Assessing for Risk and Progression of Osteoarthritis: The Nurse’s Role

Understanding pathophysiology, epidemiology, and risk will aid nurses who are seeking to expand their role in management

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OVERVIEW: The authors review the primary and secondary risk factors for osteoarthritis (OA), its pathophysiology and epidemiology, the evidence-based approaches to slowing progression, and the role of nurses encountering OA in primary care and other settings.

KEYWORDS: management, osteoarthritis, risk factors

The most common type of arthritis in the United States,1,2 osteoarthritis (OA) is a complex disease involving biomechanical, genetic, and environmental processes. Of the many risk factors for OA, genetics and aging are the most significant contributors to the primary form of the disease, while obesity and injury are the most common secondary factors. Its manifestations range from asymptomatic joint changes seen on X-ray to severe disability. While the most common problems with OA are joint pain and functional impairment, the risk factors, clinical manifestations, and radiographic findings vary for different joints. The course of OA is extremely variable, and its outcome is difficult to predict.

Although no approved disease-modifying drugs or other curative interventions are available, certain measures can have a positive impact on pain, function, and quality of life in people with OA. We’ll review the factors involved in the development of OA and the evidence-based measures that can slow its progression and consider the roles of nurses encountering OA in primary care and specialty settings.

Pathophysiology, Epidemiology, and Risk of OA

An understanding of the pathophysiology and epidemiology of OA is required to assess and manage risk factors and implement preventive measures (see Table 1).3,4 The pathologic processes behind OA result in cartilage loss and bony changes in diarthrodial joints (also called synovial joints), the freely movable unions between bones that have hyaline cartilage on their articulating surfaces.2 The joint is surrounded by a capsule and lined with a synovial membrane, which produces synovial fluid.

Hyaline cartilage consists of a matrix that includes protein and sugar compounds (proteoglycans and glycosaminoglycans such as chondroitin sulfate, keratan sulfate, and hyaluronic acid) synthesized by chondrocytes (cartilage-forming cells); it’s composed of the collagen fibers that give the cartilage structure. The glycosaminoglycan matrix of cartilage regenerates every one to three months, while the collagen structural elements are replaced even more slowly.

Synovial fluid nourishes the chondrocytes and lubricates the joint. Hyaline cartilage has no blood supply, significantly limiting its reparative, restorative capacity. OA affects the degradation–synthesis cycle of the chondrocytes, as well as of the extracellular matrix and the subchondral bone, which in turn leads to softening, fibrillation, ulceration, and loss of cartilage. OA bony changes include fractures of subchondral trabecular (spongy) bone, marginal osteophytes (bone spurs), and subchondral cysts. Hyaline cartilage has no nerves, and the pain that accompanies OA arises from the subchondral trabecular bone, the joint capsule, and the periarticular tissues. Synovial inflammation may be present, in part because of cartilage debris, which may result in joint effusion (swelling). Periarticular structures, especially the muscles, are also affected.2

While any diarthrodial joint can be involved in OA, the distal and proximal interphalangeal joints of the hands are the most commonly affected, followed by the knees and the hips.3,5 A diagnosis of OA is determined by joint...
symptoms, including pain and functional impairment or joint pathoanatomic changes, or both. A significant problem in addressing risk and progression of OA is the wide spectrum of clinical features that depend in part on demographic factors and the joint affected. Although epidemiologically OA is often described by a single joint region, in reality patients often have OA in multiple joints.

The American College of Rheumatology and other groups have developed criteria for diagnosing OA in the knee, hip, and hand; however, diagnosis is challenged by the limitations of X-ray findings in the early stages of OA and the fact that such findings do not correlate well with clinical symptoms. For example, estimates of the prevalence of knee OA vary widely according to whether clinical or radiographic criteria are used and the characteristics of the cohort studied. Computed tomography and magnetic resonance imaging can more readily detect cartilage and bony changes, but criteria for defining OA using them have not yet been established.

Data on the prevalence of OA are based primarily on X-ray surveys and self-report questionnaires. It’s estimated that 27 million people over the age of 25 in the United States have OA. The Centers for Disease Control and Prevention predicted in 2006 that 67 million people, or one-quarter of the U.S. adult population, will be diagnosted with some form of arthritis by 2030. Although uncommon under the age of 40, the prevalence of radiographic OA in all joints increases with age. People—including the aging baby boom population—are living longer now, so the burden of OA is rising sharply. Radiographic screening studies have demonstrated that more than 80% of people over the age of 55 have OA X-ray changes in some joint. Between 1993 and 2006, the prevalence of OA increased by 76%, with a 167% increase in the 45-to-64-year-old age group.

With aging, changes in the hyaline cartilage matrix and decreases in its water content influence its biomechanical properties and reduce its shock-absorbing capacity. Pathologic abnormalities found in the cartilage in OA patients significantly differ from the changes in the matrix and collagen composition seen with aging. In addition, degradative enzymes and cytokines are produced by chondrocytes and the synovium of OA patients, which can further damage the cartilage. Although the cartilage and subchondral bone make attempts at repair, the body is not able to reverse the OA disease process.

Genetic factors play a major role in the expression of OA; however, the influence of heredity varies in different joints. Susceptibility to OA has been investigated by genome-wide linkage of families with OA and candidate gene association studies in unrelated individuals. No specific genes for predicting OA have been identified yet, but those that regulate the synthesis of the various components of cartilage rather than coding for specific structural proteins offer the greatest promise. Current evidence suggests that the influence of heredity on OA development is greater in women than in men—as high as 60% for hand and hip OA, but considerably lower for knee OA (estimates range from 10% to 39%). In several epidemiologic studies, the influence of inheritance in knee OA was too low for linkage information to be determined.

### Secondary Risk Factors

In addition to age, sex, and genetics, a variety of secondary factors can influence load distribution across a joint.

#### Obesity

The relationship among obesity, joint pain, functional impairment, and physical activity levels is complex. Obesity, which now affects 33.8% of adults ages 20 years and older in the United States, is a significant risk factor for the development and progression of OA, especially in the knee but also in other joints. Increased load that obesity places on joints can exacerbate the pain and functional limitations of OA. Because of the physics of knee movement, activity magnifies the load across the knee from upper-body weight by three to seven times. Therefore, weight loss in obese patients, especially if accompanied by increased physical activity, can improve physical function and quality of life. Thirty percent of women with body mass indexes (BMIs) of less than 25 will have knee OA during their lives, compared with 60% with BMIs of 30 or higher. Weight change can have a significant modifying effect: women who lost just two points in BMI in 10 years had a 35% reduction in risk of developing symptomatic knee OA.

Being overweight may also play a role in OA in other joints, including the hips, hands, and shoulders, although in those cases the role is not well defined. For example, one study showed a moderate relationship between obesity

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**TABLE 1. RISK FACTORS FOR OSTEOARTHRITIS**

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<th>Primary (systemic) factors</th>
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<td>Genetics</td>
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and the development of hip OA, but only limited evidence of an association between obesity and bilateral hip OA.16 OA in these joints may be related to mechanical factors, abnormalities in intermediary metabolism in adipose tissue, or the production of cytokines.17

Physical activity. Light or moderate physical activity and exercise that do not cause pain or injury do not increase the risk of OA.18 Female runners and tennis players are three times more likely to develop tibiofemoral and patellofemoral knee OA than are age-matched controls.19 The risk of developing OA of the shoulder is increased by participation in overhead sports activities, while OA of the elbow is associated with weight lifting and sports involving throwing. Other joints can also be affected by OA, and significant trauma or injury to those joints is frequently a major factor. Examples include the ankle and foot in ballet dancers, the shoulder after rotator cuff injury, and the metacarpophalangeal joints in workers using jackhammers.17,20,21

A knee injury that prevents unaided walking for more than a week is a strong predictor of knee OA. Such injuries often involve rupture of the anterior cruciate ligament (ACL), which may be associated with a tearing of the meniscus or the medial collateral ligament. Both ACL damage and meniscal tears are strongly linked to early OA development.22,23 Because the incidence of ACL injury is two to eight times higher among female compared with male athletes, the rising number of women participating in sports can be expected to result in higher rates of knee OA in women.22,23 Early ACL reconstruction with meniscal preservation may help to prevent knee OA. The poorest postoperative outcomes have been seen following meniscectomy.3,5

Occupation. The influence of occupation on the development of OA is unclear and seems quite variable.24,25 Occupations that require repetitive overuse of joints, especially with impact loading, causes high biomechanical stresses and may predispose to OA. Repeated crouching, kneeling, squatting, stair climbing, and heavy lifting can result in abnormal loading across the joint and articular damage. Few occupational studies have included women, but the evidence suggests that the risk factors are similar in both sexes.24

Mechanical environment. Knee alignment has a strong impact on joint load distribution.11 Varus alignment is associated with progressive medial compartment OA (wearing of the medial meniscus) and valgus alignment with lateral compartment OA (wearing of the lateral meniscus); the degree of alignment correlates with the magnitude of the joint-space narrowing. People with varus knee alignment have a greater loss of function when walking than those with valgus alignment do.

A decrease in proprioception, which helps to establish and maintain joint stability, is present in both the involved and the uninvolved knees of patients with unilateral knee OA and may precede overt pathologic changes in the joints.5,26 Laxity of the knee ligaments is another contributing factor. With aging, varus–valgus laxity increases more often in women than in men, and it may also precede knee OA. Patients with unilateral knee OA have greater varus-valgus laxity in both the involved and the contralateral knee. Forefoot varus alignment may increase the likelihood of hip OA.1,3,5

Muscle strength. Muscles are the major supporting structures for joints and are critical for joint movement and stability.27 Studies have shown that quadriceps weakness commonly accompanies knee OA.28 This finding was attributed to disuse atrophy secondary to knee pain and abnormal biomechanics, but extensor weakness is also present with asymptomatic tibiofemoral OA, suggesting that quadriceps weakness is a risk factor for knee OA development. Individuals who developed knee OA during follow-up had 18% less quadriceps strength at baseline than those who did not. The influences of muscle strength and afferent sensory dysfunction on the progression of knee OA are uncertain.27

Other factors may also influence the risk and progression of OA.4 Estrogen has a physiologic effect on cartilage and bone metabolism: while both estrogen deficiency and OA occur with advancing age, observational studies on the influence of estrogen, including with hormone replacement therapy, have been inconclusive.3 It has been suggested that vitamin D and K deficiencies contribute to the development of OA.4 Inflammatory arthritides such as rheumatoid arthritis and systemic lupus erythematosus can injure cartilage and bone and result in secondary OA changes.17,24

Evidence-Based Management

While there is no cure for OA, the American College of Rheumatology (ACR), the European League Against Rheumatism, and others have established nonpharmacologic and pharmacologic management guidelines.29,30 Therapy is primarily directed at controlling pain and maintaining functional activity. The Osteoarthritis Research Society International (OARSI) has published evidence-based recommendations for the management of knee, hip, and hand OA.29,30 Of the nonpharmacologic therapies, relief has been demonstrated for self-management, education, exercise, acupuncture, and weight loss. Included in these measures are instruction on disease pathophysiology; use of self-management techniques such as thermal modalities and activity modification; regular telephone contact; referral to physical therapy; walking aids; knee braces; aerobic, muscle-strengthening, and water-based exercises; and footwear and insoles.

Pharmacologic therapies for OA also focus on pain management since no medications can significantly influence the disease’s course. The 2008 OARSI pharmacologic recommendations29 included acetaminophen, cyclooxygenase-2 nonselective and selective nonsteroidal antiinflammatory drugs (NSAIDs), topical NSAIDs and capsaicin, glucosamine sulphate, chondroitin sulfate, intraarticular injections of corticosteroids and hyaluronates, and the use of opioid analgesics for refractory pain. The ACR guidelines recommend acetaminophen and NSAIDs as primary agents for OA. Studies have suggested that for analgesic efficacy in OA, NSAIDs have a therapeutic advantage over acetaminophen, but NSAIDs can cause adverse gastrointestinal and cardiovascular effects.

The response to different NSAIDs is variable and clinically unpredictable. Lower NSAID doses are primarily analgesic, while larger doses are required for antiinflammatory effects. A therapeutic trial of two weeks or longer...
The Nurse’s Role in Managing OA Risk and Progression

The role of the nurse in managing OA risk and progression has been evolving, for example, in primary care, teaching, research, and other tasks and settings.32–35 (See Table 2.) With the projected increase in OA prevalence forewarning of a much larger patient population, it will be essential for facilities to have coordinated OA teams staffed with trained professionals. But there is a critical shortage of these practitioners: there are roughly 500 nurse members of the Association of Rheumatology Health Professionals, a division of the ACR, and 415 physicians were in rheumatology training in 2009. Furthermore, there are limited data on specific nursing interventions for OA and their effectiveness in primary care, rheumatology, and orthopedic surgery practice settings.33,34 Nurses assist in diagnosing and assessing the disease's functional and psychosocial impacts, provide medication and pain management,35 monitor disease progress,36 educate patients, and coordinate care with other providers (physical, occupational, and psychosocial therapists).39 Understanding the clinical manifestations of and the diagnostic criteria for OA provide the foundation for these activities.

Patient evaluation for OA should begin with taking a targeted history of the problem. The circumstances surrounding the onset of the joint problem, including precipitating factors such as trauma, and the problem’s duration are first determined, along with the impact it has on activities of daily living such as walking, bending, performing fine motor activities, and climbing stairs. Additional information, as stipulated by the diagnostic OA criteria being used, such as those of the ACR, should also be obtained.

After a diagnosis of OA, education will help patients compensate for and manage its effects by adapting their usual activities; using assistive devices; and complying with medication, exercise, and weight management programs.40–43 Nurses can impart this information to patients in formal discussions or educational materials and by giving them advice on using electronic resources to find more information. An accurate assessment of the patient’s educational level must be made before an effective program can begin. According to the 2003 National Assessment of Adult Literacy, 14% of the population in the United States has “below basic” health literacy, which limits their ability to understand health information and make informed health-related decisions.44

Studies in primary care settings demonstrate that nurses are key assessors of and coordinators in managing their OA patients’ care.37,39 There are programs and protocols designed specifically for OA that define screening tools for uniformly collecting patient data,46 provide models for treatment, and promote the integration of interventions among various health care providers.45–47 Self-report instruments such as the Western Ontario and McMaster Universities Osteoarthritis Index, the Health Assessment Questionnaire, and the Medical Outcomes Study Short Form 36-Item Health Survey are validated tools that can provide important information for assessing outcomes in OA.48 Helping patients accept their disease and encouraging them to actively participate in the care process are essential. It is also important to identify patients who are hiding their symptoms from health care professionals and trying to self-manage their problems. In addition to all of these responsibilities, orthopedic nurses must also actively engage in pre- and postoperative care.48–50

**TABLE 2. THE NURSE’S ROLE IN ASSESSING FOR RISK AND MANAGING PROGRESSION OF OA**

| • Evaluation, including a targeted history and physical examination along with laboratory and joint imaging studies to establish diagnosis |
| • Education of the patient on the manifestations of OA and its management |
| • Risk management for medications and other management activities |
| • Care coordination among the patient and health care professionals |
| • Compliance strategies, including proper medication use, weight reduction, and exercise |

**REFERENCES**