–75th) percentile	P value
MH (cm)	161.25 ± 5.85 161 (157.25–166.50)	164.44 ± 5.46 164 (160–168)	0.022*
MW-BP (kg)	58.89 ± 8.76 60 (52.25–65)	58.03 ± 10.82 56 (52–62)	0.361**
IH-B (cm)	48.89 ± 3.02 50 (47–51)	49.26 ± 1.62 49 (48–50)	0.563*
IW-B (g)	3,214.82 ± 409.78 3,250 (3,000–3,375)	3,140.93 ± 360.39 3,050 (2,880–3,460)	0.427*
M-BMI	22.70 ± 3.53 22.05 (20.30–25.23)	21.45 ± 3.68 21.30 (19.38–22.96)	0.064**
I-BMI	13.48 ± 1.61 13.18 (12.52–14.51)	12.92 ± 1.15 12.82 (12–13.50)	0.143**
MW/MH	36.54 ± 5.40 35.93 (32.56–40.53)	35.26 ± 6.20 34.59 (31.45–37.42)	0.200**
IW/IH	6.57 ± 0.71 6.49 (6.16–6.93)	6.37 ± 0.62 6.18 (5.94–6.85)	0.208*
I-BMI/M-BMI	0.61 ± 0.12 0.60 (0.52–0.68)	0.61 ± 0.08 0.61 (0.57–0.67)	0.810*
MH/IH	3.31 ± 0.18 3.30 (3.17–3.43)	3.34 ± 0.16 3.33 (3.25–3.42)	0.398*
MH/I-BMI	12.13 ± 1.55 12.31 (11.37–12.92)	12.82 ± 1.19 12.77 (12.12–13.79)	0.038*

MH, maternal height; MW-BP, maternal weight before pregnancy; IH-B, infant height at birth; IW-B, infant weight at birth; M-BMI, maternal body mass index; I-BMI, infant body mass index; MW/MH, maternal weight/maternal height; IW/IH, infant weight/infant height; I-BMI/M-BMI, infant body mass index/maternal body mass index; MH/IH, maternal height/infant height; MH/I-BMI, maternal height/infant body mass index.
*Student t test, **Mann-Whitney U test.

and MH/I-BMI were determined to be 0.54 and 0.44, respectively. MH and MH/I-BMI were found to have a strong association with DDH. An increase of 1 cm in maternal height was found to decrease the risk of DDH 1.1 fold (1/0.90) and an increase of 1 point in MH/I-BMI was found to decrease the DDH risk 1.49 fold (1/0.67).

Discussion

The etiology of DDH is complex since hormonal, genetic and mechanical factors may contribute to the deformity of the developing hip joint [9]. Although the pathogenesis of DDH is not yet fully understood, known risk factors are breech presentation, female gender, primiparity, first born, swaddling, multiple births, congenital foot deformities, high birth weight, oligohydramnios and a positive family history of DDH [4, 10–13]. However, the majority of cases have no risk factors [14, 15]. The rate of subsequent development of hip dysplasia with the existence of one or more of these risk factors in infants may range from 0.1 to 10% [13]. DDH can also be associated with other risk factors such as nationality, congenital

Table 2 Odds ratio (OR) for risk factors

		Odds Ratio	95% CI for odds ratio	*P* value
MH	Group I	0.90	0.82–0.99	0.027
	Group II	1	–	–
MW-BP	Group I	1.01	0.96–1.06	0.723
	Group II	1	–	–
IH-B	Group I	0.93	0.75–1.15	0.508
	Group II	1	–	–
IW-B	Group I	1.01	0.99–1.02	0.421
	Group II	1	–	–
M-BMI	Group I	1.10	0.96–1.26	0.166
	Group II	1	–	–
I-BMI	Group I	1.36	0.94–1.95	0.100
	Group II	1	–	–
MW/MH	Group I	1.04	0.96–1.13	0.374
	Group II	1	–	–
IW/IH	Group I	1.61	0.77–3.40	0.208
	Group II	1	–	–
I-BMI/M-BMI	Group I	0.54	0.04–70.96	0.807
	Group II	1	–	–
MH/IH	Group I	0.28	0.02–5.23	0.394
	Group II	1	–	–
MH/I-BMI	Group I	0.67	0.46–0.99	0.044
	Group II	1	–	–

muscular torticollis, or twin pregnancy [16, 17], or with other congenital postural deformities such as scoliosis, talipes equinovarus, genu recurvatum, Potter's or compression facies (associated with oligo-hydramnios), or plagiocephaly [4].

In the present study, we attempted to eliminate the influence of genetic bias and known mechanical factors in DDH, thereby allowing an in-depth study of the influence of unknown factors (maternal and fetal height and weight). Because a positive family history of DDH may be considered genetic bias, infants with a family history of DDH were not included in this study. The finding that DDH is more common in girls than in boys is attributed to greater female sensitivity to the maternal hormone relaxin, which creates ligament laxity and allows the hip to subluxate [7]. Therefore, only female infants were included in order to eliminate the gender risk factor. In addition, the incidence of DDH may be higher in firstborns because of increased

pressure from the abdominal wall [18], so only first-born infants were included, and other known mechanical factors were excluded.

Some previous studies have examined the association between a birth weight of < 2,500 g and DDH. Low birth weight has been reported to have a protective effect [4, 13, 19, 20]. In addition, very low birth weight infants have been found not to be at increased risk for DDH [21]. In a study by Chan *et al.* [4], increasing risk was seen with increasing birth weight as infants weighing 4,000–4,499 g had an OR of 1.55 (1.26, 1.91), while those weighing ≥ 4,500 g had an OR of 2.67 (1.81, 3.94). In another study, Bache *et al.* [22] report that infants with a birth weight of > 4 kg had a two-fold increase in abnormality determined by ultrasonography. It has been reported that high birth weight for gestational age is an important but minor risk factor for DDH screening policies [23]. Most authors report that large, heavy infants

have an increased incidence of DDH [2, 9, 24–27]. In contrast, Lambeek *et al.* [28] found that birth weight was not related to DDH. Additionally, Sionek *et al.* [29] report no statistically significant relationship between birth weight and Graf hip joint type. In the present study, the mean birth weight was 3,250 g (range, 3,000–3,375 g) in Group I and 3,050 g (range, 2,880–3,460 g) in Group II. The mean birth weight in Group I was within the normal range. To the best of our knowledge, there have been no reports on DDH that have investigated the mother's anthropometric characteristics before pregnancy in addition to the infant's BMI. The combination of high BMI in the fetus with short maternal height may increase the risk of DDH through abnormal positioning of the hip joint in the intrauterine period.

The present study had certain limitations. First, the power analyses for the significant parameters were smaller than 80%. It would be ideal to perform a multicenter analysis to evaluate the effect of these parameters on DDH and increase the power of this study. Second, this study was limited by its relatively small number of subjects. A larger observational study or individual patient data meta-analysis might provide the number of patients needed to support or reject the hypothesis.

The natural history of DDH is not yet fully understood, and there are many unanswered questions. According to the present findings, it could be said that short maternal height and increased BMI in the fetus can be associated with a high incidence of DDH. To the best of our knowledge, this is the first study to investigate the relationship between MH and the BMI of the fetus. The precise etiology of DDH remains unknown, but genetic and environmental factors may act as internal or external influences. Although the role of the MH/I-BMI ratio has not yet been fully explained, it may contribute to a greater understanding of the etiology of DDH. Infants with mothers of short stature should be carefully assessed in the neonatal period for signs of DDH. Better phenotypic characterization and classification will be important for future analyses. In conclusion, the present results suggest that MH and infant BMI should be considered risk factors for DDH. Studies with larger series of various population groups are required to confirm and expand these findings as well as to further clarify the natural developmental history of hip dysplasia in infants.

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