Osteoarthritis of the hip joint and acetabular dysplasia in women

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Abstract

Objective—To investigate the suggestion that osteoarthritis (OA) of the hip joint is often caused by subclinical acetabular dysplasia among elderly British women.

Methods—We examined 393 hip joints from the radiographs of a sample of women aged 60–75 years undergoing intravenous urography. Acetabular dysplasia was assessed using measurements of the centre-edge (CE) angle and acetabular depth (AD), which are both reduced in this condition. OA was assessed using an overall grade based on the Kellgren and Lawrence system and by measurement of minimum joint space (MJS).

Results—MJS was the more repeatable measure of OA, and showed a strong correlation with overall grade (Spearman rank correlation coefficient \( r_s = -0.61, p < 0.001 \)). MJS was significantly positively correlated with CE angle (Pearson correlation coefficient \( r = -0.25, p < 0.001 \)) and AD (\( r = -0.11, p < 0.05 \)). Consistent with these findings, there was a weak but significant positive correlation between overall grade of OA and one of the two measures (CE angle) of acetabular dysplasia.

Conclusions—These results do not support the hypothesis that mild degrees of acetabular dysplasia account for a substantial proportion of hip OA in elderly women. Changes in hip joint geometry as a result of OA may be responsible for the weak negative association observed.

Patients and methods

A sample of women aged 60–75 years old undergoing intravenous urography during 1988–91 was identified from the records of the radiology department at Southampton General Hospital. Radiographs were retrieved and individual hip joints were assessed from postmicturition films. Hip joints were excluded if visualisation was inadequate or total hip replacement surgery had been performed.

Observer 1 (RWS) examined the radiographs over a three month period. Angular and linear indices were measured to the nearest degree and 0.5 mm, respectively, using techniques described previously7 and the bony landmarks illustrated in figure 1. Osteoarthritis was quantified by measurements of minimum joint space (MJS)9 and according to a global grading system based on the Kellgren-Lawrence scale.10 Acetabular dysplasia was assessed by measurement of the centre-edge (CE) angle and acetabular depth (AD) as previously described.7

Correlation between measurements of acetabular dysplasia and OA was examined using Pearson's correlation coefficient and Spearman's rank correlation coefficient. A subset of 40 hip joints was reassessed by observer 1 after a three month interval and then independently by observer 2 (CC), each unaware of previous results. Repeatability of OA criteria was tested using Cohen's kappa statistic.

Osteoarthritis (OA) of the hip joint is an important cause of disability in the elderly.1 While it is generally accepted that the severe degrees of acetabular dysplasia associated with congenital dislocation of the hip (CDH) predispose to OA, the importance of mild forms of the condition remains unclear.2

Studies of hospital patients have suggested that OA is a frequent sequel of acetabular dysplasia.4–6 In contrast, a previous epidemiological study of elderly men in the general population failed to detect an association between the two disorders.7 However, the incidence of CDH in women is approximately double that in men,5 and acetabular dysplasia may play a greater role in the aetiology of OA of the hip joint in women. We have therefore investigated the relationship between OA and acetabular dysplasia in elderly women.

Figure 1. Diagrammatic reproduction of the hip joint illustrating bony landmarks and radiographic measurements used in the study. A = Minimal joint space; B = osteophyte; C = sclerosis; D = Centre-edge angle; E = acetabular depth.
Results
The intravenous urograms of 203 women were retrieved, and 393 hip joints were suitable for assessment. A total of 13 were excluded because of previous hip joint surgery (nine) or poor visualization (four). The mean MJS in the 393 female hips assessed was 3-02 mm (SD 0-70 mm). Eighteen hips (4-6%) had an overall OA grade of 3–5 (equivalent to grades 3/4 on the Kellgren-Lawrence scale). MJS was closely correlated with overall grade (Spearman rank correlation coefficient $r_s = -0.61$, $p < 0.01$), but the former measure was more repeatable both within and between observers (kappa = 0.85 and 0.42, respectively) than the latter (kappa = 0.61 and 0.37, respectively). The prevalence of osteophyte was 3-6% and the mean depth of subchondral sclerosis was 1-1 mm in the sample as a whole.

Both measures of acetabular dysplasia were highly repeatable, with Pearson correlation coefficients ranging between 0-86 and 0-97 for within and between observer variation. Mean CE angle was 38° (SD 6-5°) and mean AD was 14-4 mm (SD 2-8 mm); 3-8% of hips had a CE angle less than 25°, an AD of less than 9 mm, or both (these being previously suggested thresholds for these measurements). Among women with MJS measurements of less than 1-5 mm, none had CE angles or AD values below these thresholds. Even when the results were re-analysed assuming the nine replaced hips to all be dysplastic, the prevalence of dysplasia in the study sample increased only to 6-1%.

Figure 2 shows that MJS was significantly negatively correlated with CE angle ($r = -0.26$, $p < 0.001$) and AD ($r = -0.11$, $p < 0.05$). Consistent with this observation, overall OA grade was positively associated with CE angle ($r_s = 0.11$, $p < 0.05$) and positively, though not significantly, with AD ($r_s = 0.04$, $p > 0.10$). The table shows the measurements of acetabular dysplasia in hips with severe OA, as defined by MJS 1.5 mm or less, compared with those of the sample as a whole. The mean values of CE angle and AD in the nine severely affected hips were greater than those for the total sample.

Discussion
Our results do not support the hypothesis that acetabular dysplasia is a major cause of hip OA in elderly British women. Women undergoing intravenous urographic examination for non-musculoskeletal indications were selected for study. Urological disorders are not generally associated with arthropathy, and although inappropriate investigation of hip pain may have distorted the sample, it is unlikely to have biased any association between OA and acetabular dysplasia.

Epidemiological studies of hip OA require indices of the disorder which are simple to use and repeatable. In this study we have shown that the radiographic criteria we previously validated in men may also be applied to women. While MJS is a more repeatable measure than overall OA grade, some investigators have suggested that the addition of other radiographic features (most notably osteophyte) to measures of joint space improves prediction of hip pain and dysfunction. We therefore measured both MJS and overall OA grade using an explicitly stated scale which can be directly translated to the Kellgren-Lawrence system.

When compared with our previous data in men, mean MJS in women was slightly (around 0.5 mm) smaller, a difference readily attributable to differences in height. However, the approximate 2.5th percentile of the MJS distribution was identical ($\leq 1.5$ mm) in both sexes. Major differences between men and women included fewer osteophytes (3.5% v 14%) and lower mean depth of subchondral sclerosis (1.1 v 3.6 mm) among women.

| Measurements of acetabular dysplasia in hips with severe OA changes compared with whole study population |
|--------------------------------------------------|--------|--------|--------|
| CE angle (degrees) | AD (mm) | All hips | MJS < 1.5 mm |
| All hips | MJS < 1.5 mm | All hips | MJS < 1.5 mm |
| Number | 390 | 9 | 386 | 9 |
| Mean | 38.0 | 42.4 | 14.4 | 15.8 |
| SD | 6.5 | 12.0 | 2.8 | 4.8 |

MJS = minimum joint space.
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Developmental abnormalities of the hip are known to predispose to premature hip OA. The three most important conditions are congenital dislocation of the hip, slipped capital femoral epiphysis and Perthes’ disease. However, their estimated cumulative incidence could only explain a small proportion of all adult hip OA. The claim for their significance is based on the hypothesis that they also occur in milder forms which disturb hip biomechanics while remaining clinically silent in childhood. Such hip dysplasias are a frequent cause of OA in some heavy breeds of dog. Ecological studies suggest, for humans, that areas with a low prevalence of hip dysplasia also have low rates of hip arthroplasty, although methodological difficulties make this literature difficult to interpret. However, the most important piece of evidence in evaluating the role of human hip dysplasia in causing OA is the relationship between these disorders in individuals. Such studies have usually involved the measurement of acetabular configuration in patients awaiting surgery for advanced hip OA. Prevalence estimates of dysplasia, using various thresholds for CE angle and AD, are substantial with a range from 20 to 48%. However, in the only population based study performed in 60–75 year old men, we were unable to find any association between these widely accepted measures of dysplasia and hip OA. The results of the present study extend these findings to women.

One weakness of all such cross-sectional studies is that measurements of hip dysplasia might themselves be affected by progressive OA. We explored this issue by adjusting our CE angle measurements for the degree of superior and medial joint space loss. This adjustment reduced the previously significant negative relationship between CE angle and MJS (r = -0.01, p = 0.89), but did not unmask an association in the direction of the hypothesis being tested. Other potential biases include the age of our cohort and the exclusion of patients who had undergone arthroplasty. Our study was confined to women aged 60–75 years—an age chosen to include a reasonably high prevalence of hip OA. It remains possible that acetabular dysplasia predisposes to a greater proportion of cases of OA among younger women. Our exclusion of the nine replaced hips appears less likely to have led to biases. When the results were recalculated assuming these to have been dysplastic, the prevalence of dysplasia in the sample as a whole increased only to 6.1%.

We conclude that mild forms of acetabular dysplasia do not account for a large proportion of prevalent hip OA in elderly British women. It is difficult to explain the high prevalence of dysplasia apparent in hospital series of patients with severe hip OA. It may have arisen through inclusion of younger patients coming to surgery, a group in which the attributable risk of OA from dysplasia may be greater, or through the measures of dysplasia being altered by severe OA. These issues can be formally addressed only in a prospective population based study.

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