OPINIONS   PEOPLE   YOUR PRACTICE   MED STUDENTS   ARCHIVE   CLASSIFIEDS   CME   BLOGS

Home >> Back to the October 2010 Issue

Clinical features and pathogenetic mechanisms of osteoarthritis of the hip and knee

Issue: BCMJ, Vol. 52, No. 8, October 2010, page(s) 393-398 Articles

Manal Hasan, MBBS, MD, Rhonda Shuckett, MD, FRCPC, Diplomate ABIM

Understanding how osteoarthritis develops is critical to treating this disabling disease.

Osteoarthritis is a noninflammatory form of arthritis that accounts for 25% of visits to primary care physicians. When osteoarthritis affects the hip and knee, it can lead to major disability and compromised quality of life. Diagnosis relies on clinical symptoms, physical findings, and radiographic findings. The interplay between mechanical and systemic factors such as congenital abnormalities, obesity, and malalignment may predispose individuals to osteoarthritis of the hip and knee. We must identify these factors and the underlying causes of osteoarthritis if we are to develop more progressive early interventions for this common affliction.

Osteoarthritis (OA) is a noninflammatory form of arthritis. A common misconception is that OA is due solely to wear and tear, since OA is typically a disease of persons in the sixth decade and beyond. "Degenerative arthritis" is often used as a synonym for OA, but OA is not the result of a bland degenerative process; rather, OA involves both degenerative and regenerative processes.

OA is common and serves as the main source of chronic joint complaints in adults. The morbidity conferred by OA of the knee and hip in an ever-aging population is major. Its high prevalence and huge impact on quality of life demand that we engage in better understanding of OA by considering diagnostic, epidemiological, clinical, and radiographic features. An understanding of how OA is classified and OA risk factors is also critical.

Diagnosis and epidemiology

The diagnosis of OA relies on clinical symptoms, physical findings, and radiographic findings. Not all persons who have radiographic OA have clinical disease. Conversely, not all persons who have joint pain demonstrate plain radiographic findings of OA. Thus, there is often discordance between X-ray findings and symptoms of OA.[1]

OA accounts for 25% of visits to primary care physicians, and 50% of NSAID prescriptions.[2] It is estimated that up to 80% of the population will have radiographic evidence of OA by age 65, with 60% of those showing symptoms and thereby having clinical OA.[3]

Another study found that by age 70 to 74 years, about 33% of men and 40% of women will have OA with clinical and X-ray features.[4] The lifetime risk of developing symptomatic knee OA is about 45%, rising to 66% in obese persons. While there is variation in these numbers, it is clear that the morbidity and disability conferred by OA of the hip and knee is enormous and demands our attention.[5]

Symptoms and physical findings

The main symptoms of OA of the knee or hip are pain, stiffness, and altered function. Initially this tends to be worse with weight bearing and ambulation. Eventually this can progress to pain day and night once cartilage loss leads to bone-on-bone contact.

True hip pain is felt in the groin most commonly, but can also present in the buttock and often down the anteromedial thigh to the knee. Not uncommonly, patients may present solely with knee pain when the problem is in the hip. Pain arising from osteoarthritis of the knee is felt right around the knee joint, and unlike pain caused by hip OA, this pain does not typically radiate.

In contrast to inflammatory arthritides such as rheumatoid arthritis, with their prolonged morning stiffness and worsened pain in the morning, OA tends to worsen as the day progresses. The stiffness in OA is termed "inactivity stiffness" and contrasts with the prolonged "morning stiffness" of rheumatoid arthritis. Inactivity stiffness in osteoarthritic lower limb joints lasts about 5 to 10 minutes and occurs when the patient gets up and bears weight after prolonged immobility.

On physical examination, a small effusion with a fluid bulge sign can be present in OA of the knee. Larger effusions can occur but are less frequent than in the inflammatory arthropathies. Synovial fluid analysis after aspiration of an OA knee effusion reveals that the fluid is thick and viscous with a low synovial white blood cell count, most of which are mononuclear cells.

On examination, there may be cartilaginous crepitus or a crackling feeling on palpation of the knee with motion. Eventually there may be coarse bone-on-bone crepitus whereby the opposing bone ends, denuded of cartilage, seem to grate against one another. There is often a loss of range of motion of the involved knee or hip, particularly with progression of OA.

Loss of cartilage of the knee can lead to malalignment of the leg with a varus deformity or bow-legged positioning of the leg being evident. This angulation of the knee applies to medial compartment OA of the knees.

Less commonly, patients may present with a valgus or knock-knee deformity, indicative of more advanced disease in the lateral compartment of the knee. On occasion, and much less commonly, patients may present with isolated OA in the patellofemoral joint, which can of itself be very symptomatic.

In the case of the hip, a true capsular pattern of limitation is found with groin or buttock pain (or both) and particular pain with internal rotation of the hip. Flexion deformity of the involved hip can be present with advanced OA. Patients will often walk with a limp, and a waddling Trendelenburg gait may be evident in late stages.

X-ray findings

THIS ARTICLE

  Download PDF(0.46 MB)
  Printer-friendly version
  Create Citation
  Permissions

DISCUSSION

  Submit a Letter

  Add Comment   0

SHARE THIS ARTICLE

Standard knee X-rays should include a standing anteroposterior (AP) view of both knees, plus lateral views. In patients with suspected posterolateral OA with a mild valgus deformity, a 30 degree flexed standing posteroanterior (PA) view with the beam directed 15 degrees from cephalad to caudad may be valuable in showing the disease in the posterior aspect of the lateral compartment of the knee.[6,7]

In early cases, a standard standing AP view may appear normal or indicate very mild OA, whereas the standing flexed PA view may show bone-on-bone contact. Patellofemoral OA of the knee cap is also a common finding, best diagnosed on a skyline X-ray view.

X-rays of the hips to evaluate for OA should include a standing AP pelvis view and frog-leg views of the suspected hip joint. It is important to always order standing X-rays of both knees in the case of suspected knee OA and an X-ray of the pelvis and not just the affected hip in the case of suspected hip OA. This will allow for comparison between sides and improves the ability to diagnose mild to moderate disease.

On plain X-ray evaluation, loss of the radiolucent cartilage, termed joint space narrowing, is seen in OA. In the hip joint the joint space narrowing tends to be more in the weight-bearing superolateral aspect of the joint, again highlighting the role of mechanics in OA (Figure 1).

However, there are different patterns of OA of the hip, and it is possible to get more central wear, particularly in patients with deep sockets or protrusio acetabuli. In the knee, main involvement is often in the medial joint compartment (Figure 2), but involvement of other compartments or of the entire joint is also common.

On plain X-ray of an osteoarthritic joint, in addition to joint space narrowing, there tends to be subchondral sclerosis or an appearance of whitening of the subchondral bone. Osteophytes, which reflect a regenerative process with formation of fibrocartilaginous extensions or hooks at the joint margins, are common.

Interestingly, the presence of osteophytes in one compartment, such as the lateral compartment in a patient with medial compartment OA, is not indicative of disease in that compartment. It is simply indicative of the body's reparative response to the abnormal stresses and presence of disease in the medial compartment.

The identification of OA on plain X-rays means there is already full thickness cartilage loss and even bone-on-bone contact. These radiographic findings occur relatively late in the course of OA. It would be ideal to be able to identify OA before gross changes are apparent on radiographs.

Earlier OA detection is important in identifying disease before the progressive bone-on-bone stage. Joint ultrasound has been applied in studies to identify OA earlier, but this is more a research tool than a routine clinical application. MRI has emerged as an excellent modality for detection of OA when the plain radiographs indicate no disease or mild disease, and the patient's symptoms are out of keeping with the apparent severity of disease. MRI can detect large focal articular cartilage lesions that cannot be detected on plain films.[6-8]

Classification of OA

Traditionally OA has been classified as primary or secondary (Table 1).[9] Primary OA denotes generalized or erosive OA with no identifiable cause. Secondary OA denotes OA caused by an underlying condition, including those caused by inflammatory diseases, trauma, and mechanical factors.

In a large series of cases of so-called primary osteoarthritis of the hip, some underlying mechanical developmental variation could be found in most cases to account for the onset of the disease.[10] For instance, the subtle presence of a shallow cup of the hip, called acetabular dysplasia, is a common precursor to OA of the hip.

In middle-aged men, femoroacetabular impingement (FAI) is thought to be the most common cause of OA of the hip. FAI of the pincer type occurs most often in middle-aged women. On occasion, patients may present with symptoms of impingement prior to the development of advanced OA.

It thus appears that the term "primary or idiopathic OA" is probably a misnomer as it applies to the hip or knee, and that if we look hard enough an underlying structural cause will often be apparent.

In the 1970s Mitchell and Cruess proposed a more pathogenetic classification of OA (Table 2). This classification system assumes that osteoarthritis can arise from an intrinsic problem of the cartilage as encountered after years of chronic inflammatory arthritis.[11] Thus, OA can occur with (A) normal force on abnormal cartilage.

Alternatively, it can occur with (B) abnormal concentrations of force on normal cartilage. This would implicate mechanical aberrations such as malalignment, the post-meniscectomy knee, or a cruciate deficient knee. The abnormally formed hip mentioned above would fall into this category as well.

Mitchell and Cruess's classification system also includes situations where there is (C) stiffened subchondral bone, as in the case of the rare Paget disease, which does indeed predispose to OA of an involved joint. Alternatively, they describe situations where (D) weakened subchondreal bone, as in avascular necrosis, predisposes to OA.

Risk factors for OA

OA is best viewed as the end result of an interplay between local and systemic factors. Such factors are well outlined in the classification schema of mechanical factors proposed by Mitchell and Cruess. Several local systemic factors may be operative in predisposing patients to OA of the hip or knee.

Gender and the estrogen connection

Women are more likely than men to have OA, be it generalized OA of the hands or OA of the hips and knees.[12] The increase in OA in menopausal women has led to numerous investigations into the relationship between hormonal factors and OA. The results have been conflicting and inconclusive.[13,14] Clearly, other health issues are of concern when determining whether hormone replacement therapy is to be considered in the postmenopausal patient.

Congenital/developmental abnormalities

Local factors that affect the shape of the joint may increase local stress on cartilage and contribute to the development of osteoarthritis, especially in the hip joint.

As already mentioned, subtle and asymptomatic anatomic variations have been associated with hip osteoarthritis. These include acetabular dysplasia or epiphysiolysis, which are common milder variants of congenital hip dislocation and slipped capital femoral epiphysis, respectively.[10]

Femoralacetabular impingement is gaining increasing recognition as a major structural precursor to hip OA. These are usually asymptomatic before possible progression to OA and can be seen on a screening AP pelvis radiograph. Such pre-symptomatic X-rays, however, are not ordered routinely.

Genetic factors

The strongest association between genetic factors and OA applies to generalized osteoarthritis of the hands. Evidence for a correlation between genetics and knee or hip OA is less conclusive.[15,16]

Physical activity

Although the health of cartilage and other joint tissues requires regular joint loading, excessive loading may contribute to OA. While some studies suggest a strong positive relationship between work-related knee bending exposure and knee

OA, others have failed to find a direct relationship between the presence of knee OA and habitual physical activity or recreational running.[17]

A relationship between heavy manual work, farming in particular, and hip OA was found in different studies, but the association is still considered a weak one.[18]

Although it makes sense that high levels of impact and repeated torsional loading could increase the risk of articular cartilage degeneration, this is not borne out consistently in studies. Still, it would appear prudent to suggest that anyone with a known underlying predisposition to OA, such as abnormal hip or joint anatomy or excessive body weight, avoid repetitive impact-loading activities such as jogging.

Obesity
Every step taken in a normal gait places about three times an individual's body weight on lower limb joints. Thus it should not be surprising that obesity and high body mass index have long been recognized as potent risk factors for OA, especially medial compartment OA of the knee in females.

The Framingham Study found that women who lost about 5 kg had a 50% reduction in the risk of developing new symptomatic knee OA.[19] Weight-loss interventions have been shown to decrease pain and disability in established knee OA.

The Arthritis, Diet, and Activity Promotion Trial showed that weight loss combined with exercise, but not either weight loss or exercise alone, was effective in decreasing pain and improving function in obese elders who already had symptomatic knee OA.[20]

When patients ask their physicians how they can prevent OA of the knees, weight control is paramount. Unfortunately weight loss is challenging in established OA of the knee due to the limited physical activity possible.

The relationship between excess weight and hip OA is less clear. The evidence in hip OA is not as compelling as with knee OA.[21,22]

In addition, there is evidence that obesity predisposes to osteoarthritis in non-weight-bearing joints such as the joints of the hand. Clearly excess weight in a biomechanical sense alone does not explain this finding.

Recent studies have shown that body fat, particularly central fat deposits, are biochemically active and produce substances such as leptin and adiponectin.[23] It has also been shown that leptin can induce the formation of cytokines, such as interleukin-6, which can have a deleterious effect on chondrocytes of the cartilage.

Trauma
In general, there is a paucity of good documentation to support the contention that blunt trauma to a joint increases the risk of future osteoarthritis.

An exception to this is the presence of intra-articular fractures, that is, fractures that extend though the joint line. The disruption of the cartilage and subchondral bone with an intra-articular fracture does portend a heightened risk of OA of the involved joint in future decades.

Trauma of the knees leading to internal knee derangement such as a mensical or major ligamentous tear will predispose to osteoarthritis. In the case of the hip, acetabular labral tears, which can only be seen on MRI combined with an arthrogram, will increase the risk of future OA of the involved hip joint.

An acetabular labral tear is often an indication for hip arthroscopy to trim the torn fragment. Hip arthroscopy is not often done for diagnostic purposes because MRI is so effective at picking up lesions.

It is thought that blunt trauma such as contact with a dashboard in a motor vehicle accident can lead to patellofemoral syndrome and chondromalacia patella. However, whether these pre-OA lesions will progress in future decades to full thickness confluent cartilage loss signifying OA has not been determined.

Alignment, including leg length
Strong evidence suggests that altered mechanics play a role in OA incidence and progression, and recent studies are beginning to isolate specific mechanical factors that may be of particular importance. Such alignment problems include a leg length discrepancy of more than 1 cm, which confers an increased risk of OA of the hip on the long leg side. All patients should be assessed for this.

Leg length measurements include the apparent and the true leg length. To measure leg length, you should have the patient lie flat on his or her back on the examining table and ensure that there is no hip or knee flexion deformity that will challenge accurate leg length measurement.

It is key to place the patient's legs in proper alignment. There should be an equal distance between the medial malleoli of the ankles, and the feet should be centred in a neutral position under the corresponding hips. The apparent leg length is measured from the umbilicus to the medial malleolus on each side. A discrepancy usually signals a scoliosis.

The true leg length is measured from the anterior superior iliac spine to the medial malleolus, and a discrepancy suggests a true variation between the two legs. For a true leg length discrepancy of more than 1 cm, a shoe lift or built-up orthotic that adjusts for half of the leg length difference is typically recommended. For a large discrepancy this may not be readily attainable.

Varus deformities, valgus deformities, and cruciate ligament tears are other factors that can predispose to the development and progression of knee OA. Detailed discussion of such factors is beyond the scope of this article.

Like the medial compartment and the lateral compartment, the patellofemoral compartment of the knee is often afflicted with OA. While injury is a common factor in medial and lateral compartment OA, malalignment is a more common factor in patellafemoral OA. Most cases of chondromalacia patella that result from malalignment are nonprogressive, but some can progress to OA.[24]

Conclusions
OA of the hip and knee is a major health care issue in an ever-aging population. OA of weight-bearing joints confers major disability and compromised quality of life. At this time, medical treatment of OA is not as sophisticated as the treatment of rheumatoid arthritis.

All too often we fail with conservative treatment, and patients with hip and knee OA progress to total joint arthroplasty. Advances in joint replacement seem to overshadow advances in more conservative medical treatment of OA.

The better we understand the underlying causes and mechanisms of OA, the better we will be equipped to develop more progressive early interventions for this common affliction. As Ilardi and Sokoloff, two pioneers in the study of OA, said several decades ago, "Our treatment of osteoarthritis can be no more rational than our understanding of its pathogenesis."[25]

Competing interests
None declared.

References

1. Hannan MT, Felson DT, Pincus T. Analysis of the discordance between radiographic changes and knee pain in osteoarthritis of the knee. J Rheumatol 2000;27:1513-1517.

2. Lawrence RC, Felson DT, Helmick CG, et al. Estimates of the prevalence of arthritis and other rheumatic conditions in the United States. Part II. Arthritis Rheum 2008;58:26-35.

3. Agency for Healthcare Research and Quality. Hospitalizations for osteoarthritis rising sharply. Newswise. www.newswise.com/articles/hospitalizations-for-osteoarthritis-rising-sharply (accessed 27 July 2010).

4. Kopec JA , Rahman MM, Berthelot J-M, et al. Descriptive epidemiology of osteoarthritis in British Columbia, Canada. J Rheumatol 2007;34:386-393.

5. Murphy L, Schwartz T, Helmick CG, et al. Lifetime risk of symptomatic knee osteoarthritis. Arthritis Rheum 2008;59:1207-1213.

6. Leach RE, Gregg T, Siber FJ. Weight-bearing radiography in osteoarthritis of the knee. Radiology 1970;97:265-268.

7. Cibere J. Do we need radiographs to diagnose osteoarthritis? Best Pract Res Clin Rheumatol 2006;20:27-30.

8. Boegard TL, Rudling O, Petersson IF, et al. Correlation between radiographically diagnosed osteophytes and magnetic resonance detected cartilage defects in the patellofemoral joint. Ann Rheum Dis 1998;57:395-400.

9. Brandt KD. Osteoarthritis: Clinical patterns and pathology. In: Kelly W, Harris E, Ruddy S, et al. (eds). Textbook of rheumatology. 2nd ed. Philadelphia: Sauders; 1985:1432.

10. Stulberg SD, Harris WH. Acetabular dysplasia and development of osteoarthritis of the hip. In: Harris WH (ed). The hip. Proceedings of the Second Open Scientific Meeting of the Hip Society. St Louis: CV Mosby; 1974:82.

11. Mitchell NS, Cruess RL. Classification of degenrative arthritis. Can Med Assoc J 1977;117:763.

12. Srikanth VK, Fryer JL, Zhai G, et al. A meta-analysis of sex differences in prevalence, incidence and severity of osteoarthritis. Osteoarthritis Cartilage 2005;13:769-781.

13. Nevitt MC, Felson DT, Williams EN, et al. The effect of estrogen plus progestin on knee symptoms and related disability in postmenopausal women: The Heart and Estrogen/Progestin Replacement Study, a randomized, double-blind, placebo-controlled trial. Arthritis Rheum 2001;44:811-818.

14. Hannan MT, Felson DT, Anderson JJ, et al. Estrogen use and radiographic osteoarthritis of the knee in women. The Framingham Osteoarthritis Study. Arthritis Rheum 1990;33:525-532.

15. Zhai G, Ding C, Stankovich J, et al. The genetic contribution to longitudinal changes in knee structure and muscle strength: A sibpair study. Arthritis Rheum 2005;52:2830-2834.

16. Lian K, Zmuda JM, Nevitt MC, et al. Type I collagen alpha1 Sp1 transcription factor binding site polymorphism is associated with reduced risk of hip osteoarthritis defined by severe joint space narrowing in elderly women. Arthritis Rheum 2005;52:1431-1436.

17. Lane NE, Michel B, Bjorkengren A, et al. The risk of osteoarthritis with running and aging: A 5-year longitudinal study. J Rheumatol 1993;20:461-468.

18. Maetzel A, Makela M, Hawker G, et al. Osteoarthritis of the hip and knee and mechanical occupational exposure—A systematic overview of the evidence. J Rheumatol 1997;24:1599-1607.

19. Felson DT, Zhang Y, Anthony JM, et al. Weight loss reduces the risk for symptomatic knee osteoarthritis in women. The Framingham Study. Ann Intern Med 1992;116:535-539.

20. Messier SP, Loeser RF, Miller GD, et al. Exercise and dietary weight loss in overweight and obese older adults with knee osteoarthritis: The Arthritis, Diet, and Activity Promotion Trial. Arthritis Rheum 2004;50:1501-1510.

21. Heliovaara M, Makela M, Impivaara O, et al. Association of overweight, trauma and workload with coxarthrosis. A health survey of 7,217 persons. Acta Orthop Scand 1993;64:413-518.

22. Karlson EW, Mandl LA, Aweh GN, et al. Total hip replacement due to osteoarthritis: The importance of age, obesity, and other modifiable risk factors. Am J Med 2003;114:93-98.

23. Simopoulou T, Malizos KN, Iliopoulos D, et al. Differential expression of leptin and leptin's receptor isoform (Ob-Rb) mRNA between advanced and minimally affected osteoarthritis cartilage; effect on cartilage metabolism. Osteoarthritis Cartilage 2007;15:872-883.

24. Hunter DJ, Zhang YQ, Niu JB, et al. Patella malalignment, pain and patellofemoral progression: The Health ABC Study. Osteoarthritis Cartilage 2007;15:1120-1127.

25. Ilardi CF, Sokoloff L. The pathology of osteoarthritis: Ten strategic questions for pharmacologic management. Semin Arthritis Rheum 1981;11:3-7.

Dr Hasan is a rheumatology fellow in the Division of Rheumatology at the University of British Columbia, sponsored by the Kingdom of Saudia Arabia. Dr Shuckett is a clinical associate professor in the Division of Rheumatology at UBC.