Original Research Article

HIP International

HIP International
2019, Vol. 29(5) 527–534

DOI: 10.1177/1120700018813829
journals.sagepub.com/home/hpi
SAGE

Diabetic hip arthropathy is associated with a higher prevalence of femoral head chondromalacia: a case-controlled study

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Abstract

Introduction: No previous studies have characterised hip joint disease in diabetic patients undergoing hip arthroscopy. The purpose of our study was to evaluate intra-articular hip pathology and surgical variables in patients with diabetes compared to matched, non-diabetic controls. We hypothesised that diabetic patients would demonstrate a higher prevalence and severity of hip chondral pathology.

Methods: We retrospectively reviewed 795 consecutive hip arthroscopies performed by a single surgeon between 2010 and 2015. Patients ⩾18 years of age without a history of diabetes served as controls and were matched based on age, sex, body mass index, duration of symptoms, and operative side. Clinical symptoms, preoperative physical examination, and radiologic and intraoperative findings were assessed. The primary outcomes were the acetabular and femoral head chondromalacia index (CMI), calculated as the product of the Outerbridge chondromalacia grade and surface area (mm^2*severity).

Results: 15 diabetic patients were matched to 137 non-diabetic controls. Diabetic patients demonstrated a higher prevalence of femoral head chondromalacia compared to controls both on magnetic resonance imaging (45.5% vs. 7.5%, $p = 0.002$) and during arthroscopy (100% vs. 75.9%, $p = 0.042$). Femoral head chondromalacia in diabetic patients had higher Outerbridge grade (2.4 vs. 2.0, $p = 0.030$) but similar CMI (513.0 vs. 416.4, $p = 0.298$) compared to controls.

Discussion: Femoral head chondral pathology was more prevalent and of higher severity grade in diabetic patients. The prevalence, size, and severity of acetabular chondral disease were similar between diabetic and non-diabetic patients. Multivariate analysis demonstrated that diabetic status was independently associated with the presence of femoral head chondromalacia.

Keywords

Chondromalacia, diabetic arthropathy, diabetes mellitus, hip arthroscopy, hip pathology, hip preservation

Date received: 23 February 2018; accepted: 23 July 2018

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Introduction

Diabetes mellitus is a largely prevalent disease in the United States that leads to microvascular damage and systemic disease with end organ involvement.[1] Diabetes is an important risk factor for cartilage degradation and osteoarthritis in the hip and knee joints,[2–4] and basic science studies support more chondral pathology in diabetic models.[5] Advanced glycation end-products (AGEs) accumulate in cartilage and joint tissues during normal aging,[6] but AGE formation may occur at an accelerated rate in diabetic patients.[7]

In patients with diabetes, hip arthropathy remains poorly described and presents diagnostic and treatment challenges. Several factors may contribute to diabetic arthropathy of the hip, including intra-articular chondral damage and neuropathic etiology. To our knowledge, no studies have characterised the pattern of hip joint disease in the diabetic patient undergoing hip arthroscopy. The purpose of this study was to evaluate intra-articular hip pathology and surgical variables in patients with diabetes compared to a matched, non-diabetic group. We hypothesised that diabetic patients would demonstrate a higher prevalence and greater degree of hip chondral pathology.

Methods

A prospective database of 795 consecutive hip arthroscopies performed between 2010 and 2015 by a single, fellowship-trained orthopaedic surgeon was reviewed retrospectively. Patients were enrolled in an Institutional Review Board-approved study prior to surgery. Patients over 18 years of age were included for analysis if they presented with unilateral hip pain due to chondrolabral pathology that failed to improve after 12 weeks of conservative treatment. Patients under 18 years of age, those who had a previous ipsilateral hip surgery, or those with a history of developmental dysplasia of the hip, avascular necrosis of the hip, Legg-Calvé-Perthes disease, or slipped capital femoral epiphysis were excluded. Diabetic patients (type 1 or 2) were matched to a control, non-diabetic group. After running initial descriptive statistics of the diabetic group, parameters for the continuous variables of age, body mass index (BMI), and duration of pain symptoms were applied to the control group for matching. Secondary matching based on categorical variables of sex, and operative side was performed to further ensure that the 2 groups were well-matched.

Information regarding subjective pain symptoms (location, presence of mechanical symptoms, timing of the pain, and exacerbating factors), analgesic medications, preoperative physical examinations, radiographic markers, and intraoperative findings was collected for each patient. Patient-reported pain symptoms were characterised by location, timing, exacerbating variables, and the presence of mechanical symptoms. Subjective pain symptoms and preoperative physical examination findings were prospectively collected by the senior author in a standardised fashion during the initial consultation. On plain radiographs, patients who demonstrated evidence of hip osteoarthritis (Tönnis grade > 1) were excluded. Magnetic resonance imaging (MRI) was performed on all patients prior to hip arthroscopy and interpreted by both a musculoskeletal radiologist and the senior author. Chondromalacia, even partial thickness lesions, on MRI is demonstrated by focal areas of hyperintensity in the cartilage on non-fat-suppressed fast spin echo sequence. Our primary outcome variable was the chondromalacia severity index (CMI), a previously described metric that is calculated as a product of the Outerbridge chondromalacia grade during hip arthroscopy and the affected surface area of the lesion estimated using an arthroscopic ruler (mm^2*severity) (Figure 1).[8] In patients with multiple chondral defects, the summation of the calculated CMI of each lesion was used for analysis.

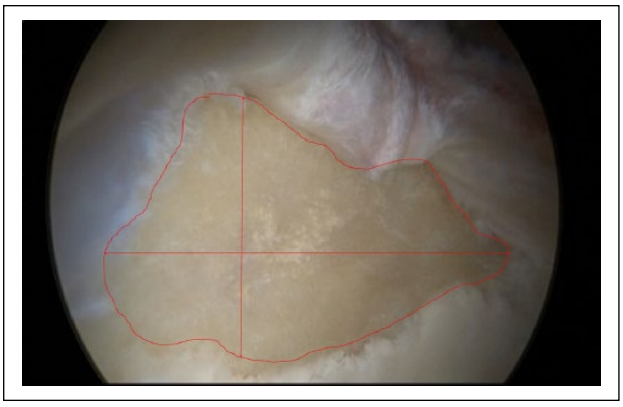

Figure 1. Arthroscopic view of an acetabular chondral lesion. The affected surface area is estimated using an arthroscopic ruler and multiplied by the Outerbridge grade of the lesion to obtain the chondromalacia severity index (CMI).

Statistical analysis

Univariate analysis was performed for patient demographics, pain symptoms, and physical examination/radiographic/intraoperative findings. Fisher's exact test was performed for categorical variables when appropriate, and Student's *t*-test was performed for continuous variables. Spearman's rho was used to determine correlations. A stepwise multivariate linear regression analysis was performed to account for potential differences and confounders in demographics between the diabetic and non-diabetic groups. Results were reported as means $\pm$ standard deviations, odds ratios, 95% confidence intervals, and p values. A *post hoc* power analysis was performed to minimise beta error. Statistical significance was denoted by $p \leqslant 0.05$.

Analysis was conducted using SPSS software (Version 22.0, IBM Corp., Armonk, NY).

Results

A total of 152 patients met inclusion criteria, with 15 diabetic patients (8 Type 1 and 7 Type 2) and 137 non-diabetic controls matched for continuous variables (age, BMI, and duration of symptoms) (Figure 2) (Table 1). Secondary matching based on categorical variables demonstrated similar ratios of sex and operative side.

Both groups reported similar pain symptoms with respect to activity and exacerbation and similar rate of analgesic usage with respect to nonsteroidal anti-inflammatory drugs (NSAIDs) and opioids (Table 2). A greater number of diabetic patients reported posterolateral hip pain compared to control patients, while other pain locations and exacerbating symptoms were grossly similar between the 2 groups.

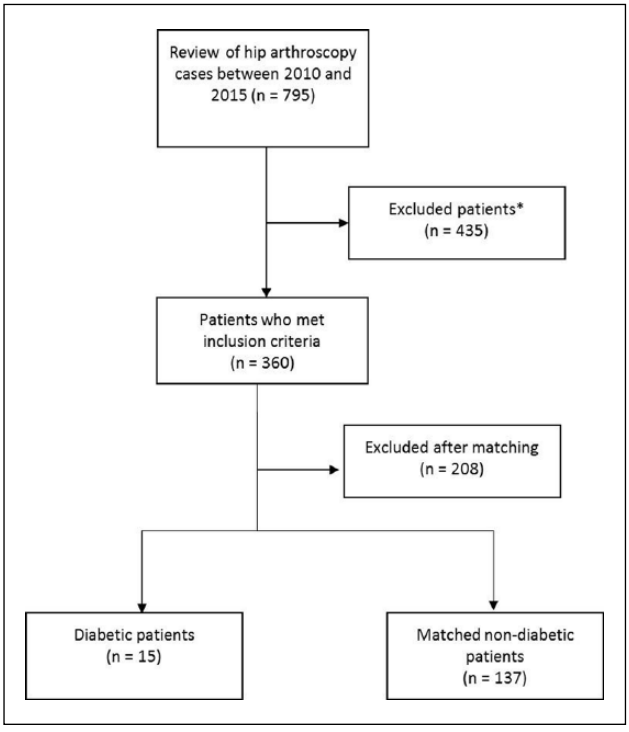

Figure 2. Flow diagram of patients who were included for analysis. *Patients were excluded for age under 18 years, previous ipsilateral hip surgery, congenital hip pathology, prior history of hip avascular necrosis or slipped capital femoral epiphysis, and Tönnis grade > 1.

On physical examination, the 2 groups exhibited similar rates of sacroiliac and greater trochanter tenderness (Table 3). Diabetic patients demonstrated significantly greater perpendicular distance from the lateral geniculate line to the exam table on FABER figure-4 testing.[9] Hip internal rotation on the operative extremity was greater in the control group, but this was not statistically significant.

Radiographic evaluation demonstrated significantly greater lateral and anterior center edge angles and smaller Sharp's angle in diabetic patients in comparison to controls (Table 4). The Tönnis grade and alpha angle did not significantly differ between the 2 groups. On MRI, diabetic patients demonstrated a significantly higher prevalence of femoral head chondral pathology compared to controls. The correlation between diabetes and femoral head chondromalacia on MRI demonstrated a positive Spearman's coefficient ($r = 0.352$, $p < 0.001$). Conversely, nearly all patients in the control group demonstrated acetabular chondromalacia on MRI, which negatively correlated with diabetic status ($r = -0.379$, $p < 0.001$). Intraoperatively, however, acetabular chondromalacia was seen in all patients in both groups (Table 5). The Outerbridge grade, surface area, and the calculated CMI were similar between the groups on intraoperative findings.

The higher prevalence of femoral head chondromalacia in diabetic patients observed on MRI was confirmed during arthroscopy (Table 5). The mean Outerbridge grade of the major femoral head chondral lesion was significantly greater in diabetic patients; however, the calculated CMI was statistically similar between the 2 groups of patients. Multivariate regression modeling demonstrated that diabetic status was a significant positive predictor of the presence of femoral head chondromalacia independent of all other patient-centered variables (age, sex, BMI, operative side, duration of symptoms) ($r = 0.175$, $p = 0.033$). Multivariate regression analysis also demonstrated that diabetic status and BMI were significant positive predictors of femoral head Outerbridge chondromalacia grade

Table 1. Diabetic and control group demographics comparison.

	Diabetic ($n = 15$)	Non-diabetic ($n = 137$)	p value
Age (years, mean ± SD)	37.7 ± 9.5	37.8 ± 9.3	0.971
Sex (% female)	60.0	62.0	1.000
BMI (kg/m^2, mean ± SD)	28.8 ± 5.2	29.4 ± 6.0	0.695
Operative extremity (% right)	60.0	54.0	0.787
Duration of pain symptoms (months, mean, range)	31.7 (4–111)	33.4 (1–192)	0.841

SD, standard deviation; BMI, body mass index.

Table 2. Patient-reported pain symptoms and analgesic usage.

	Diabetic (%)	Non-diabetic (%)	*p* value
Anterior hip pain	66.7	50.4	0.283
Anterolateral hip pain	46.7	48.1	1.000
Lateral hip pain	20.0	15.3	0.708
Posterolateral hip pain	46.7	14.8	**0.007**
Posterior hip pain	13.3	14.6	1.000
Groin pain	13.3	6.0	0.268
Mechanical symptoms	60.0	60.9	1.000
Pain with sitting	86.7	91.9	0.620
Pain with walking	80.0	88.2	0.406
Pain with rising from seated position	85.7	73.7	0.519
Pain with crossing legs	92.9	89.4	1.000
Pain at night	86.7	72.6	0.356
NSAIDs use	80.0	90.7	0.196
Narcotic use	40.0	25.6	0.236

NSAIDs, non-steroidal anti-inflammatory drugs.

Table 3. Comparison of physical examination findings between diabetic and control groups.

	Diabetic	Non-diabetic	*p* value
Operative side			
Sacroiliac tenderness (%)	73.3	72.6	1.000
Greater trochanter tenderness (%)	73.3	66.4	0.774
Terminal hip flexion (degrees, mean ± SD)	85.3 ± 9.0	89.2 ± 11.6	0.213
Internal rotation (degrees, mean ± SD)	1.7 ± 3.1	5.6 ± 10.0	0.131
FABER test (cm, mean ± SD)	33.6 ± 10.7	27.6 ± 9.9	**0.040**
Straight leg raise (maximum 5, mean ± SD)	3.9 ± 0.7	4.0 ± 0.7	0.598
Nonoperative side			
Sacroiliac tenderness (%)	13.3	25.2	0.524
Greater trochanter tenderness (%)	20.0	19.3	1.000
Terminal hip flexion (degrees, mean ± SD)	97.5 ± 5.7	99.0 ± 7.8	0.460
Internal rotation (degrees, mean ± SD)	6.7 ± 4.9	10.9 ± 9.9	0.112
FABER test (cm, mean ± SD)	17.8 ± 7.7	18.2 ± 8.3	0.876
Straight leg raise (maximum 5, mean ± SD)	5.0 ± 0	4.8 ± 0.4	0.079

SD, standard deviation.
Note: FABER test was measured as the perpendicular distance from the lateral femoral condyle to the exam table with the affected extremity in the figure-4 position.

($r = 0.294$, $p = 0.006$). With respect to arthroscopic procedures, a greater percentage of diabetic patients underwent femoral head microfracture compared to controls.

With respect to hip morphology, more diabetic patients were diagnosed with pincer pathology compared to controls (100% vs. 72.9%, $p = 0.037$). Intraoperatively, acetabuloplasty was performed in 93.3% of the diabetic patients. Significantly fewer diabetic patients were preferentially treated with acetabular microfracture (6.7% vs. 31.4% of controls, $p = 0.035$) for acetabular chondral pathology, while control patients were preferentially treated with acetabular chondroplasty (93.3% vs. 67.9% of diabetics, $p = 0.031$). Similar rates of labral tears were found in both the diabetic group (93.3%) and the control group (92.7%, $p = 1.000$). In the diabetic group, 93.3% of patients were found to have at least partial tearing of the labral cartilage compared to 92.7% of patients in the control group ($p = 1.000$). The mean grade of the labral tears (1 = partial tear, 2 = complete tear) was not significantly different between the diabetic (1.4 ± 0.5) and control groups (1.3 ± 0.5, $p = 0.250$). There were no significant differences between the diabetic and non-diabetic groups with respect to the rates of labral repair (86.7% vs. 87.6%, $p = 1.000$), labral debridement (13.3% vs. 13.1%, $p = 1.000$), femoral head chondroplasty (53.3% vs. 70.1%, $p = 0.242$), ligamentum teres debridement (66.7% vs. 80.3%, $p = 0.313$), loose body excision (0% vs. 10.9%, $p = 0.364$), and iliopsoas release (13.3% vs. 19.0%, $p = 0.739$).

Table 4. Comparison of radiological findings between diabetic and control groups.

	Diabetic	Non-diabetic	*p* value
Tönnis grade (mean ± SD)	0.9 ± 0.3	0.8 ± 0.4	0.236
Lateral center edge angle (mean ± SD)	40.3 ± 11.4	31.1 ± 7.8	**0.008**
Anterior center edge angle (mean ± SD)	40.3 ± 10.1	34.3 ± 8.9	**0.017**
Sharp's angle (mean ± SD)	36.0 ± 4.1	40.1 ± 4.0	**<0.001**
Alpha angle (mean ± SD)	63.7 ± 7.2	64.9 ± 5.6	0.548
Acetabular chondromalacia on MRI (%)	63.6	96.4	**0.002**
Femoral head chondromalacia on MRI (%)	45.5	7.5	**0.002**
Presence of acetabular subchondral cyst (%)	0	3.3	0.826
Presence of acetabular paralabral cyst (%)	9.1	2.2	0.178
Presence of femoral subchondral cyst (%)	9.1	0	0.099

SD, standard deviation; MRI, magnetic resonance imaging.

Table 5. Evaluation of chondromalacia during hip arthroscopy in diabetic and control patients.

	Diabetic	Non-diabetic	*p*-value
Acetabular chondromalacia (%)	100	100	n/a
Grade of primary acetabular chondral lesion (mean ± SD)	2.3 ± 0.6	2.7 ± 0.9	0.131
Area of primary acetabular chondral lesion (mm^2, mean ± SD)	136.7 ± 59.8	141.1 ± 80.0	0.835
Acetabular CMI (mm^2*severity)	364.2 ± 263.4	442.6 ± 344.1	0.394
Microfracture of acetabulum (%)	6.7	31.4	0.035
Chondroplasty of acetabulum (%)	93.3	67.9	0.031
Femoral head chondromalacia (%)	100	75.9	0.042
Grade of primary femoral head chondral lesion (mean ± SD)	2.4 ± 1.0	2.0 ± 0.7	0.030
Area of primary femoral head chondral lesion (mm^2, mean ± SD)	204.5 ± 127.9	165.2 ± 105.4	0.199
Femoral head CMI (mm^2*severity)	513.0 ± 285.3	416.4 ± 316.6	0.298
Microfracture of femoral head (%)	33.3	2.9	<0.001
Chondroplasty of femoral head (%)	53.3	70.1	0.242

SD, standard deviation; CMI, chondromalacia severity index.

Post hoc power analysis for 2-tailed *t*-test comparing 2 independent means with the following parameters: effect size of 0.8, $\alpha \leqslant 0.05$, sample sizes of 15 and 137 yielded power of 0.832. *Post hoc* power analysis for contingency table chi-square test comparing 2 independent ratios with the following parameters: effect size of 0.3, $\alpha \leqslant 0.05$, total sample sizes of 152, and degrees of freedom of 5 yielded power of 0.829.

Discussion

The purpose of this study was to examine the presence of intra-articular hip pathology at the time of hip arthroscopy in diabetic patients. Secondarily, we aimed to compare the degree of chondral damage within the hip joint in diabetic patients compared to non-diabetic controls. Most significantly, our results demonstrated that at the time of hip arthroscopy, diabetic patients demonstrated higher prevalence and Outerbridge grade of femoral head chondromalacia compared to controls. Controlling for all other patient-centered variables, diabetic status was found to be a positive independent predictor of the presence and grade of femoral head chondromalacia. This finding supports our hypothesis that chondral pathology is more common in diabetic patients who present with hip pain compared to a matched group of non-diabetic patients. The size and the calculated severity of the chondral lesions were not statistically different between groups, but overall, diabetic patients who present with hip pain should be identified as higher risk for the presence of chondromalacia of the femoral head and treated accordingly. With respect to arthroscopic intervention, the diabetic patients in the present study more frequently underwent microfracture of the femoral head compared to control patients.

Diabetes is known to result in systemic disease and can lead to chondral pathology.[10] On clinical presentation, pain symptoms were generally found to be similar in diabetic and non-diabetic patients in this study. Diabetic patients

more frequently reported pain in the posterolateral aspect of the hip and demonstrated more limited mobility on the FABER test, as indicated by a greater perpendicular distance from the leg to the table, compared to controls. The localisation of their pain symptoms may be related to acetabular overcoverage as reflected by the greater lateral and anterior center edge angles and smaller Sharp's angle in the diabetic group. This pincer-type pathology was confirmed in all 15 diabetic patients intraoperatively; however, given the nature of the present study, it is difficult to explain the difference in acetabular coverage between the 2 groups. Hip morphology is known to affect the pattern of damage to articular cartilage.[11,12] Given the acetabular overcoverage in the diabetic patients, all but 1 patient underwent acetabuloplasty. This study further supports the concept that pincer lesions are effectively chondroprotective of the acetabular surface, because the overhang limits femoral motion against the chondral surface.

The effect of diabetes mellitus in the pathogenesis of cartilage disease is difficult to elucidate.[4] Type 2 diabetes is strongly associated with increased age and obesity, 2 important factors in the development of osteoarthritis.[13–16] It has long been established that a combination of metabolic factors and patient characteristics contribute to osteoarthritis. A recent multicentre study demonstrated sex differences in hip morphology and presentation in patients undergoing hip arthroscopy.[17] Recent evidence further suggested that age and sex are predictive of outcome after hip arthroscopy and conversion to total hip arthroplasty.[18,19] By matching the groups based on non-modifiable patient factors (age, sex) and BMI, we hoped to evaluate diabetes as an independent factor in the presentation of symptomatic hip pathology requiring arthroscopic intervention. The use of a stepwise multivariate linear regression model controlled for other associated patient-centred variables and demonstrated that the risk of femoral head chondromalacia increased with diabetes independently. On both MRI and intraoperative assessment, the diabetic group demonstrated higher rate of chondral damage in the femoral head. The use of MRI was helpful in the preoperative assessment of chondral involvement and correlated with intraoperative findings at the femoral head; however, MRI detected femoral head chondromalacia in less than half of the diabetic patients, whereas the diagnosis was confirmatory in all diabetic patients intraoperatively. Acetabular chondromalacia was more frequently diagnosed in control patients on MRI; however, intraoperative assessment revealed that all patients in this study showed some evidence of acetabular chondromalacia. Gold et al.[20] previously demonstrated that the sensitivity of detection of chondral damage is variable on MRI but is significantly improved with MR arthrography. Pathologic findings on MRI, however, can be asymptomatic in certain individuals, as Duthon et al.[21] demonstrated in female professional ballet dancers.

Various pre-clinical studies have demonstrated that elevated blood glucose levels adversely affect chondrocyte metabolism, leading to destruction of joint cartilage.[22–24] In the present study, only patients with Tönnis grade 0 or 1 were included for analysis to exclude advanced osteoarthritis as a confounding variable. The natural progression of chondral pathology and osteoarthritis in the diabetic patient population was beyond the scope of this study, because only patients who underwent hip arthroscopy were analysed. The extent of the chondral defect often dictates the surgical procedures used during hip arthroscopy.[25,26] As expected with the intraoperative findings, microfracture of the femoral head was performed with greater frequency in diabetic patients. On the acetabular side, diabetic patients underwent chondroplasty at a higher rate, while control patients underwent microfracture at a higher rate. Although not statistically significant, the greater Outerbridge grade of acetabular chondromalacia in control patients influenced the intraoperative decision-making. Numerous authors have demonstrated that the presence of full thickness chondral defects with associated labral tears is associated with poor clinical outcomes after hip arthroscopy,[12,27–29] which suggest a worse prognosis for the femoral head in patients with diabetes than non-diabetic patients. In our study, however, diabetes was not associated with greater incidence or severity of labral tears. A recent longitudinal study also suggested that diabetes may be a predictive factor in the development of OA requiring hip and knee arthroplasty independent of age, sex, and BMI,[2] further highlighting the need to counsel diabetic patients preoperatively with respect to surgical treatment expectations. This is further supported by the results of our multivariate regression model, demonstrating diabetic status as independent predictive factors for femoral head chondromalacia.

The molecular pathways leading to diabetic arthropathy of the hip have not been as well studied as in the knee.[10] At the molecular level, excess intracellular glucose in chondrocytes leads to secondary pathways that induce oxidative stress via formation of reactive oxygen species (ROS).[1,30] Advanced glycation end-products result from persistently elevated glucose and lead to a perpetual inflammatory state including cartilage degradation,[7,24,31,32] which may explain the increased prevalence of femoral head chondromalacia; however, this association was beyond the scope of the present study and warrants further investigation to better understand the association between symptomatic hip pain and chondral pathology in diabetic patients.

Our study carries inherent limitations due to the retrospective nature of the study and small size of the diabetic group. While hip pain affects a wide range of patients, diabetic patients made up only a minority of the patient population presenting to our institution for hip arthroscopy. The small sample size of diabetic patients increases the risk of

beta error; however, our *post hoc* power analysis revealed that our study was adequately powered for comparing continuous and categorical data, which included frequency of femoral head chondromalacia. A larger diabetic group may allow for the detection of other findings (hip flexion and internal rotation deficits in diabetic patients) that did not reach statistical significance in the present study. The nature of the study prevented us from capturing diabetic patients with hip pain who did not undergo arthroscopy, which may provide, at minimum, additional clinical and radiographic data to assess hip range of motion, pain characteristics, and MRI findings. No information was collected regarding the extent of each patient's diabetes (e.g. aetiology, time of onset, level of glycemic control and disease management, etc.), and therefore, we could not control for these factors in the multivariate analysis. Consequently, there may be sampling bias based on these and other factors, which may have confounded our results. Lastly, the present study was limited by the lack of postoperative outcomes, due to loss of patient followup, which would have provided further prognostic information for patients with symptomatic hip pain and diabetic arthropathy of the hip.

In conclusion, this study supports our hypothesis concerning diabetic hip arthropathy; specifically, that chondral pathology of the femoral head was more prevalent and of higher severity grade in diabetic patients compared to non-diabetic patients. However, the prevalence, lesion size, and severity of acetabular chondral disease was statistically similar between diabetic and non-diabetic patients. Multivariate analysis demonstrated that diabetic status was independently and significantly associated with the presence of femoral head chondromalacia.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship and/or publication of this article.

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