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Research article

Risk factors for long-term hip osteoarthritis in patients with hip dysplasia without surgical intervention

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ABSTRACT

Hip dysplasia is a common cause of hip pain and a known risk factor for hip osteoarthritis (OA) and early total hip arthroplasty (THA). Unfortunately, little is known about the specific factors associated with an increased risk of OA. The purpose was (i) to report the overall rate of symptomatic hip OA and THA and (ii) to identify radiographic features and patient characteristics associated with the development of symptomatic hip OA. A geographic database was used to identify all patients aged 14–50 years old diagnosed with symptomatic hip dysplasia between 2000 and 2016. Kaplan–Meier analysis was used to determine the rate of symptomatic hip OA, defined as a Tönnis grade of $\geq$1 on hip radiograph. Univariate and multivariate proportional hazard regression models were performed to determine risk factors for OA. One hundred and fifty-nine hips (144 patients) with hip dysplasia (52 F:107 M) out of 1893 patients with hip pain were included. Of these, 45 (28%) had severe hip dysplasia with a lateral center-edge angle $\leq$18°. Mean age at time of presentation was 26.1 ($\pm$10.1) years. Mean follow-up time was 8.2 ($\pm$5) years. The rate of OA was 20%. THA was performed in 11% of patients. Body mass index >29 ($P = 0.03$) and increased age ($P < 0.01$) were risk factors for OA. Patients with symptomatic hip dysplasia are at significant risk of developing hip OA. Body mass index >29 and age $\geq$35 years at the time of presentation with hip pain were risk factors for hip OA.

INTRODUCTION

Hip dysplasia results from a shallow acetabulum that can lead to hip instability and joint cartilage overload as the femoral head is not fully covered. The labrum is often hypertrophied to compensate for the shallow acetabulum. This lack of coverage can result in damage to the articular cartilage and labrum [1, 2]. Multiple studies have reported that hip dysplasia is among the most common causes of early-onset osteoarthritis (OA) [3–5]. These patients typically present with groin pain or feelings of hip instability and can be diagnosed based on anteroposterior pelvis radiographs demonstrating a Tönnis angle >10° and/or a lateral center-edge angle (LCEA) <25° [6]. Although it is well established that hip dysplasia is a common cause of hip OA, little is known about the specific factors associated with an increased risk of OA in this patient population.

Many risk factors for primary hip OA have been established. For example, increased body mass index (BMI) significantly increases the risk [7]. Conversely, smoking was shown to decrease the risk of OA in at least one study [8]. Structural deformities of the hip can lead to early-onset OA [6]. It has been estimated that hip dysplasia is the cause of hip OA in up to 20–40% of cases [1]. A recent study evaluating patients with femoroacetabular impingement found that BMI >29, increased age and male sex were associated with an increased risk of OA [9]. Hip dysplasia is more of a risk for OA than Femoroacetabular Impingement (FAI), yet specific risk factors for OA within a population of patients with hip dysplasia have not been evaluated [4]. Certain radiographic parameters and their association with OA have been evaluated within the hip dysplasia population. Murphy *et al.* [5] evaluated 286 hips and found that no patient with a lateral center-edge angle of <16°, an acetabular index of >15° or uncovering of the femoral head of >31% avoided a total hip arthroplasty (THA) into their 60s. Wyles *et al.* [4] compared patients with hip dysplasia, FAI and a control group in patients with contralateral THA. They found that patients with hip dysplasia were more likely to develop OA and do so at an accelerated rate. These studies did have limitations that are important to note. Both involved a cohort of patients that had undergone contralateral THA and had already shown a propensity progress to hip OA.

A study evaluating a large cohort of patients with hip dysplasia, treated nonoperatively and without contralateral THA, and identifying specific factors within this population that increase the risk of hip OA would be a valuable addition to the literature. This would prove helpful when counseling patients with hip dysplasia regarding their risk of developing OA. Therefore, the purposes

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of this study were (i) to report the overall rate of symptomatic hip OA and THA in patients with hip dysplasia without surgical intervention and (ii) to identify radiographic features and patient characteristics associated with hip OA.

METHODS

Institutional Review Board approval (17-004959, 030-OMC-17) was obtained prior to performing this study. We used the Rochester Epidemiology Project (REP) to study the patient cohort. The REP medical record database covers all patients in Olmsted County, MN, USA, who presented to a physician, regardless of the institution where the care was provided (population of 144 260 in 2010) [10]. The REP was queried to identify all patients between the ages of 14 and 50 years who presented with an International Classification of Diseases (ICD) 9 or 10 code (Table I) for hip pain between January 2000 and December 2016. Only patients with hip radiographs were included. Each patient's medical information was individually examined and logged using REDcap software (v9.1.0, Vanderbilt University, Nashville, TN, USA). Patients with a history of avascular necrosis, neuromuscular disorder, trochanteric bursitis, hip fracture, pelvic fracture, previous hip surgery and/or hip dislocation were excluded. All patients had radiographs evaluating for dysplasia at initial presentation to their physician with hip pain. Subsequent radiographs were obtained at the discretion of the evaluating physician. If patients were not symptomatic enough to present for additional medical care and radiographs, it was assumed that they did not develop symptomatic OA.

Each patient underwent anterior-posterior and lateral (cross-table, 45° Dunn, or frog-leg) view radiographs, and they were individually reviewed by an attending surgeon or senior level orthopedic surgery resident (JZ, HPM). Both reviewers evaluated the first 100 radiographs to standardize radiographic measurements. Hip dysplasia was diagnosed if patients had a Tönnis angle >10° and/or a LCEA <25°, which was based off of prior parameters established by Clohisy *et al.* [6].

Once hip dysplasia was confirmed, patients were followed by chart and/or radiographic review to determine whether they developed symptomatic hip OA. Patients who underwent hip preservation procedures were excluded. Subsequent physician visits were typically on an as-needed basis. Symptomatic hip OA was defined as hip symptoms significant enough to present to a physician and a Tönnis grade of ≥1 on hip radiograph. Tönnis grade was determined on any radiographic image. Patient factors, such as BMI, sex, smoking status, anti-inflammatory use, physical exam findings, diabetes, physical therapy participation and eventual surgery, were recorded during the chart review. Time from hip pain to follow-up and development of hip OA was recorded.

Table I. ICD 9 and 10 codes

Code	*Code description*
ICD-9	
719.45	Hip pain
719.85	Femoroacetabular impingement
719.95	Unspecified disorder of joint; pelvic region and thigh
ICD-10	
M25.559	Pain in unspecified hip
M25.551	Pain in hip
M25.859	Other specified joint disorders, unspecified hip
M25.852	Other specified joint disorders, hip

Statistical analysis

Patient comorbidities, demographics and radiographic measurements and their associated standard deviations and percentages of the population were reported for descriptive statistical purposes. Time to event was calculated as date of event or last follow-up date minus date of hip pain onset. Kaplan–Meier analysis was used to determine the rate of OA development. Kaplan–Meier analyses were then sub-analyzed by sex using a Wilcoxon test. Data were censored by OA event.

Proportional hazard regressions were used to determine risk factors associated with OA following the diagnosis of hip dysplasia. To determine the effect of comorbidity on OA development, comorbidity variables (sex, BMI >29, smoking status and diabetes) were analyzed with a multivariate hazard regression model. After being adjusted for comorbidities, univariate hazard models were performed on individual radiographic and procedural variables to determine individual risk factors for OA progression. Variables included in the analysis were as follows: LCEA ≤18° (severe dysplasia), LCEA 18°–24° (mild dysplasia), Tönnis angle ≥ 10°, Alpha angle > 55°, crossover sign, posterior wall sign, ischial spin sign and age of patient at time of pain ≥35 years. All variables were nominal and risk factors were presented as positive criteria for the variable.

A multivariate proportional hazard regression was performed on all variables to determine risk factors for OA. Kaplan–Meier analyses were then performed on the significant risk factors associated with OA. Alpha values were set to <0.05. Jmp Pro 14 (SAS Institute) was used for all analyses.

RESULTS

There were 159 hips (144 patients), out of 1893 patients evaluated for hip pain, with hip dysplasia (107 F:52 M) that had no surgical intervention. Twenty patients who had undergone hip preservation surgery were excluded. Mean follow-up time was 8.2 (±5) years. Patient characteristics are shown in Table II and radiographic findings are shown in Table III.

Rate of hip OA

Overall, 159 patients with hip dysplasia were analyzed and 32 hips (20%) developed symptomatic OA. Overall, 18 patients (11%) underwent THA. Mean survival time from presentation with pain until development of OA was 11.3 years (Fig. 1).

Risk factors for hip OA

Table IV shows the nonradiographic factors associated with OA. BMI >29 and increased age were risk factors for OA (BMI > 29: HR 2.52, $P = 0.03$; increased age: HR 4.79, $P < 0.01$.). Smoking, sex and diabetes mellitus were not significant risk factors. Radiographic risk factors for OA are shown in Table V. Patients with severe dysplasia and a Crossover Sign (COS) were found to be statistically significant.

Table II. Patient characteristics

	Total cohort (n = 159)	*OA (n = 32)*
Age at date of pain (years)	26.1 (10.1)	35.2 (4.7)
Contralateral diagnosis	15 (9%)	2 (6%)
Sex		
Male	52 (33%)	15 (47%)
Female	107 (67%)	17 (53%)
Laterality		
Left	65 (41%)	17 (53%)
Right	94 (59%)	15 (47%)
Comorbidities		
BMI	27.4 (5.1)	30.1 (4.5)
Smoker	27 (17%)	10 (31%)
Diabetes	6 (4%)	2 (6%)
Follow-up time after pain (yr.)	8.2 (5.2)	5.3 (3.9)
Follow-up time after first radiograph (yr.)	25.9 (12.1)	31.7 (12.3)

Values in parenthesis are presented as mean (SD) or *n* (% of cohort).

Table III. Radiographic findings

	Total cohort (n = 159)	*OA (n = 32)*
LCEA > 24° (Tönnis ≥ 10°)	26 (16%)	5 (16%)
LCEA 18°–24° (mild dysplasia)	87 (55%)	7 (22%)
LCEA ≤ 18° (severe dysplasia)	45 (28%)	20 (63%)
Tönnis angle ≥10°	124 (78%)	28 (88%)
Alpha angle >55°	116 (73%)	26 (81%)
Positive crossover sign	99 (62%)	16 (50%)
Positive posterior wall sign	119 (75%)	25 (78%)
Positive ischial spine sign	66 (42%)	14 (44%)

Values in parenthesis are presented as mean (SD) or *n* (% of cohort).

DISCUSSION

Hip dysplasia is a known risk factor for OA, and many patients require THA early in life. There is a paucity of research recognizing risk factors within this population that increase the risk of OA. The present study identified nonradiographic and radiographic risk factors for OA in patients with hip dysplasia. BMI >29 and increased age at the time of presentation with hip pain were risk factors for hip OA. Patients with severe hip dysplasia (LCEA ≤ 18°) were also at an increased risk of hip OA.

In this study, the rate of hip OA was 20%. Overall, 11% of patients underwent a THA. A few studies have evaluated the natural history of the dysplastic hip [4, 5]. Murphy *et al.* [5] evaluated 286 patients with hip dysplasia who had a contralateral THA. They followed the contralateral hip to determine the rate of severe OA before the age of 65 years. Ultimately, around 40% of patients developed severe OA before 65 years. This cohort was older than the cohort in our study and many patients demonstrated hip OA at the initiation of the study. In addition, all patients had a contralateral THA, which is a clear risk factor for contralateral hip OA and need for THA. Another notable study by Wyles and colleagues [4] also evaluated a cohort of patients with hip dysplasia and contralateral THA. They found that 29% of patients went on to THA at 20 years and 43% went on to THA at 30 years. Their rate of OA is slightly higher than that found in our study. This can likely be attributed to longer follow-up, increased mean age and the fact that all patients in their study had undergone contralateral THA, which is an inherent risk factor. With longer follow-up, our study may find similar rates of OA.

The present study identified nonradiographic characteristics that increased the risk of OA in patients with hip dysplasia. BMI >29 and increased age at the time of presentation with hip pain were risk factors for hip OA. These are not surprising findings, but, to our knowledge, this had not yet been demonstrated in patients with hip dysplasia. In the general population, increased BMI has been shown to increase the risk for hip OA and our study is consistent with those findings [7, 11]. Patients

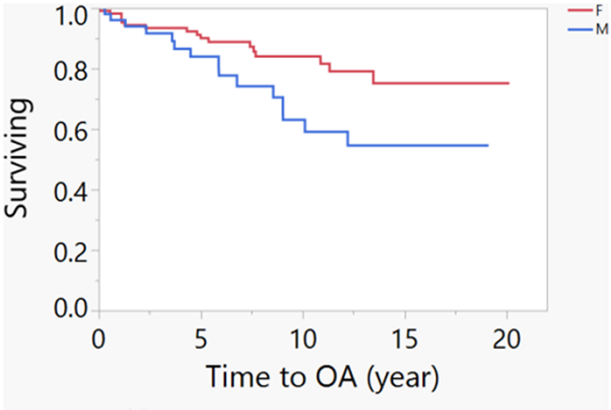

Fig. 1. Kaplan–Meier survival analysis for OA by sex [female (F) is top line, male (M) is bottom line].

Table IV. Nonradiographic risk factors for hip OA

	Hazard ratio	*95% CI*		*P-value*
Male sex	1.54	0.73	3.25	0.26
BMI > 29	**2.52**	**1.10**	**5.75**	**0.03**
Smoker	1.73	0.81	3.71	0.16
Diabetes	0.86	0.20	3.64	0.83
Age ≥ 35	**4.79**	**2.23**	**9.89**	**0.00**

[a]Boldface signifies statistical significance.

Table V. Radiographic risk factors for hip OA

	Hazard ratio	*95% CI*		*P-value*
Radiographic				
LCEA > 24° (Tönnis ≥ 10°)	0.82	0.31	2.12	0.68
LCEA 18°–24° (Mild dysplasia)	0.21	0.09	0.51	0.00
LCEA ≤ 18° (Severe dysplasia)	**4.94**	**2.40**	**10.17**	**0.00**
Tönnis angle ≥ 10°	2.00	0.71	5.80	0.18
Alpha angle > 55°	1.007	0.41	2.49	0.99
Positive crossover sign	0.37	0.19	0.75	0.01
Positive posterior wall sign	1.23	0.53	2.87	0.62
Positive ischial spine sign	1.17	0.58	2.365	0.6576

[a]Boldface signifies statistical significance.

who presented with hip pain at a later age were at an increased risk of OA. It is possible that older patients were less likely to be offered hip preservation intervention and had further progression of hip damage by time of presentation. However, Sohatee *et al.* reported increasing age as a risk factor for increased rates of conversion to THA in patients with dysplasia following hip preservation surgery [12]. Smoking was not found to be a risk factor for OA. Similar findings have been found in general population studies as smoking has been shown to be protective against hip OA [8]. It should be noted that a recent study evaluating patients with FAI also found that increased age and BMI were risk factors for hip OA [9]. Interestingly, the study on FAI patients found that males were at an increased risk of OA; however, that was not the case in this dysplasia cohort. The findings in the present study can be used to counsel patients with hip dysplasia on relative risk factors for hip OA.

Patients with severe hip dysplasia, defined as a LCEA $\leq 18^{\circ}$, were at an increased risk of hip OA. This was the only radiographic finding to significantly increase the risk of OA. This is an expected finding as prior studies have demonstrated that patients with more severe radiographic features of dysplasia are at an increased risk of hip OA [4, 5]. Notably, a contemporary systematic review reporting on the radiographic factors associated with hip OA identified acetabular undercoverage as the most strongly associated factor cited in the literature for both the development and progression of hip OA [13]. We believe this may have important therapeutic implications. For example, if a patient with a LCEA $\leq 18^{\circ}$ underwent a hip preservation procedure to increase their LCEA, their risk of OA could be decreased. In fact, a recent study did demonstrate that PAO effectively decreased the risk of progression of hip dysplasia to OA [14].

Limitations of this study must be considered. There was no control group and it was retrospective. As the follow-up time and mean age of the cohort increases, we would expect the rate of OA to increase. In addition, there were likely patients who developed OA yet had not become symptomatic, so they would not have been captured in this study. Some patients did have OA at initial evaluation, and we did not track the progression of OA through Tönnis grades. Unfortunately, there were not enough patients who went on to THA to allow for further analysis of the specific risk factors for THA. Although the fact that these patients were treated nonoperatively allows us to follow the natural history of their hips, it is likely true that many of them should have been treated surgically. We suspect that hip dysplasia was only recognized or diagnosed on our retrospective review in many cases. Despite these limitations, this study identified both radiographic and nonradiographic risk factors for hip OA in a cohort of patients with hip dysplasia. To our knowledge, it is the first large cohort study to evaluate the rate of hip OA in a group of hip dysplasia patients who had not undergone contralateral THA.

CONCLUSION

This large geographic cohort study demonstrated that patients with symptomatic hip dysplasia are at significant risk of developing hip OA. At mean follow-up of 8 years, 20% of hips had symptomatic OA and 11% underwent THA. BMI >29 and age ≥ 35 years at the time of presentation with hip pain were risk factors for hip OA. Patients with LCEA $\leq 18^{\circ}$ were also at an increased risk of hip OA.

DATA AVAILABILTY

The data underlying this article are available in the article and in its online supplementary material.

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CONFLICT OF INTEREST STATEMENT

None declared.

REFERENCES

1. Gala L, Clohisy JC, Beaule PE. Hip dysplasia in the young adult. *J Bone Joint Surg Am* 2016; **98**: 63–73.
2. LaPrade MD, Melugin HP, Hale RF *et al.* Incidence of hip dysplasia diagnosis in young patients with hip pain: a geographic population cohort analysis. *Orthop J Sports Med* 2021; **9**: 2325967121989087.
3. Jacobsen S, Sonne-Holm S. Hip dysplasia: a significant risk factor for the development of hip osteoarthritis. A cross-sectional survey. *Rheumatology* 2005; **44**: 211–8.
4. Wyles CC, Heidenreich MJ, Jeng J *et al.* The John Charnley Award: redefining the natural history of osteoarthritis in patients with hip dysplasia and impingement. *Clin Orthop Relat Res* 2017; **475**: 336–50.
5. Murphy SB, Ganz R, Muller ME. The prognosis in untreated dysplasia of the hip. A study of radiographic factors that predict the outcome. *J Bone Joint Surg Am* 1995; **77**: 985–9.
6. Clohisy JC, Carlisle JC, Beaule PE *et al.* A systematic approach to the plain radiographic evaluation of the young adult hip. *J Bone Joint Surg Am* 2008; **90 Suppl 4**: 47–66.
7. Jiang L, Rong J, Wang Y *et al.* The relationship between body mass index and hip osteoarthritis: a systematic review and meta-analysis. *Joint Bone Spine* 2011; **78**: 150–5.
8. Allen KD, Golightly YM. State of the evidence. *Curr Opin Rheumatol* 2015; **27**: 276–83.
9. Melugin HP, Hale RF, Zhou J *et al.* Risk factors for long-term hip osteoarthritis in patients with femoroacetabular impingement without surgical intervention. *Am J Sports Med* 2020; **48**: 2881–6.
10. Rocca WA, Yawn BP, St Sauver JL *et al.* History of the Rochester epidemiology project: half a century of medical records linkage in a US population. *Mayo Clin Proc* 2012; **87**: 1202–13.
11. Lievense AM, Bierma-Zeinstra SM, Verhagen AP *et al.* Influence of obesity on the development of osteoarthritis of the hip: a systematic review. *Rheumatology* 2002; **41**: 1155–62.
12. Sohatee MA, Ali M, Khanduja V *et al.* Does hip preservation surgery prevent arthroplasty? Quantifying the rate of conversion to arthroplasty following hip preservation surgery. *J Hip Preserv Surg* 2020; **7**: 168–82.
13. Shapira J, Chen JW, Bheem R *et al.* Radiographic factors associated with hip osteoarthritis: a systematic review. *J Hip Preserv Surg* 2020; **7**: 4–13.
14. Wyles CC, Vargas JS, Heidenreich MJ *et al.* Natural history of the dysplastic hip following modern periacetabular osteotomy. *J Bone Joint Surg Am* 2019; **101**: 932–8.