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ORIGINAL PAPER

Acetabular dysplasia as an aetiological factor in development of hip osteoarthritis

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Abstract The objective of this study was to investigate the relation between acetabular dysplasia (AD) of the hip and risk for development of hip osteoarthritis (OA) in a Sámi population with very high prevalence of AD (38%). A population-based survey was conducted in a main Sámi area in Norway. A total of 315 middle-aged subjects were examined clinically and radiologically and were regarded as a random sample of 836 responders from a general health survey. The results demonstrated that 6.8% of the Sámi population had OA. The logistic regression analyses did not show a significant association between AD and OA. Only age had a statistically significant influence on OA. In this middle-aged Sámi population, there is no evidence for the influence of AD on the development of hip OA.

Résumé Objectif: de façon à évaluer les relations entre la dysplasie acétabulaire (AD) de la hanche et le risque d'évolution vers une coxarthrose (OA), la population Sami qui présente un haut niveau de dysplasie de la hanche (38%) a été analysée. Méthode: la région Sami est une région de Norvège. 315 sujets d'âge moyen ont été examinés cliniquement et radiologiquement sur 836 questionnaires généraux de santé. Résultats: 6,8% de la population de la région Sami présentait une coxarthrose. L'étude avec courbe de régression a montré que le facteur statistiquement significatif influençant l'évolution vers une coxarthrose était l'âge. En conclusion: dans la population d'âge moyen de la région de Sami, il n'y a pas à l'évidence d'influence de la dysplasie de hanche sur le développement de la coxarthrose.

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Introduction

Osteoarthritis (OA) of the hip is considered a multifactorial disease. It has been assumed that systemic factors are the main cause, but that local biomechanical factors play the final role in determining the severity [2]. Evidence of acetabular dysplasia (AD) has been found on radiographs of many patients who have degenerations of the hip joint [24, 6], and marked AD has therefore been considered a major aetiological factor [22]. However, the relationship between the severity of AD and the rate of OA development is less clear. Because of advances in reconstructive hip surgery [18, 13], the need for a clear understanding of the natural course of AD is more important than ever. Acetabular dysplasia is probably polygenetically inherited [25], but few population studies have been published on this subject [9, 7, 8 ,17]. Also, studies of the natural history of untreated dysplastic hips are few, and those available do not provide objective prognostic guidelines [6, 1]. In the available literature on the influence of hip dysplasia on the development of hip OA, only some studies investigated the influence of this parameter in a prospective cohort design with a long follow-up period [1, 6, 15, 19].

The risk for development of hip OA varies widely between different populations, from 1- 4% of adult black populations to 5–11% in white populations, with the

Table 1 Persons invited to participate in the population study

	Sámi attending the screening			Sámi responders			Examined by X-ray		
Age groups(years)	Males	Females	Total	Males	Females	Total	Males	Females	Total
20–39	146	146	272	112	100	212	25	35	60
40–49	209	205	414	157	145	302	48	58	106
50–64	207	122	429	155	167	322	77	72	149
Total	562	573	1135	424	412	836	150	165	315

highest prevalence among older people [17, 20]. It is well known that in the Sámi population there is a high prevalence of hip problems. Wessel [23] performed systematic skeletal research among Sámi in the county of Finnmark in 1918, and he found that 5% of the population had dislocated hips. Getz [4] studied skeletal material of approximately 300 Sámi in 1956, and he found that 4% had dislocated hips. Recently we described the centre edge (CE) angle in the Sámi population [14]. The CE angle was described in 1939 by Wiberg to quantify acetabular dysplasia in adults [24]. According to Wiberg, CE angles greater than 24° are normal, angles between 20 and 24° are subnormal or slightly dysplastic while angles below 20° are defined as dysplastic. We found dysplastic hips in 17% and slightly dysplastic hips in 21% of the adult Sámi population. Thus 38% had more or less acetabular dysplasia based on the CE angles. The Sámi population, then, is considered to have a risk for development of hip OA. Therefore, in this population study among adult Sámi we have investigated the association between radiographic evidence of AD and risk for development of OA.

The Sámi are indigenous people of northern Scandinavia. The Sámi people are regarded as an extreme outlier among European populations and their large genetic separation from other European populations is best explained by assuming that Sámi are descendants of a narrow, distinctive subset of Europeans. The Sámi stem, the mitochondrial (mtDNA) lineage of the Sámi people, largely from a hunter-gatherer population that resided in south-western Europe and the population split between Sámi and other European populations is estimated to have taken place more than 10,000 years ago [21]. In Norway approximately 50–70,000 inhabitants are Sámi. The Sámi are to some extent genetically mixed with other Scandinavian populations, but genetic distinctions can still be traced.

Materials and methods

Geographic and population characteristics

The Sámi are an ethnic minority in Norway, but in the county of Finnmark in the municipalities Karasjok and Kautokeino more than 80% are of Sámi origin.

In Karasjok and Kautokeino 1,723 individuals were invited to participate in a general health survey in 1987, and 1,347 persons (78.2%) attended the screening [10]. A questionnaire was distributed to those who were invited to attend the screening. It was to be filled in at home and returned by mail. It included questions concerning ethnicity and symptoms of back problems. Of 1,347 participants, 1,135 (84.3%) were defined as Sámi (when two or more of the grandparents were Sámi), of whom 424 men and 412 woman (73.7) returned the questionnaire (Table 1).

Back problems were reported by 210 persons (25.1%) while 626 had no back complaints. All 210 persons with back problems were invited to a radiographic examination, of whom 158 (75.2) turned up [10]. Furthermore, a random sample of 206 persons among those with no back complaints were invited, of whom 157 (76.2%) came for examination. The controls were matched for age and gender.

In an earlier paper from the same work, we showed that the mean CE angle in those with back pain and those without (controls) was statistically similar [14].

A standard standing anteroposterior radiograph was taken, with the feet pointing straight forward. Respondents were given a bowel-emptying regime (Toilax, ERCO, Denmark) before the radiographic examination. The same radiology technicians recorded all pictures. The CE angle was defined by two lines from the centre of the femoral head, one parallel with the long axis of the body and the other running through the most lateral point of the acetabular roof as described by Wiberg [24]. Osteoarthritis was classified according to Kellgren and Lawrence (K/L) with a range from minor formation of osteophytes on the joint margins (grade 1) to disintegration of the hip (grade 5) [12].

Table 2 Weighted prevalence of osteoarthritis (OA) in the Sámi population

Age	*N*	Male (%)	Female (%)	Total (%)
20–39	210	5.2	-	2.1
40–49	302	3.9	6.3	5.2
50–64	322	8.8	11.1	10.0
All	836	6.5	7.1	6.8

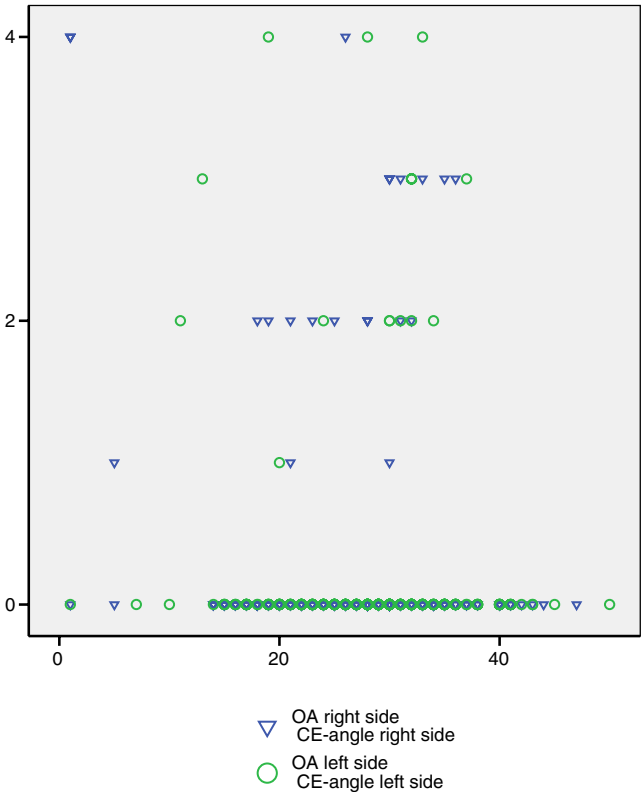

Fig. 1 Osteoarthritis (OA) score (K/L) versus Wiberg angle/CE angle. Y-axis: hip osteoarthritis graded 1–5 according to Kellgren and Lawrence. 0 = no osteoarthritic changes (no subjects with grade 5). X-axis: CE angle according to Wiberg

The films were read by an orthopaedic specialist who was unaware of the clinical status of the subjects (O.R.). According to Wiberg [24] we defined a CE angle greater than 24° as normal, angles between 20 and 24° as subnormal or slightly dysplastic, while below 20° was defined as pathological.

Statistics

The statistical analysis was performed with SPSS v12. Logistic regression was performed with osteoarthritis as the dependent variable. OA was defined as positive if a patient had OA on at least one side grade 2 or more according to K/L. The model contained four independent variables (gender, age, smallest CE angle of left or right and back pain) Separate analyses were also run for left and right hips. *P* values <0.05 were considered statistically significant.

Finally, we performed multiple linear regression analysis. The dependent variable, OA, as ordinal variable, graded from 0 to 5, on both sides, with the same independent variables as mentioned above.

Ethics

The study was approved in the Regional Ethics Committee for Medical Research in North Norway. The national Data Inspectorate (Datatilsynet) gave us permission to establish personal records. This work was supported by "Medisinsk forskning i Finnmark", University of Tromsø.

Results

All the persons examined by radiography were regarded as a random sample of the back pain group (210 persons) and no back pain group/controls (626 persons). This was accounted for in a material estimated analysis that showed a prevalence of OA (grade=>1, according to K/L) in this Sámi population to be 6.8% (Table 2).

The patterns did not show a clear correlation between AD as expressed by the CE angle and OA (Fig. 1). Most of the observations indicated an osteoarthritis score of nil, but the CE angles varied from 0 to 50°. On the other hand, the observed CE angles in subjects with an OA score of 1–5 tended to correlate positively with a high CE angle, which is opposite to what was expected.

The logistic regression analysis did not detect any significant effect of CE angle on OA. The full model containing all predictors was statistically significant: $P=0.003$. The model had a pseudo R^2 of between 5% (Cox and Snell) and 11% (Nagelkerke). As shown in Table 3, of the

Table 3 Logistic regression

	B	SE	Wald	*P*	Odds ratio (OR)	95.0% CI for OR	
						Lower	Upper
CE angle	−0.032	0.029	1.269	0.260	0.968	0.916	1.024
Sex	−0.450	0.424	1.124	0.289	0.638	0.278	1.465
Age	0.074	0.027	7.437	0.006	1.077	1.021	1.135
Back pain	0.763	0.452	2.859	0.091	2.146	0.886	5.199
Constant	-5.108	1.668	9.383	0.002	0.006		

The model was built with osteoarthritis as the dependent variable; the independent variables were entered in one step. The non-significant terms sex and back pain were retained for confounder adjustment. The model is significant ($P=0.003$)

independent variables, only age made a statistically significant contribution to the model. The separate analyses for right and left side did not show any significant association between presence of OA and low CE angle.

The separate multiple linear regression analysis for right and left hips, with the dependent variable OA, graded from 0 to 5, also showed no significant association between OA and AD.

Discussion

In this study we used the CE angle of Wiberg [24] to quantify acetabular deficiency of the hip. In a previous article we introduced the results of the mean CE angle (26±7°) and AD rates in this Sámi population [14]. The CE angle was far below values which previously had been presented as the normal range from other populations [18, 3]. This difference may reflect genetic variations in the anatomy of the human hip in agreement with previous observations [6, 25, 8, 16]. However, one possible risk factor in the Sámi population for developmental dysplasia of the hip (DDH) was the earlier tradition of wrapping up the child and laying it in the Sámi/Lapp cradle with the hips extended and adducted. Also, the Sámi population examined in this study live north of the Arctic circle, where the sun totally disappears for some months yearly, and a possible link between vitamin D and DDH can be suspected [11].

The relationship between DDH and later development of OA of the hip has been assumed since the work of Wiberg, and it has been proposed that in some patients with primary hip OA the disease occurs as a consequence of AD that persists into adult life. Support for this theory comes from radiographic observations in patients with OA of the hip [6].

Then, if there is an association between AD and degenerative hip joint disease, it should been assumed that the Sámi population is at greatest risk for hip OA, because of the very high prevalence of AD [14]. However, in this study, we could not find any evidence that AD in general is associated with increased risk of incident hip OA.

Also, two cross-sectional studies that evaluated hip radiographs for changes in hip OA and AD in a setting similar to ours failed to identify an association between these disorders [8, 5]. Inoue et al. [8] presented also a theory that low CE angle can be a possible favourable factor for acetabular cartilage, which is associable with this study, showing that those subjects with the highest grades of osteoarthritis had relatively high CE angles, indicative for a slightly negative correlation (Fig. 1). However, the sample size is relatively small as only 27 of 315 possible subjects showed some degree of OA.

On the other hand, our observations are in contrast to other previous studies: one cross-sectional study from Denmark [9] and two follow-up studies reporting that AD is associated with incident radiographic OA of the hip [15, 19]. In the study from Denmark, a small, but significant correlation was found between AD and OA (odds ratio: 1.07–1.6). However, the two prospective studies were performed in elderly patients with a mean age of 65 and 70 years, in contrast to a mean age of 48 years in our study. Furthermore, it was only found as a risk factor in women. In the Reijman et al. study [19], when CE angle for men was below 25°, the odds ratio (OR) was not significant (OR: 2.6, 95% confidence interval: 0.8–8.6). In addition, in the Lane et al. study [15], the CE angle below 30° for women had a OR=3.3 with a wide 95% confidence interval from 1.1 to over 10, which is an indication of low power in the study.

Taken together, these different observations indicate that, if AD is a risk factor, it is mostly of relevance in elderly women. Subclinical AD, then, persists into adult life in the majority of people without predisposing to hip OA. This is compatible with the authors' clinical impressions that the Sámi people have few problems with their hips, compared with other populations in the area.

Viewed against this background and all works with ethnical and geographic differences on the prevalences of DDH and OA and the differences in the relations between DDH and OA, the aetiology of OA must be multifactorial, and likely the main reason is still unknown. This is supported by the findings of this study, showing no significant relation between AD and hip OA.

In conclusion, among Sámi, a hip with DDH has low predictive value of concurrent radiographic osteoarthritis. Thus, radiographic indices can probably not predict the rate at which the hip joint will develop osteoarthritis. Supposedly other factors of hip development may be of importance for the development of OA, including hereditary, ethnic and other factors yet to be found.

References

1. Cooperman DR, Wallensten R, Stulberg SD (1983) Acetabular dysplasia in the adult. Clin Orthop Relat Res 175:79–85
2. Felson DT, Zhang Y (1998) An update on the epidemiology of knee and hip osteoarthritis with a view to prevention. Arthritis Rheum 41:1343–1355
3. Fredensborg N (1976) The CE angle of normal hips. Acta Orthop Scand 47:403–405
4. Getz B (1956) Congenital dislocation of the hip in Lapps (in Norwegian). Tidsskr Nor Laegeforen 76:812–815
5. Goker B, Sancak A, Haznedaroglu S (2005) Radiographic hip osteoarthritis and acetabular dysplasia in Turkish men and women. Rheumatol Int 25:419–422
6. Goodman DA, Feighan JE, Smith AD, Latimer B, Buly RL, Cooperman DR (1997) Subclinical slipped capital femoral epiphysis. Relationship to osteoarthrosis of the hip. J Bone Joint Surg Am 79:1489–1497

7. Inoue K, Shichikawa K, Ota H (1999) Prevalence of hip osteoarthritis and acetabular dysplasia in Kamitonda: from a longitudinal population-based epidemiological study of rheumatic diseases in Japan. Rheumatology (Oxford) 38:793–794
8. Inoue K, Wicart P, Kawasaki T, Huang J, Ushiyama T, Hukuda S, Courpied J (2000) Prevalence of hip osteoarthritis and acetabular dysplasia in french and japanese adults. Rheumatology (Oxford) 39:745–748
9. Jacobsen S, Sonne-Holm S, Soballe K, Gebuhr P, Lund B (2005) Hip dysplasia and osteoarthrosis: a survey of 4151 subjects from the Osteoarthrosis Substudy of the Copenhagen City Heart Study. Acta Orthop 76:149–158
10. Johnsen K, Gran JT, Dale K, Husby G (1992) The prevalence of ankylosing spondylitis among Norwegian Samis (Lapps). J Rheumatol 19:1591–1594
11. Kapoor B, Dunlop C, Wynn-Jones C, Fryer AA, Strange RC, Maffulli N (2007) Vitamin D and oestrogen receptor polymorphisms in developmental dysplasia of the hip and primary protrusio acetabuli–a preliminary study. J Negat Results Biomed 6:7
12. Kellgren JH, Lawrence JS (1957) Radiological assessment of osteo-arthrosis. Ann Rheum Dis 16:494–502
13. Kim YJ, Ganz R, Murphy SB, Buly RL, Millis MB (2006) Hip joint-preserving surgery: beyond the classic osteotomy. Instr Course Lect 55:145–158
14. Knut J, Goll R, Reikerås O (2008) Acetabular dysplasia in the Sami population: a population study among Sami in north Norway. Int J Circumpolar Health 67(1):147–153
15. Lane NE, Lin P, Christiansen L, Gore LR, Williams EN, Hochberg MC, Nevitt MC (2000) Association of mild acetabular dysplasia with an increased risk of incident hip osteoarthritis in elderly white women: the study of osteoporotic fractures. Arthritis Rheum 43:400–404
16. Lanyon P, Muir K, Doherty S, Doherty M (2000) Assessment of a genetic contribution to osteoarthritis of the hip: sibling study. BMJ 321:1179–1183
17. Lau EM, Symmons DP, Croft P (1996) The epidemiology of hip osteoarthritis and rheumatoid arthritis in the Orient. Clin Orthop Relat Res 323:81–90
18. Murphy SB, Kijewski PK, Millis MB, Harless A (1990) Acetabular dysplasia in the adolescent and young adult. Clin Orthop Relat Res 261:214–223
19. Reijman M, Hazes JM, Pols HA, Koes BW, Bierma-Zeinstra SM (2005) Acetabular dysplasia predicts incident osteoarthritis of the hip: the Rotterdam study. Arthritis Rheum 52:787–793
20. Roux CH, Saraux A, Mazieres B, Pouchot J, Morvan J, Fautrel B, Testa J, Fardellone P, Rat AC, Coste J, Guillemin F, and Euller-Ziegler L (2007) Screening for hip and knee osteoarthritis in the general population: predictive value of a questionnaire and prevalence estimates. Ann Rheum Dis Dec 12 [Epub ahead of print]
21. Tambets K, Rootsi S, Kivisild T, Help H, Serk P, Loogvali EL, Tolk HV, Reidla M, Metspalu E, Pliss L, Balanovsky O, Pshenichnov A, Balanovska E, Gubina M, Zhadanov S, Osipova L, Damba L, Voevoda M, Kutuev I, Bermisheva M, Khusnutdinova E, Gusar V, Grechanina E, Parik J, Pennarun E, Richard C, Chaventre A, Moisan JP, Barac L, Pericic M, Rudan P, Terzic R, Mikerezi I, Krumina A, Baumanis V, Koziel S, Rickards O, De Stefano GF, Anagnou N, Pappa KI, Michalodimitrakis E, Ferak V, Furedi S, Komel R, Beckman L, Villems R (2004) The western and eastern roots of the Saami–the story of genetic "outliers" told by mitochondrial DNA and Y chromosomes. Am J Hum Genet 74:661–682
22. Wedge JH, Wasylenko MJ (1978) The natural history of congenital dislocation of the hip: a critical review. Clin Orthop Relat Res 137:154–162
23. Wessel AB (1918) Laaghalte slegter i Finmarken. Tidsskr Nor Laegeforen 38:337
24. Wiberg G (1939) Studies on dysplastic acetabulae and congenital subluxation of the hip joint. Acta Orthop Scand 58:1–132
25. Wynne-Davies R (1970) A family study of neonatal and late-diagnosis congenital dislocation of the hip. J Med Genet 7:315–333