Osteoarthritis and Knee Pain

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Osteoarthritis (OA), also known as degenerative joint disease, is the most common form of arthritis. OA is a clinical syndrome in which low-grade inflammation results in pain in the joints. This pain is caused by abnormal wearing of the cartilage that acts as a cushion inside joints or by destruction or decrease of synovial fluid that lubricates those joints. Once the articulating surfaces become less protected by cartilage, the patient experiences pain with weight-bearing activities such as standing, walking, and going up and down stairs. Due to decreased movement because of pain, regional muscles may atrophy, resulting in weakness and loss of stability. Ligaments might become lax, increasing joint deformity, instability and pain.

OA affects approximately 21 million people in the U.S. and accounts for 25 percent of visits to primary care physicians and half of all nonsteroidal anti-inflammatory drug prescriptions. Projections are that 80 percent of the population will have radiographic evidence of OA by age 65, although only 60 percent will be symptomatic. OA accounts for more mobility disability in the elderly than any other disease.

OA often is thought of as a disease that affects the aged of our society, but this is not necessarily true. There are people in their 80s and 90s who have no clinical or functional signs of the disease. To understand this phenomenon, one must know the difference between primary and secondary OA. Primary OA is a chronic degenerative disorder related to, but not caused by, aging.

As people age, the water content of cartilage decreases as a result of reduced proteoglycan content, causing the cartilage to become less resilient. The collagen fibers of the cartilage become more easily degraded and degenerated. As the breakdown products of the cartilage are released into the synovial space, mild inflammation results and the cells lining the joint attempt to remove them. New bone outgrowths, called spurs or osteophytes, can form on the margins of the joints. These bony changes, together with inflammation, can lead to the signs and symptoms associated with OA, particularly pain and disability. Secondary OA is initiated by various disease processes. Although the cause is different with secondary OA, the patient presentation and clinical picture are the same as primary OA.
Some of the more common causes include:

- congenital disorders (joint dysplasia, unequal leg length and improperly formed joint structure);
- endocrine conditions (diabetes, hypothyroidism and hyperparathyroidism);
- inflammatory joint diseases (gout and rheumatoid arthritis);
- changes associated with post traumatic injury (macrotrauma and microtrauma);
- infections;
- ligamentous instability;
- obesity; and
- postural stress (muscle imbalances, pes planus and hyperpronation).

Diagnosis routinely is made after a careful history and physical examination accompanied by X-rays. The standard loss of cartilage, subchondral sclerosis, subchondral cysts, narrowing of the joint space and bone spur formation clearly are identified on standard X-ray studies. While X-ray visualizes mainly bone, MRI studies are capable of visualizing all of the structures within the knee. CT studies also visualize mainly bone, but in greater detail than plain films.

Pain is the main symptom of OA. The quality of OA pain often is described as a sharp ache or a burning sensation in the related muscles and tendons. The pain associated with OA often results in decreased function, which further results in joint stiffness. There often is a cracking sound associated with movement in joints afflicted with OA. This noise is called crepitus and represents the roughening of the articulating surfaces. Occasionally, the joint might fill with fluid and it’s a common experience for patients with OA to have increased pain in humid weather. Eventually, the effects of OA cause the joint to appear larger, stiffer and more painful and patients generally feel worse as the joint is worked throughout the day. This is a distinguishing feature from rheumatoid arthritis. OA also is the most common cause of water on the knee.

OA typically affects joints in a non-uniform manner; the most frequently affected component of the knee is the medial compartment. This is due to the high medial-compartment forces generated during weight-bearing activities. Walking is the most common daily activity that exerts the greatest repetitive forces through the knee. Normal forces acting on the leg produce a varus torque. This varus torque is directly associated with the compressive force across the medial aspect of the knee, which is nearly two-and-a-half times the force through the lateral aspect of the knee. Varus torque is believed to be responsible for the progression of knee OA and is supported by data from both animal and clinical studies.
An animal study showed that excessive varus torque directly induced osteoarthritic changes. Clinical surgical data support the proposal that reducing the knee varus torque effectively decreases both knee pain and disease progression.

Sasaki and Yasuda first reported the potential of a laterally wedged insole in treating osteoarthritis of the knee. They showed that when subjects stood on a board that was angulated five degrees laterally, there was a change in the spatial position of the lower limb. This change caused them to conclude that loading of the medial knee joint would be reduced. Given the importance of varus torque in the progression of medial-compartment knee osteoarthritis, conservative means to reduce this torque constitute a logical treatment approach.

The use of orthotic devices with a six-degree lateral-wedge insole has been found to make knee joint varus movement significantly smaller, whereas the subtalar joint valgus movement was significantly greater, thereby creating a lateral shift in the center of pressure during the stance phase of gait. This alteration in biomechanics could explain a significant reduction in knee joint varus moment gained by using orthotics with a lateral-wedge insole. The increased valgus angle of the subtalar joint contributed to the reduced load in the medial compartment of the knee joint. This results in a reduction of knee pain in OA patients.

Improved mobility in patients who wear orthotics has been commensurate with those patients who have received arthroplasty surgery. A loss of joint space in the medial compartment alters the biomechanics, causing a continual progression in the destructive effects of arthritis on the body. Patients with OA of the knee would clearly be helped by the use of orthotics, which would interrupt this vicious cycle of pain and continued knee joint deterioration. There is ample evidence to suggest a simple lateral-wedge orthotic is useful in interrupting and slowing the progression of OA and its associated pain and disability.

Besides orthotics, there are several other approaches to treating OA that share the common goal of decreasing pain and increasing function. These treatment options are:

- Encourage weight control. This can relieve joint stress.
- The use of support devices, including knee braces, a cane or walker. Decreasing the compression and strain of weight bearing on the knee can help reduce symptoms and possibly slow the continuing degenerative process.
- Regular exercise such as walking or swimming. Regular exercise will support the goal of weight control and offer other benefits specifically to increase lower-extremity strength, encourage more
normal range of motion and contribute an aerobic component into the treatment plan. Close-chained exercise in the form of mini squats and leg presses will decrease shearing forces on the knee and increase the stabilizing forces.

- Low-level aerobic conditioning is easier for some patients to maintain than moderate forms of exercise. If there is discomfort performing this activity, aquatics are an alternative. Going to a pool where body weight would be reduced by buoyancy might decrease the joint compression sufficiently to allow the patient to receive a good aerobic workout without discomfort and still receive all the benefits.

- Recommending dietary supplementation; a common conservative treatment approach that might offer some additional patient benefits. Some of these more commonly recommended supplements include: omega-3 fatty acid, boswella, bromelain and antioxidants (vitamins C and E may provide pain relief). Vitamin D and calcium are frequently recommended for bone strength. Standard daily doses of calcium are 1,000-1,200 mg. The current guideline for vitamin D is 400 IU per day. These doses may vary depending on individual circumstance.

- Reducing saturated fat intake.

Heat-producing modalities (especially moist heat) may be beneficial in treating the pain, increasing circulation and decreasing the stiffness that accompanies OA of the knee. Electric stimulation, lasers and soft-tissue techniques will help decrease pain and increase mobility, thereby allowing patients to increase their functional capacity, leading to improved health and wellness.

These therapies should be applied on a patient-by-patient basis only after careful and complete evaluation and a thorough decision-making algorithm has been accomplished.

References


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