Osteoarthritis
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Osteoarthritis (OA) also known as degenerative arthritis or degenerative joint disease or osteoarthrosis, is a group of mechanical abnormalities involving degradation of joints,[1] including articular cartilage and subchondral bone. Osteoarthritis is not an inflammatory disease even though the name suggests one. Symptoms may include joint pain, tenderness, stiffness, locking, and sometimes an effusion. A variety of causes—hereditary, developmental, metabolic, and mechanical deficits—may initiate processes leading to loss of cartilage. When bone surfaces become less well protected by cartilage, bone may be exposed and damaged. As a result of decreased movement secondary to pain, regional muscles may atrophy, and ligaments may become more lax.[2]

Treatment generally involves a combination of exercise, lifestyle modification, and analgesics. If pain becomes debilitating, joint replacement surgery may be used to improve the quality of life. OA is the most common form of arthritis,[2] and the leading cause of chronic disability in the United States.[3] It affects about 1.9 million people in Australia,[4] 8 million people in the United Kingdom and nearly 27 million people in the United States.[5]

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Osteoarthritis

Classification and external resources

MRI of osteoarthritis in the knee, with characteristic narrowing of the joint space.

ICD-10


ICD-9


OMIM

165720 (http://omim.org/entry/165720)

DiseasesDB

9313 (http://www.diseasesdatabase.com/ddb9313.htm)

MedlinePlus

000423 (http://www.nlm.nih.gov/medlineplus/ency/article/000423.htm)

eMedicine

Signs and symptoms

The main symptom is pain, causing loss of ability and often stiffness. "Pain" is generally described as a sharp ache or a burning sensation in the associated muscles and tendons. OA can cause a crackling noise (called "crepitus") when the affected joint is moved or touched and people may experience muscle spasms and contractions in the tendons. Occasionally, the joints may also be filled with fluid.[6] Some people report increased pain associated with cold temperature, high humidity, and/or a drop in barometric pressure, but studies have had mixed results.[7]

OA commonly affects the hands, feet, spine, and the large weight bearing joints, such as the hips and knees, although in theory, any joint in the body can be affected. As OA progresses, the affected joints appear larger, are stiff and painful, and usually feel better with gentle use but worse with excessive or prolonged use, thus distinguishing it from rheumatoid arthritis.

In smaller joints, such as at the fingers, hard bony enlargements, called Heberden's nodes (on the distal interphalangeal joints) and/or Bouchard's nodes (on the proximal interphalangeal joints), may form, and though they are not necessarily painful, they do limit the movement of the fingers significantly. OA at the toes leads to the formation of bunions, rendering them red or swollen. Some people notice these physical changes before they experience any pain.

OA is the most common cause of a joint effusion of the knee.[8]

Causes

Damage from mechanical stress with insufficient self repair by joints is believed to be the primary cause of osteoarthritis.[9] Sources of this stress may include: misalignments of bones caused by congenital or pathogenic causes; mechanical injury; excess body weight; loss of strength in the muscles supporting a joint; and impairment of peripheral nerves, leading to sudden or uncoordinated movements.[9] However exercise, including running in the absence of injury, has not been found to increase the risk.[10] Nor has cracking one's knuckles been found to play a role.[11]

Primary
A number of studies have shown that there is a greater prevalence of the disease among siblings and especially identical twins, indicating a hereditary basis. Although a single factor is not generally sufficient to cause the disease, about half of the variation in susceptibility has been assigned to genetic factors.

As early human ancestors evolved into bipeds, changes occurred in the pelvis, hip joint and spine which increased the risk of osteoarthritis. Additionally genetic variations that increase the risk were likely not selected against because usually problems only occur after reproductive success.

The development of OA is correlated with a history of previous joint injury and with obesity, especially with respect to knees. Since the correlation with obesity has been observed not only for knees but also for non-weight bearing joints and the loss of body fat is more closely related to symptom relief than the loss of body weight, it has been suggested that there may be a metabolic link to body fat as opposed to just mechanical loading.

Changes in sex hormone levels may play a role in the development of OA as it is more prevalent among post-menopausal women than among men of the same age. A study of mice found natural female hormones to be protective while injections of the male hormone dihydrotestosterone reduced protection.

Secondary

This type of OA is caused by other factors but the resulting pathology is the same as for primary OA:

- Alkaptonuria
- Congenital disorders of joints
- Diabetes
- Ehlers-Danlos Syndrome
- Hemochromatosis and Wilson's disease
- Inflammatory diseases (such as Perthes' disease), (Lyme disease), and all chronic forms of arthritis (e.g. costochondritis, gout, and rheumatoid arthritis). In gout, uric acid crystals cause the cartilage to degenerate at a faster pace.
- Injury to joints or ligaments (such as the ACL), as a result of an accident or orthopedic operations.
- Ligamentous deterioration or instability may be a factor.
- Marfan syndrome
- Obesity
- Septic arthritis (infection of a joint)

Pathophysiology
While OA is a degenerative joint disease that may cause gross cartilage loss and morphological damage to other joint tissues, more subtle biochemical changes occur in the earliest stages of OA progression. The water content of healthy cartilage is finely balanced by compressive force driving water out & swelling pressure drawing water in.[21] Collagen fibres exert the compressive force, whereas the Gibbs–Donnan effect & cartilage proteoglycans create osmotic pressure which tends to draw water in.[21] However during onset of OA, the collagen matrix becomes more disorganized and there is a decrease in proteoglycan content within cartilage. The breakdown of collagen fibers results in a net increase in water content.[22][23][24][25][26] This increase occurs because whilst there is an overall loss of proteoglycans (and thus a decreased osmotic pull),[23][27] it is outweighed by a loss of collagen.[21][27] Without the protective effects of the proteoglycans, the collagen fibers of the cartilage can become susceptible to degradation and thus exacerbate the degeneration. Inflammation of the surrounding joint capsule can also occur, though often mild (compared to what occurs in rheumatoid arthritis). This can happen as breakdown products from the cartilage are released into the synovial space, 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, possibly in an attempt to improve the congruence of the articular cartilage surfaces. These bone changes, together with the inflammation, can be both painful and debilitating.

**Diagnosis**

Diagnosis is made with reasonable certainty based on history and clinical examination.[28][29] X-rays may confirm the diagnosis. The typical changes seen on X-ray include: joint space narrowing, subchondral sclerosis (increased bone formation around the joint), subchondral cyst formation, and osteophytes.[30] Plain films may not correlate with the findings on physical examination or with the degree of pain.[31] Usually other imaging techniques are not necessary to clinically diagnose OA.

In 1990, the American College of Rheumatology, using data from a multi-center study, developed a set of criteria for the diagnosis of hand OA based on hard tissue enlargement and swelling of certain joints.[32] These criteria were found to be 92% sensitive and 98% specific for hand OA versus other entities such as rheumatoid arthritis and spondyloarthropathies.[33]

Related pathologies whose names may be confused with OA include pseudo-arthrosis. This is derived from the Greek words pseudo, meaning "false", and arthrosis, meaning "joint." Radiographic diagnosis results in diagnosis of a fracture within a joint, which is not to be confused with OA which is a degenerative pathology affecting a high incidence of distal phalangeal joints of female patients. A polished ivory-like appearance may also develop on the bones of the affected joints, reflecting a change called eburnation.[34]
Classification

A number of classification systems are used for gradation of osteoarthritis:

- WOMAC scale
- Kellgren-Lawrence grading scale

OA can be classified into either primary or secondary depending on whether or not there is an identifiable underlying cause.

Both primary generalized nodal OA and erosive OA (EOA, also called inflammatory OA) are sub-sets of primary OA. EOA is a much less common, and more aggressive inflammatory form of OA which often affects the distal interphalangeal joints of the hand and has characteristic articular erosive changes on x-ray.[35]

Management

Lifestyle modification (such as weight loss and exercise) and analgesics are the mainstay of treatment. Acetaminophen (also known as paracetamol) is recommended first line with NSAIDs being used as add on therapy only if pain relief is not sufficient.[36] This is due to the relative greater safety of acetaminophen.[36]

Lifestyle modification

For overweight people, weight loss may be an important factor.[37] Patient education has been shown to be helpful in the self-management of arthritis.[37] It decreases pain, improves function, reduces stiffness and fatigue, and reduces medical usage.[37] Patient education can provide on average 20% more pain relief when compared to NSAIDs alone in patients with hip OA.[37]

Physical measures

Moderate exercise is beneficial with respect to pain and function in those with osteoarthritis of the knee and hip.[38][39] These exercises should occur at least three times per week.[40] While some evidence supports certain...
Treatment recommendations by risk factors

<table>
<thead>
<tr>
<th>GI risk</th>
<th>Stroke and heart risk</th>
<th>Option</th>
</tr>
</thead>
<tbody>
<tr>
<td>low</td>
<td>low</td>
<td>NSAID, or paracetamol[49]</td>
</tr>
<tr>
<td>Moderate</td>
<td>Low</td>
<td>Paracetamol, or low dose NSAID with antacid[49]</td>
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<tr>
<td>Low</td>
<td>moderate</td>
<td>Paracetamol, or low dose aspirin with an antacid[49]</td>
</tr>
<tr>
<td>Moderate</td>
<td>Moderate</td>
<td>Low dose paracetamol, aspirin, and antacid. Monitoring for abdominal pain or black stool[49]</td>
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Medication

The analgesic acetaminophen is the first line treatment for OA.[36][50] For mild to moderate symptoms effectiveness is similar to non-steroidal anti-inflammatory drugs (NSAIDs), though for more severe symptoms NSAIDs may be more effective.[36] NSAIDs such as naproxen while more effective in severe cases are associated with greater side effects such as gastrointestinal bleeding.[36] Another class of NSAIDs, COX-2 selective inhibitors (such as celecoxib) are equally effective to NSAIDs with lower rates of adverse gastrointestinal effects but higher rates of cardiovascular disease such as myocardial infarction.[51] They are also much more expensive. Oral steroids are not recommended in the treatment of OA because of their modest benefit and high rate of adverse effects.

There are several NSAIDs available for topical use including diclofenac. Topical and oral diclofenac work equally well with topical having a greater risk of mild skin reactions but no greater risk of gastrointestinal adverse effects.[52] Opioid pain medications such as morphine and fentanyl reduce pain a small amount, but this benefit is outweighed by frequent adverse events and thus they should not routinely be used.[53] Topical capsaicin is controversial with some reviews finding benefit[54][55] and others not.[56]

Injection of glucocorticoids (such as hydrocortisone) leads to short term pain relief that may last between a few weeks and a few months.[57] Joint injections of hyaluronic acid have not been found to lead to significant improvement.[56][58] Hyaluronic acid injections have been associated with significant harm.[58] Nevertheless another study about hyaluronic acid injections says efficacy on pain and function, and no adverse effect when compared to saline injections.[59] Injections of platelet rich plasma improves function but not pain and is associated with increased risk.[60]

Surgery

If disability is significant and more conservative management is ineffective, joint arthroplasty surgery or resurfacing may be recommended. Evidence supports joint replacement for both knees and hips as it is both
clinically effective,[61][62] and cost-effective.[63][64]

Arthroscopic surgical intervention is largely not recommended or not addressed by most major orthopedic and rheumatological associations.[65]

Alternative medicine

Many dietary supplements are sold as treatments for OA and some of them have been found to be effective.

Since glucosamine is a precursor for glycosaminoglycans, and glycosaminoglycans are a major component of cartilage, some have hoped that supplemental glucosamine could beneficially influence cartilage structure, and alleviate arthritis. Its use as a therapy for osteoarthritis appears safe. The effectiveness of glucosamine is controversial.[66][67] Most recent reviews found it to be equal to[68][69] or only slight better than placebo.[70][71] A difference may exist between glucosamine sulfate and glucosamine hydrochloride, with glucosamine sulfate showing a benefit and glucosamine hydrochloride not.[72] The Osteoarthritis Research Society International recommends that glucosamine be discontinued if no effect is observed after six months[73] and the National Institute of Clinical Excellence no longer recommends its use.[2] Despite the difficulty in determining the efficacy of glucosamine, it remains a viable treatment option.[74]

Phytodolor,[54] SAMe,[75] and SKI 306X (a Chinese herbal mixture)[55] may be effective in improving pain, and there is some evidence to support the use of cat's claw as an anti-inflammatory.[76] There is tentative evidence to support avocado/soybean unsaponifiables (ASU),[55][77] MSM[54] and rose hip.[54] A few high-quality studies of *Boswellia serrata* show consistent, but small, improvements in pain and function among people with osteoarthritis.[78]

There is little evidence supporting benefits for some supplements, including: the Ayurvedic herbal preparations with brand names Articulin F and Eazmov, collagen, devil's claw, Duhuo Jisheng Wan (a Chinese herbal preparation), fish liver oil, ginger, the herbal preparation Gitadyl, glucosamine, hyaluronic acid, omega-3 fatty acids, the brand-name product Reumalax, stinging nettle, turmeric, vitamins A, C, and E in combination, vitamin E alone, vitamin K and willow bark. There is insufficient evidence to make a recommendation about the safety and efficacy of these treatments.[54][76]

While acupuncture leads to improvements in pain relief, this improvement is small and may be of questionable importance. Waiting list-controlled trials for peripheral joint osteoarthritis do show clinically relevant benefits, but these may be due to placebo effects.[79] Acupuncture does not seem to produce long-term benefits.[80] While electrostimulation techniques such as TENS have been used for twenty years to treat osteoarthritis in the knee, there is no conclusive evidence to show that it reduces pain or disability.[81]

Epidemiology

Globally approximately 250 million people have osteoarthritis of the knee (3.6% of the population).[83] OA affects nearly 27 million people in the United States, accounting for 25% of visits to primary care physicians, and half of all NSAID prescriptions. It is estimated that 80% of the population have radiographic evidence of OA by age 65, although only 60% of those will have symptoms.[84] In the United States, hospitalizations for OA increased from 322,000 in 1993 to 735,000 in 2006.[85]

As of 2004, OA globally causes moderate to severe disability in 43.4 million people.[86]
In the United States, there were approximately 964,000 hospitalizations for osteoarthritis in 2011, a rate of 31 stays per 10,000 population.[87] With an aggregate cost of $14.8 billion ($15,400 per stay), it was the second-most expensive condition seen in U.S. hospital stays in 2011. By payer, it was the second-most costly condition billed to Medicare and private insurance.[88][89]

### Etymology

OA is derived from the Greek word part *osteo-*-, meaning "of the bone", combined with *arthritis: arthr-*, meaning "joint", and -*itis*, the meaning of which has come to be associated with inflammation.[90] The -*itis* of OA could be considered misleading as inflammation is not a conspicuous feature. Some clinicians refer to this condition as *osteoarthosis* to signify the lack of inflammatory response.

### History

Evidence for OA found in the fossil record is studied by paleopathologists, specialists in ancient disease and injury. OA has been reported in fossils of the large carnivorous dinosaur *Allosaurus fragilis*.[91]

### Research

There are ongoing efforts to determine if there are agents that modify outcomes in OA. Sprifermin is one candidate drug. There is also tentative evidence that strontium ranelate may decrease degeneration in OA and improve outcomes.[92][93]

Gene transfer strategies aim to target the disease process rather than the symptoms.[94]

### References


**External links**

- American College of Rheumatology Factsheet on OA (http://www.rheumatology.org/practice/clinical/patients/diseases_and_conditions/osteoarthritis.asp)
- Clinical features of osteoarthritis (http://www.doctor121.com/2014/07/clinical-features-of-osteoarthritis.html)


Categories: Arthritis | Skeletal disorders

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