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CORR Insights

CORR Insights®: The 2015 Frank Stinchfield Award: Radiographic Abnormalities Common in Senior Athletes With Well-functioning Hips but Not Associated With Osteoarthritis

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Where Are We Now?

The current understanding of the etiology of hip osteoarthritis (OA) is that certain variations in hip anatomy predispose the joint to the development of OA [7]. Natural history studies have shown that hips with dysplasia and femoroacetabular impingement (FAI) are more likely to develop arthritis [1, 17]. When analyzing the etiology of idiopathic OA, up to 79% of hips can be attributed to underlying hip deformity, with dysplasia implicated in 39% to 49% and CAM deformity in 40% to 63% [4, 8, 16].

During the past 30 years, the innovation and adoption of surgical techniques such as periacetabular osteotomy (PAO) [6] and hip arthroscopy have allowed surgeons to alter the mechanical environment of the hip at risk for OA with the goal of decreasing the likelihood that arthritis will develop. There is some evidence that these interventions can delay or even prevent OA [12, 15]. Consequently, the biggest challenge for surgeons is to identify the hip at mechanical risk for degeneration and to accurately correct the anatomical cause of the mechanical dysfunction.

However, dysplastic and FAI anatomy are spectra of deformity that are modulated by many nonanatomical factors such as genetics, activity level, and age, making it difficult to predict which hips with dysplasia or FAI will eventually become symptomatic and develop OA. Previous estimates of radiographic prevalence in the general population range from 3% to 13% for dysplasia [9, 10] and 15% to 25% for FAI [8, 14] Although prior studies demonstrated that a large portion of arthritic hips had underlying dysplasia or FAI, it remains unknown how many hips with radiographic signs of dysplasia or FAI stay asymptomatic and continue to function into old age.

In the current study, Anderson and colleagues reported the prevalence of hip dysplasia and FAI morphology in a group of highly functional senior athletes. Although other studies have

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looked at the prevalence of hip deformities in the general population, this study uniquely looks at hips that have not only survived into the sixth decade of life, but also remained healthy enough to allow participation in sports.

A total of 1081 hips in 547 individuals of an average age of 67 years were included in this cohort. Researchers found that 83% (898 of 1081) of hips had radiographic abnormalities consistent with FAI; of those hips, 67% had CAM morphology. A total of 10% (103 of 1081) of hips had radiographic evidence of dysplasia.

Where Do We Need To Go?

A couple of factors may explain why the prevalence of FAI was higher in this population of senior athletes than in previous studies. Tönnis Grades 2 and 3 OA were present in 17% (186 of 1081) of hips, which is higher than the average 8% incidence estimated in the general population [5]. Of those 186 arthritic hips, 93% had evidence of FAI. Radiographic measurements of FAI can be confounded by the presence of reactive femoral head osteophytes masking as CAM deformities and ossified labrums appearing as acetabular overcoverage. Given that a substantial portion of the study group had OA, the radiographic prevalence of FAI may have been overestimated.

Additionally, bilateral hips from 534 individuals were used in the study. More than 75% of patients with FAI can have bilateral deformity [2, 4] and 61% to 84% of patients with acetabular dysplasia have signs of dysplasia in the contralateral hip [11, 13]. Inclusion of bilateral hips in any population study may therefore overestimate the prevalence of the deformity of interest. Future population studies may consider assessing the hip anatomy of unique individuals instead of individual hips.

After controlling for age and sex, neither radiographic signs of FAI nor dysplasia were predictive of OA in this select population of senior athletes with asymptomatic hips. However, the study group only included hips that had successfully survived past the age of 50 and excluded the senior population of hips that had failed before this age, which may have had more substantial deformity. More severe FAI and dysplastic deformity is associated with increased intraarticular damage and higher risk of developing OA [1, 3, 17]. Therefore, we cannot conclude that dysplasia and FAI are not associated with the development of OA overall; only in this particular active senior population.

Perhaps the most interesting question that is raised by this study is: What are the other nonanatomic protective factors that have allowed these senior hips to flourish despite the presence of radiographic risk factors, and indeed in 17% of participants, the occurrence of actual osteoarthritis?

How Do We Get There?

Given that hip mechanics are a complex interplay between acetabular and femoral anatomy modulated by activity, a thorough anatomic evaluation of the hip is necessary before attributing mechanical dysfunction to any particular anatomic abnormality. In addition to careful radiographic evaluation for dysplasia and FAI, torsional variations need to be considered in the pathomechanics of the hip [18]. Impingement can be the result of femoral retroversion especially in coexistence with acetabular retroversion; conversely, instability of the hip can be caused by increased combined femoral and acetabular anteversion. Investigating the torsional profile in combination with radiographic signs of FAI and dysplasia would help to elucidate additional anatomical risk factors and protective aspects for development of OA.

The mechanical disease of impingement or instability of the hip is still being diagnosed by static imaging studies that only reveal to us the anatomic risk factors for mechanical misbehavior. Ultrasound, dynamic MRI and computer modeling may give us better views of the hip in its active state and allow us to assess the mechanical pathology.

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From a clinical perspective, while radiographic screening may not be warranted, patients with risk factors for FAI or dysplasia such as a family history of hip disease and decreased internal rotation of the hip may benefit from counseling regarding the signs and symptoms of hip pathology. There are clearly additional genetic, biochemical and behavioral factors that affect whether hip OA becomes symptomatic, and future research will aim to identify which elements are predictive for and protective against the development of symptomatic OA.

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