

Relationship Between Hip Dysplasia, Pain, and Osteoarthritis in a Cohort of Patients with Hip Symptoms

Johanne Morvan, Ronan Bouttier, Bernard Mazieres, Evelyne Verrouil, Jacques Pouchot, Anne-Christine Rat, Dewi Guellec, Francis Guillemin, Joel Coste, and Alain Saraux, for the KHOALA Cohort Study Group

ABSTRACT. Objective. The relationship between acetabular dysplasia (HD) and hip osteoarthritis (OA) remains unclear, especially for mild forms of dysplasia. Our objectives were to estimate the prevalence of HD in a population-based sample with symptoms and to evaluate potential associations linking HD, hip OA, and hip pain.

Methods. Individuals 40 to 75 years of age with symptoms in 1 or both hips were recruited during a multiregional prevalence survey. All study participants underwent examination and radiographs. Radiographs were evaluated using Kellgren-Lawrence staging (with stages ≥ 2 indicating hip OA) and HD measures [center-edge (CE) angle, acetabular inclination angle (HTE), acetabular depth (AD), and vertical center-anterior margin angle].

Results. We studied both hips of 842 individuals (1684 hips), among whom 203 had hip OA. Compared to left hips, right hips had significantly smaller CE angles and significantly greater AD and HTE values ($p \leq 0.001$). Overall, the prevalence of HD ranged from 7.6% to 22.2% of the hips depending on the measure used. The prevalence of HD was higher in individuals with hip OA, with significant differences for abnormal HTE (19.1% vs 11.4%; $p < 0.0001$) and abnormal CE (11.3% vs 7.5%; $p = 0.04$). By logistic regression, only abnormal HTE remained associated with OA. Same-side hip pain was not statistically more common in individuals with HD after stratification on OA status ($p = 0.12$).

Conclusion. Our study confirms the relationship between OA and HD, particularly as defined based on the HTE angle. (First Release July 15 2013; J Rheumatol 2013;40:1583–9; doi:10.3899/jrheum.121544)

Key Indexing Terms:

OSTEOARTHRITIS

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Osteoarthritis (OA) of the hip is a major cause of morbidity and disability, especially among the elderly. Hip OA is viewed as a complex multifactorial disease. Acetabular

dysplasia (HD) increases the mechanical stresses on the cartilage matrix and may therefore make a substantial contribution to the development of hip OA. Radiological

From the Medicine Department, Laennec Hospital, Quimper; Radiology Department, Cavale Blanche University Hospital, Brest; Rheumatology Centre, Purpan University Hospital, Toulouse; Faculty of Medicine, Paul-Sabatier University, Toulouse; Centre Hospitalier Universitaire (CHU) Nancy, Paul Verlaine Metz, Paris Descartes, Nancy; Department of Internal Medicine, Hôpital Européen Georges Pompidou, Assistance Publique-Hôpitaux de Paris (APHP); Université de Lorraine, Lorraine; Faculty of Medicine, School of Public Health, Vandoeuvre-lès-Nancy; INSERM, CIC-EC CIE6, and Rheumatology Department, CHU de Brabois, Vandoeuvre-lès-Nancy; Rheumatology Department, Cavale Blanche University Hospital and Université Bretagne Occidentale, Brest; Unité de Biostatistique et d'Épidémiologie, Hôpital Cochin, Paris; Rheumatology Department and EA 2216, Cavale Blanche University Hospital; and Université Bretagne Occidentale, Brest, France.

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J. Morvan, MD, MSc, Rheumatologist, Medicine Department, Laennec Hospital; R. Bouttier, MD, Radiology Department, Cavale Blanche University Hospital; B. Mazieres, MD, Professor of Rheumatology, Rheumatology Centre, Purpan University Hospital, Faculty of Medicine,

Paul-Sabatier University; E. Verrouil, MD, Assistant Rheumatologist, Rheumatology Centre, Purpan University Hospital; J. Pouchot, MD, PhD, Professor of Internal Medicine, Nancy-Université, Paul Verlaine Metz, Paris Descartes and Hôpital Européen Georges Pompidou, Department of Internal Medicine, APHP, Faculté de Médecine Paris Descartes; A-C. Rat, MD, PhD, Associate Professor of Rheumatology, Université de Lorraine, Université Paris Descartes, Faculty of Medicine, School of Public Health, INSERM, CIC-EC CIE6, and Rheumatology Department, CHU de Brabois; D. Guellec, MD, Rheumatology Department, Cavale Blanche University Hospital and Université Bretagne Occidentale; F. Guillemin, MD, PhD, Professor of Epidemiology, Université de Lorraine, Paris Descartes, and School of Medicine, School of Public Health, and INSERM, CIC-EC CIE6, CHU de Brabois; J. Coste, MD, PhD, Professor of Biostatistics, Nancy-Université, Paul Verlaine Metz, Paris Descartes, and Unité de Biostatistique et d'Épidémiologie, Hôpital Cochin; A. Saraux, MD, PhD, Professor of Rheumatology, Rheumatology Department and EA 2216, Cavale Blanche University Hospital and Université Bretagne Occidentale.

Address correspondence to Prof. A Saraux, Service de rhumatologie, Hôpital de la Cavale Blanche, BP 824, F 29609 Brest Cedex, France. E-mail: alain.saraux@chu-brest.fr

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observations in patients with hip OA and followup studies of patients with hip dysplasia support this possibility^{1,2,3,4,5,6,7}. However, the relationship between hip OA and hip dysplasia, particularly in its mild forms, remains unclear, and several studies found no association between the 2 conditions^{8,9,10,11,12,13}. Moreover, most studies failed to distinguish individuals with and those without hip pain. In a 2003 study of HD in primary-care patients presenting with hip pain, the prevalence of hip dysplasia was considerably increased (up to 32%) compared to that found in population-based surveys, suggesting an association between HD and hip pain¹⁴.

Our main objective was to estimate the prevalence of HD in a population-based sample of individuals with symptoms in the hip area. Radiographs were obtained routinely to look for hip OA and to assess 4 different hip dysplasia measures. Associations were sought between hip OA, hip dysplasia, and hip pain.

MATERIALS AND METHODS

Population. Between April 2007 and October 2009, a multiregion 2-phase survey was conducted in the general population to estimate the prevalence of symptomatic hip and knee OA in France¹⁵. Screening for hip and knee symptoms was performed by telephone interview in 6 geographic regions [Brittany (west), Côte d'Azur (southeast), Lorraine (east), Picardie (north), Paris (north-center), and Toulouse (southwest)]. The first phase of sampling conducted by telephone involved use of a random selection of telephone numbers in each selected area. Telephone numbers for enterprises, businesses, and seniors' residences, as well as second homes, were excluded. We randomly selected 1 adult in each household by using the next birthday method. The person first answering the phone was asked to name the household residents who were between 40 and 75 years old. The person with a birthday closest to the interview date (eligible subject) was then invited to answer the screening questionnaire.

The screening questionnaire comprised 4 items about the hip and 4 about the knee and was developed from a literature review and the American College of Rheumatology criteria for OA. The screening questions concerning the hip were (1) During the last 4 weeks, have you had pain in the hip, groin, or in the upper thigh? (2) Do you have pain in the hip, groin, or in the upper thigh while climbing stairs or walking down slopes? (3) Do you have a limitation in the range of motion of one or both hips? and (4) Do you have hip OA?

Of 27,632 eligible subjects, 9621 had a positive screening with at least 1 hip symptom and/or self-reporting OA diagnosis (question 1 to 4). A total of 4640 patients agreed to participate (Figure 1).

We included the survey participants who were 40 to 75 years of age and who had had physical examinations and radiographs. For each participant, an anterior-posterior pelvic radiograph and a Lequesne false-profile radiograph were obtained. We excluded individuals with a history of hip surgery (e.g., arthroplasty) and those in whom both hips were not visible on the radiographs.

Radiographic definition of hip OA. All radiographs were read centrally by 2 authors (BM and EV) working together. The Kellgren-Lawrence (KL) stage was determined based on the degree of osteophyte formation, joint space narrowing, sclerosis, and joint deformity¹⁶. We defined radiological hip OA as a KL stage of 2 or higher.

Radiographic measures of hip dysplasia. All radiographs were read independently by 2 trained observers (JM and RB). Acetabular morphology was assessed using 4 measures: the center-edge (CE) angle [formed by the vertical line through the center of the femoral head (C) and the CE line from C to the lateral edge of the dense subchondral line along the roof of

the acetabulum (E)]¹; the acetabular inclination angle [or horizontal toit externe (HTE) angle or Tonnis angle: horizontal line through the medial edge of the acetabular roof (T) and the TE line], acetabular depth (AD; distance from the deepest point of the acetabular cavity to the EP line, where P is the superior pubic angle), and the vertical center-anterior margin (VCA) angle, formed by the vertical line through the center of the femoral head (VC line) and the CA line, where A is the most anterior point of the linear subchondral density along the acetabular roof. HD was defined as 1 or more of the following: CE angle $\leq 20^\circ$, HTE angle $> 12^\circ$, AD < 9 mm, or VCA angle $\leq 20^\circ$. They were not evaluated when 1 point was not clearly defined, explaining differences of denominators, particularly for AD with superior pubic angle. These cutoffs have been extensively validated by Lequesne and de Seze¹⁷ and others^{18,19}. We also measured the centrum-collum-diaphyseal angle (CCD or neck-shaft angle formed by the neck axis through the center of the femoral head and the axis of the femoral diaphysis; a value $< 130^\circ$ indicates coxa vara and a value $> 140^\circ$ coxa valga). The CCD angle is used to detect metaphyseal dysplasia, described as a common concomitant of HD¹⁹. Interobserver and intraobserver agreements were assessed using the intraclass correlation coefficients (ICC), Cohen's kappa coefficients (κ), and Bland-Altman plots²⁰. Intraobserver agreement for CE, VCA, HTE, and AD ranged from 0.72 to 0.94 and interobserver agreement from 0.68 to 0.74²¹.

Statistical analysis. First, we described the prevalence of hip dysplasia and OA. Then, we evaluated the correlation between hip dysplasia measures for the right and left hips to identify the best statistical unit (participant or hip) and measure (CE, VCA, HTE, or AD) for further analysis. HD measures were found not to be highly correlated. Finally, we evaluated associations linking hip dysplasia to same-side OA and hip dysplasia to same-side pain. The individual was the statistical unit. For each patient we evaluated the association between right dysplasia and right OA and the association between left dysplasia and left OA. Then we evaluated the association between hip dysplasia defined as an abnormal HTE and hip pain after stratification on OA status using the Mantel-Haenszel chi-square test at the right hip and at the left hip.

Differences between continuous measures were assessed using the Student t test or paired t test. For dichotomous data, we chose the chi-square test (Mantel-Haenszel test in case of stratification). All dysplasia measures associated to OA in the univariate analysis with $p < 0.1$ were included in a multivariate logistic regression, with backward selection using the likelihood ratio test to evaluate associations linking hip dysplasia to OA.

Correlations and agreement between measures were evaluated using the Pearson correlation coefficient and Bland-Altman plots, respectively.

RESULTS

Population characteristics. We included 842 individuals (i.e., 1684 hips), 235 men and 607 women, with a mean age of 57.9 ± 9.2 years (Figure 1). Among them, 676 (80.3%) had hip pain during the visit: 449 (53.3%) had pain in 1 hip (right hip in 242 and left hip in 207) and 227 (27.0%) had pain in both hips (Table 1). Prevalence of hip OA was slightly higher in male (right hip 52/235; left hip 60/235) than in female patients (right hip 88/607; left hip 83/607; $p = 0.008$ and 0.0001 for the right and left hip, respectively).

Comparison of the right and left hips. Mean CE angle and AD were significantly lower in the right hips than in the left hips (CE angle: 29.0° vs 30.4° , $p \leq 0.0001$; and AD: 12.1 mm vs 13.3 mm, $p \leq 0.0001$). Mean HTE angle was greater in the right hips than in the left hips (7.1° vs 5.3° , $p \leq 0.0001$). Similarly, the proportion of patients with hip dysplasia was higher in the right hips than in the left hips

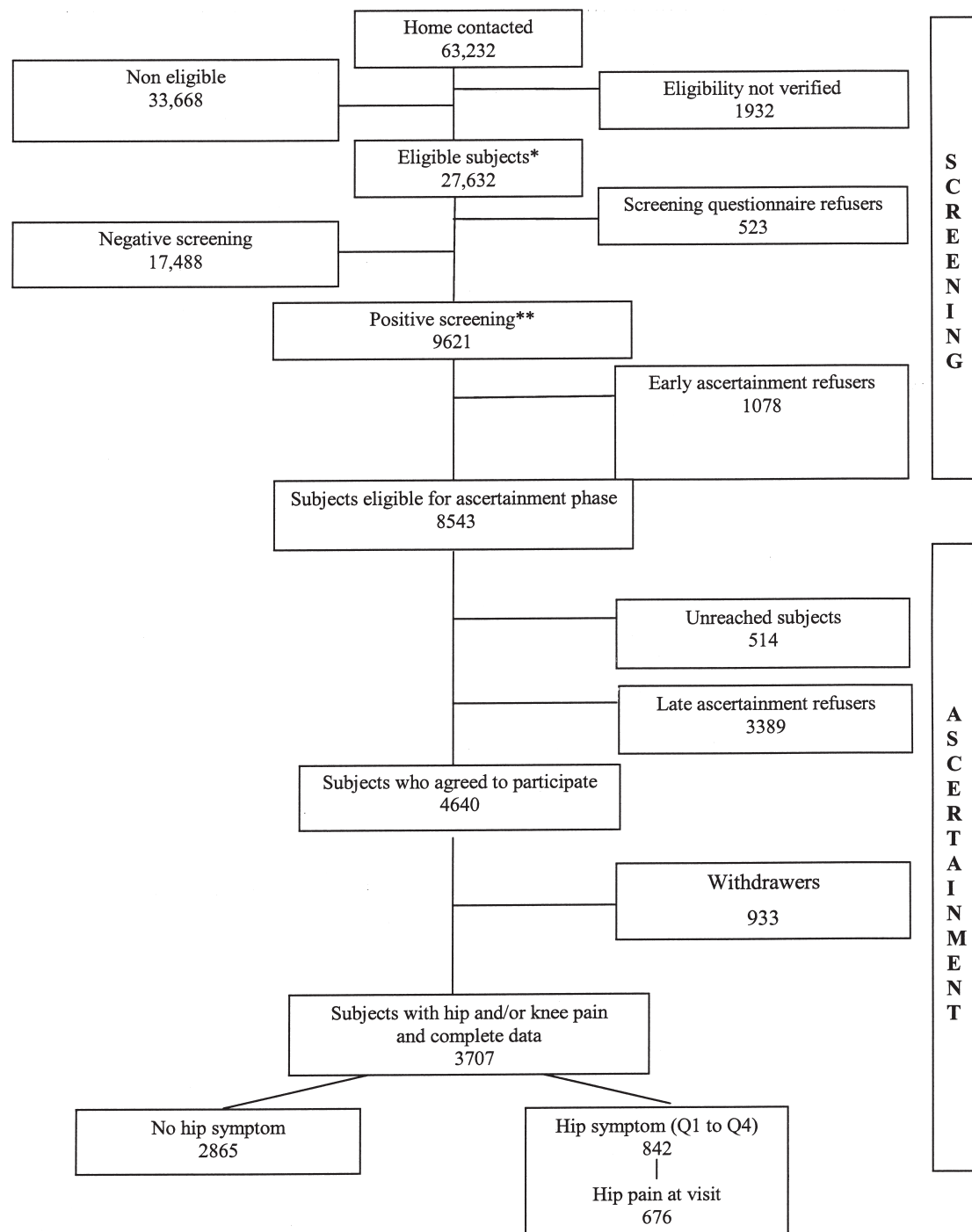


Figure 1. Selection of subjects in the study. *Household residents between 40 and 75 years old with a birthday closest to the interview. **At least 1 hip symptom and/or self-reporting osteoarthritis diagnosis.

using either VCA (86 vs 51) or HTE (115 vs 98), suggesting the pertinence of these differences. No significant side-to-side difference was found for the VCA angle (30.5° vs 30.7°, $p = 0.51$).

Strong correlations were detected between the right and left hips for CE ($r = 0.64$), VCA ($r = 0.68$), and HTE ($r =$

0.59). The correlation was very strong for AD ($r = 0.92$). The Bland-Altman plots confirmed that there is no systematic bias of differences between the right and left hips (Figure 2).

Comparisons of male-female characteristics. Prevalence of hip dysplasia was not higher in female than in male patients

Table 1. Prevalences of pain, osteoarthritis (OA), and hip dysplasia in 842 individuals. Numbers in parentheses are percentages.

Feature	Unilateral*		Bilateral*	Total	Total
	Right	Left	(/no. individuals)	(/no. individuals)	(/no. hips)
Pain	242/842 (28.7)	207/842 (24.6)	227/842 (27.0)	676/842 (80.3)	676 (53.6)
OA	60/842 (7.1)	63/842 (7.5)	80/842 (9.5)	203/842 (24.1)	283 (16.8)
CE dysplasia	62/842 (7.4)	27/842 (3.2)	24/842 (2.9)	113/842 (13.4)	137 (8.1)
VCA dysplasia	32/816 (3.9)	40/809 (4.9)	26/809 (3.2)	98/809 (12.1)	124 (7.6)
HTE dysplasia	74/840 (8.8)	57/840 (6.8)	41/840 (7.9)	172/840 (20.5)	213 (12.7)
AD dysplasia	72/656 (11.0)	20/657 (3.0)	100/656 (15.2)	192/656 (29.3)	292 (22.2)

* Not all measures could be obtained on all radiographs, resulting in differences in the denominators. CE: center-edge angle; HTE: acetabular inclination angle; AD: acetabular depth; VCA: vertical-center-anterior margin angle.

for VCE (right 65/607 vs 21/214, $p = 0.45$; left 37/607 vs 14/235, $p = 0.94$), HTE (right 81/605 vs 34/235, $p = 0.94$; left 69/605 vs 29/235, $p = 0.70$), or VCA (right 47/586 vs 11/230, $p = 0.10$; left 50/584 vs 16/225, $p = 0.50$).

Correlations among radiographic measures of hip dysplasia. The CE angle correlated strongly with the VCA and HTE angles (right side: $r = 0.65$, $p \leq 0.0001$, and $r = 0.75$, $p \leq 0.0001$, respectively; left side: $r = 0.66$, $p \leq 0.0001$, and $r = 0.77$, $p \leq 0.0001$, respectively). The CE angle showed weak correlations with AD and CCD angle (right side: $r = 0.35$, $p \leq 0.0001$, and $r = -0.14$, $p \leq 0.0001$, respectively; left side: $r = 0.40$, $p \leq 0.0001$, and $r = -0.09$, $p = 0.007$, respectively).

Prevalence of hip OA and dysplasia. The routine radiographs showed hip OA in 203 (24.1%) participants. Hip OA was bilateral in 80 (39.4%) of these 203 individuals. Table 2 reports the KL stages on the right and left sides.

The prevalence of HD was 31.0% of the hips when HD was defined as having at least one abnormal measure among the 4 used in the study. When a single one was selected, prevalence was lowest (7.6% of the hips) for abnormal VCA and highest (22.2% of hips) for abnormal AD. Prevalence was higher on the right side than on the left side (Table 1). The prevalence of HD was higher in the subgroup with hip OA than in those without hip OA (Table 3).

In participants with unilateral OA of the right hip, the prevalence of hip dysplasia in the left hip compared to participants without hip OA was increased, except when VCA was used to define hip dysplasia (CE: 5.0% vs 13.3%, $p = 0.02$; VCA: 7.6% vs 12.3%, $p = 0.2$; HTE: 9.0% vs 26.7%, $p < 0.0001$; and AD: 16.7% vs 29.2%, $p = 0.05$). No increase in right hip dysplasia was seen in participants with unilateral OA of the left hip.

On both sides, among the hip dysplasia definitions (VCE, HTE, VCA, and AD), HTE was the hip dysplasia measure that was most strongly associated with hip OA (Table 3). By logistic regression, HTE was the only measure that remained associated (right hip, $p = 0.038$; left hip, $p = 0.017$) with hip OA (right hip, OR 1.86, 95% CI 1.17–2.97; left hip, OR 1.81, 95% CI 1.10–2.98).

Hip dysplasia based on HTE was found in 125 of 903 painful hips and 88 of 777 pain-free hips. After stratification on hip OA status, hip dysplasia was not associated with hip pain (Table 4).

DISCUSSION

By design, our study included only individuals with hip symptoms. However, the participants were recruited within a representative population-based sample. We studied 4 measures of hip dysplasia. CE correlated strongly with HTE and VCA and weakly with AD. In 2010, McWilliams, *et al*⁶ reported that CE and AD were independent risk factors for OA, despite a relatively strong correlation between these 2 measures. Both CE and AD had good interobserver reliability in our previous study²⁰, regardless of the method used to assess this variable (ICC, kappa, or Bland-Altman plot). These data may warrant the use of CE and AD as the only measures when investigating hip dysplasia. HTE has been studied less extensively, but has shown good reliability in several studies^{22,23}. This measure may have independent effects, because it was significantly associated with hip pain in our study. Therefore, evaluation of HTE in future studies would be of interest. The role for VCA requires further investigation.

CE was significantly smaller and AD and HTE significantly greater on the right side than on the left side in our study, in keeping with previous data⁶. The prevalence of dysplasia based on CE, HTE, and/or AD was higher on the right side than on the left side. Although all 4 measures showed positive correlations between the 2 sides, the correlation was strong only for AD. We consequently conducted separate analyses of the right and left hips. In contrast, because we did not observe any difference between the sexes, male and female patients were pooled for analysis.

Unilateral OA of the right hip was associated with an increased prevalence of dysplasia in the left hip. Similarly, McWilliams, *et al*⁶ reported significantly lower mean CE and AD values in the unaffected contralateral hips of participants with unilateral hip OA than in the right and left hips of controls without hip OA. OA may develop eventually in the

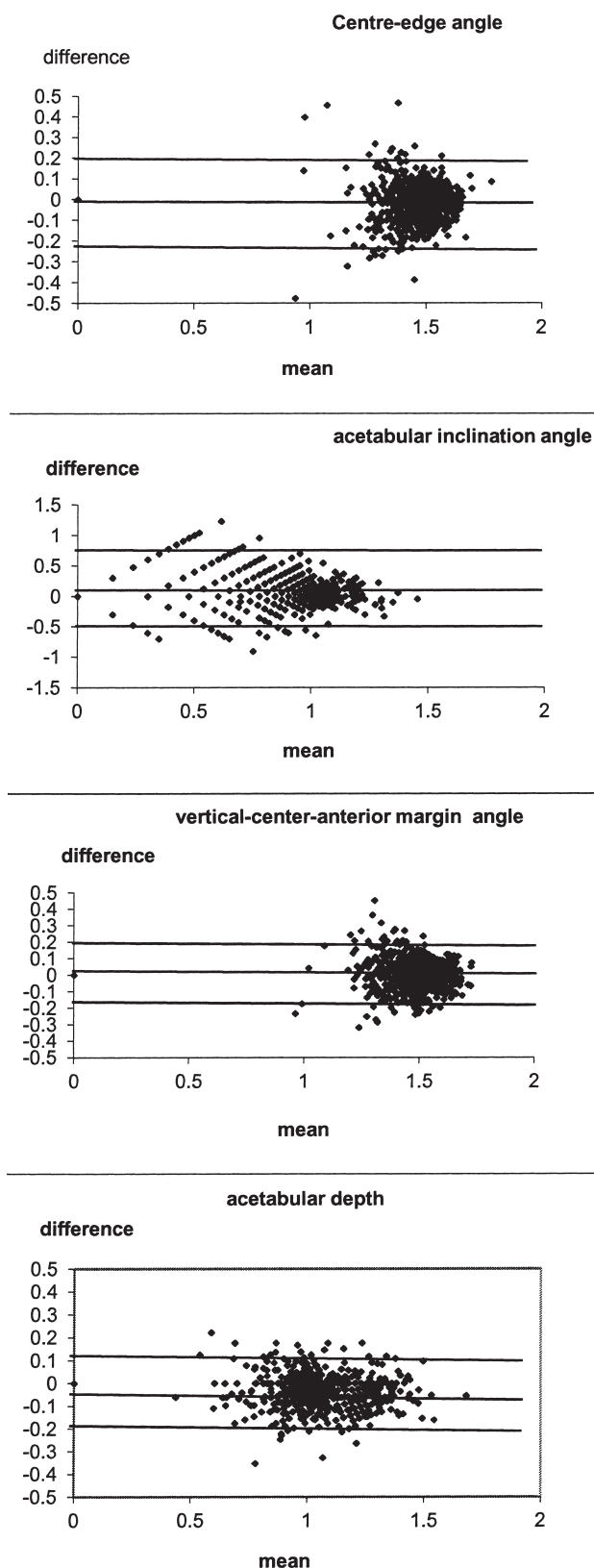


Figure 2. Bland-Altman graphs for dysplasia measures: agreement between the right and left sides.

Table 2. Kellgren-Lawrence (K-L) stages of osteoarthritis.

		Left					
Right	KL = 0	KL = 1	KL = 2	KL = 3	KL = 4	Total	
KL = 0	384	63	20	9	2	478	
KL = 1	79	113	28	3	1	224	
KL = 2	21	28	52	8	0	109	
KL = 3	4	7	14	5	0	30	
KL = 4	0	0	0	1	0	1	
Total	488	211	114	26	3	842	

unaffected hips, but only a prospective study would be able to assess this possibility.

The prevalence of HD in our study was high, up to 31% when defined as an abnormality in any of the 4 measures (CE, HTE, VCA, and AD), but we evaluated patients with hip symptoms and the prevalence in our population does not represent the prevalence in a population without hip pain. Similarly, in patients presenting with a new episode of hip pain, the prevalence of HD was 32%¹⁴. Lower prevalence of 1% to 15% was found in population-based surveys^{4,5,9,10,11,24,25}, including 1 conducted in France¹³, but both ethnic distribution and detection methodology (evaluation of general population with or without hip pain, definition of dysplasia) may explain why the prevalence can vary across studies. HD was more common in our study in hips with OA than in those without OA, although the differences were significant only for abnormal CE and HTE. Because of the cross-sectional design of our study, our data indicate an association between OA and dysplasia but do not provide information on causality. Few longitudinal studies have been published, and to our knowledge, only 3 used population-based samples^{4,5,24}. All 3 studies found a positive association between HD and hip OA. Similar results were obtained in a population without hip OA at study initiation²⁴.

In our study, after stratification on OA status, hip dysplasia was not associated with hip pain. Similarly, a previous study found no consistent association between hip dysplasia and self-reported hip or groin pain⁴. By contrast, a cross-sectional study showed an increased prevalence of dysplasia in participants with hip pain, suggesting that dysplasia may be a major cause of hip pain [symptomatic adult acetabular dysplasia (SAAD) syndrome]¹⁴. Data from the Rotterdam cohort identify HD as a stronger predictor of hip OA development during followup in individuals with hip pain at baseline but no radiographic evidence of hip OA, compared to individuals without hip pain⁵. Thus, in patients with hip pain and hip dysplasia, close monitoring may be in order to ensure the early detection of hip OA. Hip pain, hip dysplasia, and OA are associated but the association between hip dysplasia and pain diminishes when OA is taken into account, suggesting that pain is generally associated to OA because of dysplasia.

Table 3. Prevalence of dysplasia according to the presence or absence of osteoarthritis (OA).

Type of Dysplasia	Either Hip or Both Hips			Right Hip			Left Hip		
	OA–	OA+	p	OA–	OA+	p	OA–	OA+	p
CE (%)	105/1401 (7.5)	32/283 (11.3)	0.04*	65/702 (9.3)	21/140 (15.5)	0.05*	40/699 (5.7)	11/143 (7.7)	0.34
VCA (%)	103/1350 (7.6)	21/275 (7.6)	0.92	47/678 (6.9)	11/138 (8.0)	0.72	54/672 (8.0)	12/137 (8.8)	0.73
HTE (%)	159/1397 (11.4)	54/283 (19.1)	<0.0001*	86/700 (12.3)	29/140 (20.7)	0.01*	73/697 (10.5)	25/143 (17.5)	0.02*
AD (%)	235/1099 (21.4)	57/214 (26.6)	0.10	141/548 (25.7)	31/108 (28.7)	0.55	98/550 (17.8)	22/107 (20.6)	0.5

* Statistically significant. CE: center-edge angle; HTE: acetabular inclination angle; AD: acetabular depth; VCA: vertical-center-anterior margin angle.

Table 4. Association between hip dysplasia defined as an abnormal acetabular inclination angle [horizontal toit externe (THE) angle or Tonnis angle] and hip pain after stratification on osteoarthritis (OA) status (chi-square Mantel-Haenszel test).

Feature	OA+			OA–			Total		
	Pain–	Pain+	p	Pain–	Pain+	p	Pain–	Pain+	p
Right HTE dysplasia (%)	8/50 (16.0)	21/90 (23.3)	0.14	33/321 (10.3)	53/379 (14)	0.30	41/371 (11.1)	74/469 (15.8)	0.07
Left HTE dysplasia (%)	9/50 (18.0)	16/93 (17.2)	0.90	38/356 (10.7)	35/341 (10.3)	0.86	47/406 (11.6)	51/434 (11.8)	0.79

Our study has several limitations. The cross-sectional design precludes an evaluation of the association between hip dysplasia and hip OA. Prospective cohort studies are needed to determine whether hip dysplasia increases the risk of hip OA. The prevalence of OA may have been underestimated, because detection relied only on standard radiographs. Some of our study participants with no radiographic hip OA may have had subtle changes that would have been detectable by magnetic resonance imaging. Interobserver assessment was not perfect, although it was comparable overall to that reported in earlier studies. The radiographic evaluation of hip dysplasia is known to have limited reliability^{22,26}. However, in our study, antero-posterior pelvic and Lequesne false-profile radiographs were obtained from all participants, whereas in most of the previous studies the hips were evaluated on intravenous urography views, which were obtained without leg-position standardization, a factor likely to affect the measurement of hip dysplasia variables. It is sometimes difficult to determine angles based on radiographs in advanced OA. The C point is fairly easy to position in practice, in the absence of alterations in femoral head shape (because of advanced hip OA), which complicates the positioning of points E and A. In our study, only 4 hips were grade 4 and this potential difficulty did not complicate our interpretation. Finally, our limited sample size may explain the

absence of statistically significant differences in the comparisons of prevalence across subgroups.

The prevalence of HD is high in individuals with hip symptoms. This prevalence is higher in cases of hip OA, confirming reports of an association between hip OA and dysplasia. Hip dysplasia assessment based on CE, AD, and HTE should be performed routinely in patients presenting with hip symptoms.

APPENDIX

List of study group members: Joel Coste, L. Euler-Ziegler, P. Fardellone, B. Fautrel, Francis Guillemin, Bernard Mazieres, Johanne Morvan, Jacques Pouchot, Anne-Christine Rat, C.H. Roux, Alain Saraux, and Evelyne Verrouil.

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