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Epidemiology of Posttraumatic Osteoarthritis

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Osteoarthritis is a leading cause of disability whose prevalence and incidence continue to increase. History of joint injury represents an important risk factor for posttraumatic osteoarthritis and is a significant contributor to the rapidly growing percentage of the population with osteoarthritis. This review will present the epidemiology associated with posttraumatic osteoarthritis, with particular emphasis on the knee and ankle joints. It is important to understand the effect of posttraumatic osteoarthritis on the population so that sufficient resources can be devoted to countering the disease and promoting optimal long-term health for patients after joint injury.

Key Words: injuries, arthritis, knee, ankle

Osteoarthritis (OA) is a leading cause of mobility-related disability in the United States. Due in large part to the aging population and the increasing rate of obesity, the prevalence of OA is expected to double by the year 2020.[1] Another factor that warrants consideration in the increasing rate of OA is joint trauma. Individuals who sustain a joint injury are known to be at substantially increased risk of developing OA compared with uninjured persons.[2] Osteoarthritis that develops after joint injury is deemed *posttraumatic OA* (PTOA).

As of 2005, treating lower extremity PTOA cost $11.79 billion, with direct costs exceeding $3 billion annually.[3] As injury rates rise and PTOA becomes more prevalent, the financial burden on the health care system will likely increase.

Although a number of reviews on the epidemiology of OA are available,[4–6] we aim to detail the prevalence and risk factors associated with PTOA, particularly of the knee and ankle joints.

DEFINITIONS OF OA

Osteoarthritis is characterized by degeneration of the articular cartilage and subchondral bone, often leading to pain, joint stiffness, and disability. A number of definitions of OA exist, including both radiographic and symptomatic versions. Commonly, OA is graded radiographically using the Kellgren-Lawrence (K-L) scale.[7] The K-L scale ranges from 0 to 4 based on the presence and degree of osteophytes, joint-space narrowing, sclerosis, and deformity, with grades of 2 or higher indicating the presence of radiographic OA. The K-L scale does not consider symptoms when defining OA severity. The use of magnetic resonance imaging to detect the presence of cartilage and bone marrow lesions, osteophytes, and effusion is gaining popularity, though no standard magnetic resonance imaging–based definition of OA exists.

Symptomatic OA is defined as the presence of radiographic OA plus symptoms including pain, aching, stiffness, and disability in the affected joint.[5,8] It should be noted that not all individuals with radiographic OA present with symptomatic OA.

Posttraumatic OA develops after joint injury. Injury may be in the form of fracture, cartilage damage, acute ligament sprain, or chronic ligamentous instability (or a combination of these).

INCIDENCE AND PREVALENCE OF OA

Approximately 27 million adults in the United States aged 25 years and older have a clinical diagnosis of OA of any joint.[1] Persons with PTOA account for nearly 12% of all cases of symptomatic OA, or approximately 5.6 million cases of lower extremity OA in the United States.[3] At the knee, an estimated 13 million adults aged 60 years and older in the United States have radiographic OA, with approximately 4 million of those individuals classified as having symptomatic knee OA. Persons who sustain a knee injury are 4.2 times more likely to develop OA than those without a history of knee injury.[9] Authors[3] of a retrospective medical record review suggested that PTOA accounts for approximately 10% of all cases of knee OA.

Idiopathic OA of the ankle is rare. In fact, only 1% of the global population is estimated to have any form of ankle OA.[10] Further, patients are 10 times more likely to be diagnosed with knee OA than ankle OA.[11] However, among individuals with ankle OA, prior joint trauma is the most common cause, with PTOA accounting for between 20% and 78% of all cases of ankle OA.[3,12,13]

Posttraumatic OA may present in any joint after trauma, though limited epidemiologic data are available regarding PTOA in joints other than the knee and ankle. Posttraumatic OA of the hip, for example, represents approximately 2% of all cases of hip OA.[3] The prevalence of hip PTOA is higher among military personnel, with rates reaching 20%.[14] At the shoulder, PTOA prevalence ranges from 8% to 20% in patients scheduled to undergo a variety of

surgical stabilization procedures for anterior glenohumeral instability.[15–17]

RISK FACTORS FOR PTOA

Joint Injury

Knee Injury. Despite the popularity of injury-prevention programs for youth through professional athletes, the knee and ankle remain among the most commonly injured joints in the body. Sprains and strains to the knee or leg make up 11% of all musculoskeletal injuries treated by physicians in the United States.[18] Further, knee injuries account for 15% of all high school sport-related injuries.[19] Among knee injuries, 23% involve the meniscus and 25% involve the anterior cruciate ligament (ACL), with isolated injuries to the meniscus accounting for 11% and the ACL, 12% of all knee injuries.[20] Specifically, nearly 250 000 ACL injuries occur annually in the United States,[21] with approximately 175 000 of those patients undergoing ACL reconstruction.[22] Anterior cruciate ligament injuries are frequently accompanied by damage to other structures within the knee joint, including the articular cartilage and subchondral bone, collateral ligaments, and menisci. In fact, concurrent meniscal damage occurs in up to 75% of all ACL injuries.[23] Incidence rates for meniscal injuries range from 0.33 to 0.61 per 1000 person-years[24,25] in physically active individuals but are as high as 8.27 per 1000 among active-duty military personnel.[26] Unfortunately, incidence rates for meniscal injuries among physically active persons are believed to be underestimated because of people not seeking medical treatment.[27]

Both ACL and meniscal injuries carry a high risk of PTOA development. The prevalence of PTOA after ACL injury is conflicting because of the different classification methods for defining OA in the literature.[28] For patients with isolated ACL injuries, PTOA prevalence ranges from 0% to 39%,[29–36] whereas prevalence is higher among individuals with combined ACL and meniscal injuries (21%–100%).[31,34,36–38] However, Oiestad et al[28] suggested that poor methodologic quality of studies has led to overestimation of PTOA rates and that prevalence may be closer to 13% in patients with isolated ACL injuries and between 21% and 48% in those with combined ACL and meniscal injuries who are at least 10 years postinjury.[28]

A systematic review by Luc et al[39] compared PTOA prevalence among patients after ACL reconstruction with those who were ACL deficient. Overall rates of OA development were higher in patients after ACL reconstruction (44%) than in those who remained ACL deficient (37%), with an odds ratio (OR) of 1.29 (95% confidence interval [CI] = 1.06, 1.52).[39] Luc et al[39] further observed that time since injury affected PTOA prevalence. Specifically, the prevalence of PTOA was greater in ACL-reconstructed individuals through the first 2 decades after injury; however, PTOA prevalence was 34% greater in ACL-deficient compared with -reconstructed patients in the third decade after injury.[39] When concomitant meniscal injury was considered, 52% of patients who underwent ACL reconstruction plus meniscectomy demonstrated PTOA, whereas 59% of patients who underwent meniscectomy but remained ACL deficient developed PTOA.[39] However, these results should be interpreted with caution because of the small numbers of patients included in the ACL-deficiency studies, particularly by the third decade after injury.[39] Though the incidence rates vary, the majority of studies suggest that surgical reconstruction of the ACL does not protect against future OA development.

As noted, meniscal injuries and related surgeries are also associated with PTOA development. Data from the Osteoarthritis Initiative indicate that meniscal injuries were not significantly associated with PTOA development at 2-year follow up.[40] Yet these same data reveal that patients who did go on to develop PTOA within 2 years were more likely to have sustained medial meniscectomy (OR = 3.03; 95% CI = 1.4, 6.5), complex meniscal tears (OR = 5.0; 95% CI = 1, 25), or radial tears of the meniscus (OR = 5.92; 95% CI = 1.7, 7.5).[40] Pengas et al[41] followed patients for an average of 40 years after meniscectomy, observing a relative risk of PTOA development of 4.5 (95% CI = 1.8, 11.2). This value is similar to that reported by Englund and Lohmander[42] at 15- to 22-year follow up (relative risk = 5.4; 95% CI = 2.5, 13).

Considerable data suggest that complete meniscal resection is associated with higher rates of PTOA than meniscal repair or partial meniscectomy.[43] Stein et al[44] reported radiographic changes in 19.2% of patients after medial meniscal repair compared with 60% of patients after partial medial meniscectomy at 8-year follow up. Andersson-Molina et al[45] found that 33% of patients developed PTOA after partial meniscectomy compared with 72% of individuals after total meniscectomy. Similarly, Englund and Lohmander[42] demonstrated greater odds of PTOA development after total meniscectomy compared with partial meniscectomy (OR = 3.6; 95% CI = 1.4, 9.4). Collectively, these data indicate a greater risk of PTOA development as the amount of meniscus that is removed increases.

What causes PTOA after ACL and meniscal injury remains unknown. Regression analyses from several studies suggest a number of factors may contribute, among them ACL reconstruction, medial meniscectomy at the time of ACL reconstruction,[46] higher body mass index,[47] poor performance (<90% compared with the contralateral limb) on a single-legged hop test 12 months after surgery,[48] and decreased knee extension and increased joint laxity at 13 years after surgery.[46] It remains possible that trauma from the large forces required to tear the ACL causes sufficient tissue damage to initiate the degenerative process.[49] In fact, up to 90% of ACL injuries are accompanied by osteochondral lesions, suggesting that trauma to the articular cartilage occurs with ACL rupture.[4] Increased presence of inflammatory markers and biomarkers of cartilage degradation have also been reported after ACL injury and reconstruction[50] and meniscal injury.[51] However, the prognostic importance of these biomarkers is presently unknown, as it is unclear if the elevated concentrations of these markers are a healthy or pathologic adaptation to injury.[50]

Altered loading about the injured joint has also been suggested to contribute to PTOA development. Biomechanical changes in ACL-deficient and -reconstructed individuals may change the regions where tibiofemoral joint contact occurs, thereby loading areas of cartilage that were previously unloaded and decreasing loads to areas of cartilage normally experiencing higher loads during weight bearing.[52] Similar biomechanical alterations have been observed after meniscectomy that may contribute to PTOA

development.[53] It has also been suggested that quadriceps weakness and central activation deficits that arise after knee-joint injury contribute to PTOA.[54] Quadriceps function is important to energy absorption about the knee. When the quadriceps are weak, as is often the case years after ACL injury and reconstruction or meniscectomy,[55] they cannot adequately absorb the energy of impact. This results in greater magnitudes of loading on the tibiofemoral articular cartilage and, ultimately, joint degeneration. This association was demonstrated by Tourville et al,[56] who examined knee-flexion and knee-extension strength and tibiofemoral joint-space width in patients after ACL injury but before reconstruction and at 1 and 4 years postoperatively. Tourville et al[56] observed that baseline quadriceps strength was less in patients with narrow joint-space width compared with patients with normal joint-space width. These deficits persisted 4 years postoperatively.[56]

Intra-articular fractures also contribute substantially to PTOA of the knee. An estimated 23% to 44% of intra-articular fractures at the knee will lead to PTOA.[57,58] Both acute mechanical damage and chronic abnormal joint loading contribute to cartilage breakdown after intra-articular fracture.[59] Though the precise contributions of these factors to PTOA development are unknown, mounting evidence suggests that acute mechanical damage predominates and that the energy absorbed by the articular surface at the time of injury dictates how the cartilage will tolerate chronic changes in joint mechanics.[59]

Ankle Injury. Ankle injuries are common and account for roughly 20% of all emergency department visits each year[60] and 23% of all high school sport-related injuries.[61] The majority (85%) of these injuries are lateral ankle sprains,[62] with an estimated 25 000 ankle sprains occurring daily.[63] However, 37% of all cases of ankle PTOA are the result of fractures.[12,13] Lubbeke et al[64] examined 102 patients at an average of 18 years after ankle fracture treated with open reduction and internal fixation, noting a K-L grade 3 to 4 OA in 36% of patients. Further, ankle PTOA was present in 60% to 70% of individuals who had at least 3 of the following risk factors: (1) Weber C or medial malleolus fracture, (2) age 30 years or older at the time of injury, (3) overweight or obese at the time of injury, and (4) longer-duration follow up since surgery.[64]

Recurrent ankle injuries are the second leading cause of ankle PTOA, accounting for 13% to 16% of all cases,[12,13,65] although this figure has been reported to be as high as 78%.[66] Individuals with a history of a lateral ankle sprain frequently develop chronic ankle instability (CAI), or lifelong symptoms, recurrent injury, and disability after ankle sprain. In fact, 75% of individuals with a history of ankle sprain may have CAI.[67] How recurrent ankle instability contributes to PTOA development is unknown, though mechanical factors may contribute. Similar to patients after knee injury, individuals with CAI frequently exhibit muscle weakness, joint laxity, and altered biomechanics. These impairments may alter the load distribution about the ankle joint, leading to breakdown of the articular cartilage,[68,69] as chondral lesions have been found in 95% of ankles with chronic ligamentous injuries.[69]

A history of a single ankle sprain with persistent pain represents the third leading cause of ankle PTOA development, accounting for 13.7% of all cases.[12] Taga et al[69] observed that 89% of patients with acute lateral ankle sprains presented with chondral lesions. It has been reported that cartilage damage anywhere in the ankle joint is an independent risk factor for ankle PTOA development,[70] supporting the notion that cartilage damage sustained at the time of ankle sprain may contribute to joint degeneration.

Other Joint Injury. Hip PTOA is frequently caused by acetabular fracture, with upward of 25% of patients sustaining acetabular fractures going on to develop PTOA.[71,72] As with idiopathic OA, obesity augments PTOA development in these patients. In fact, 68% of patients who were morbidly obese at the time of acetabular fracture fixation developed PTOA.[73] These data provide evidence of a link between obesity and PTOA development, but more studies are necessary to better define the incidence and prevalence of PTOA in obese and nonobese individuals.

Similarly to ankle PTOA, glenohumeral PTOA is associated with recurrent joint instability.[74] Buscayret et al[16] suggested that age at the time of the initial episode of instability, increased time from initial injury to surgery, rotator cuff tears, and bony lesions to the glenoid or humerus increase risk of OA development. Further research is needed on all joints, but particularly the hip and shoulder, to deepen our knowledge of the prevalence of and risk factors for PTOA.

Other Potential Risk Factors

Genetics. One important factor that cannot be ignored in the discussion of PTOA is genetics. Genetic factors are a large contributor to OA development, accounting for 50% or more of the variation in susceptibility to the disease.[75] In the hand, the heritability of OA is more than 60%.[75] Previously, investigators[76] examined the association between hand OA and the risk of knee OA development after meniscectomy performed on average 20 years before study enrollment. The presence of hand OA was associated with a higher rate of knee OA development (OR = 3.0; 95% CI = 1.2, 7.5), thereby suggesting that knee OA development after meniscectomy may not be entirely due to joint trauma and that the patients who developed OA could have been genetically predisposed to develop the disease.[76] Similarly, Valdes et al[77] researched the influence of genetic risk factors on total knee and total hip arthroplasty rates among individuals with or without a history of joint injury. Genetic factors contributed to the risk of total knee and hip arthroplasty nearly equally among individuals with or without a history of joint injury.[77] Collectively, these studies seem to suggest that genetic risk factors for OA development may contribute to PTOA. Thus, it may be inaccurate to deem cases of OA that develop after known joint injury solely posttraumatic in origin.

Physical Activity. Physical activity to strengthen the musculature surrounding an injured joint is often recommended to decrease symptoms and improve function. However, repetitive use of a joint is associated with an increased OA risk[4] and previous data indicate that muscle strengthening may increase joint-space narrowing in patients with tibiofemoral OA.[78] This may be particularly problematic after joint injury when normal biomechanics and load distribution are disrupted, unloading areas of cartilage that are normally loaded and loading areas of

cartilage that are not normally loaded during weight-bearing activity. Conversely, many patients experience continued pain after joint injury. Pain may promote a reduction in physical activity, which may lead to the person becoming overweight or obese, both of which are associated with the development and progression of OA. For every 5-unit increase in body mass index, the risk of knee OA development increases 35%.[79] Additionally, the link between obesity and hip PTOA has been discussed earlier. Although physical activity is important for overall health, it may compromise joint health after injury. Finding strategies to remain physically active across the lifespan while minimizing repetitive joint stress and pain is necessary.

Patient Sex. It has been clearly established that females are more susceptible to OA development and present with more severe OA than males.[6] These findings may be related to hormonal factors, though evidence of a hormonal link is conflicting.[6] In regard to PTOA, an association between patient sex and disease prevalence has yet to be established.

CONCLUSIONS

Posttraumatic OA affects more than 5 million adults in the United States. Posttraumatic OA arises after joint injury and repetitive joint trauma associated with recurrent instability and primarily affects the knee and ankle joints. Joint injury alters neuromuscular control and biomechanics around the affected joint, which may contribute to cartilage degradation. Given the large number of knee and ankle injuries that occur annually and the strong association between joint injury and OA development, PTOA represents a significant public health burden. Developing treatment strategies to delay or prevent PTOA (or both) and promote optimal long-term health after joint injury is imperative.

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