Hip Osteoarthritis: A Primer

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ABSTRACT

The objective of this article is to deliver a concise up-to-date review on hip osteoarthritis. We describe the epidemiology (disease distribution), etiologies (associated risk factors), symptoms, diagnosis and classification, and treatment options for hip osteoarthritis. A quiz serves to assist readers in their understanding of the presented material.

INTRODUCTION

Please see the Sidebar: Quiz to Assess Knowledge of Hip Osteoarthritis (True/False/Depends) with Answers.

Osteoarthritis (OA), often referred to as “wear-and-tear” arthritis, age-related arthritis, or degenerative joint disease, is the most common form of joint disorder in the US, and it is estimated that more than 27 million Americans are affected.1 As a degenerative disorder, OA can involve any joint, and it primarily affects the articular cartilage and surrounding tissues.2 OA can be broadly classified into primary and secondary types. In primary OA, the disease is of idiopathic origin (no known cause) and usually affects multiple joints in a relatively elderly population. Secondary OA usually is a monoarticular condition and develops as a result of a defined disorder affecting the joint articular surface (eg, trauma). This review will focus on primary hip OA with a discussion of secondary hip OA.

The hip joint is one of the body’s largest weight-bearing joints, only secondary to the knee joint, and is commonly affected by OA.3 The current accepted understanding of hip OA is that although articular cartilage is mainly affected, the entire joint also is affected. The OA process involves progressive loss of articular cartilage, subchondral cysts, osteophyte formation, periarticular ligamentous laxity, muscle weakness, and possible synovial inflammation.2,4 There is a growing consensus that OA is not the result of a singular process affecting the joints but rather results from a number of distinct conditions, each associated with unique etiologic factors and possible treatments that share a common final pathway. The effects of OA on the large joints of the lower extremities, including the hips, can result in reduced mobility and marked physical impairment that can lead to loss of independence and to increased use of health care services. As such, OA may have a profound effect on activities of daily living and lead to substantial disability and dependency in walking, stair climbing, and rising from a seated position. Several risk factors are linked to the development of hip OA including age, gender, genetics, obesity, and local joint risk factors. However, the exact primary hip OA etiology remains unknown,4 and a universal protocol is lacking for its diagnosis and treatment. In this report, we describe hip OA epidemiology (disease distribution), etiologies (associated risk factors), symptoms, diagnosis and classification, and treatment options.

PREVALENCE

The difference between the clinical and radiographic prevalence of hip OA remains unclear; however, most epidemiologic studies of hip OA involve radiographic parameters to establish disease prevalence.9,10 Research suggests that hip OA is epidemiologically distinguishable from OA affecting other joints.11 For example, only a small percentage of patients who underwent total hip arthroplasty (THA) to address primary hip OA required a total knee arthroplasty (3%–7%) and vice versa.12 In a prominent US-based population study,13 prevalence of symptomatic hip OA was reported at 9.2% among adults age 45 years and older, with 27% showing radiologic signs of disease; prevalence was slightly higher among women. A systematic review of radiographic hip OA prevalence demonstrated an increase in mean prevalence with advancing age for both men and women.14 Men have a higher prevalence of hip OA before age 50, whereas women have a higher prevalence thereafter.15 Caucasian populations also have a higher hip OA prevalence that ranges between 3% and 6% as compared with 1% or less in Asians, blacks, East Indians, or native Americans,15,16 suggesting a genetic predisposition. According to the Centers for Disease Control and Prevention, lifetime risk for symptomatic hip OA is 18.5% for men and 28.6% for women.5

ETIOLOGIES AND RISK FACTORS

OA is a chronic disorder affecting synovial joints. Although sometimes referred to as “degenerative joint disease,” this term is a misnomer. The degenerative process manifested by progressive loss of articular cartilage is accompanied by a reparative process with reactive bone formation, osteophyte growth, and remodelling.3 The dynamic process of destruction and repair determines the final disease picture. OA is not primarily an inflammatory process, and synovial inflammation, when found, usually is not accompanied by a systemic rise in inflammatory markers. Primary OA (also termed idiopathic), generally is a diagnosis of exclusion and is believed to account for...
the majority of all hip OA. Aging is assumed to contribute to the development of hip OA mainly because of the inability to specifically define an underlying anatomic abnormality or specific disease process leading to the degenerative process.

Genetic factors also may play a role in hip OA, possibly by the inheritance of an anatomical abnormality such as acetabular dysplasia. A sibling study demonstrated a higher risk for hip OA among those who had an affected sibling, as demonstrated by structural changes noted on hip radiographs. Secondary (from a known cause) OA results from conditions that change the cartilage environment. These conditions include trauma, congenital or developmental joint abnormalities, metabolic defects, infection, endocrine disease, neuropathic conditions, and disorders that affect the normal structure and function of hyaline cartilage. Secondary hip OA occurs when a condition results in an anatomic abnormality, which can be relatively subtle, that predisposes the hip to mechanical factors that lead to degenerative changes.

Risk factors associated with hip OA can be divided into local risk factors that act on the joint level and more general risk factors.

Local Risk Factors
Joint Dysplasia
Conditions such as acetabular dysplasia and other developmental disorders leading to structural joint abnormalities are believed to play a major role in development of hip OA later in life. Mild dysplastic changes often can go unnoticed and predispose to hip OA.

Trauma
Fractures involving the joint articular surface can lead to secondary posttraumatic arthritis. It is unclear whether isolated labral tears contribute to hip OA.

General Risk Factors
Age
The Research on Osteoarthritis/Osteoporosis Against Disability study, which prospectively followed 745 Japanese men and 1470 Japanese women for 3 years, revealed that age greater than 60 years is an important risk factor for radiographic OA. However, it is also clear that aging of joint tissues and OA development are distinct processes. Chondrocalcinosis, an age-related matrix change observed in radiographs of arthritic joints, may contribute to OA by stimulating production of proinflammatory mediators.

Sex
Hip OA prevalence is higher among men younger than age 50 years, whereas women have the highest prevalence after age 50 years. This finding may be attributable to postmenopausal changes and is supported by observations from multiple studies that report protective effects of estrogen replacement therapy and hip OA.

Obesity
Excess body weight is a risk factor for OA not only in weight-bearing joints, but also in the hand. Excess weight produces increased load on the joint, but there is growing evidence for a metabolic contribution to OA as well.

Genetics
Several studies suggest that genetics have an important role in the etiopathogenesis of hip OA, and a twin study reported on a 60% risk for hip OA attributable to genetic factors. Another study demonstrated that having a first-, second-, or third-degree relative who undergoes THA for hip OA increases a person’s risk for having the procedure.

Occupation
Certain occupations involving heavy manual work and high-impact sports activities are linked to OA in the hip and other joints later in life. Repetitive stress and biomechanical overload, especially in the setting of a preexisting hip joint anatomical abnormality, are likely causes. Farmers are particularly prone to hip OA. However, no credible evidence demonstrates that exercise and physical activity are directly related to hip OA in the general population.

Quiz to Assess Knowledge of Hip Osteoarthritis (True/False/Depends) with Answers:

1. Aging and other risk factors contribute to hip osteoarthritis (OA).
   Answer: True. However, not all hip OA is related to the aging process. Young people can develop secondary hip OA from trauma, congenital dysplasia and developmental disorders, infection, metabolic conditions, and other causes.

2. Patients should wait as long as possible before undergoing total hip arthroplasty (THA).
   Answer: False. Patients who fail nonsurgical treatment should not delay undergoing THA because delay correlates with worse clinical outcomes even after surgery is performed.

3. Hip OA primarily is a disease of cartilage.
   Answer: True. Progressive loss of articular cartilage often is accompanied by a reparative process that involves sclerosis and osteophyte formation.

4. Joint stiffness in hip OA may not improve for several hours, or it may last throughout an entire day.
   Answer: False. Morning stiffness helps to differentiate OA from rheumatoid arthritis. In rheumatoid arthritis, joint stiffness may not improve for several hours or it may last throughout the entire day. In OA, stiffness typically lasts for only a few minutes and subsides in 30 minutes or less. Movement and physical activity that loosens the joint generally improve OA.

5. Certain radiographic parameter measurements as described by Kellgren and Lawrence can help clinicians assess hip OA severity.
   Answer: False. Currently, there is no gold standard with which to measure and report the prevalence of radiographic primary hip OA. The Kellgren and Lawrence method of diagnosis is the most common method with which to measure radiographic OA severity. A limitation associated with this system is its reliance on the presence of osteophytes, which correlate poorly with hip pain.

6. Studies demonstrate that viscosupplementation injections slow OA symptom progression.
   Answer: False. Most clinical studies show that these treatments are no more effective than a placebo and are not recommended as hip OA treatment.

7. Nonsteroidal anti-inflammatory drugs (NSAIDs) are effective first-line hip OA treatments.
   Answer: True. Both topical NSAIDs (such as capsaicin) and oral NSAIDs may be considered as an adjunct for symptomatic pain relief in addition to core treatments for patients with OA. Diclofenac and etoricoxib are the most efficacious NSAIDs for pain relief in hip OA, producing a moderate to large effect size. However, NSAIDs should be used with caution to avoid potential complications associated with long-term use.

SYMPTOMS

The most common symptom of hip OA is pain around the hip joint (generally located in the groin area). The pain can develop slowly and worsening over time (most common) or pain can have a sudden onset. Pain and stiffness can develop in the morning or after sitting or resting. Stiffness typically lasts for only a few minutes and subsides over 30 or fewer minutes. Movement and activity that loosen the joint generally improve OA symptoms. Later in the progression of the disease, painful symptoms may occur more frequently, including during rest or at night (see Sidebar: Common Hip Osteoarthritis Symptoms).

DIAGNOSIS AND CLASSIFICATION

Hip OA often can be diagnosed upon clinical presentation alone, although imaging investigations can be useful to both confirm a diagnosis and to monitor disease progression (Figure 1A-C). After taking a careful medical history that includes a review of associated hip OA risk factors, a clinician should perform a focused clinical examination of the affected hip. The examination should include an inspection and comparison of leg length between the affected and opposite sides, an evaluation of a possible joint fixed position denoting deformity, and a gait assessment. These steps should be followed by palpation of bony prominences and tendons to assess for tenderness and/or injuries. A neurovascular assessment of both lower extremities and range of motion of the affected joint should be performed with a comparison to the contralateral side. Additional tests may provide more information regarding underlying conditions that lead to hip OA.

In 1957, Kellgren and Lawrence described a grading scale for the radiologic assessment of OA that remains the most widely used classification system; however, this scale is not specific for hip OA grading. In 1963, Kellgren described four grades of hip OA based on the degree of joint space narrowing, osteophyte formation, arthritic changes affecting the bone margins, and gross deformity as the following: Grade 1, doubtful OA with possible joint space narrowing medially and subtle osteophyte formation around the femoral head. Grade 2, mild OA with definite joint space narrowing inferiorly with definite osteophyte formation and slight subchondral sclerosis. Grade 3, moderate OA with marked narrowing of the joint space, small osteophytes, some sclerosis and cyst formation, and deformity of the femoral head and acetabulum. Grade 4, obliterated joint space with features seen in grades 1 to 3, large osteophytes, and gross deformity of the femoral head and acetabulum. Several other radiographic classification systems exist such as Croft’s grade and the Tönnis classification. Other imaging studies such as computerized tomography and magnetic resonance imaging typically are not required for diagnosis and usually are reserved for the identification of secondary causes or presurgical planning. Blood tests may be ordered to help confirm a diagnosis and to rule out other inflammatory conditions such as rheumatoid arthritis, especially if joint symptoms are associated with morning stiffness and synovial inflammatory changes. Complete blood count, erythrocyte sedimentation rate, C-reactive protein, rheumatoid factor, and cyclic citrullinated peptide antibody tests are among the most common laboratory studies ordered; when testing for hip OA, however, these test results are expected to fall within defined limits. The American College of Rheumatology has established clinical criteria and radiologic parameters that are commonly used for hip OA diagnosis in clinical practice.

An important contrast between patient symptoms and radiographic findings may be observed. Patients with marked radiographic changes may not necessarily demonstrate severe correlating clinical symptoms and vice versa. Some patients with high-grade radiographic hip OA may be asymptomatic.

TREATMENT OPTIONS

Nonpharmacologic Treatments

Exercise

An exercise program that does not involve high-impact activities usually is advocated and is associated with pain reduction. Aquatic exercises also improve function. Exercises that strengthen and stretch the muscles around the hip can support the hip joint and ease hip strain. Certain activities and exercises...
that can aggravate the hip joint should be recognized and avoided (see Sidebar: Common Activities that Exacerbate Osteoarthritis Hip Pain). Activities that necessitate twisting at the hip such as golf or are high impact such as jogging should be replaced with activities that exert less stress on the hip joint such as gentle yoga, cycling, or swimming. Manipulation and stretching should be considered as adjuncts to core treatments, particularly for hip OA.46

Physical Therapy
Physical therapy is the mainstay of treatment in mild and early hip OA and is aimed at strengthening hip muscles and maintaining joint mobility. Physical therapy that is provided during the later stages of hip OA may provide little or no benefit.40

Weight Reduction
Gaining 10 pounds can exert an extra 60 pounds of pressure upon a hip with each step.41 Unloading the joint through weight loss can slow cartilage loss and decrease joint impact. Weight recommendations that address hip OA are based upon findings from many cohort studies.42–45 An individualized exercise program combined with effective behavioral strategies aimed at weight loss may be most beneficial in reducing pain for overweight patients.

Transcutaneous Electrical Nerve Stimulation
Transcutaneous electrical nerve stimulation should be considered as an adjunct to core treatments for pain relief patients with hip OA.46

Temperature Extremes
Hot and cold treatments sometimes are effective pain relief modalities. Heat treatments enhance circulation and soothe stiff joints and tired muscles. Cold treatments slow circulation, reduce swelling, and alleviate acute pain. A patient may need to experiment and/or alternate use of heat and cold therapies to determine which is most effective.

Proper Footwear and Bracing/Joint Supports/Insoles
Patients should be educated about appropriate footwear that features shock-absorbing properties to address lower limb OA.46 Patients with OA who have biomechanical joint pain or instability may be considered for assessment of bracing/joint supports/inoles as an adjunct treatment.46 Bracing may have a role in modifying biomechanics to treat hip OA, although more research in this area is necessary.5

Assistive Devices
Walking sticks, tap turners, canes, and other devices should be considered as adjuncts to core treatments for people with OA who have specific problems with activities of daily living. If needed, patients can be referred for further evaluation and treatment from occupational and physical therapists and/or specialized disability device and equipment companies.46

Acupuncture is not recommended as OA treatment. Patient education can help to incorporate multiple approaches into hip OA treatment and minimize risk factors.

Pharmacologic Treatments
Acetaminophen and Nonsteroidal Anti-Inflammatory Drugs
Acetaminophen typically is recommended as a first-line medication for OA.47 However, the role of acetaminophen for short-term relief of hip OA pain remains equivocal.47 Topical Nonsteroidal anti-inflammatory drugs (NSAIDs) (such as capsaicin) may be considered as an adjunct therapy for pain in addition to core treatments. Acetaminophen and topical NSAIDs should be considered ahead of oral NSAIDs, cyclooxygenase 2 inhibitors, or opioids.46 Topical capsaicin should be considered as an adjunct to core treatments for knee or hand OA but has limited use in hip OA because of hip joint depth.46 If acetaminophen is insufficient for pain relief, NSAIDs may be more efficacious.46 Diclofenac and etoricoxib are the most efficacious NSAIDs for pain relief in hip OA, producing moderate to large effects.46 However, NSAIDs should be used with caution to avoid potential complications such as gastrointestinal tract bleeding and adverse cardiovascular events associated with long-term use.46,47 If acetaminophen and/or NSAIDs provide insufficient pain relief, opioid analgesics may be considered. Opioid medications, however, are not routinely used because of concerns regarding their side effects and long-term addiction potential.48 Risks and benefits should be considered, particularly for older patients.46

Rubefacients
Topical rubefacients should not be used to treat OA.46

Glucosamine/Chondroitin
Use of glucosamine or chondroitin products for OA treatment is not recommended.46

Intra-Articular Injections
Corticosteroids; hyaluronic acids; and, relatively recently, platelet-rich plasma injections, are the most common modalities to treat pain associated with hip OA. Corticosteroids offer short-term pain relief,46 and guidelines recommend their use as an adjuvant to other nonsurgical treatment modalities.45 Although the literature in this area is scarce and data are weak, recent evidence suggests that caution should be exercised when using multiple intra-articular steroid hip injections before THA because multiple injections have been associated with a significantly higher risk for prothetic joint infection than a single injection administered before THA.44,49,50 Clinical trials do not provide strong support for the clinical use and value of hyaluronic acid injections.46,47 The use of platelet-rich plasma remains under investigation in clinical trials, and data available from small studies do not provide substantial evidence for a clear clinical role.5

Surgical Treatments
Hip Arthroscopy
Studies on the use of arthroscopy in hip OA are not high quality. Arthroscopy, which primarily is performed during early OA stages, provides temporary relief and is associated with a high conversion rate to THA (9.5%-50%).71

Total Hip Arthroplasty
THA is today’s surgical modality for patients with intractable pain, for those who have failed nonsurgical treatment, and for those with severe functional impairment. Approximately 1 million THA procedures are performed globally each year for patients with advanced hip OA.52 This procedure repeatedly demonstrates cost-effectiveness in clinical trials.53 Hip implant longevity has
been demonstrated, with as many as 95% of prostheses remaining functional at 10 years, which is consistent in certain populations where the patient has good overall general physical health, ability to exercise, remains active and maintains a good weight for which more than 80% of prostheses can remain functional at 25 years.65-67 Primary care providers should advise symptomatic patients who fail nonsurgical treatment to avoid waiting unnecessarily to undergo THA because evidence demonstrates that prolonged delays correlate with worse clinical outcomes after THA.69 Progressive pain, disability, and functional impairment can cause further unnecessary damage to tissues and joints that affect the biomechanical environment in other joints. Interference with usual activities of daily living can be unnecessarily affected; this can be especially problematic for younger patients who work and are more socially and physically active.

**Hip Resurfacing**

Although originally developed as a substitute for THA for younger patients who failed nonsurgical treatment, current evidence indicates that hip resurfacing is suitable for a very specific subset of patients, usually young active men with large femoral heads, as an alternative to THA.54-57

**CONCLUSION**

OA is a chronic disorder affecting synovial joints and a leading cause of disability in the US and worldwide. Current thought is that hip OA results from a number of distinct conditions, each associated with unique etiologic factors and possible treatments that share a common final pathway. The most common symptom of hip OA is pain around the hip joint (generally located in the groin area). Most of the time, the pain develops slowly and worsens over time, or pain can have a sudden onset. Aging and genetic factors are important contributing causes of hip OA. The European League against Rheumatism 2005 Recommendations for the Management of Hip Osteoarthritis advocate a multidisciplinary approach for the management of hip OA (see Sidebar: European League against Rheumatism 2005 Recommendations for the Management of Hip Osteoarthritis).

**Disclosure Statement**

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