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Pincer deformity does not lead to osteoarthritis of the hip whereas acetabular dysplasia does: acetabular coverage and development of osteoarthritis in a nationwide prospective cohort study (CHECK)

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SUMMARY

Objective: Determining the relation between acetabular coverage, especially overcoverage which may lead to pincer impingement, and development of osteoarthritis (OA) of the hip.
Design: From a prospective cohort study of 1,002 individuals with symptoms of early OA (Cohort Hip and Cohort Knee, CHECK), 720 participants were included. Standardized anteroposterior pelvic radiographs and false profile lateral radiographs were obtained at baseline and 5 years follow-up. Acetabular undercoverage (mild dysplasia) and overcoverage (pincer deformity) were measured by a centre edge angle of $<25^\circ$ and $>40^\circ$ respectively in both radiographic views. The strength of association between those parameters at baseline and development of incident OA (Kellgren and Lawrence (K&L) grade >2 or total hip replacement), or joint space narrowing within 5 years was expressed in odds ratio (OR) adjusted for K&L grade, age, body mass index (BMI), and sex using generalized estimating equations.
Results: At baseline, 76% of the included hips had no signs of radiographic OA (K&L = 0) whereas 24% had doubtful OA (K&L = 1). Within 5 years, 7.0% developed incident OA. Acetabular dysplasia was significantly associated with development of incident OA with ORs between 2.62 (95% confidence interval (CI) 1.44–4.77) and 5.45 (95% CI 2.40–12.34), dependent on the radiographic view. A pincer deformity was not associated with any outcome measure, except for a significantly protective effect on incident OA when a pincer deformity was present in both radiographic views OR 0.34 (95% CI 0.13–0.87).
Conclusion: Acetabular dysplasia was significantly associated with development of OA. However, a pincer deformity was not associated with OA, and might even have a protective effect on its development, which questions the supposed detrimental effect of pincer impingement.

Introduction

The aetiology of osteoarthritis (OA) is mainly unclear, though both systemic factors and local biomechanical factors are known to play a role[1]. OA of the hip often occurs without the presence of OA in other susceptible joints, indicating that local biomechanical factors may predominate[2].

Growing evidence supports the theory that these local factors are mainly explained by bone shape variants of the hip, causing OA by an altered biomechanical loading pattern[3–6]. These bone shape variants can be located at the femoral side, acetabular side, or both. An example of a femoral sided morphological abnormality is a non-spherical femoral head (cam deformity) which may lead to a motion dependent abnormal contact between the femoral head and the acetabulum, also known as cam-type Femoroacetabular Impingement (FAI)[3]. A cam deformity is thought to develop during growth and is an important risk factor for OA[7–9]. An abnormal shape of the acetabulum may also lead to OA by either acetabular undercoverage, also known as (mild)

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dysplasia, or acetabular overcoverage, known as a pincer deformity.

In hips with mild acetabular dysplasia, a decreased contact area between femur and acetabulum results in higher static loads on the anterosuperior acetabular cartilage. In hips with a pincer deformity, the mechanism leading to OA is much less understood. The proposed mechanism is that of a dynamic abnormal linear contact between the overcovered acetabular rim and the femoral neck during terminal motion of the hip, which is known as pincer-type FAI[3]. When vigorous hip motion causes repetitive impingement events, the soft tissue structures within the hip joint might gradually damage, leading to hip OA.

This hypothesis is supported by intra-operative findings in symptomatic patients with a pincer deformity, where acetabular cartilage damage was found throughout the acetabulum in a small thin strip around the labrum[10]. Also, cartilage damage at the posterior-inferior site has been described as a result of a 'countrecoup lesion' by the femoral head, due to the leverage effect of the neck when it abuts against the anterior acetabular rim (Fig. 1).

Evidence for the relation between mild dysplasia and development of OA provided by cross-sectional or retrospective studies is inconsistent, but prospectively designed studies generally show a moderate increased risk for hip OA[11–15]. In contrast, the relation between pincer deformities and development of OA is conflicting[16–23]. However, these studies are often limited by a retrospective or cross-sectional design, making it difficult to draw conclusions on causality. As in mild dysplasia, prospective studies might identify an association, but no such studies are available for pincer deformities.

The aim of this study was to examine the relation between baseline anterior and lateral acetabular coverage, specifically pincer deformities, and the risk of developing OA after 5 years follow-up. We further investigated whether acetabular coverage was associated with pain and decreased hip function.

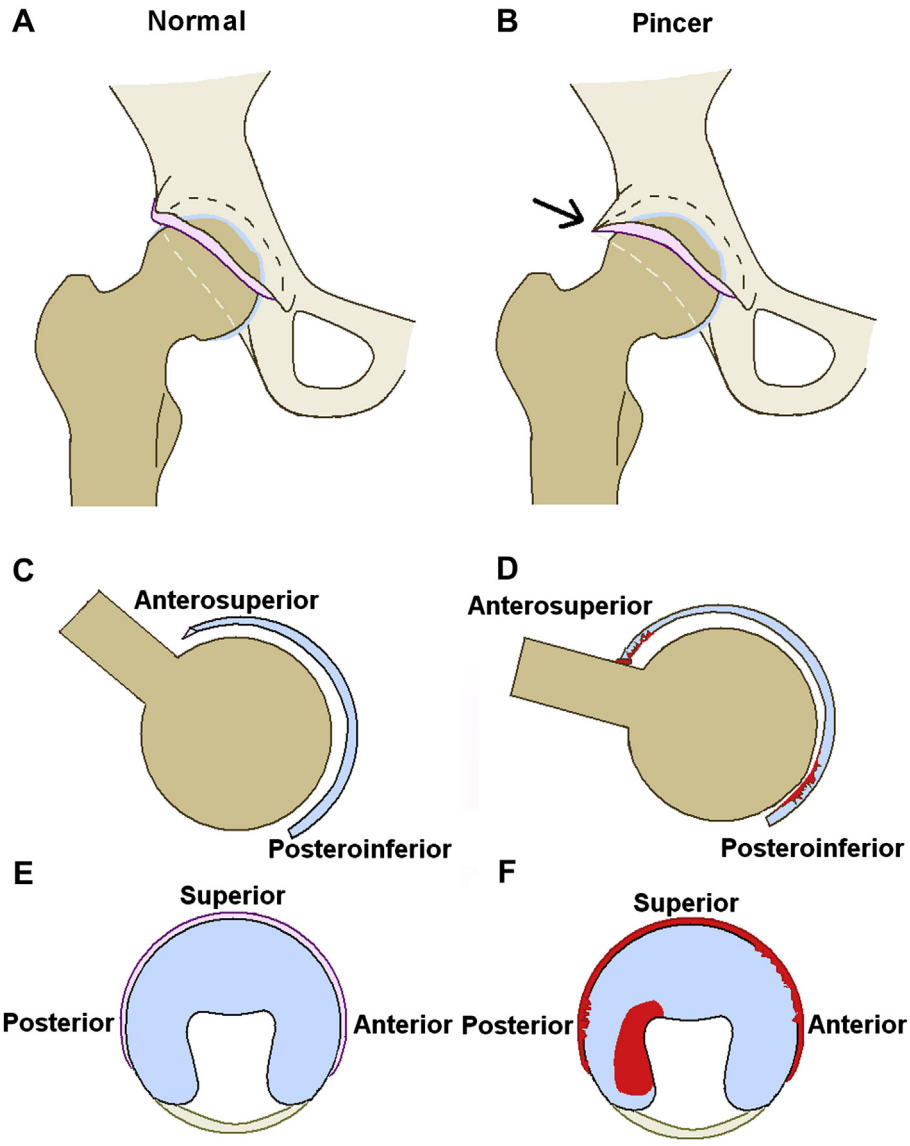

Fig. 1. The mechanism of pincer impingement. A normal hip (A) and a hip with a pincer deformity (B) are shown. The anatomy of the normal hip provides the hip a physiological ROM (C) whereas a pincer deformity (arrow) is proposed to lead to an abnormal linear contact between the overcovered acetabular rim and the femoral neck during terminal motion of the hip, which is known as pincer impingement (D). When vigorous hip motion causes repetitive impingement events, the acetabular cartilage might gradually damage throughout the acetabulum in a small thin strip around the labrum. Also, the leverage of the femoral head in the acetabulum might lead to a contrecoup lesion posteroinferiorly (F).