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Abstract

Far more publications are available for osteoarthritis of the knee than of the hip. Recognizing this research gap, the Arthritis Foundation, in partnership with the Hospital for Special Surgery, convened an in-person meeting of thought leaders to review the state of the science of and clinical approaches to hip osteoarthritis. This article summarizes the recommendations and clinical research gaps gleaned from 5 presentations given in the “how hip osteoarthritis begins” session of the 2023 Hip Osteoarthritis Clinical Studies Conference, which took place on February 17 and 18, 2023, in New York City.

Keywords

osteoarthritis, arthritis, hip

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Introduction

Osteoarthritis (OA) is a major health burden that affects over 500 million adults or 15% of adults across the globe [22,31]. Hip OA has been found to be epidemiologically distinguishable from OA affecting other joints, such as the knee and hand [17]. In the United States, hip OA accounts for most of total hip arthroplasty (THA) procedures, which are projected to increase by 284% between 2014 and 2040 [66].

Yet far more publications are available for OA of the knee than of the hip. Recognizing this research gap, the Arthritis Foundation, in partnership with the Hospital for Special Surgery, convened an in-person meeting of thought leaders to review the state of the science of and clinical approaches to hip OA. This article summarizes the recommendations gleaned from 5 presentations given in the “how hip osteoarthritis begins” session of the 2023 Hip Osteoarthritis Clinical Studies Conference, which took place on February 17 and 18, 2023, in New York City.

Epidemiology of Hip OA and the Johnston County Osteoarthritis Project

Presented by Amanda E. Nelson, MD

Estimates suggest a prevalence of around 25% for radiographic hip OA, and 5% to 10% for symptomatic hip OA in the U.S. adult population [36,38,61]. The prevalence of hip OA among demographic subgroups is not well characterized [69]. Although previous studies suggested a lower prevalence of hip OA in African Americans [1,67], the Johnston County Osteoarthritis (JoCoOA) Project found that hip OA is at least as common among Black and White Americans, with a similar burden in Hispanics.

The JoCoOA Project is a longitudinal community-based study that followed 4000 unique participants from 1991 to 2018, performing measurements at baseline and 4 main follow-ups including an extensive questionnaire, imaging, clinical data, and biospecimens [35,53]. The population-based

study design allows generalizable estimates of prevalence and incidence relevant to the broader U.S. population [36,48,54]. The burden of symptomatic hip OA was emphasized to be substantial, with 1 in 4 people developing this condition by age 85 years. This was higher among those who are women, identify as White, are obese, or have prior hip injury [49]. Black and White Americans showed differences in progression patterns. For example, Black Americans reported progressive pain and disability, while White Americans had more radiographic hip OA progression [23]. Diabetes was associated with symptom development, and diabetes and cardiovascular disease made symptoms more persistent. Despite evidence that obesity predicts increased risk of hip OA and THA, an association between body mass index and lifetime risk of hip OA was not found. Racial disparities in THA could not be attributed to differences in disease occurrence.

While hip OA and knee OA differ, many OA management guidelines focus on knee OA and extrapolate this information to hip OA [3,39]. In fact, hip OA is more difficult to diagnose. The American College of Rheumatology criteria for the classification of hip OA requires hip pain most days of the prior month in combination with (1) erythrocyte sedimentation rate ≤ 20 mm/h, (2) femoral and/or acetabular osteophytes, and (3) joint space narrowing [2]. The prevalence of radiographic hip OA is approximately 10% of the population [30,36].

Challenging Kellgren and Lawrence: Epidemiology of Hip OA in White Women and Men

Presented by Nancy E. Lane, MD

Initial study of hip OA was slow due to a lack of a good radiographic definition of the disease. In its early stages, radiographic changes in hip OA include both joint space narrowing and femoral head osteophytes [37]. This differs from knee OA, in which radiographic changes are initially focused on osteophytes; joint space narrowing is only considered much later in the disease. Lane and colleagues at the University of California San Francisco developed a novel

scoring method for the hip that included an equal weighting of femoral osteophytes and joint space narrowing, the modified Croft Score, and used that to evaluate the epidemiology of prevalent, incident, and progression hip OA [16,40,57]. In addition, they determined that mild changes in the femoral head or acetabulum could increase the risk of incident hip OA, and they pioneered active shape modeling to provide a more comprehensive assessment of hip shape, ultimately defining the femoral head shapes that increased the risk of hip OA [41,46,55,56]. After defining radiographic hip OA, Lane and colleagues identified a number of risk factors—including higher total hip bone mineral density, height, weight, and polymorphisms of the Wnt/ β -catenin signaling pathway—that were significant predictors of radiographic hip OA in elderly White women. In elderly men, radiographic hip OA was associated with higher total hip bone mineral density [11,43,57]. Recently, it was found that radiographic hip OA was a strong risk factor for all-cause mortality and cardiovascular disease mortality in both elderly women and men [4,42].

Development of Hip OA Through a Pediatric Orthopedics Perspective

Presented by Young-jo Kim, MD, PhD

Developmental hip abnormalities (dysplasia or femoral head deformities such as pistol grip, femoral head tilt, or cam deformity) may cause 20% to 40% of hip OA [24,50,51,68]. Of the roughly 1% of infants born with hip instability [20], 10% will not have their hip instability spontaneously resolved during infancy [5]. In young patients, hip OA potentially caused by acetabular dysplasia, Legg-Calve-Perthes disease, slipped capital femoral epiphysis (SCFE), or femoroacetabular impingement (FAI) is a major cause (48%) of premature hip failure and subsequent THA [13]. Acetabular dysplasia is an insufficiency of coverage by the acetabulum of the femoral head that may result from developmental dysplasia of the hip (DDH) [28]. In babies and children with DDH, the hip has not developed properly and causes instability, dislocation, or subluxation in the joint [19].

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Early diagnosis and brace treatment can lead to normal hip development and prevent hip OA in adulthood [72]. Selective ultrasound screening of infants for DDH offers improved diagnostic accuracy over physical examination [32,59,65]. The Pavlik harness, potentially with supplemental bracing techniques, is first-line treatment for reducing a dislocated or subluxated hip and encouraging proper acetabular development [52,60]. Surgical techniques to alter and possibly normalize hip joint structure are available and valuable, although controversial [8,12,27,62]. In patients receiving surgical treatment for DDH, 31% required THA 45 years after the procedure [70]. Arthroplasty is very effective in young adults, but there is often a time in young adulthood when joint damage is minor but the affected hip is very symptomatic [18].

Perspective on pediatric hip OA is important due to the significant implications of growth and development in normal hip morphology. Developmental conditions such as infant hip dysplasia and SCFE are model systems that should be further explored to better understand the role of mechanics in hip OA. Adolescent cam deformity differs from SCFE deformity, yet it has similar mechanical effects in the development of joint damage. Surgical treatment of hip deformity has demonstrated efficacy in improving symptoms, but it is still difficult to show disease-modifying effect. Interventions that can be applied prior to cartilage tissue disruption may prevent disease progression and should be further examined.

Mechanobiology of Hips Prone to OA Using Computational Models

Presented by Sandra Shefelbine, PhD

Computational models can aid in the understanding of the mechanobiology of growing bone and cartilage in the pediatric population; this helps define the mechanical causes of malformed joints, a strong predictor of hip OA [7,14]. Models simulate the endochondral ossification process that occurs during growth by proposing that hydrostatic stress maintains cartilage and shear stress results in hypertrophy and ossification.

In the prenatal hip, finite element models demonstrate how abnormal forces influence bone morphology and the development of DDH [64]. Dynamic mechanobiological simulation showed that fetal movements affect femoral head sphericity and neck-shaft angle, indicating the manifestation of DDH [26]. Simulations also indicated that early treatment in a Pavlik harness [72] is critical to ensuring proper bone growth and joint shape.

Further, children with altered gait may be affected by deformed bone growth due to abnormal stresses on the developing bones; subsequently, they may be at higher risk

of hip OA [10,15,21]. Specifically, children with cerebral palsy frequently exhibit proximal femoral deformities, such as anteversion and coxa valga [9].

Cam morphology—a bump on the anterosuperior portion of the femur that forms during skeletal growth in elite adolescent athletes in specific sports (ice hockey, basketball, and soccer)—is a strong risk factor for the development of hip OA [58]. In addition, those with cam FAI tend to walk with more anterior pelvic tilt [34,45]. Musculoskeletal modeling was used to determine if pelvic tilt could change muscle and joint forces impacting the loading on the hip and subsequent growth.

Biomechanical loading during growth and development affects hip morphology. Altered loading (pathologic or elite sports) may alter forces sufficiently to create morphologies at high risk for hip OA. A better understanding of the “proper” forces critical for hip development during growth may enable the prevention of some morphological causes of hip OA and inform planning treatment strategies to preserve correct loading on the bone at a young age.

First Results From the World Collaboration on OA Prediction for the Hip

Presented by Jos Runhaar, PhD

The current “one size fits all” management approach to OA—in which the needs of hip OA are often co-opted from what we know about knee OA—should be challenged through better understanding of determinants and risk factors. The Worldwide Collaboration on OsteoArthritis prediction for the Hip: World COACH consortium was initiated for this purpose, as well as to develop an informed risk prediction model. The consortium includes all the prospective cohort studies worldwide that have longitudinal (at least 4 years apart) hip imaging data available (Table 1). The studies included information such as physical examination, family history, fractures/falls, comorbidities, medication, lifestyle/diet, and Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) questionnaires, as well as biospecimens and radiographs.

The World COACH consortium is a comprehensive set of data used to identify risk factors for hip OA and to drive personalized prevention strategies. World COACH is divided in the following work packages: methodology, hip morphology, genetics, clinical measures, and prediction models. Harmonization of study data across cohorts can lead to uniform assessments of hip morphology and high-quality data to study hip OA development. Hip morphological data based on automatized statistical shape modeling and predefined radiological measures were analyzed. The odds of developing radiographic hip OA within 4 to 8 years

Table 1. Description of cohorts included in the world COACH consortium.

Cohort	No. of participants	No. of baseline radiographs	Years of age at inclusions	Maximum follow-up (years)
Cohort Hip and Cohort Knee (CHECK) [6,73]	1002	1002	46–65	10
Chingford 1000 Women Study [29]	1003	1003	44–67	19
Johnston County Osteoarthritis (JoCoOA) Projects [35]	4337	3697	35–70	21
Multi-center Osteoarthritis Study (MOST) [63]	3026	3008	50–79	7
Osteoarthritis Initiative (OAI) [44]	4796	4771	45–79	8
Rotterdam Study (RS) [33]	14,926	11,147	45+	25
Tasmanian Older Adult Cohort (TASOAC) [24]	1099	1099	50–80	10
Study of Osteoporotic Fractures (SOF) [47, 71]	10,366	8291	65+	8
Total	40,555	34,018	35–80	7–25

are 1.24 times higher in hips with acetabular dysplasia than in hips without acetabular dysplasia. The odds of developing radiographic hip OA within 4 to 8 years are 1.59 times higher in hips with pincer morphology than in hips without pincer morphology. Study of the prevention of abnormal hip joint morphology or risk factors among individuals with abnormal hip joint morphology is warranted to further the field of hip OA prevention.

Conclusion

Hip OA must be recognized as a research target with origins, risk factors, and patient populations distinct from those of knee OA. In children, developmental conditions such as DDH and SCFE should be studied for their role in the mechanics of hip OA. Computer modeling offers better understanding of the biomechanical forces critical for proper hip development during growth and may inform the morphological causes of hip OA. Preventing disease progression with early interventions before cartilage tissue is disrupted is a tantalizing goal.

In adults, hip OA is associated with an increased risk of all-cause mortality, and understanding the impact of THA on mortality will be important in guiding public health policy. Racial disparities exist in THA, but insufficient data are available on underrepresented minorities. Other chronic conditions, such as diabetes and cardiovascular disease, are linked to and seem to exacerbate hip OA symptoms. Exploring differences between hip OA and other types of OA could provide insight into the pathophysiology of the whole disease. Since increased bone mineral density is associated with increased rates of radiographic hip OA, further research is needed on the potential role of a high bone turnover phenotype in the development of hip OA. Further research is also needed on the role of bone metabolism variants in hip OA. Overall, expanding research in hip OA could accelerate the development of evidence-based interventions that could be translated into community and clinical settings to prevent hip OA, improve physical function, and lower mortality rates.

Declaration of Conflicting Interests

The author(s) declared the following potential conflicts of interest with respect to the research, authorship, and/or publication of this article: Young-jo Kim, MD, PhD, reports a relationship with Cytex. Nancy E. Lane, MD, reports a relationship with Biosplice. Michael B. Millis, MD, reports relationships with the Peabody Foundation, Elsevier, and University of Minnesota. Amanda E. Nelson, MD, reports relationships with the NIH/NIAMS, CDC (National Institutes of Health / National Institute of Arthritis and Musculoskeletal and Skin Diseases K23AR061406, L30AR056604, P60AR064166, and P30AR072580; Centers for Disease Control and Prevention U01DP006266), University of Alabama, and Osteoarthritis Research Society International. Mathias P. Bostrom, MD, reports a relationship with Smith + Nephew. The other authors declared no potential conflicts of interest.

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Human/Animal Rights

All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as revised in 2013.

Informed Consent

Informed consent was not required for this review article.

Level of Evidence

Level V: Review Article/Expert Opinion.

Required Author Forms

Disclosure forms provided by the authors are available with the online version of this article as supplemental material.

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