Assessing the risk of asymptomatic dysplasia in parents of children with developmental hip dysplasia

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Abstract
Objective: The aim of this study was to investigate whether being the parents of children with developmental hip dysplasia (DDH) is a risk factor for asymptomatic dysplasia.

Methods: Asymptomatic parents of children who were diagnosed with DDH were assessed for presence of dysplasia by examining their anteroposterior pelvis radiographs at the neutral position. Eighty-six hips of 43 participants were included in the study group and 98 hips of 49 participants were included in the control group. Presence of hip dysplasia over the anteroposterior pelvis radiographs was analyzed for Wiberg’s angle, acetabular index of the weight-bearing zone (the Tönnis angle), acetabular depth/width index, femoral head coverage ratio (FCHR) and femoral neck/shaft angle.

Results: The mean acetabular depth/width ratio was 44.3% in the study group and 53.5% in the control group. And, the mean FCHR was 80% in the study group and 82% in the control group. There was a statistically significant difference between the two groups in terms of mean acetabular depth/width ratio (p < 0.05) and FCHR (p < 0.05). In addition, 21 participants in the study group and 2 in the control group had a pathological acetabular depth/width ratio. And, the number of participants with a pathological FCHR was 22 in the study group and 13 in the control group. A statistically significant difference was found between the two groups regarding the number of pathological measurements of acetabular depth/width ratio (p < 0.05) and FCHR (p < 0.05).

Conclusion: Having a parent with DDH is a definitive risk factor for the development of hip dysplasia in childhood. In addition, being a parent of a child with DDH is a risk factor for asymptomatic dysplasia. These parents should be screened by roentgenogram.

Level of Evidence: Level III, Diagnostic Study.

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Introduction

Developmental dysplasia of the hip (DDH) is a debilitating condition characterized by incomplete formation of the acetabulum and femoral head that occurs in different shapes at different ages. The most commonly accepted reason for the etiology, is the excessively loose joint capsule and the failure of ensuring the continuity of the femoral head within the acetabular cavity. There is not a single cause of DDH, but different predisposing factors have been described. These factors are: ligament laxity, intrauterine causes, prenatal and postnatal position, genetic causes, racial difference and environmental factors. Numerous studies have been conducted on hereditary transition of DDH. In genetic studies on DDH, it has been shown that the presence of DDH in one of the family members increases the risk five times for other family members. It can be said that when these hereditary factors are taken into consideration, parents of children with DDH also constitute a risky population.

In ideal treatment of DDH, early diagnosis can be made in presence of concentric reduction without causing epiphysis

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destruction and continuation of this reduction. If DDH is detected in the first years of life, the treatment can be often done easily and the development of the affected hip may exhibit normal results.\(^5\)\(^6\) However, DDH can progress silently until around 40–50 years of age and can be diagnosed when it shows symptoms such as pain, limitation of movement. In a radiology study on 2152 trauma patients, acetabular dysplasia was found as 9.3%.\(^7\)

A newborn should be examined physically and radiologically in more detailed way because of this familial transition. However, in our daily practice, the asymptomatic parent of a child with hip dysplasia is not screened physically and radiologically in terms of asymptomatic hip dysplasia. We realized that this reverse transition was not studied in the literature and wanted to initiate awareness toward this risky population.

What would be the benefit of this study? The current treatment approach for symptomatic elderly patients is often arthroplasty. On the other hand, arthroplasty is an invasive procedure for patients and it is economically very demanding in some countries. Early diagnosis of acetabular dysplasia may not be cured. However, in case dysplasia is detected in the asymptomatic phase, measures by lifestyle changes, weight control, exercise programs may be taken to delay the being symptomatic or reduce the need for surgical treatment in the future. Thus, the aim of this study was to investigate whether being the parents of children with DDH is a risk factor for asymptomatic dysplasia.

**Patients and methods**

The study protocol was approved by the Ethics Committee of Clinical Investigations (2/10/2014). This study was conducted in accordance with the principles of the Declaration of Helsinki.

The inclusion criteria were having a child diagnosed with Stage 2B, 3 or 4 DDH by ultrasonography, being between the ages of 20 and 40 or not having symptoms such as hip pain or joint motion limitation. Patients with a history of trauma or steroid use, DDH, metabolic bone disease, Perthes disease and symptoms such as hip joint pain or decreased joint motion were excluded.

Asymptomatic parents of the children who were diagnosed with DDH were assessed for presence of dysplasia between the years 2015 and 2017 by examining the anteroposterior pelvis radiographs at the neutral position. Eighty-six hips of 43 participants were included in the study group and 98 hips of 49 participants were included in the control group. The control group consisted of patients who presented with soft tissue trauma, lumbago and trauma. Anteroposterior pelvis radiographs of all participants from both groups were taken at the neutral position.

Presence of hip dysplasia over the anteroposterior pelvis radiographs was analyzed for Wiberg’s angle, acetabular index of the weight-bearing zone (the Tönnis angle), acetabular depth/width index, femoral head cover ratio (FHCR) and femoral neck/shaft angle (FNSA). The measurements were made by two radiologists, and the averages of the measurements were taken into account.

For Wiberg’s angle measurement, the line passing through both femur heads is plotted first. Then, the angle between the vertical line passing through the center of the femoral head and the line connecting the outer edge of the acetabulum to the center of the femoral head was measured as the Wiberg angle (Fig. 1). A value of lower than 20° has been associated with acetabular dysplasia.\(^8\)

For acetabular depth/width index measurement, a line was drawn connecting the superolateral and inferomedial edge of the acetabulum (width). Then, another line perpendicular to the first line was drawn (depth). Depth/Width*100 formula was used, and values greater than 38° were considered normal (Fig. 3).\(^10\)

Three vertical lines must be drawn for FHCR. The line passing through the medial edge of the weight-bearing surface of the

![Fig. 1. Calculation of the Wiberg’s angle.](image)

![Fig. 2. Calculation of the acetabular index (the Tönnis angle).](image)
acetabulum (1), the line passing through the lateral edge of the acetabulum (2), and the line passing through the lateral edge of the femoral head (3) (Fig. 4). The FHCR is then calculated by dividing the distance between the medial and lateral edge of the acetabulum to the distance between the medial edge of acetabulum and the lateral edge of femoral head, multiplied by 100. Values higher than 75% are considered normal.

The FNSA measurement is calculated by measuring the angle between the line parallel to the femoral head-neck and the line parallel to the femur shaft (Fig. 5). Values between 120° and 135° are considered normal.

Statistical analyses were performed using the SPSS v.21.0 software. The Mann–Whitney U and Wilcoxon signed-rank tests were used for mean values. The chi-square test was used to assess participants with pathological results. P values less than 0.05 were considered significant.

Results

The mean age of children at the DDH diagnosis was 2.2 (range, 2–3) months. 17 of these children were stage 2b, three children were stage 3 and two children were stage 4.

The mean age of 22 male participants in the study group was 34.6 (range, 25–39) years, while the mean age of 21 female participants was 33.1 years (range, 24–36) years.

In control group, the mean age of 25 male participants was 34.1 (range, 24–37) years, while the mean age of 24 female participants was 33.7 years (range, 25–36) years.

The mean acetabular depth/width ratio was 44.3% in the study group and 53.5% in the control group. There was a statistically significant difference between the two groups (p = 0.00) (Table 1).

Twenty-one participants in the study group and two in the control group had a pathological acetabular depth/width ratio and a statistically significant difference was found between the two groups (p = 0.00) (Table 2).

The mean FHCR was 80% in the study group and 82% in the control group, with a statistically significant difference between the two groups (p = 0.04) (Table 1). The number of participants with a pathological FHCR was 22 in the study group and 13 in the control group. A statistically significant difference was also found between these groups (p = 0.03) (Table 2).

The mean Wiberg’s angle was 34.2° in the study group and 33.8° in the control group. No statistically significant difference was found between the two groups (p = 0.25) (Table 1). Five
participants in the study group and three in the control group
had pathological Wiberg's angle. No statistically significant
difference between the two groups was detected (p = 0.32)
(Table 2).

The mean acetabular index was 6.8° in the study group and 7.6°
in the control group, with no statistically significant difference
between the groups (p = 0.06) (Table 1). Three participants in
the study group and five in the control group had pathological
acetabular indices. Again, no statistically significant difference was
detected between the two groups (p = 0.21) (Table 2).

The mean FNSA was calculated as 140.8° in the study group
and 134.5° in the control group. There was no statistically significant
difference between the two groups (p = 0.85) (Table 1). Thirty-five
participants in the study group and 33 in the control group had a
pathological FNSA, with no statistically significant difference being
detected between the two groups (p = 0.31) (Table 2).

Discussion

Developmental dysplasia of the hip covers a wide range of
anomalies ranging from a shallow acetabulum to a pseudo-
acetabulum. The natural course of DDH is well described in the
literature. It may show a silent progress up to around 40
acetabulum. The natural course of DDH is well described in the
anomalies ranging from a shallow acetabulum to a pseudo-

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