OSTEOARTHRITIS GENERAL

Version 2 Final

Document control

Version history

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Changes since last version

General Information

(Osteoarthrosis, degenerative joint disease, hypertrophic arthritis)

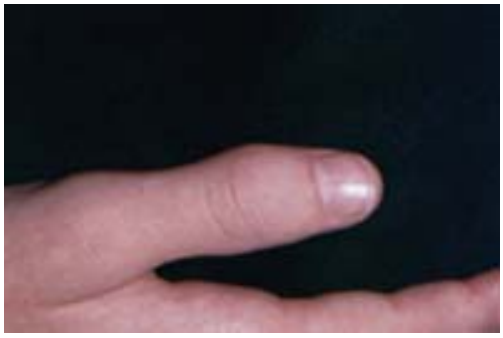
Description

Osteoarthritis (OA) is the most common condition to affect human joints and the most frequent of all the arthritic disorders occurring in nearly 100% of the population in at least one site by the eighth decade of life. It is present in every population world-wide although geographic differences do occur. However clinically significant disease is estimated to affect a smaller number of 10 – 20% of people. [1]

There is no widely accepted definition of OA, but it may be regarded as a disorder of synovial joints involving degeneration of articular cartilage with involvement of the subchondral bone. The suffix, "-itis", is a misnomer as it implies an inflammatory component, which is not a necessary aspect of the condition.

Osteoarthritis is a hetereogeneous condition for which the prevalence, risk factors, clinical manifestations and prognosis vary according to the joints affected.

The most frequently affected joints are knees, hips, spinal apophyseal joints and certain joints of the hands - especially the distal interphalangeal joints and the base of the thumb [2].



It is usually defined by pathological or radiological criteria rather than clinical features and is characterised by focal areas of damage to the cartilage surfaces of synovial joints, associated with remodelling of the underlying bone and mild synovitis.

When severe there is characteristic joint space narrowing and osteophyte formation, with visible subchondral bone changes on radiography. [1]

The changes lead to bone deformity, mal-alignment of the joint and consequent internal derangement of the joint.
There is poor correlation between objective measurement of disease severity (especially radiological) and symptoms. Any decisions regarding treatment are based largely on symptomatology.

Aetiology

Osteoarthritis is a heterogeneous disorder and may consist of a group of overlapping distinct diseases occurring in response to genetic, age and environmental factors. Rarely can a single factor be identified as wholly responsible, and many are best regarded as “associations”. There is frequently of interplay between a number of these, including:

- Age
- Genetic predisposition
- Exposure to repetitive physical stress/trauma
- Gender
- Race
- Obesity
- Generalised joint laxity (hypermobility)

Age is the strongest determinate for this condition. Radiographic change in the small joints of the hand has been identified in 80% of the population over the age of 70 [3].

Obesity has been strongly linked to osteoarthritis of the knee and to a lesser extent the hip.

The mechanism by which obesity and age predisposes to osteoarthritis is unclear. Obesity may act by increasing the mechanical stresses in weight bearing joints but this would not explain a role as a risk factor for osteoarthritis of the hand. Similarly the biochemical changes in ageing cartilage may predispose it to damage and evidence is emerging of change in cartilage composition which reduces compressive stiffness and predisposes to erosion. [4]

A hereditary component to osteoarthritis, particularly the generalised arthritis with Heberden’s nodes has long been recognised. Genetic factors are now said to account for at least 50% of cases of osteoarthritis in the hands and hips and a smaller percentage in the knees. It is unlikely that there is a single gene for a structural component of the cartilage that could fully explain any genetic contribution to osteoarthritis although the mutations in the vitamin D receptor gene which is near the locus for type II collagen (COL2A1) is considered a strong candidate. [1]

Studies in England and Sweden have focused on the role of occupation [5] in the development of osteoarthritis (with workers whose jobs involve physical labour having high rates of knee osteoarthritis, farmers showing high rates of hip osteoarthritis [6] and drill operators having osteoarthritis of the upper extremities [7]). Sporting activity (sports where there are high levels of practice with overuse injury e.g. running) [8] are noted risk factors for the hip and knee joints, although the evidence for this is not conclusive [9].

Poor or sub-optimal intake of vitamins C and D may play a part in both development and progression. [10, 11]

Secondary osteoarthritis

A number of specific conditions have been associated with OA. When such a disease is identified, the resultant OA is said to be “secondary”. These include:

- trauma
- septic arthritis
- inflammatory arthritis (rheumatoid, psoriatic)
- crystal deposition disease (gout, pseudogout)
- childhood hip disease (developmental dysplasia, Perthes, slipped upper femoral epiphysis)
- previous meniscectomy or ACL insufficiency in the knee
- avascular necrosis (which may be idiopathic or secondary to steroid use)
- radiation damage
- haemophilia
- haemochromotosis
- alkaptonuria (ochronosis)

All these risk factors appear to act through two major pathogenic mechanisms.

1. Factors influencing a generalised predisposition:
   inherited susceptibility, gender, race.

2. Factors affecting biomechanical loading on or at specific joints:
   Joint shape and laxity
   Trauma (commonest at the knee and includes fractures, cruciate ligament and meniscal tears)
   Occupation.
   Disease altering the articular cartilage

N.B. osteoporosis has a NEGATIVE association with osteoarthritis [12].

Most epidemiological studies have defined osteoarthritis on the presence of typical radiographic features and therefore although much is known about the principal risk factors for radiographic osteoarthritis (age, female sex, obesity and joint trauma) these are not the same risk factors as those in patients reporting joint pain (psychosocial factors, general health status).

Prevalence

Radiographic evidence of osteoarthritis increases with age the disease becoming almost universal in the elderly but often asymptomatic. However X-ray evidence alone is not sufficient to make a diagnosis. 12% of adults between the ages of 24-74 have radiological features of osteoarthritis in at least one joint [13] while conversely post-mortem studies have shown that, even in the presence of significant degeneration, joints can exhibit a normal X-ray appearance.

The joints primarily affected by osteoarthritis have changed within the last two centuries. A study of adult skeletons excavated from the crypt of Christ Church in east London in 1991 [14] found the acromioclavicular, facet joints of the spine and the hands to be the principle sites involved while osteoarthritis of the large joints, hips and knees, was uncommon.

In Caucasians osteoarthritis of the hip is rare under the age of 40 years and has an equal sex incidence in contrast to the knee and hand where there is an increased female incidence of 2:1 post-menopausally.

A principal source of the data on the frequency of osteoarthritis in Great Britain is the morbidity surveys conducted by the Royal College of General Practitioners, however, as these studies give no indication of the diagnostic criteria used some caution is required in the interpretation of the data [15].

Diagnosis

The diagnosis of OA is based on a combination of typical symptoms in association with appropriate radiological signs.

The clinical manifestations differ in different joints.

Characteristic presenting symptoms are those of (a) pain (b) stiffness (c) deformity. The clinical criteria include reduced range of motion, pain on passive movement and muscle wasting in the affected limb.

Pain

The cause of pain is unclear. Hyaline cartilage contains no nerve fibres. Pain may be a result of stretched joint capsule, inflammation of surrounding soft tissue, muscle spasm, or increased pressure within juxta-articular bone [16].

Pain can be a transient feature of osteoarthritis and may be absent in spite of severe joint damage [17].

The pain is related to activity, is characteristically worse on loading and begins within seconds or minutes of onset of use. It can continue for many hours after the activity has ceased. The description varies with the patient and ranges from a constant ache to discrete stabbing pains; pain is often associated with tenderness around the joint margin and associated soft tissues. It may be referred to a different site (eg hip OA is commonly felt in the knee).

Stiffness

This tends to occur imperceptibly often causing patients to subconsciously restrict their activities without being aware of it. The reduced range of movement is due to the formation of osteophytes, which stretch the joint capsule or directly impinge on each other.

Joint stiffness causes difficulty with initiating movement and problems with the range of movement. It is often used to describe ache or pain.

Characteristically joints affected by osteoarthritis 'gel' after inactivity.
The short lasting morning stiffness (<30 minutes) is in contrast to that of Rheumatoid Arthritis.

Deformity

In more advanced stages of OA, periarticular bone becomes enlarged due to osteophyte formation. Joints are swollen with effusion, and may become malaligned.

This is caused by asymmetric osteophyte formation and capsular stretching, and direct bone erosion.

Osteoarthritis is also associated with crepitus.

Radiology

X-rays normally display changes in joint space (reduction), osteophyte formation and subchondrial bone sclerosis, as well as the presence of subchondral bone cysts.

Imaging of joints by X-ray examination is the current ‘gold standard’ for detecting changes in joint structure but even with improved and standardised techniques plain X-ray examination remains an indirect measure of cartilage destruction. MRI has the advantage over plain radiography in that it can monitor changes in cartilage and other soft tissues but also remains only an indirect measure of the disease process while documenting the consequences of it.

MRI may also be useful in identifying avascular necrosis of underlying bone.

The natural history of osteoarthritis is highly variable and it is now clear that although radiographic progression is usual it is not inevitable and a proportion (20-30%) of patients with established osteoarthritis do not show progression on follow-up at five years [18] and some may apparently appear to improve.

It is important to note that the previously outlined radiographic features are often not in accord with clinical symptoms and many articles emphasise that there is no correlation between radiographic changes (particularly radiographic signs of deterioration)[19] and clinical symptoms and signs (pain and disability) [20].

Laboratory investigations

Laboratory investigations are usually only used to exclude or identify associated disease. Unless there is a coexisting inflammatory process, there is no elevation in ESR, plasma viscosity, or CRP, and Rheumatoid Factor is within normal limits.

The identification of biological markers for disease activity in osteoarthritis has proved complex (unlike Paget’s disease) and currently several markers including hyalurononate and cartilage oligomeric complex protein are the most promising candidates.

Classification

The heterogeneity of this disease has led to many attempts to classify it.

The classification which divides osteoarthritis into two forms, **primary** (idiopathic/unknown aetiology) and **secondary** (as a result of joint damage) continues to be utilised despite its many shortcomings, not least the considerable overlap that occurs within a single individual,

Pragmatically the best approach in describing a patient's disease may be purely descriptive, detailing the main joint site/s affected together with any other underlying disorders (e.g. metabolic) or history of trauma.

Treatment

Treatment for osteoarthritis varies slightly from joint to joint (particularly the point of surgical intervention) but generally is aimed at reducing pain, improving/maintaining joint mobility and limiting functional disability

The joint changes which occur in osteoarthritis cannot be reversed and therefore the aim is to modify a process that cannot be eliminated.

In general, the initial strategy is oral analgesia, with activity modification. Weight loss and range of movement exercises to prevent stiffness may be beneficial. Attempts to delay the degenerative process with oral agents such as cod liver oil and chondroitin sulphate, or injections of hyaluronic acid, are still the subject of much debate. Intra-articular glucocorticoid injection may improve symptoms, but repeated injection may worsen articular cartilage destruction [21].

Surgical strategies include re-alignment of the limb to redistribute the biomechanical forces (e.g. high tibial osteotomy, proximal femoral osteotomy) and prosthetic replacement. Despite increasing success and longevity, all surgical procedures have a limited duration of success, and the decision to operate must be taken with the expectation of eventual failure.

Specific current recommendations are detailed in the relevant protocols**.**

Main disabling effects

Most patients present with pain/stiffness and joint abnormalities which on direct questioning are found to impair their ability to perform the normal activities of daily living to varying degrees.

Pain and reduced function are the cardinal symptoms in osteoarthritis and represent the subjective and behavioural consequences of the disease. Anxiety, coping style and possibly depression are associated with the pain and observed disability in osteoarthritis [22].

The bone deformity and mal-alignment of joints limits function with reduced range of movement and pain at the extremes of movement. This is particularly noticeable in joints of locomotion.

Functional limitation causes secondary muscle weakness and wasting thereby further exacerbating instability in severe cases.

Pathological joint changes may have an effect on physical function that is independent of symptoms. Currently little is known about the relationship between these variables and the resultant apparent disability. It is therefore dangerous to conclude that pain alone correlates with disability.

Pain is the most common symptom in the hip and knee particularly while walking and negotiating stairs and less often while sitting and lying.

Pain while at rest is an indication of more severe disease and is usually preceded by a history of pain when joints are active.

Many individuals demonstrate poor correlation between objective clinical findings (examination and X-rays) and functional performance; although in general greater degrees of impairment are associated with the progression of osteoarthritis in any particular joint [23].

Other major predictors for disability need to be considered including, poor general health, muscle weakness and atrophy, psychological problems, occupational demands and multiple joint involvement.

Performance based methods of evaluating physical function in patients with chronic diseases such as osteoarthritis have been in existence since the 1980s [24]. However these early methods of assessment had questionable validity and reliability.

Recently more sophisticated ‘multidimensional’ methods of assessing functional limitations have been developed which, in addition to assessing difficulties in performing ‘Activities of Daily Living’ (ADLs) now allow the evaluation of social and psychological function.

In the arthritis literature there are several well accepted measures of functional limitation including, indices for assessing the severity of hip (ISH) and knee (ISK) disease [25] and the Western Ontario and McMaster University Osteoarthritis Index (WOMAC)[26].

These can be found as appendices to the protocols on osteoarthritis of the hip and osteoarthritis of the knee.

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