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Pathomorphologic Alterations Predict Presence or Absence of Hip Osteoarthrosis

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Abnormal morphology of the hip has been associated with primary osteoarthrosis. We evaluated the morphology of 464 consecutive hips contralateral to hips treated by THA. We excluded all hips with known diagnoses leading to secondary osteoarthritis and all hips with advanced arthrosis to eliminate the effect of arthritic remodeling on the morphologic measurements. Of the remaining 119 hips, 25 were in patients aged 60 years or older who had no or mild arthrosis (Tönnis Grade 0 or 1) and 94 hips had Tönnis Grade 2 osteoarthrosis. We quantified morphologic parameters on plain radiographs and CT images and simulated range of motion using virtual bone models from the CT data. The nonarthritic hips had fewer pathomorphologic findings. High alpha angles and high lateral center edge angles were strongly associated with the presence of arthritis; decreased internal and external rotation in 90° flexion showed lesser correlation. The data confirm previous observations that abnormal hip morphology predates arthrosis and is not secondary to the osteoarthritic process. Hips at risk for developing arthrosis resulting from pathomorphologic changes may potentially be identified at the cessation of growth, long before the development of osteoarthrosis.

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Although the concept of primary systemic osteoarthrosis (OA) has been postulated,[18,38] Stulberg et al[31] in 1975 suggested the majority of OA of the hip occurs in association with developmental abnormalities and in the absence of systemic OA. Developmental abnormalities, including hip dysplasia, Legg-Perthes disease, slipped capital femoral epiphysis, and "slip-like" deformities are the most common readily identified causes of secondary hip OA, leading Harris to conclude "it seems clear that either osteoarthritis of the hip does not exist at all as a primary disease entity or, if it does, is extraordinarily rare."[8] The fact that OA of the hip frequently occurs in the absence of OA of other large joints has led authors of both clinical and epidemiologic studies to assume OA of the hip is a distinct entity that behaves differently from OA in other synovial joints.[2,4,9,10] This finding suggests there are morphologic factors specific to the hip that led to its destruction in the majority of cases in the absence of a systemic arthritic process. Certain malformations of the hip have been recognized as primary causes of OA of the hip in North American and European populations.[23,30,31]

Specifically, a number of reports postulate malformations that cause femoroacetabular impingement are a common cause of OA of the hip.[5,13,15,19,29] Malformations of the acetabulum resulting from acetabular overcoverage causing pincer impingement, or incongruous femoral head-neck junctions associated with cam impingement, cause uneven loading and shear stresses within the joint, leading to labral abnormalities, cartilage damage, and ultimately arthrosis.[5] Many authors suggest relief of femoroacetabular impingement may improve joint function and delay or prevent the progression of secondary OA.[3,5,13,16,19,29] If such specific abnormalities predict future OA we may then identify and treat hips at risk before the development of advanced arthrosis.

We asked whether hips that had OA had similar morphology and simulated range of motion compared to hips that did not have OA.

MATERIALS AND METHODS

We retrospectively examined 464 consecutive hips (383 patients) contralateral to hips that had been treated by THA. We excluded patients whose hip arthroplasty had been performed for known developmental deformities (dysplasia [110]), osteonecrosis [21], posttraumatic arthritis [20], Legg-Perthes disease [6], slipped capital femoral epiphysis [4]), or that had inflammatory or polyarticular arthritis (33) to focus the study strictly on hips that were replaced for so-called OA of unknown etiology ("primary osteoarthrosis"). We also excluded 83 hips because the patients had bilateral hip arthroplasty and 31 hips with advance OA Tönnis Grade III or IV OA[38] (Table 1). Finally, we excluded 37 hips that had not developed arthrosis but were in patients younger than 60 years of age because they were deemed too young to ensure they would not subsequently develop arthrosis. Of the 119 remaining hips, we compared the 25 hips (21%) that had not developed OA in patients aged at least 60 (nonarthritic group) were compared with the 94 (79%) that had developed Tönnis Grade II osteoarthritis (arthritic group).

Patients in the nonarthritic group were older ($p < 0.01$) as a result of the fact that we predetermined the absence of arthritis at a minimum age of 60 years was the criterion for inclusion in the nonarthritic group (Table 2). There was a preponderance ($p = 0.04$) of men in the arthritis group. None of the patients had undergone previous hip surgery and there was a similar distribution of left and right hips. All hips in both groups had conventional radiographs as well as computed tomography (CT) studies that had been performed for preoperative planning and computer-assisted surgical navigation of the contralateral THA.

We compared morphologic features in the 25 hips that had no OA (nonarthritis group) with those in the 94 hips that had developed Tönnis Grade II OA (arthritis group).

One author (TME), not blinded to the patients' diagnoses, analyzed the plain radiographs and CT studies for the presence or absence of deformities of the hip by assessing established radiographic parameters[3,7,11,20,21,24–26,34,35,37] (Table 3). In addition, femoral antetorsion was measured on CT scans with the method suggested by Murphy et al.[21] The alpha angles were measured (Fig 1). We used the crossover sign (Fig 2) as a general qualitative marker of local anterior acetabular overcoverage as described by several authors.[5,26]

We calculated the predicted range of motion of each hip using three-dimensional models derived from the CT images and using a software algorithm that had previously been introduced and validated by Kubiak-Langer et al[15] and Tannast et al.[32] The software allows for simulation of motion of the joint based on rotation through the center of rotation of the spherical portion of the joint and can calculate range of motion and the location of impingement in any desired extreme of motion.

For statistical analysis, we used the Kolmogorov-Smirnoff test to determine normal distribution of the continuous morphologic variables. Normally distributed parameters were assessed with the unpaired t-test and nonnormally distributed parameters were assessed with the Mann-Whitney U-test. We used Fisher's exact test to compare nonparametric variables. Probability values below 0.05 were considered significant. After adjusting for multiple comparisons with a Bonferroni test, the significant parameters were included in a hierarchic logistic regression analysis to investigate for correlation between the significant variables and the presence of OA. Three isolated analyses were performed controlling for gender, significant radiographic parameters, and significant range of motion parameters. Finally, a full model analysis was conducted, including all the significant parameters from the prior isolated analyses.

TABLE 1. Exclusion Criteria and Numbers Excluded

Reason for Exclusion	Number Excluded (n = 345)
Developmental dysplasia	110
Bilateral THA	83
Nonarthritic younger than 60 years	37
Tönnis Grade III or higher	31
Systemic arthritis	26
Osteonecrosis	21
Posttraumatic arthritis	20
Rheumatoid arthritis	7
Legg-Calvé-Perthes disease	6
Slipped capital femoral epiphysis	4

TABLE 2. Demographic Data

Parameter	No Arthritis Group (n = 25)	Arthritis Group (n = 94)	p Value
Gender			
Male	13 (52%)	69 (73.4%)	0.04
Female	12 (48%)	25 (26.6%)	
Age (years)*	65.8 ± 5.7 (range, 60.2–82.1)	57.5 ± 10.9 (range, 36.7–85.1)	< 0.01
Side			
Left	16 (64%)	54 (57.4%)	0.4
Right	9 (36%)	40 (42.6%)	

*Mean ± standard deviation

RESULTS

The radiographic parameters on plain radiographs and CT scans were generally different between the group of hips that had OA and the group of hips that did not. The alpha angles were lower ($p < 0.0001$) in the nonarthritic than OA group (mean 51.2° versus 65.6°, respectively) (Table 4). Fewer ($p < 0.0001$) nonarthritic hips had an alpha angle higher than 50° compared to the arthritic group (40% versus 83%, respectively). More hips ($p = 0.03$) in the OA

TABLE 3. Explanation of Radiographic Parameters

Parameter	Definition	Author	Radiographic Means	Normal Value
Lateral center edge angle	Angle formed by a line parallel to the longitudinal pelvic axis and by the line connecting the center of the femoral head with the lateral edge of the acetabulum according to Wiberg	Wiberg[37]	AP pelvic radiograph	Greater than 25°
Radiographic grade of osteoarthritis	Grading system according to Tönnis	Tönnis and Heinecke[35]	AP pelvic radiograph	Grade 0
Acetabular index	Angle formed by a horizontal line and a tangent from the lowest point of the sclerotic zone of the acetabular roof to the lateral edge of the acetabulum	Tönnis and Heinecke[35]	AP pelvic radiograph	Less than 10°
ACM angle of Idelberger and Frank	Angle constructed by different points on the acetabular rim indicating the depth of the acetabulum (exact construction of the angle; see reference)	Idelberger and Frank[11]	AP pelvic radiograph	More than 39°, less than 51°
Coxa profunda	The floor of the fossa acetabuli touches the ilioischial line	Beck et al[3]	AP pelvic radiograph	No coxa profunda
Acetabular retroversion Crossover sign	The anterior rim runs more laterally in the most proximal part of the acetabulum and crosses the posterior rim distally	Reynolds et al[26]	AP pelvic radiograph	No retroversion
Extrusion index	Percentage of uncovered femoral head in comparison to the total horizontal head diameter	Murphy et al[20]	AP pelvic radiograph	Unknown
Neck shaft angle	Angle formed by the axis of the femoral neck and the proximal femoral diaphyseal axis	Tönnis and Heinecke[35]	AP pelvic radiograph	More than 125°, less than 135°
Herniation pit	Round to oval radiolucency surrounded by a thin zone of sclerosis in the proximal superior quadrant of the femoral neck	Pitt et al[25]	AP pelvic radiograph	No herniation pit
Femoral antetorsion	Angle between the femoral neck axis through the center of the base of the femoral neck and the condylar axis	Murphy et al[21]	CT of pelvis and distal femur	No retrotorsion
Pistol grip deformity	Aspherical configuration of the femoral head-neck junction in the lateral aspect of the femoral head	Goodman et al[7]	AP pelvic radiograph	No pistol grip deformity
Alpha angle	Angle formed by the femoral neck axis and a line connecting the center of the femoral head with the point of beginning asphericity	Nötzli et al[24]	3D CT of distal femur (crosstable lateral view)	Less than 50°

ACM = angle of Idelberger and Frank[11]; AP = anteroposterior; 3D = three-dimensional; CT = computed tomography

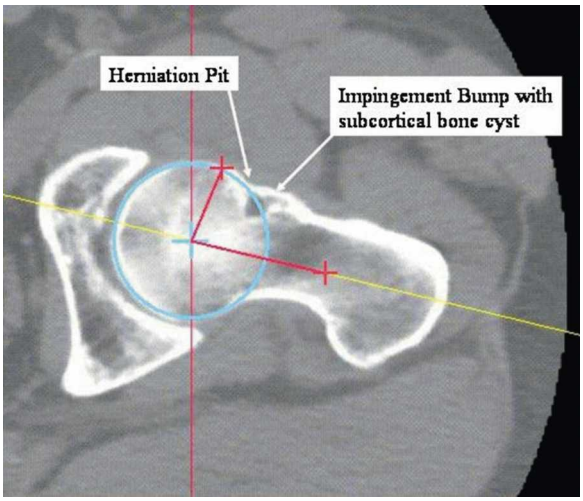

Fig 1. This image shows the aspheric head-neck junction at the femur causing cam impingement and leading to an increased alpha angle. Progressive prearthritic changes are shown by a herniation pit and concomitant subcortical bone cyst.

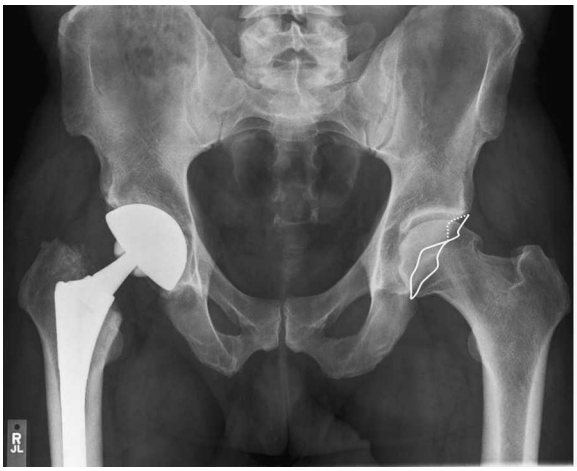

Fig 2. The crossover sign is a radiographic marker demonstrating acetabular retroversion and local acetabular overcoverage. It is formed by the anterior acetabular rim overlapping the posterior acetabular rim as shown on this anteroposterior pelvis radiograph.

group (n = 20) had femurs with a pistol grip deformity compared with the nonarthritic group (n = 1). More (p = 0.006) hips in the arthritis group had a bony prominence at the head-neck junction (cam impingement; Fig 1) than hips in the nonarthritic group (38 versus three hips, respectively). The anteversion angle of the OA group was lower (p = 0.12) than that in the nonarthritis group. None of the nonarthritic hips had an anteversion less than 0°. The neck shaft angle was similar in the groups.

The lateral center edge angles for the osteoarthritis group were higher (p = 0.0001) (Table 5) suggesting excessive acetabular coverage. Although the acetabular index was similar (p = 0.07) in the two groups, the angle of Idelberger and Frank (ACM angle) and extrusion index were lower (p = 0.05 and 0.01, respectively) in the arthritic group. The number of hips with a positive crossover sign was higher (p = 0.008) in the arthritic than nonarthritic group (31 versus 2, respectively). The occurrence of rim fractures was similar among both groups (16 in the arthritic hips and three in the nonarthritic hips). In our range of motion simulation, the hips in the nonarthritic group had more flexion (Fig 3), abduction, internal rotation in extension, and internal and external rotation in 90° of flexion (Fig 4) (Table 6). All hips that survived to age 60 without developing arthrosis had at least 100° of flexion and a minimum of 21° of internal rotation in 90° of flexion.

High alpha angles and high lateral center edge angles were correlated with presence of OA (odds ratio [OR], 1.09, p = 0.003; OR, 1.14, p = 0.02) (Table 7) as did decreased internal and external rotation in 90° of flexion (OR, 0.92, p = 0.008; OR, 0.94, p = 0.008; Table 7). We observed no gender-specific increase in risk for having OA (OR, 0.95, p = 0.94).

DISCUSSION

Abnormal morphology of the hip has been associated with primary osteoarthrosis. The work of some authors suggests primary OA may be rare.[8,23,30,31] To confirm this sugges-

TABLE 4. Femoral Radiographic Parameters

Parameter	Normal Group (n = 25)	Arthritis Group (n = 94)	p Value
Alpha angle*	51.2° ± 11.2° (range, 36.8°–80.1°)	62.8° ± 12.7° (range, 31.9°–87°)	< 0.0001
Neck shaft angle*	130.5° ± 3.9° (range, 121.9°–137.8°)	129.8° ± 5.5° (range, 115°–144.8°)	0.5
Pistol grip deformity	1 (4%)	20 (21.3%)	0.03
Herniation pits	3 (12%)	24 (25.5%)	0.12
Femoral antetorsion*	17.2° ± 8.1° (range, 2.7°–33.5°)	14.3° ± 8.1° (range, –6.1°–34.8°)	0.12
Cam finding	3 (12%)	38 (40.4%)	0.006

*Mean ± standard deviation

TABLE 5. Pelvic Radiographic Parameters

Parameter	Normal Group (n = 25)	Arthritis Group (n = 94)	p Value
Lateral center edge* angle	32.9° ± 5.5° (range, 24°–46.6°)	38.8° ± 8.4° (range, 22.5°–63°)	0.0001
Acetabular index*	8.5° ± 4.7° (range, −4°–16.4°)	6.2° ± 5.7° (range, −12°–16.8°)	0.07
ACM angle*	46° ± 3.5° (range, 37.7°–52.5°)	44.1° ± 4.2° (range, 33.2°–53.3°)	0.05
Coxa profunda	1 (4%)	13 (13.8%)	0.16
Extrusion index*	16.5 ± 6.2 (range, 2.2–32.7)	12.6 ± 7 (range, 1.9–29.2)	0.01
Crossover sign	2 (8%)	31 (33%)	0.008
Rim fractures	3 (12%)	16 (17%)	0.4

*Mean ± standard deviation; ACM = angle of Idelberger and Frank[11]

tion we asked whether hips that have OA and hips that do not have OA have similar morphology and simulated range of motion.

There are several limitations of this study to consider. First, we did not attempt to investigate the pathophysiological mechanisms and the natural history whereby these deformities occur or the point in time when these deformities develop. Although we found a higher incidence of morphologic factors that are putting patients at risk for developing arthritis, it remains unclear whether they are congenital or whether there is any other developmental incident or genetic predisposition responsible for their appearance. Second, we only looked at a sequential group of patients treated in North America and the conclusions of this study may not apply to other patient populations, because it is known there is a geographic predominance of certain pathologies of the hip[12,40] and there are marked ethnic differences in the incidence of hip OA.[17] The analysis of the radiographs was conducted by a single, nonblinded examiner. Thus, an interobserver correlation cannot be provided. Also, measurements from plain radiographs are susceptible to errors.[27] The influence of pelvic tilt or rotational malposition is known to influence reliability of measurements performed on conventional radiographs.[1,6,14,22,28,33,39] Thus, assessment of parameters such as the crossover sign might be influenced by these errors.

With the study limitations in mind, the data demonstrate differences between the morphology of hips that do not have OA at an age of at least 60 years and those that do. After logistic regression analysis, it seems evident that especially high alpha angles and high lateral center edge angles, malformations that cause early impingement in flexion and flexion-internal rotation are putting patients at high risk to develop arthritis. Specifically, no hip survived to age 60 years without arthrosis with flexion of less than 100° or internal rotation in 90° of flexion of less than 21°, and the regression analysis also suggested a correlation between the decreased range of motion in internal and external rotation in 90° flexion and the occurrence of arthritis. Our findings support the theory that femoroacetabular impingement, whether arising from the femoral side leading to cam impingement, the acetabular side leading to pincer impingent, or both, is a major cause of primary OA of the hip.[5,13,19,29,36] These findings of limited motion in our patients with arthrosis are also consistent with prior reports on limited motion reported in patients with symptomatic anterior femoroacetabular impingement.[15,32]

Because none of the hips we analyzed were in patients who had arthrosis of other large joints, it is unlikely the etiology of arthrosis in the hip that was replaced was the result of a systemic form of OA. Furthermore, since we only excluded 5.6% of patients from the current study

TABLE 6. Range of Motion Calculation

Parameter	Nonarthritic Group (n = 25)*	Arthritic Group (n = 94)*	p Value
Flexion	123.6° ± 10.5° (range, 100°–142°)	109.1° ± 17.3° (range, 65°–141°)	< 0.001
Extension	68° ± 15.7° (range, 35°–101°)	63.8° ± 22.1° (range, 10°–110°)	0.37
Adduction	36.8° ± 9° (range, 12°–51°)	34.9° ± 10.8° (range, 9°–56°)	0.32
Abduction	67.4° ± 8.5° (range, 54°–84°)	56.6° ± 13.4° (range, 22°–83°)	< 0.001
Internal rotation	116.7° ± 17.4° (range, 87°–151°)	89.1° ± 28.1° (range, 17°–159°)	< 0.001
External rotation	49.8° ± 8.7° (range, 31°–66°)	48.7° ± 14.5° (range, 4°–78°)	0.62
Internal rotation in 90° flexion	38.5° ± 10.3° (range, 21°–58°)	25.5° ± 12.9° (range, 0°–61°)	< 0.001
External rotation in 90° flexion	106.5° ± 10.9° (range, 88°–134°)	93.9° ± 21.1° (range, 18°–128°)	0.0002

*Mean ± standard deviation

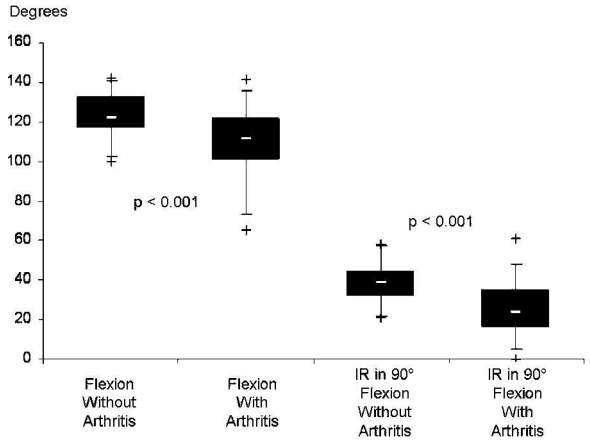

Fig 3. This boxplot shows the results of the simulated range of motion. Note the differences between the two groups were found in flexion and internal rotation in 90° of flexion.

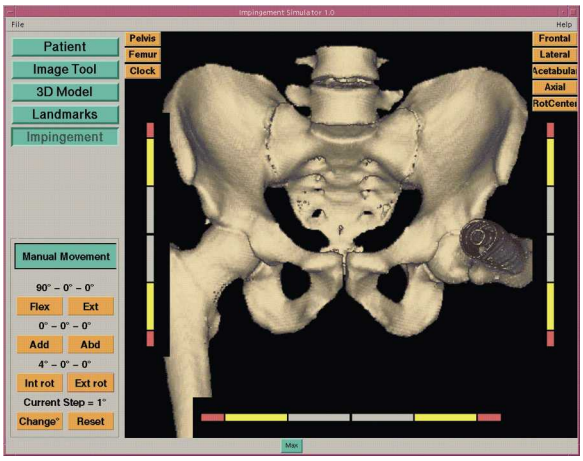

Fig 4. This screenshot of the simulation algorithm shows the reconstructed models of the pelvis and femur. The restricted internal rotation in 90° of flexion is clearly visible.

because of arthrosis of other large joints, it appears that a rather low number of hip osteoarthrosis in our patient population is attributable to systemic forms of osteoarthrosis. Rather, the current study supports the theory that morphologic factors specific to the hip are responsible for the vast majority of OA. Thus, the theory that primary OA of the hip is commonly the result of systemic causes of arthrosis appears unsound. Furthermore, attempts at systemic treatment of OA after skeletal maturity and after these deformities developed would unlikely provide any substantial benefit. Additionally, the higher number of pistol grip lesions and crossover signs as well as the presence of femoral anteversion angles below 0° in the arthritis group might suggest these deformities are not compatible with long-term health of the hip.

Our findings suggest efforts to prevent OA of unknown etiology in the hip should focus on promoting the development of normal hip morphology during growth and identifying the structural abnormalities at an early age. Early recognition of morphologic abnormalities and even-

TABLE 7. Hierarchic Logistic Regression Analysis for Factors Predicting Development of Osteoarthritis

Model	Parameter	Odds Ratio	Confidence Interval	p Value
Gender				
	Gender	0.41	0.17–1.02	0.06
Range of motion				
	Flexion	0.79	0.94–1.08	0.79
	Abduction	0.97	0.91–1.03	0.28
	Internal rotation	0.98	0.95–1.01	0.19
	Internal rotation in 90° flexion	0.92	0.86–0.99	0.03
	External rotation in 90° flexion	0.95	0.91–0.99	0.02
Radiographic findings				
	LCE angle	1.13	1.04–1.23	0.003
	Crossover sign	2.48	0.48–12.78	0.28
	Cam	4.49	1.14–17.7	0.03
	Alpha	1.08	1.03–1.13	0.002
Full model				
	Gender	0.95	0.26–3.5	0.94
	Internal rotation in 90° flexion	0.92	0.87–0.98	0.008
	External rotation in 90° flexion	0.94	0.90–0.98	0.008
	Cam	4.37	0.87–21.87	0.07
	Alpha angle	1.09	1.03–1.15	0.003
	LCE angle	1.14	1.02–1.27	0.02

LCE = lateral center edge

tual treatment with joint-preserving surgical techniques might increase survival of predisposed joints and delay the necessity for prosthetic arthroplasty, although such procedures will require long-term studies to confirm validity of the concept and minimal risk.

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