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# The Relationship of Acetabular Dysplasia and Femoroacetabular Impingement to Hip Osteoarthritis: A Focused Review

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## Abstract

Hip osteoarthritis (OA) leads to significant functional limitations and economic burden. If modifiable risk factors for hip OA are identified, it may be possible to implement preventative measures. Bony abnormalities associated with acetabular dysplasia (AD) and femoroacetabular impingement (FAI) have been recently implicated as risk factors for hip osteoarthritis (OA). The purpose of this focused review is to summarize the available evidence describing the relationship between bony abnormalities and hip OA. A librarian-assisted database search using PubMed, Embase and Central was performed. Relevant articles were identified and assessed for inclusion criteria. The authors reviewed cohort and case control studies that reported on the association between abnormal hip morphology and hip OA. The available literature suggests that an association exists between bony abnormalities found in AD and FAI and hip OA and preliminary evidence suggests that AD is a risk factor for OA, however these conclusions are based on limited evidence. Prospective, longitudinal studies are needed to confirm the causal relationship between abnormal hip morphology and the future development of hip OA.

## Introduction

Hip osteoarthritis (OA) leads to significant functional limitations in middle-aged to older individuals and substantial economic burden due to medical costs and lost productivity.[1] If modifiable risk factors for hip OA are identified, it may be possible to implement preventative measures. To date, evidence suggests that age,[2] trauma,[2–4] physical workload,[5] sporting activities,[6] genetics[7–10] and being overweight[3, 5] are risk factors for hip OA. Another proposed risk factor of interest is the role of abnormal bony morphology in the development of OA. [11,12, 1314, 15]

It is generally accepted that gross bony abnormalities, such as congenital hip dislocation (CHD), Legg-Calvé-Perthes disease and slipped capital femoral epiphysis (SCFE) contribute to OA. More recently, subtle variations in the morphology of the acetabulum and femoral



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head have been implicated as an underlying cause of idiopathic or primary OA.[16] More specifically, bony abnormalities associated with acetabular dysplasia (AD) and femoroacetabular impingement (FAI) are of recent interest. Insufficient acetabular coverage of the femoral head associated with AD, excessive acetabular coverage of the femoral head associated with pincer FAI or an abnormally shaped proximal femur associated with cam FAI are proposed to contribute to abnormal stress patterns within the hip joint, leading to degenerative changes in the articular cartilage and eventually OA. The purpose of this focused review is to summarize the published literature describing the relationship between bony abnormalities of the hip joint and the incidence of hip OA.

## Literature Search

Publications were selected by searching the PubMed, Embase and CENTRAL databases through October 2010. The literature search was performed by a librarian employed by *blinded* University School of Medicine with assistance of the authors for key words. Various combinations of the following key words and related MESH terms were used: hip; osteoarthritis; arthrosis; dysplasia; impingement; femoroacetabular impingement; imaging; radiography; cadaver inspection; risk factor. A complete list is provided as an appendix. The authors searched the reference lists of selected literature for additional articles. To be included, the study had to 1) report on the association of a bony abnormality and presence of OA in humans, 2) be available in English and full text and 3) be a cohort or case-control study. Articles reporting on the progression of OA were not included in this review.

The search resulted in 279 articles from PubMed, 887 articles from EMBASE and 1 article from CENTRAL. After removing duplicates and screening titles and abstracts, 67 articles remained to be reviewed. Full text papers were screened and 46 additional articles were excluded for not meeting the criteria outlined above. The reference lists of the selected papers were screened, and 6 additional articles were included. A total of 27 articles were included in this focused review. Twenty articles reported on AD and nine articles reported on FAI. Two articles reported on both AD and FAI.[17, 18]

### ACETABULAR DYSPLASIA

Hip dysplasia is a developmental disorder in which the hip joint forms incorrectly during childhood, resulting in abnormalities of the femoral head, acetabulum, or both. Proposed risk factors for developmental hip dysplasia include fetal positioning during pregnancy, vaginal delivery with breech presentation, and genetic predisposition.[19] Current treatments aim to “reduce” the hip by repositioning it into proper alignment by using a harness, cast, or surgery. While full recovery is possible, lack of or delayed treatment allow mild forms of dysplasia to persist into adulthood. In this section, we will focus on literature specific to the dysplastic or shallow acetabulum.

Compared to the normally shaped acetabulum, acetabular dysplasia (AD) results in a smaller weight-bearing surface (Figure 1) and thus increased contact stresses that may contribute to direct labral or articular cartilage damage.[20] In addition, AD is proposed to result in structural instability that allows the femoral head to migrate into regions of less coverage. This migration may place increased stresses on the supporting soft tissues, leading to injury of the acetabular labrum, articular cartilage and joint capsule. Acetabular labral tears in association with AD are proposed to further contribute to injury. The acetabular labrum provides a seal around the hip joint that prevents fluid expression from the joint space and also provides stabilization of the hip joint. A tear of the labrum compromises this seal[21] which may result in additional negative effects on hip stability and the distribution of forces on the cartilage.[22, 23] While it has been accepted that severe forms of AD lead to osteoarthritis, less is known about mild or moderate AD.

Twenty articles were reviewed that reported the relationship between AD and OA (Table 1). Two were prospective cohort studies with a follow up period that ranged from an average of 6.6 to 22 years.[24, 25] Three were prospective case control studies with 8–10 years follow up.[26–28] Fifteen were cross-sectional studies.[17, 18, 29–41] A synopsis of these reports highlights the variability in methodology and reporting, and emphasizes the challenges in interpreting the available literature.

Three studies included only women subjects,[27, 36, 40] two included only men,[30, 35] seven included a greater majority of women,[18, 24, 26, 29, 32, 33, 38] two included a greater majority of men,[31, 39] and five included equal numbers of men and women.[17, 25, 34, 37, 41] A number of countries were represented including the United States,[27, 38, 40] United Kingdom,[29, 30, 36, 41] Denmark,[18, 26, 32, 33] Sweden,[24] Korea,[17] Japan,[31, 37] Norway,[34] Switzerland,[28] France,[31] Turkey,[39] China[35] and the Netherlands.[25] Eleven studies included only subjects greater than 40 years of age,[17, 25, 27, 29, 30, 35–37, 39–41] one study included only subjects less than 50 years[38] and seven studies included subjects across a broad age span.[18, 24, 26, 31–34] One study did not specify the sex or age of their subjects.[28]

The most common measurement to represent AD was the lateral center edge angle (CEA) of Wiberg[42] (Figure 1). All 20 studies used the CEA, however they used different values to define AD. Seven studies defined AD as CEA ≤ 20°,[17, 18, 24, 26, 32–34] six used CEA ≤25°,[29, 31, 35–37, 40] one used CEA≤30°[27] and three studies analyzed their data using more than one value, CEA ≤ 20° and ≤25°,[39] CEA ≤25° and ≤30°[25] and CEA ≤20° and a study-specific definition.[41] Three studies did not specify a particular CEA value to represent acetabular dysplasia.[28, 30, 38] Eleven studies used the acetabular depth index[43](Figure 2).[25, 27, 29–31, 35–37, 39–41] Two studies[30, 31] did not provide the criteria to define AD, however all others used an acetabular depth index of <9 mm to define AD. In addition, one study used a study-specific definition of the acetabular depth index.[41] Four studies defined AD as acetabular depth ratio[26] (Figure 2) ≤250%.[24, 26, 32, 33] Five studies used the femoral head extrusion index[44] (Figure 3).[24, 26, 28, 32, 33] One study[28] did not provide criteria to define AD, however all others used a head extrusion index as ≤25%. Other parameters to determine AD used less frequently included the Tonnis angle,[28, 38] a qualitative judgment of acetabular inclination,[24] anterior center edge of Lequesne,[38] and acetabular angle of Sharp.[24, 32]

The definition of OA varied across studies. Twelve studies used minimum joint space width,[17, 18, 26, 29–33, 35–37, 41] two used joint space narrowing,[25, 26] six used the Kellgren-Lawrence (K\L)[45] grading system,[25, 28, 31, 32, 34, 39] four used the Croft modification of the K/L system,[29, 32, 36, 37] three described their own customized scoring system[24, 27, 40] and one study used delayed Gadolinium Enhanced Magnetic Resonance Imaging of Cartilage (dGemeric), an imaging technique used to assess the biochemical properties of the articular cartilage.[38]

**Study Outcome Measures—**The studies reported their results in varying ways. Nine reported an odds ratio,[17, 18, 25, 27, 32–34, 40, 41] six reported a correlation coefficient[30, 31, 36, 37] and twelve reported the differences in the mean AD measures or the prevalence of AD between their defined cases and controls.[25–29, 31, 35, 38–41] One study reported the rate of OA onset.[24] To assess the findings across studies, articles were classified as “positive” if they demonstrated a positive association between the presence of AD and OA and “negative” if they demonstrated no association or a negative association. Eight studies were classified as positive.[17, 25, 27, 28, 32, 33, 38, 41] Six positive studies reported odds ratios. Using CEA, acetabular depth or femoral head extrusion index, these studies estimate that people with AD have 1.1 to 10.2 higher risk of developing OA than people who do not have AD.

Twelve studies were classified as negative. Two studies reported a trend relationship between AD and OA, however statistical significance was not met.[18, 26] Six additional studies found no association[24, 29, 34, 35, 39, 40] and four studies reported a negative correlation between minimum joint space and CEA (r = −0.18 to −0.39)[30, 31, 36, 37] and acetabular depth index (r =−0.11 to −0.15)[30, 31, 36]

**Qualitative Analysis—**Using the criteria described by the Centre for Evidence-Based Medicine, Oxford, United Kingdom,[46] articles were rated on a scale from one to five, one indicating a high quality study and five indicating expert opinion. Of the negative studies, two were prospective studies with small samples so were rated level three.[24, 26] The remaining articles were all cross-sectional studies and therefore rated level four. [18, 29–31, 34–37, 39, 40] The strength of these cross-sectional studies are limited due to the fact that the measures taken to represent the presence of AD, e.g. the CEA, may be affected by the presence of OA. Six studies[30, 31, 35–37, 39] used radiographs taken for non-skeletal purposes making it impossible to control variables such as pelvic rotation. Six studies did not account for a probable confounding factor, the possible relationship between a large CEA and OA. [30, 31, 34, 36, 37, 40] In these six negative studies, the authors assumed a linear relationship between the CEA and OA in their statistical analyses. Recently, it has been suggested that excessive acetabular coverage may also be a risk factor for OA, suggesting a curvilinear relationship. If the potential curvilinear relationship had been considered, the results of these studies may have been different. All four articles finding a negative correlation between CEA and OA made this assumption.[30, 31, 36, 37]

Three positive articles using a prospective cohort or case control design were rated level two,[25, 27, 28] one study had a relatively large sample.[25] Prospective studies allow measurement of the variables representing AD prior to the onset of OA and may assist in establishing the temporal relationship between AD and OA. The remaining five studies were rated level four, demonstrating some of the same limitations previously described.[17, 32, 33, 38, 41]

The studies reviewed reported conflicting results about the relationship between AD and OA. When prioritized based on the articles' level of evidence, however, there is limited evidence to suggest a positive relationship between AD and OA. Although there were a greater number of negative articles, none of the negative articles rated higher than level three. In addition, six of the negative articles did not account for a confounding factor that could have potentially reversed their results. Three of the positive articles were rated level two and were best designed to address the temporal relationship between AD and OA.

Our conclusions are similar to those reported by Lievense et al.[47] Lievense et al reviewed nine case-control and cohort studies that were reported prior to 2000. They concluded that a correlation exists between AD and OA, however due to a lack of well designed, prospective studies the evidence was limited. Since their review, eight studies have been published, six reporting a positive relationship between AD and OA.

Despite years of research on AD as a risk factor for OA, inconsistencies in the diagnostic criteria of both conditions have prevented a consensus from being reached. Although AD is commonly diagnosed using the CEA, the definition of an abnormal CEA is not consistent across studies, ranging from 20° to 30°. Defining radiographic OA is even more challenging, with most studies using some combination of minimal joint space, joint space narrowing, and presence of sclerosis, osteophytes, or cysts as evidence of OA. Abnormal values of minimal joint space are equally inconsistent with authors using values that range from 1.5–2.5 mm. In addition, morphological differences in joint space exists between men and women, further complicating the definition of OA.[48] The most commonly used scoring

system was the Kellgren-Lawrence[45] grading system, a system that has been shown to have high interobserver variability and inconsistent application across studies.[49] No studies used the Tonnis scale that is specific to the hip, which may have affected the study findings. Finally, previous studies have shown that radiographic findings may not be associated with the amount of cartilage damage in the joint, in particular in those with less advance disease.[50] Despite inconsistencies in the methods used, there is evidence to suggest that AD is associated with OA and preliminary evidence that AD may be a risk factor for OA. Clearly however, more studies are needed to establish the temporal relationship between AD and OA.

## FEMORACETABULAR IMPINGEMENT

Femoroacetabular impingement (FAI) results from one or more bony abnormalities that lead to abnormal contact between the acetabulum and the femoral head or neck. Based on the cause and location of the impingement, FAI can be categorized as cam-type, pincer-type or more commonly a combination of both. Cam FAI results from an aspherical femoral head, often referred to as a pistol-grip or post-slip deformity, or a loss of the concavity of the anterosuperior femoral head-neck junction (Figure 4). Pistol-grip refers to the radiologic appearance of the proximal femur, and post-slip describes a possible etiology of the impingement. The femoral abnormality is proposed to cause compression and shear stresses in the region between the labrum and cartilage, anterosuperiorly. These stresses cause a separation between the labrum and cartilage as the labrum is pushed outwards and the cartilage is pushed centrally. This eventually leads to articular degeneration and eventually global hip OA.[13, 14]

Pincer FAI results from a general or a localized acetabular over-coverage of the femur (Figure 4). General over-coverage may be caused by global acetabular retroversion or a deep acetabulum, represented by coxa profunda[14] or protrusio acetabuli. Local over-coverage may be caused by a small overgrowth of the acetabulum located at the roof of the acetabulum. This local overcoverage has also been termed acetabular retroversion, however more appropriately is described as cranial or superioanterior acetabular retroversion. Pincer FAI is proposed to compress the labrum and increase stresses on the underlying acetabular rim in the area of acetabular over-coverage. With repeated microtrauma, bone growth is induced and ossification may occur at the underlying rim, further deepening the acetabulum and worsening the overcoverage. The abutment, most commonly anterior, may cause a leveraging of the femoral head in the acetabulum and lead to ‘contre-coup’ chondral injury in the posterior region. Over time, the repetitive microtrauma may lead to OA.[1413]

Although these impingement patterns differ, they often co-exist in people with intra-articular pathology.[14] Recent literature has provided increasing evidence that cam and pincer FAI cause distinct patterns of articular cartilage and labral damage and may serve as an etiologic factor of “idiopathic” osteoarthritis.[14, 17, 18, 51–57]

Nine articles were reviewed that reported the relationship between FAI and OA (Table 2). One study included a prospective design with a six year follow up.[58] Eight studies had a cross-sectional design.[17, 18, 51–56]

Four studies included a greater majority of women,[18, 51, 52, 58] two included a greater majority of men[55, 56] and two included equal numbers of men and women.[17, 53] One study did not report the sex of their subjects.[54] Countries represented in these studies include United States,[52, 56] or North American continent,[55] United Kingdom,[53] Switzerland,[54] Denmark,[18] Japan,[51] Korea,[17] and the Netherlands.[58] Two studies included only subjects greater than 55 years of age,[17, 58] and six studies included subjects across a broad age

span.[18, 51, 53–56] The remaining study did not report their sample's age range, however reported the mean age of 72 and 59 years for cases and controls respectively.[52]

Four studies reported the relationship between pincer FAI (excessive acetabular coverage) and OA,[17, 51–53] three studies reported on the relationship between cam FAI (shape of the femoral head and neck) and OA[54, 56, 58] and two studies reported on a combination of bony abnormalities.[18, 55] Pincer FAI was defined in various ways. Three studies used the crossover sign[59] determined by radiograph (Figure 5).[51, 52, 55] Three studies used the CEA[42] by radiograph (Figure 1);[17, 18, 55] one study defined pincer FAI as CEA ≥40°,[17] another defined it as CEA ≥45[18] and one study did not specify a cutoff value.[55] One study used global acetabular retroversion[60] assessed by CT (Figure 6)[53] and one used coxa profunda to represent pincer impingement.[55]

Cam FAI was also variably defined. Two studies used visual inspection of cadaveric specimen[54, 56] or intra-operative findings.[54] Two studies used the presence of a post slip deformity[55] or pistol grip deformity[18] and one used the alpha angle[61] (Figure 7).[55] The remaining study used a custom-designed active shape modeling to assess the shape of the femoral head and neck.[58]

The definition of OA also varied across studies. Two studies used minimum joint space width[17, 18] and one used the mean joint space width.[53] Two studies used grading criteria, either the Kellgren-Lawrence[58] or the Tonnis grading system.[55] Two studies defined their OA cases as those hips that were awaiting total hip arthroplasty (THA).[51, 52] One study used visual inspection of the articular surfaces of cadaveric specimen[56] and the remaining study used histological analysis of tissue obtained intra-operatively from subjects with cam FAI or undergoing a THA and cadaveric specimen.[54]

**Study Outcome Measures—**Studies reported results in various ways. Four studies reported odds ratios,[17, 18, 55, 58] five studies reported the differences in the mean FAI measures or the prevalence of FAI between their defined cases and controls[51, 52, 54–56] and one study reported the mean differences in OA measures between people with and without FAI and a correlation coefficient.[53] To assess the findings across studies, articles were classified as "positive" if they demonstrated an association between the presence of FAI and OA and "negative" if they demonstrated no association. All nine studies were classified as positive. Specific to pincer FAI, Chung et al[17] reported that people with a CEA ≥ 40° have 2.3 higher risk of developing OA than people with a CEA angle of between 20° and 40°. Gosvig et al[18] similarly reported that those with CEA ≥45° have a 2.4 higher risk of developing OA. Kim et al[53] reported a positive correlation between mean joint space and global acetabular version (r = 0.46 right hip and 0.31 left hip) indicating that a decrease in acetabular version, as is the case in retroversion, is correlated with smaller mean joint space.

Specific to cam FAI, Ecker et al[55] concluded that people with a high alpha angle have a 1.09 higher risk of developing OA. According to Gosvig et al,[18] those who demonstrate a pistol grip deformity have a 2.2 higher risk of developing OA compared to those without deformities. Gregory et al[58] demonstrated that a less pronounced curve between the femoral head and neck measured at baseline was associated with a higher risk of OA at 6 year follow up. It is unclear how the measurement described by Gregory et al relates to the measurements of femoral head-neck deformities described in other studies.

**Qualitative Analysis—**While the evidence is limited, there is sufficient literature to suggest a relationship between FAI and OA. All of the studies we reviewed reported an association between FAI and OA, however eight studies were cross-sectional studies that were rated level four. [17, 18, 51–56] Similar to acetabular dysplasia, measures taken to

represent FAI, such as alpha angle or CEA, may be affected by the disease process of OA. One study was rated level two.[58] This prospective study used a customized method to assess the shape of the femoral head and neck, which limits our ability to compare to other studies. The studies reviewed suggest a relationship between FAI and OA, however the evidence was limited, due to a lack of well designed, prospective studies.

Inconsistencies in definitions and measures of bony abnormalities and OA were also found across studies linking FAI to OA, making it difficult to compare studies. For example, acetabular retroversion has various descriptions all relating to the shape and orientation of the anterior and posterior walls of the acetabulum. However, despite the differences, the literature thus far appears to support an association between FAI and OA.

## Discussion

Idiopathic or primary OA has long been a common diagnosis for those with hip joint degeneration. Research over the past decade reveals that some cases of idiopathic OA may be due to abnormal hip anatomy. Based on the articles reviewed, there is limited evidence to suggest that bony abnormalities found in AD and FAI are associated with OA. Preliminary evidence suggests that AD may be a risk factor for OA. Additional research is needed to confirm the causal relationship and the proposed mechanism underlying OA development.

While idiopathic OA was previously thought to be an inevitable consequence of aging, a more complete understanding of its underlying causes may allow preventative measures to be taken early in life. If bony abnormalities are found to increase the risk of OA, then screening programs to assess impairments related to bony morphology can be implemented and preventative strategies provided. For instance, Wyss et al[62] demonstrated that clinically-measured hip internal rotation with the hip flexed is strongly correlated with MRI-measured free space between the acetabulum and femur in this same position. Their study suggests that people with limited hip internal rotation are likely to have mechanical impingement, even if symptoms are not present. Once identified, the person with limited hip internal rotation may be educated in the impingement mechanism, and given methods to modify activities that place the hip in an at risk position, such as end-range hip flexion. However, more definitive studies investigating the presence of mechanical impingement and the development of hip pathology are needed to justify the use of screening programs.

In the studies reviewed, some subjects with bony abnormalities did not develop OA, indicating that other factors such as age,[2] trauma,[2–4] physical workload,[5] sporting activities,[6] being overweight[3, 5] and genetic factors[7–10] must all be taken into account when assessing the risk of developing OA. To find a causal relationship between any one factor and OA incidence, longitudinal studies capturing the many variables proposed to be associated with OA are necessary. Following individuals over the course of ten years or more is difficult. In addition, the progression of OA can alter the bony morphology and thus change the variables representing these bony abnormalities. Each of these problems underscores the challenge of determining the cause of OA.

An additional challenge, which prevents absolute consensus about the causal relationship between bony abnormalities and OA, is the inconsistency in measurements and definitions when studying hip morphology and OA. Among the papers reviewed, differing criteria for the diagnosis of AD, FAI and OA were used. In addition, radiographs have been shown to be insensitive in detecting cartilage damage.[50] Despite these limitations, the investigators of the studies reviewed in this paper have attempted to address the question about causes of idiopathic OA and present evidence that bony abnormalities and OA are linked.

There are limitations to our review. We reported only on bony abnormalities of the acetabulum and femoral head and neck; we did not include abnormalities of the femoral shaft, such as femoral version and femoral neck to shaft angle. We chose to focus our review to the bony abnormalities that have received recent interest in the treatment of people with joint-related pain and pre-arthritic disease. This increased attention is evident in the number of surgical procedures described to correct the bony abnormalities with the goal of reducing symptoms and preventing or delaying the onset of OA.

Our inclusion criteria were developed to assemble a homogeneous set of studies to compare; we therefore limited our review to articles that reported on the association between bony abnormalities and the presence of OA. We did not include OA progression in our search. A thorough review of articles reporting on the relationship between bony abnormalities and OA progression may provide additional information.

The criteria for our search are provided to give the reader insight into our methods, however, we recognize that some relevant articles may have been missed. In addition, the authors did not use a specific rating checklist to assess the quality of the studies. To our knowledge, there is no gold standard to evaluate observational studies.[63] We chose to use the rating system described by the Centre for Evidence-Based Medicine, because this system is familiar to many clinicians and investigators. A validated, standardized checklist to assess the quality of observational studies would improve systematic reviews of observational literature.[63]

To improve our understanding of the relationship of bony abnormalities and OA, prospective, longitudinal studies to assess joint structure prior to changes secondary to OA are needed. These studies will allow measurement of bony morphology prior to any osteoarthritic changes and confirm the temporal relationship of bony morphology and OA. In addition, future studies need to include the multiple factors thought to contribute to OA, such as age, body mass index, sex and activity level.

Reliable and valid radiological measurements are needed to define the abnormalities and the presence of OA. There is substantial variability in the measures described including the imaging used, e.g. CT versus radiograph, and the definition of abnormality. Advanced imaging techniques to assess articular cartilage integrity may improve the ability to assess the relationship of bony abnormalities and cartilage damage prior to bony changes caused by the osteoarthritic process and assist in reaching a consensus on the measurement techniques and definition of abnormality.

In conclusion, there is limited evidence suggesting that bony abnormalities found in AD and FAI are associated with OA and that AD is a possible risk factor for the development of the disease. Further research is needed to confirm the temporal relationship and specific mechanism underlying OA.

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## APPENDIX. Literature search strategy

The following search strategy was used to search PubMed. Similar search strategies were used to search Embase and CENTRAL databases.

Hip Osteparthritis

("Hip Osteoarthritis" OR "Coxarthrosis" OR "Coxarthroses" OR "hip osteoarthritis" OR "hip arthrosis" OR "hip joint arthrosis" OR "cox arthrosis" OR "hip osteoarthrosis" OR "malum coxae senilis" OR "dysplasia")

AND

Radiography/Imaging/Cadaver Inspection

"radiography"[Mesh] OR "radiography" OR "electroradiography" OR "pneumoradiography" OR "radiogram" OR "radiographic magnification" OR "radiographic method" OR "dual-energy scanned projection" OR "radioimaging" OR "radiophotography" OR "roentgen photography" OR "roentgenography" OR "roentgenoscopy" OR "X ray imaging" OR "x ray photography" OR "diagnostic X-Ray" OR "diagnostic X-Rays" OR "diagnostic X Ray" OR "diagnostic X Rays" OR "diagnostic radiology" OR "anteroposterior pelvic radiograph" OR "cross-table lateral radiograph" OR "frog-leg lateral radiograph" OR "false-profile radiograph" OR "radiographic view" OR "plain radiographic assessment" OR "plain radiographic evaluation" OR "X-Ray Computed Tomography" OR "Transmission Computed Tomography" OR "X Ray Computerized Tomography" OR "X-Ray Computer Assisted Tomography" OR "X Ray Computer Assisted Tomography" OR "Computed X Ray Tomography" OR "X-Ray Computerized Tomography" OR "X-Ray CT Scan" OR "X-Ray CT Scans" OR "CT X Ray" OR "CT X Rays" OR "Computed X-Ray Tomography" OR "Cine-CT" OR "Cine CT" OR "Electron Beam Computed Tomography" OR "Electron Beam Tomography" OR "Four Dimensional Computed Tomography" OR "4D Computed Tomography" OR "Four-Dimensional CT" OR "Four Dimensional CT" OR "Four-Dimensional CT Scan" OR "Four Dimensional CT Scan" OR "Four-Dimensional CT Scans" OR "4D CT Scan" OR "4D CT Scans" OR "Spiral Computer-Assisted Tomography" OR "Spiral Computerized Tomography" OR "Spiral CT Scan" OR "Spiral CT Scans" OR "Helical CT" OR "Helical CTs" OR "Spiral Computed Tomography" OR "Spiral CT" OR "Spiral CTs" OR "Spiral CAT Scan" OR "Spiral CAT Scans" OR "Helical Computed Tomography" OR "Ultrasonic Imaging" OR "Ultrasonic Imagings" OR "Medical Sonography" OR "Echography" OR "Echotomography" OR "Echotomographies" OR "Computer Echotomography" OR "Ultrasonic Tomography" OR "Ultrasonic Diagnoses" OR "Ultrasonic Diagnosis" OR "Diagnostic Imaging" OR "Magnetic Resonance Imaging" OR "MRI" OR "Echo-Planar Imaging" OR "autopsy" OR "necropsy" OR "obduction" OR "post mortem examination" OR "postmortem examination" OR "Autopsies"

AND

Risk Factors

("Risk Factors"[Mesh] OR "Epidemiologic Factors"[Mesh] OR "epidemiology" [Subheading] OR "Disease Susceptibility"[Mesh] OR "Risk factor" OR "Risk Factor" OR "Causalities" OR "Causality" OR "Causation" OR "Reinforcing Factors" OR "Reinforcing Factor" OR "Causations" OR "Enabling Factors" OR "Enabling Factor" OR "Predisposing Factors" OR "Predisposing Factor")

NOT

(("Animals"[Mesh]) NOT ("Animals"[Mesh] AND "Humans"[Mesh]))

Limits: English, All Adult: 19+ years, Adolescent: 13–18 years

Figure 1a

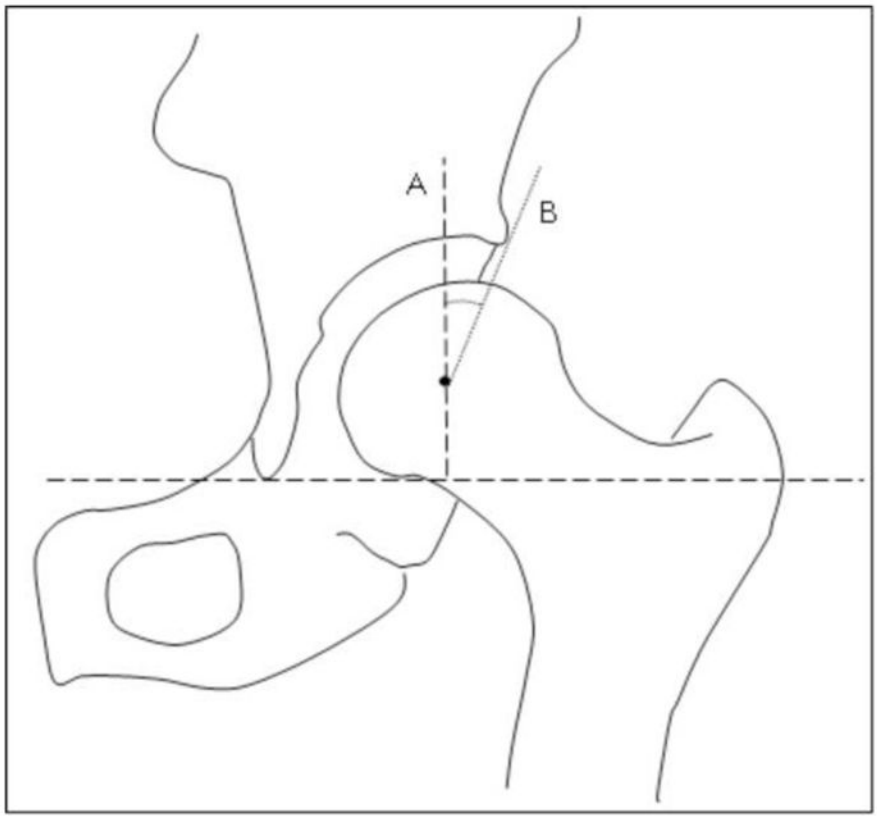

Figure 1b

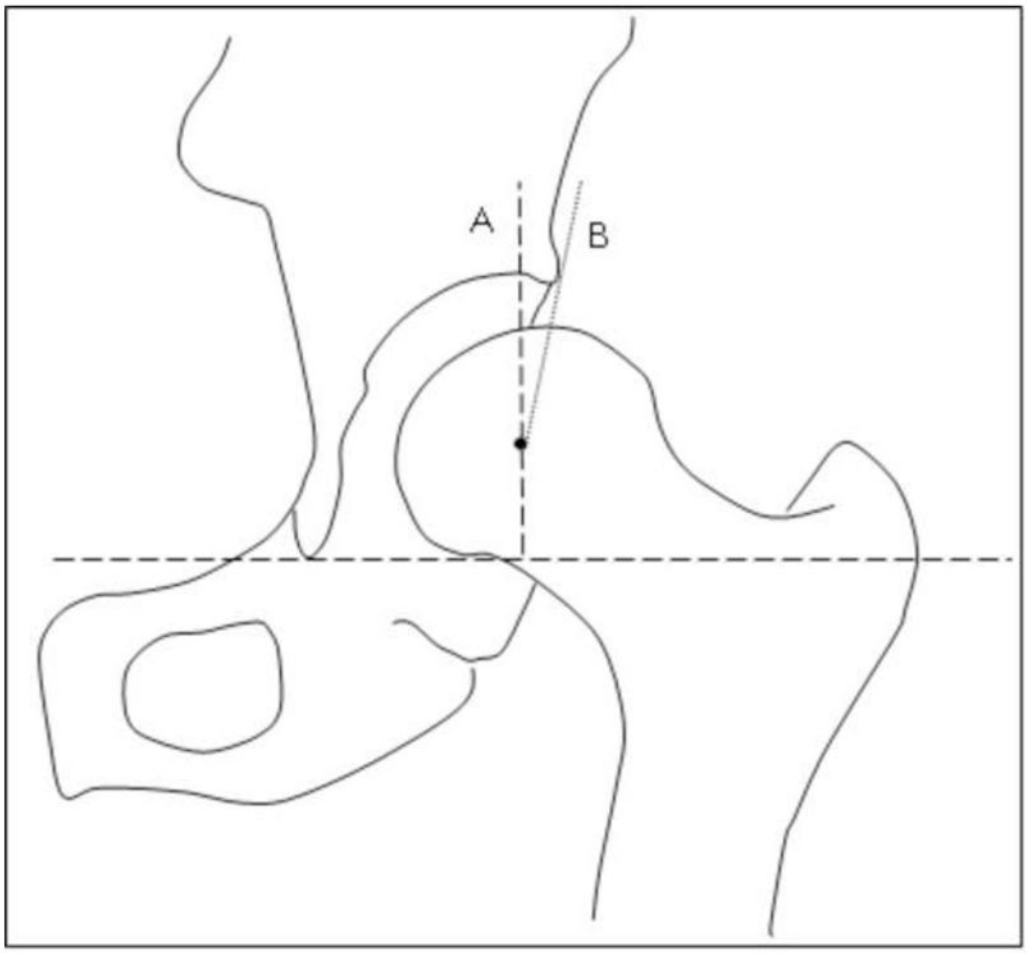


**Figure 1.**

**Figure 1a. Acetabulum without dysplasia.** One measurement to represent acetabular dysplasia is **lateral center edge angle of Wiberg (CEA).** The CEA is formed by (A) line perpendicular to the transverse axis of the pelvis drawn from femoral head center and (B) line from the femoral head center to the superolateral point of acetabulum. A range of CEA values are reported to define acetabular dysplasia including ≥ 20°, ≥ 25°≥ 30°. A range of CEA values is also reported to define pincer impingement including ≥ 40°, ≥ 45°.

**Figure 1b. Acetabular dysplasia** represented by a small lateral center edge angle of Wiberg (CEA).

Figure 2a

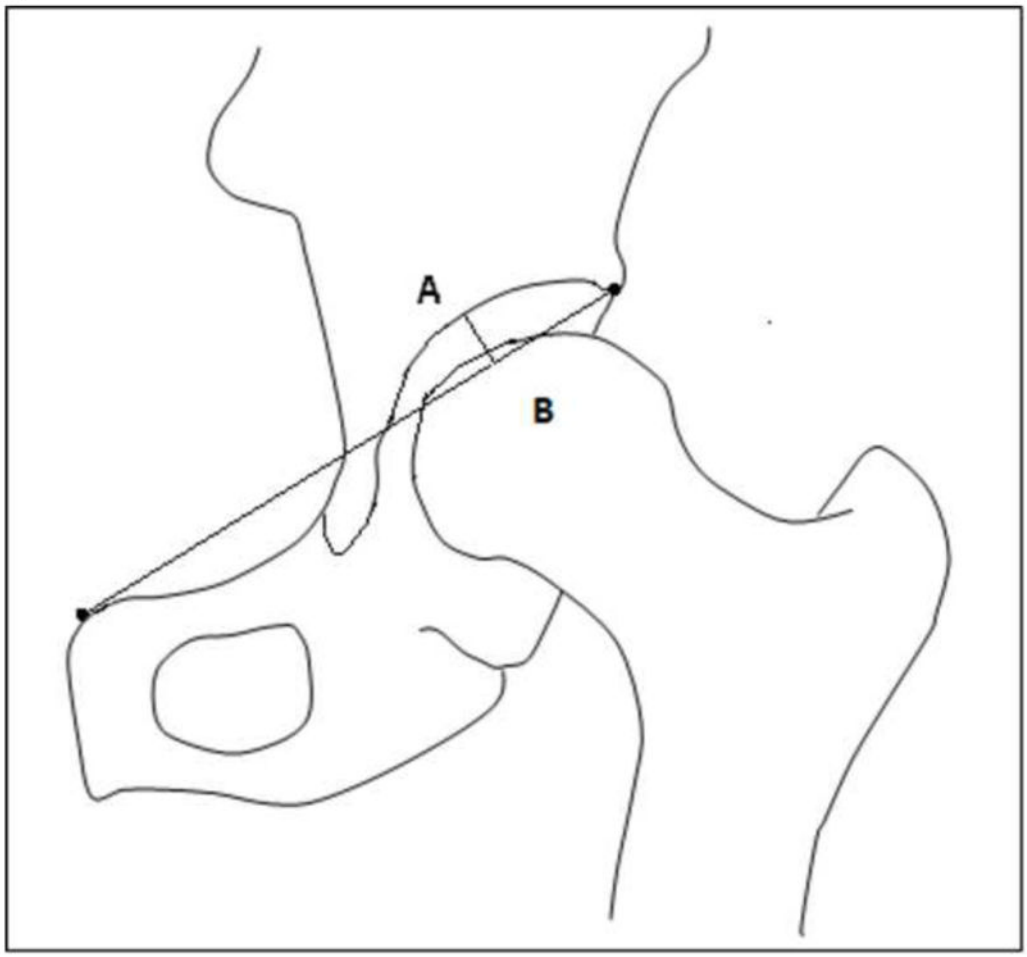

Figure 2b

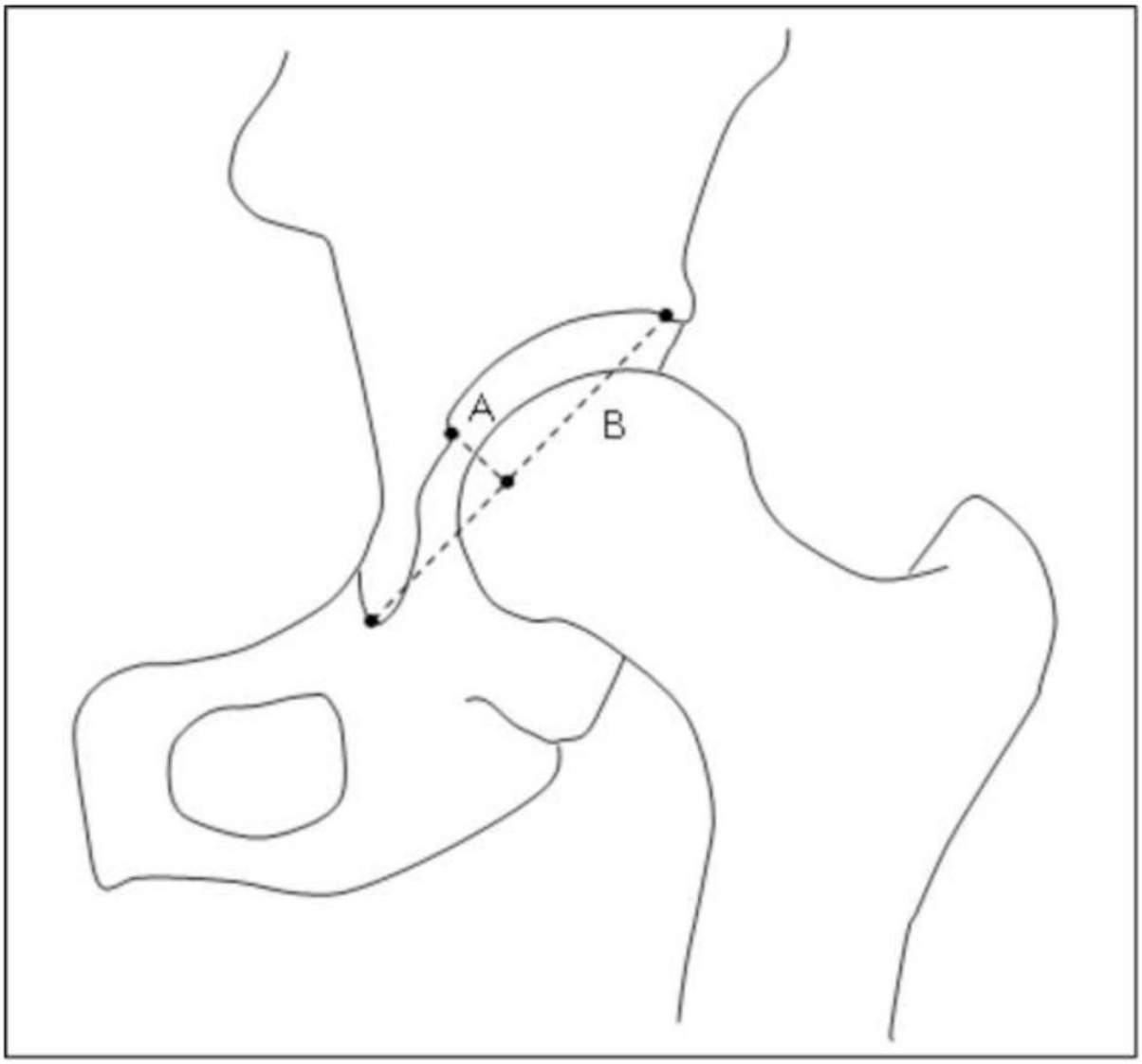


**Figure 2.**

**Figure 2a. Acetabular depth index (ADI).** The ADI is the length of line A. Line (B) is drawn from the edge of the articular surface of the acetabulum and the upper corner of the ipsilateral symphysis pubis. Line A is the distance from the deepest part of the acetabular margin to line B. Acetabular dysplasia is often defined an ADI as < 9 mm.

**Figure 2b. Acetabular depth ratio (ADR).** The ADR is calculated by dividing (A) perpendicular line from line B to the medial corner of the acetabular weight-bearing surface by (B) acetabular width and multipliing by 1000 (A/B × 1000). Acetabular dysplasia is often defined an ADR≤ 250.

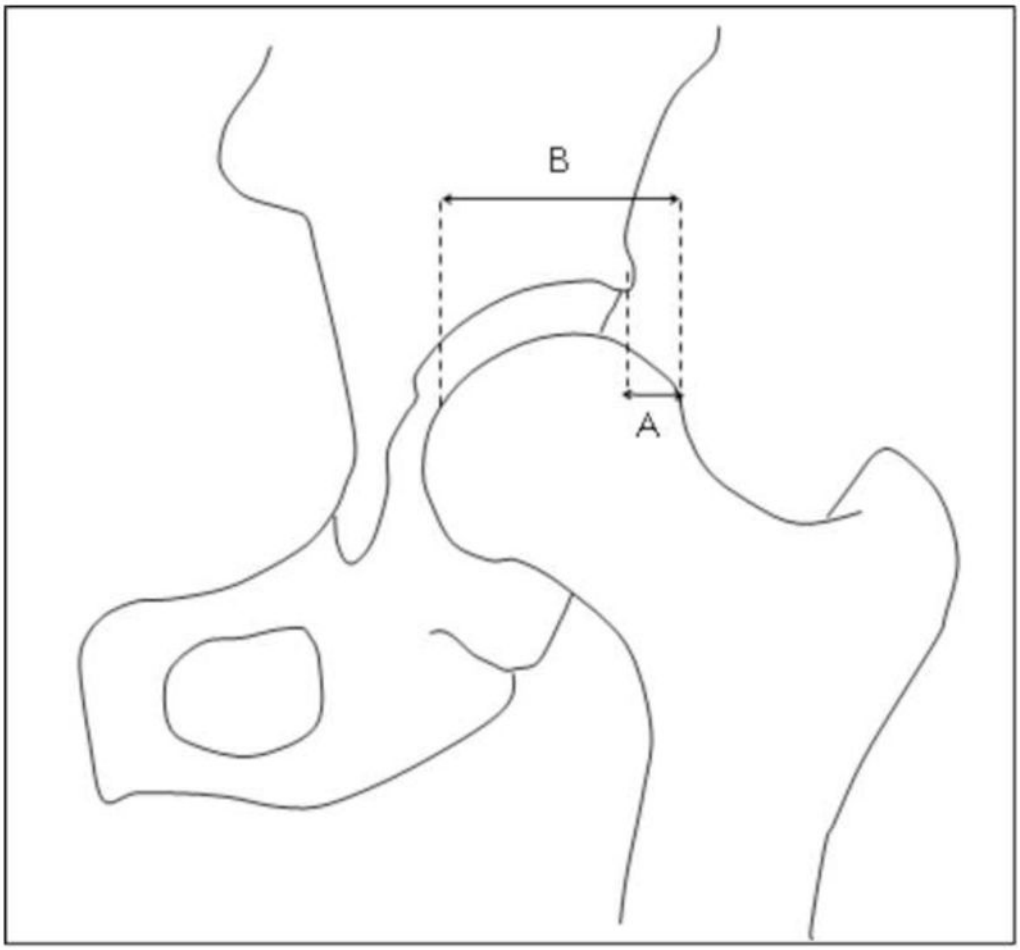


**Figure 3. Femoral head extrusion index (FHEI)**
Percentage calculated by dividing (A) the amount of the femoral head lateral to the acetabulum by (B) the total width of the head and multiplying by 100. A/B × 100. Acetabular dysplasia is often defined as FHEI ≥25%

Figure 4a

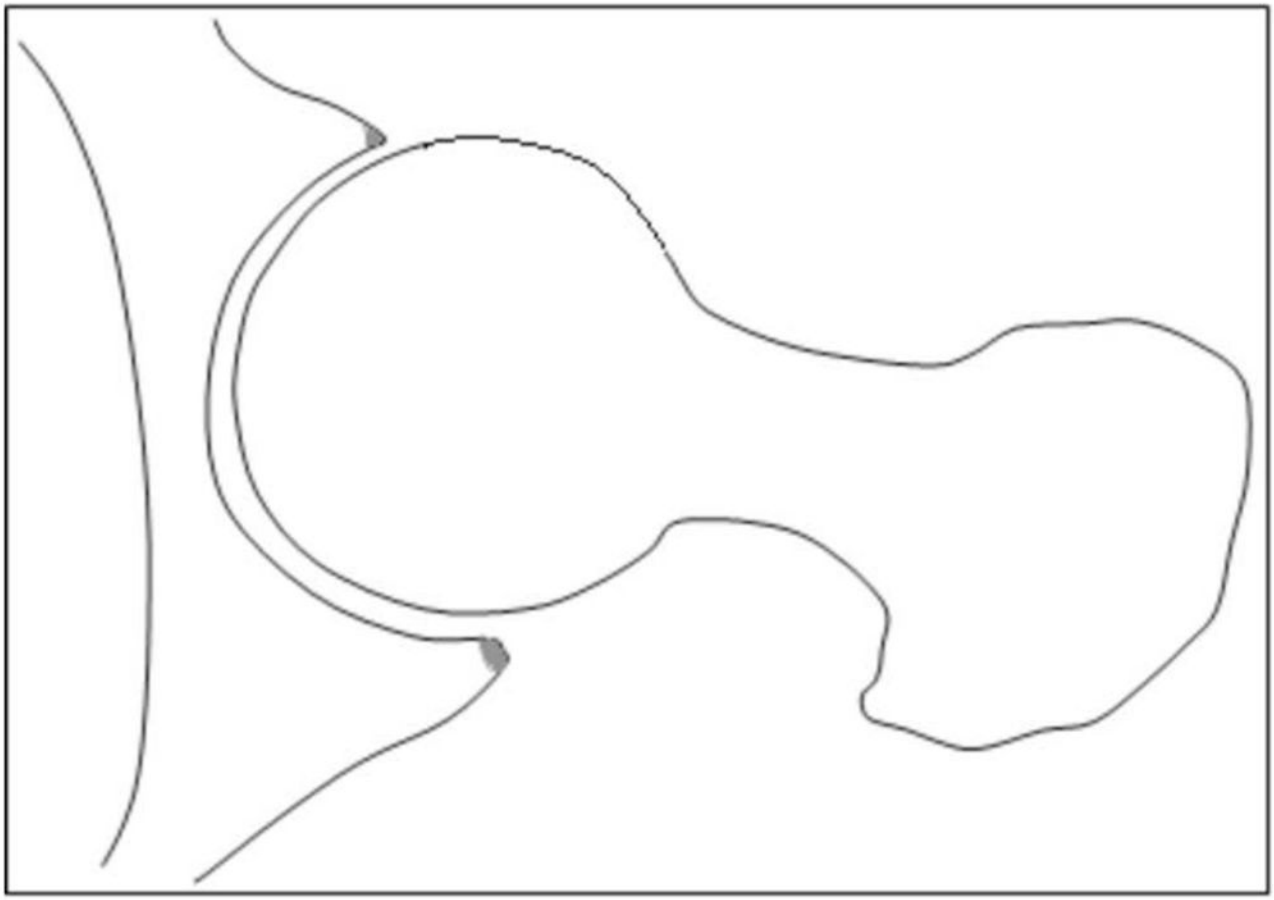

**Figure 4b**

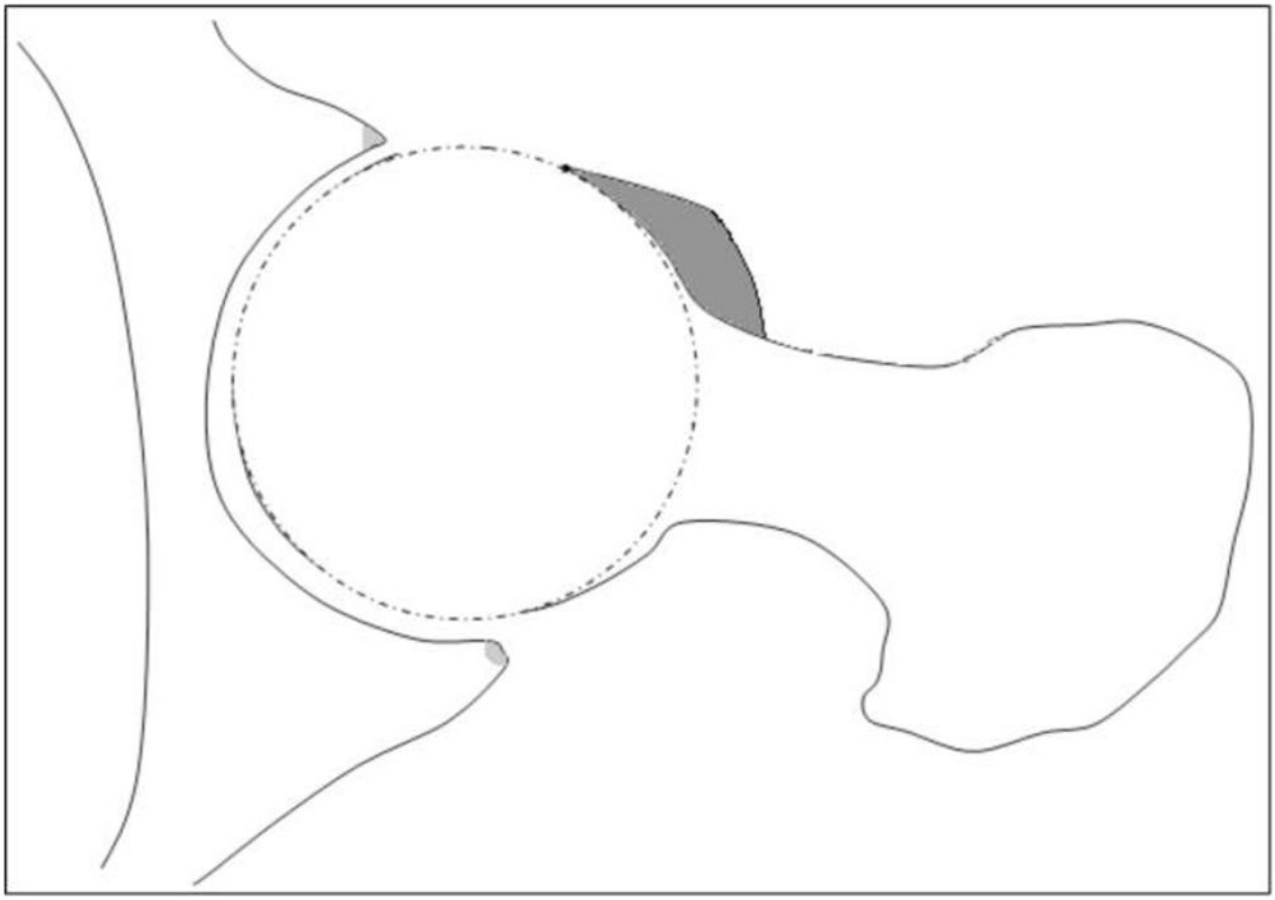

Figure 4c

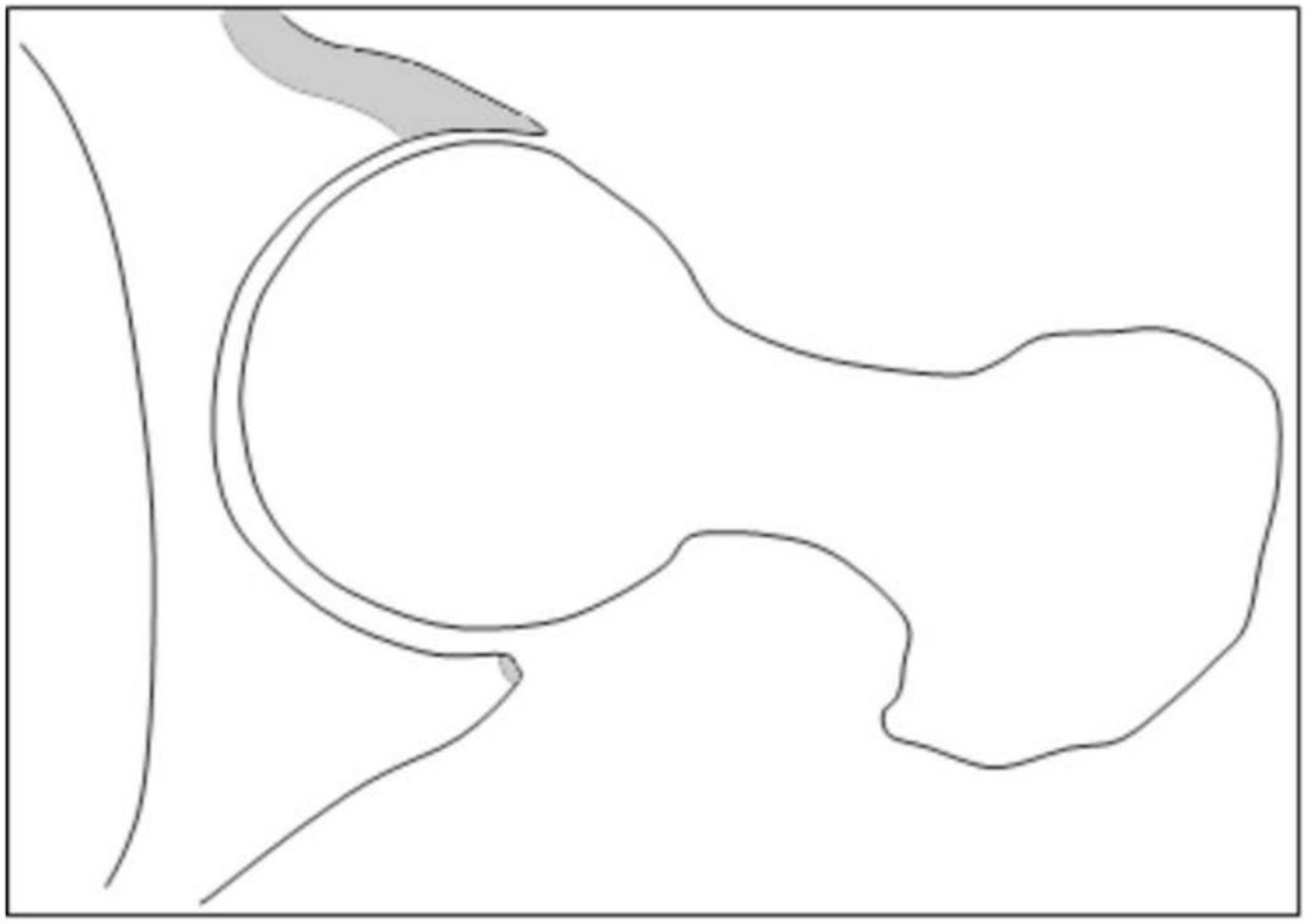

**Figure 4.**

Figure 4a. Hip joint without femoroacetabular impingement

Figure 4b. Cam femoroacetabular impingement. Shaded area denotes the loss of concavity of the head-neck junction in cam impingement.

**Figure 4c. Pincer femoroacetabular impingement.** Shaded area denotes the acetabular overcoverage of the femur.

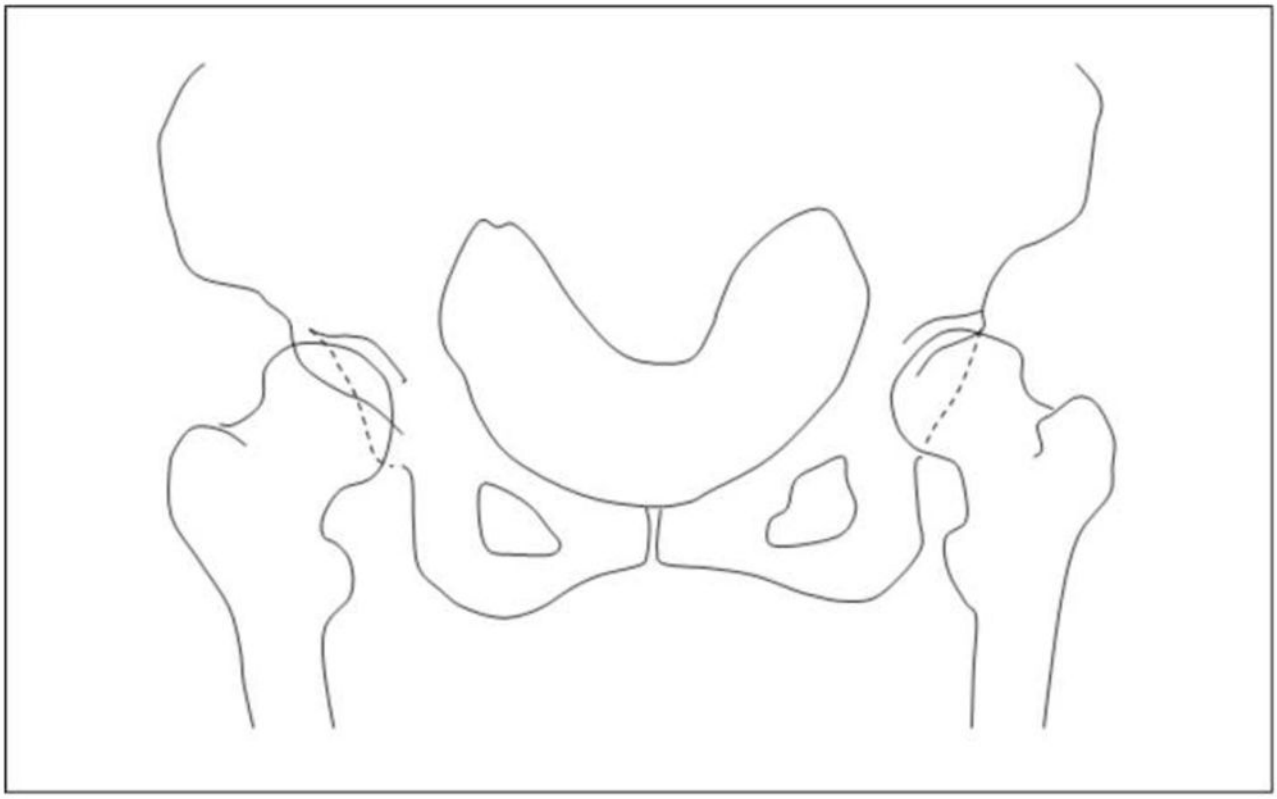

**Figure 5. Crossover sign**

The broken line represents the posterior acetabular wall and the solid line represents the anterior acetabular wall. Crossover sign noted on the right hip where the anterior wall crosses the line of the posterior wall before reaching the lateral edge of the sourcil.

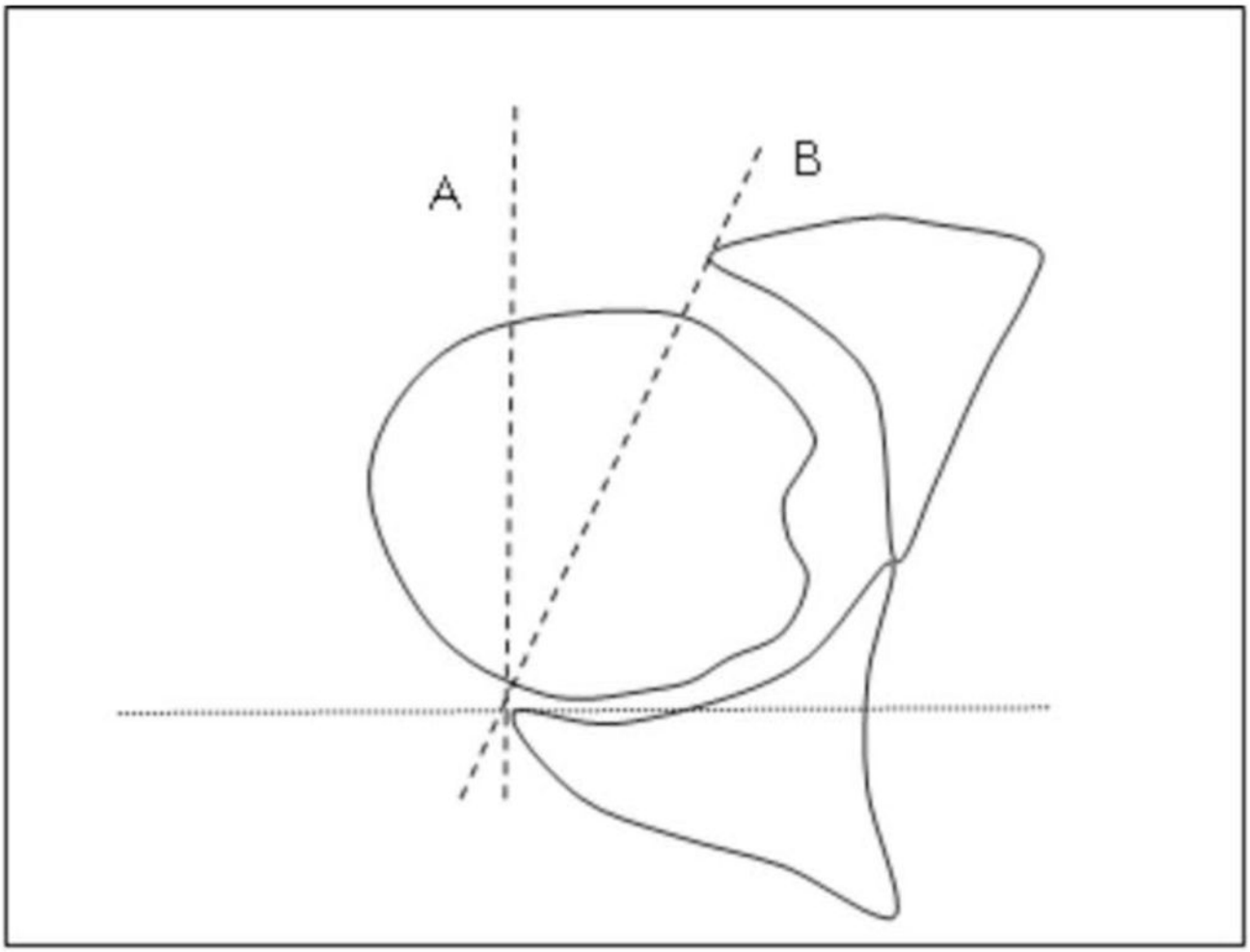


**Figure 6. Global acetabular retroversion**

Axial image of the pelvis. Angle formed by (A) line perpendicular to the coronal axis of the pelvis (B) line connecting the anterior and posterior acetabular rims. Pincer impingement was defined as angle ≤14°.

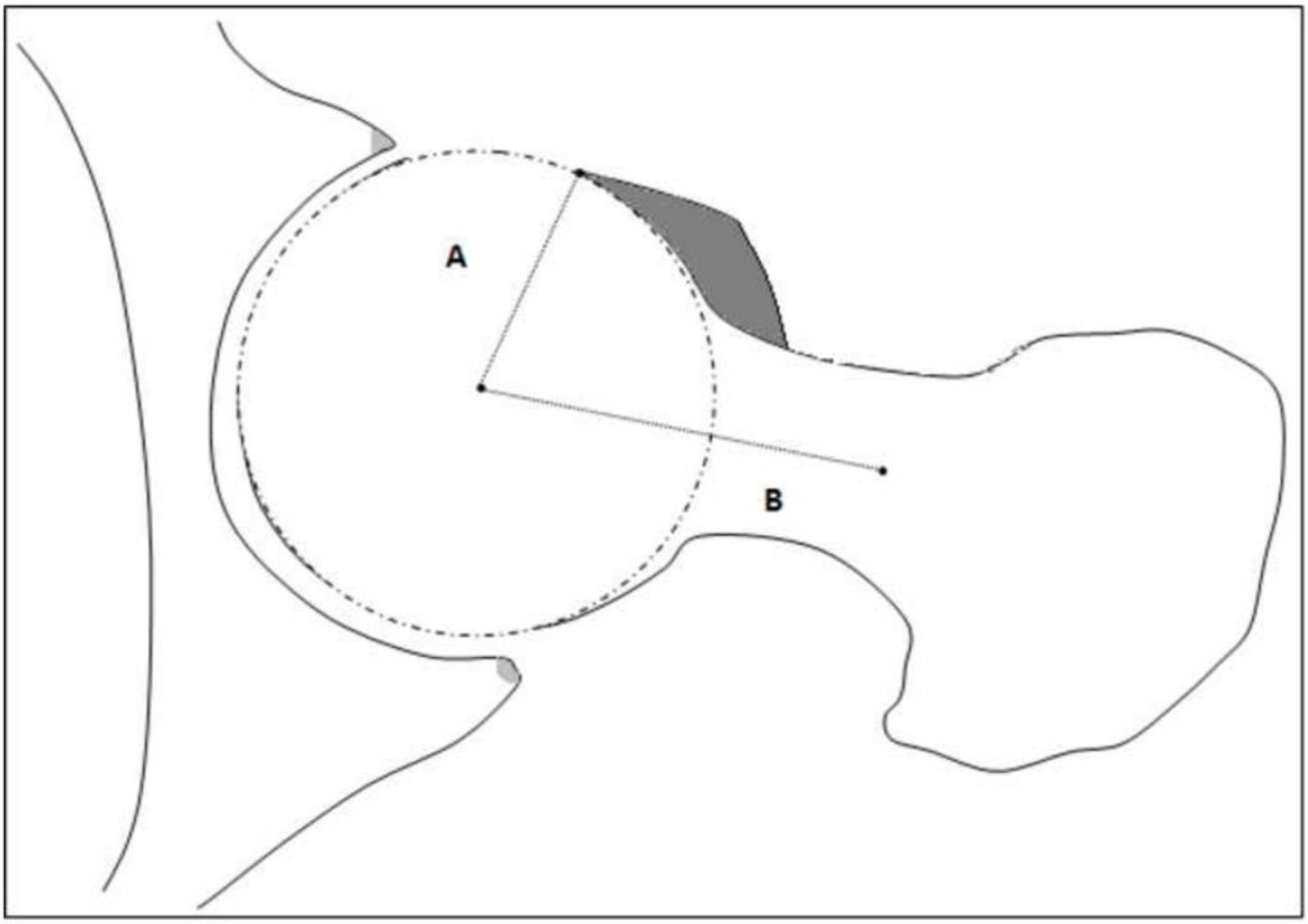


**Figure 7. Alpha angle**

Angle is formed by (A) line from the femoral head center to the point on the anterolateral head-neck junction where the radius of the femoral neck first becomes greater than the radius of the femoral head and (B) line drawn from the femoral head center through the center of the femoral neck. Cam impingement was defined as an alpha angle >50°.

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**Table 1**

Details of studies reporting the association between acetabular dysplasia and hip osteoarthritis.

| Author | Study Design | Participants | Definition Acetabular dysplasia (AD) | Definition Hip Osteoarthritis (OA) | Study Results | CEBM Level evidence | Is AD associated with OA |
|---|---|---|---|---|---|---|---|
| Lane[27] | Prospective nested case control with 8 year follow up | 176 participants (100% F)<br>58 cases developed OA<br>118 controls did not develop OA<br>Mean age 70.3 (SD 4.7)<br>United States | CEA <30°<br>ADI <9mm | Customized radiographic method using JSN, osteophytes, sclerosis and neck deformity | Association between AD and incident OA<br>CEA<30°:<br>aOR = 3.3 (1.1–10.1)<br>presence of dysplasia:<br>aOR = 2.8 (1.0–7.9) | II | Yes |
| Murphy[28] | Prospective nested case control with follow up (time not specified) | 117 patients with THA due to AD, contralateral hip assessed<br>74 cases (K/L ≥3)<br>43 control (K/L≤2)<br>Age ≥65 years<br>Switzerland | CEA<br>FHEI<br>Tonnis angle | K/L | CEA and Tonnis angle smaller in cases compared to controls<br>FHEI larger in cases compared to controls | II | Yes |
| Reijman[25] | Prospective cohort with 6.6 year follow up | 835 participants (57% F)<br>Age ≥55 years The Netherlands | CEA<25°<br>CEA<30°<br>ADI<9mm | JSN ≥1.0mm<br>K/L ≥2 | ***Those with AD had increased risk of JSN***<br>CEA<25°:<br>OR 4.3 (2.2–8.7)<br>CEA<30°:<br>OR 2.8 (1.9–4.2)<br>AD <9 mm:<br>OR 2.8 (1.8–4.5)<br>***Those with AD had increased risk of K/L ≥2***<br>CEA<25°:<br>OR 2.4 (1.2–4.7)<br>CEA<30°:<br>OR 1.7 (1.2–2.5)<br>AD <9 mm:<br>OR 2.3 (1.5–3.5) | II | Yes |
| Cooperman[24] | Prospective case cohort with 22 year follow up | 20 patients (75% F); 32 hips with CEA<20°<br>Age range 27–57 years<br>Sweden | CEA<20°<br>ADR<250<br>FHEI<br>Sharp's angle | Customized radiographic method using JSN, osteophytes, sclerosis and femoral or acetabular cysts | 30 of 32 hips developed OA. OA developed in an equal proportion of patients in their 6th and 7th decades as in the 4th and 5th, therefore the authors concluded that no AD measure correlated with onset rate of OA. | III | No |
| Jacobsen[26] | Prospective Case-control with 10 year follow up | 217 participants<br>81 cases(67% F) with hip CEA<20°<br>136 controls (51%F) with CEA≥25°<br>Mean age 48–53 years<br>Denmark | ADR ≤250<br>CEA≤20°<br>FHEI ≥20% | JSN<br>Minimum JSW ≤2mm | No difference in JSN between cases and controls. Trend of greater JSN in cases.<br>Six subjects developed OA (5 controls and 1 case) | III | No |

| Author | Study Design | Participants | Definition Acetabular dysplasia (AD) | Definition Hip Osteoarthritis (OA) | Study Results | CEBM Level evidence | Is AD associated with OA |
|---|---|---|---|---|---|---|---|
| Birrell[29] | Cross-sectional | 195(68%F) patients with new onset of hip pain<br>Age ≥40 years<br>United Kingdom | CEA<25°<br>AD<9mm | K/L (Croft modified)<br>Minimum JSW ≤1.5mm or >1.5≤2.5mm | No relationship between the prevalence of AD and presence of OA.<br>Higher prevalence of AD in those with most severe OA. | IV | No |
| Chung[17] | Cross-sectional | 674(57%F)<br>Age ≥ 65 years<br>Korean | CEA<20° | Minimum JSW ≤2.0mm or ≤2.5 mm | AD risk factor of OA, when OA defined as JSW ≤2.5mm<br>CEA<20°: aOR 10.2 (1.8–56.7) | IV | Yes |
| Croft[30] | Cross-sectional | *1516 patients getting urograms (100% M)<br>Age range 60–75 yrs<br>United Kingdom | CEA<br>ADI | Minimum JSW ≤ 1.5 mm | JSW had a weak negative correlation with ADI (r =−0.15) and CEA (r = −0.37) | IV | No |
| Inoue[31] | Cross-sectional | 1183 getting urograms<br>401 (29% F) France<br>782 (47% F) Japan<br>Age range 20–79 years | CEA <25°<br>ADI | K/L ≥3 in at least 1 hip<br>Minimum JSW | JSW had a weak negative correlation with ADI (r = −0.02; −0.13 range) and CEA (r = −0.18; −0.39 range) | IV | No |
| Goker[39] | Cross-sectional | 92 patients (29% F) getting urograms<br>Age range 55–97 years<br>Turkey | CEA <25°<br>ADI <9mm | K/L ≥3<br>K/L = 2 | No subjects had K/L ≥3<br>No difference in mean CEA or ADI when K/L ≤1 compared to K/L = 2. | IV | No |
| Gosvig[18] | Cross-sectional | 3620 (63%F)<br>Copenhagen OA Substudy of Copenhagen Heart Study<br>Age 21–90 years<br>Denmark | CEA≤20° | Minimum JSW ≤2.0mm | CEA≤20° was not a significant risk factor for OA<br>RR = 1.6 (0.98–2.5)<br>RR = “Corrected Risk Ratio ” due to >10% prevalence of OA | IV | No |
| Jacobsen[33] | Cross-sectional | 3568 participants (63% F)<br>Copenhagen OA Substudy of Copenhagen Heart Study<br>Age range 20–90 years<br>Denmark | ADR ≤250<br>CEA≤20°<br>FHEI ≥25% | Minimum JSW ≤2.0mm | OA associated with CEA ≤20 in Females<br>Left: OR=1.1 (1.0–1.1)<br>Right: OR=1.1 (1.0–1.6)<br>Males<br>Left: OR=1.6 (1.2–2.1)<br>Right: OR=1.1 (1.0–1.2 | IV | yes |
| Jacobsen[32] | Cross-sectional | 3859 participants (63% F)<br>Copenhagen OA Substudy of Copenhagen Heart Study<br>Age range 20–90 years<br>Denmark | ADR ≤250<br>CEA≤20<br>FHEI≥25%<br>Sharp’s angle≤45° | Croft (K\L modified) ≥3<br>K/L ≥2<br>Minimum JSW ≤2.0mm | Measures of OA associated with measures of AD<br>CEA: OR = 2.0–3.4<br>FHEI: OR = 3.1–6.2<br>ADR: OR = 1.9–2.8 | IV | Yes |
| Jessel[38] | Cross-sectional | 74 patients(84% F); 96 hips with CEA<16°<br>Mean age 28–32 years<br>United States | CEA<br>Tonnis angle<br>Anterior center edge angle | <390 msec<br>dGEMERIC = early OA | Average dGEMERIC index in the sample was lower than reported normal range.<br>Severity of AD measured by CEA and anterior center edge | IV | Yes |

| Author | Study Design | Participants | Definition Acetabular dysplasia (AD) | Definition Hip Osteoarthritis (OA) | Study Results | CEBM Level evidence | Is AD associated with OA |
|---|---|---|---|---|---|---|---|
| | | | | | angle is associated with early OA | | |
| Johnsen[34] | Cross-sectional | 315 participants (52% F)<br>Age range 20–65 years<br>Norway (Sami population) | CEA<br><20 = AD<br>20–24 = slight AD<br>>24 = normal | OA if K/L ≥ 2 in at least 1 hip | No association between OA and CEA OR = 0.968 (.0916–1.024 | IV | No |
| Lau[35] | Cross-sectional | 999 patients (100 % M) getting urograms<br>Age range 60–75 years<br>Hong Kong | CEA<25°<br>ADI<9 mm | Minimum JSW ≤1.5 mm | No difference in the prevalence of AD between hips with JSW ≤1.5 and hips with JSW ≥4.0 | IV | No |
| Smith[36] | Cross-sectional | *203 patients (100% F) getting urograms; 393 hips<br>Age range 60–75 years<br>United Kingdom | CEA<25°<br>ADI< 9 mm | Croft (K\L modified)<br>Minimum JSW ≤1.5 mm | JSW had a weak negative correlation with<br>ADI: r = −0.11<br>CEA: r = −0.25<br>K/L grade had a weak positive correlation with CEA: r = 0.11 and no correlation with ADI | IV | No |
| Yoshimura[37] | Cross-sectional | 198 participants (50% F)<br>Age range 60–79 years<br>Japan | CEA<25°<br>ADI< 9 mm | Croft (K\L modified) ≥3<br>Minimum JSW | JSW had a weak negative correlation with CEA (r =−0.26; −0.39 range)<br>No correlation to ADI<br>No differences in presence of AD between K\L ≥3 and K\L ≤2 | IV | No |
| Lane[40] | Cross-sectional | 253 participants (100% F)<br>165 cases with OA<br>88 controls without OA<br>Age ≥ 65 years<br>United States | CEA <25°<br>ADI <9 mm<br>Presence of either CEA <25° or ADI <9mm | Customized radiographic method using JSN, osteophytes, sclerosis and neck deformity | Hips with OA small, but not significant prevalence of<br>CEA<25°:<br>OR = 1.43 (0.46–4.46)<br>ADI< 9 mm:<br>OR = 1.47 (0.78–2.77)<br>AD (CEA or AD):<br>OR = 1.33 (0.74–2.4) | IV | No |
| McWilliams[41] | Cross-sectional | 1674 participants<br>Nottingham Genetics OA and Lifestyle study<br>Pts with 566(47.9%F) cases = unaffected hip in those with unilateral OA<br>1108(46.3%F) controls<br>Age >45<br>United Kingdom | CEA<20°<br>ADI<9 mm<br>In addition calculated study-specific cutoff values calculated from control group (1.96 × SD) | Minimum JSW ≤2.5mm | Significantly higher risk of OA in people with AD<br>CEA<20°: aOR = 10.05 (2.89–35.01)<br>CEA study specific:<br>aOR = 8.06 (4.87–13.35<br>ADI< 9 mm: aOR = 3.61 (2.47–5.27)<br>ADI study-specific:<br>aOR = 2.53 (1.28–5.00) | IV | Yes |

*Study by Yoshimura also reports on data related to British cohort from same population as Croft and Smith, however the authors of the study report the correlation between AD and OA in Japanese subjects only.

CEBM = Centre for Evidence Based Medicine

SD = standard deviation

F = females

M = males

CEA = Lateral center edge angle of Wiberg

ADI = Acetabular depth index

JSN = Joint space narrowing

aOR = Adjusted odd ratio

THA = Total hip arthroplasty

K/L = Kellgren-Lawrence grade

FHEI = Femoral head extrusion index

JSW = Joint Space Width

**Table 2**

Details of studies reporting the association between femoroacetabular impingement and hip osteoarthritis.

| Author | Study Design | Participants | Definition Femoroacetabular impingement (FAI) | Definition Hip Osteoarthritis (OA) | Study results | CEBM Level evidence | Is FAI associated with OA |
|---|---|---|---|---|---|---|---|
| Gregory[58] | Prospective case control 6 year follow up | 110 participants<br>55 cases (75%F)<br>Developed OA (K/L ≥3)<br>55 controls (76%F) (K/L=0)<br>Age range 55–80 years<br>Age and sex matched<br>Netherlands | ***Cam FAI***<br>Active shape model (ASM) to quantify femoral head shape<br>Modes of variation(1–10) | K\L ≥3 = OA | *Cases had a significantly lower baseline Mode 6 score compared to controls.<br>aORs = 1.62 (1.08–2.45)<br>Those getting THA has a significantly lower baseline Mode 6 score compared to those with OA without THA.<br>aORs = 2.35 (1.15–4.82)<br>Low Mode 6 = less pronounced curve from femoral head to neck | II | Yes |
| Chung[17] | Cross-sectional | 674(57%F)<br>Age ≥ 65 years<br>Korean | ***Pincer FAI***<br>CEA≥40 | Minimum JSW ≤2.0mm or ≤2.5 mm | Pincer FAI risk factor of OA, when OA defined as JSW ≤2.5mm<br>aOR = 2.3 (1.5–3.4) | IV | Yes |
| Gosvig[18] | Cross-sectional | 3620 (63%F)<br>Copenhagen OA Substudy of Copenhagen Heart Study<br>Age range 21–90 years<br>Denmark | ***Pincer FAI***<br>CEA≥45°<br>***Cam FAI***<br>*Pistol grip deformity using triangular index≥0* | Minimum JSW ≤2.0mm | Pincer FAI was a significant risk factor for OA<br>RR = 2.4 (2.0–2.9)<br>Cam FAI was a significant risk factor for OA<br>RR = 2.2 (1.7–2.8)<br>RR = “Corrected Risk Ratio” due to >10% prevalence of OA | IV | No |
| Kim[53] | Cross-sectional | 117 patients (55% F)getting a CT virtual colonoscopy<br>Age range 20–64 years<br>United Kingdom | ***Pincer FAI***<br>Global acetabular version retroversion ≤14° | Mean JSW | Mean JSW in hips with acetabular retroversion was narrower (1.6 mm)compared to those with normal acetabular retroversion (2.35mm)<br>Correlation between acetabular retroversion and OA<br>Right hip:<br>r = 0.46 (0.23–0.64)<br>Left hip:<br>r = 0.31 (0.05–0.53) | IV | Yes |
| Ecker[55] | Cross-sectional | 119 participants; contralateral hips to THA<br>94 cases (27%F)<br>Tonnis Gr 2 | ***Pincer FAI***<br>CEA – upper limited not provided Coxa profunda | Tonnis Gr 2 | FAI was associated with presence of OA.<br>***Pincer FAI***<br>High CEA:<br>OR = 1.14 (1.02–1.27) | IV | Yes |

| Author | Study Design | Participants | Definition Femoroacetabular impingement (FAI) | Definition Hip Osteoarthritis (OA) | Study results | CEBM Level evidence | Is FAI associated with OA |
|---|---|---|---|---|---|---|---|
| | | Age range 37–85 years<br>25 controls (48% F)<br>Tonnis Gr ≤1<br>Age 60–82 years<br>North America | Presence of crossover sign for Acetabular Retroversion<br>***Cam FAI***<br>Presence of pistol grip deformity Alpha angle>50° | | ***Cam FAI***<br>High alpha angle:<br>OR = 1.09 (1.03–1.15) | | |
| Ezoe[51] | Cross-sectional | 250 participants;<br>342 hips<br>66 cases (86% F);<br>70 hips<br>Underwent THA<br>Age 54–79 years<br>56 controls (61% F);<br>112 hips<br>Tonnis gr 1<br>Age 15–54 years<br>Remaining subjects had other hip disorders<br>Japan | ***Pincer FAI***<br>Presence of crossover sign for Acetabular Retroversion | Underwent THA | Significantly higher prevalence of acetabular retroversion in cases (20%) compared to controls (6%). | IV | Yes |
| Giori[52] | Cross-sectional | 230 participants<br>131 cases (65% F)<br>Underwent THA<br>Mean age 72 years<br>99 controls (61% F)<br>Patients with imaging for nonorthopedic reasons<br>Mean age 59 years | **Pincer FAI**<br>Presence of crossover sign for Acetabular Retroversion | Underwent THA | Significantly higher prevalence of acetabular retroversion in cases (20%) compared to controls (5%). | IV | Yes |
| Goodman[56] | Cross-sectional Cadaveric | 2665 skeletons (16% F)<br>Age range 1–90 years at time of death<br>United States<br>215 cases; 306 hips with post-slip deformity<br>215 controls; 306 hips without post-slip deformity matched by age, sex and race | ***Cam FAI***<br>Post-slip morphology determined by visual inspection | Grading scale based on visual inspection.<br>Criteria included presence of osteophytes, erosions, flattening or exposure of trabelcular bone | Severe OA is associated with post-slip morphology.<br>OA was more prevalent in cases (38%) than in controls (26%).<br>In skeletons with unilateral post-slip, severe OA was more prevalent in the affected hip (37%) than in the contralateral hip (22%) | IV | *yes* |
| Wagner[54] | Cross-sectional<br>Intraoperative and Cadaveric tissue | 42 samples<br>22 samples from patients undergoing surgical resection of cam FAI<br>Age range 19–45 years<br>6 samples from matched controls (deceased) | ***Cam FAI***<br>Clinical presentation of FAI and nonspherical head by AP radiograph | Mankin criteria to rank cartilage morphology by histological staining<br>Immunochemistry staining<br>*In situ* hybridization to assess gene expression and distribution of collagen II/I | Hyaline cartilage of FAI samples was significantly different from controls and from THA, however less distinct than THA Mean Mankin criteria<br>Controls: 1.33 (0–2)<br>FAI: 5.71 (2–12)<br>THA: 8.63 (3–14) | IV | Yes |

| Author | Study Design | Participants | Definition Femoroacetabular impingement (FAI) | Definition Hip Osteoarthritis (OA) | Study results | CEBM Level evidence | Is FAI associated with OA |
|---|---|---|---|---|---|---|---|
| | | Age range 19–37 years<br>14 samples from patients undergoing THA<br>Age range 35–86 years<br>Switzerland | | | No statistical difference in immunochemistry or *in situ* hybridization, but may show a trend | | |

*Additional data reported in Gregory et al related to mode change over time was not reviewed for this paper

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