

(Osteoarthrosis, hypertrophic arthritis, degenerative joint disease)**DEFINITION**

1. Osteoarthritis is the most common condition to affect human joints and a frequent cause of disability. Recent advances in knowledge of the biochemistry of cartilage mean that concepts of osteoarthritis are still changing. In consequence there is at present no universally agreed definition.
2. It is characterised by focal loss of articular cartilage accompanied by hypertrophy of underlying and marginal bone, mild synovitis and capsular thickening.
3. The condition is best thought of as a process rather than a disease, with osteoarthritis reflecting the inherent reparative process of the synovium of the joint. If the process keeps pace with the damage, the function of the joint is maintained. If the repair process fails to cope, then osteoarthritis results. How osteoarthritis progresses is less well understood than its initiation. Both are current subjects of research interest and thought to involve different mechanisms.

CLASSIFICATIONS

4. Various classifications of osteoarthritis have been produced, none being ideal. The most commonly used classification is clinical with osteoarthritis considered to be of two types. **Primary** (or idiopathic), where no cause can be identified, and **Secondary**, where there is an obvious local cause such as previous joint trauma or congenital abnormality. This classification is useful but also has limitations.
 - 4.1. Because the primary form is a diagnosis of exclusion and the term is used in **any** situation where no local cause is identified, the term primary osteoarthritis refers to a large heterogeneous group of conditions.
 - 4.2. Primary and secondary forms can co-exist in one individual eg where an individual with generalised primary osteoarthritis undergoes knee surgery and subsequently develops secondary post-traumatic osteoarthritis in the knee.
5. Early diagnosis of osteoarthritis is difficult because patients tend to consider the associated pain and disablement as an inevitable part of ageing. The clinical diagnosis of primary osteoarthritis is supported by:
 - involvement of multiple joints
 - clinical onset in middle age or older ie 45-50 years and above and where
 - the degree of degenerative change in the involved joints is broadly the same - and in keeping with the patient's age and gender

6. Secondary osteoarthritis should be suspected where:
 - there is a history of earlier damage or abnormality in the joint now affected by osteoarthritis
 - there is involvement of non-weight bearing joints eg gleno-humeral, elbow, wrist and ankle
 - there is single joint involvement
 - clinical onset occurs before the expected age and
 - the degree of degenerative change is greater than expected from the individual's age and gender
7. Diagnosis of primary or secondary osteoarthritis is not made by the presence of one characteristic but depends on the majority of the suggested criteria being met.

CLINICAL MANIFESTATIONS

8. Osteoarthritis becomes more common with advancing age. The characteristic symptoms are pain, loss of function (often experienced as stiffness), and creaking and cracking of the joints. Clinical signs include joint tenderness, bony swelling along the joint line, pain and crepitus on movement, reduced range of movement and mild signs of inflammation. There may be soft tissue swelling around the joint. In advanced disease there may be destruction of bone and subluxation of the joint.
9. Any synovial joint may be the site of osteoarthritis. In general the most severe degeneration occurs in joints subjected to the greatest compression. These include the weight bearing joints - that is, the lower spine, hips and knees and the joints affected by strong repetitive muscle forces such as the first metatarso-phalangeal, the first carpo-metacarpal and the mid cervical joints.

Radiology

10. Radiological evidence of osteoarthritis is very common in adults - increasing in frequency with age. There is a characteristic radiological appearance; reduced joint space, subchondral sclerosis, cyst formation, spurring of the joint margins. The X-ray findings, however, do not correlate well with the degree of pain or disability in the individual.

AETIOLOGY

General factors

11. The precise aetiology and pathogenesis of osteoarthritis remain unknown. A particular difficulty is the different definitions of the condition used in the various studies. In particular some studies identify cases on clinical signs and symptoms and others use X-ray change. A number of general factors influencing the development and course of osteoarthritis have been identified:

12. **Age.** The process appears to begin in the second or third decade of life but does not usually become clinically apparent until a decade later. Radiological change becomes apparent when the process is well established. By the age of 55 to 65 years, 85% of the population have some radiological evidence of osteoarthritis.
13. **Gender.** There is an equal prevalence in both genders of mild osteoarthritis. A female preponderance becomes apparent for severe grades of osteoarthritis, in older age groups and for osteoarthritis of the hands and knees.
14. **Ethnic group.** Overall there seems to be uniformity in the prevalence of the condition across the peoples of the world. However the joints affected vary in different ethnic groups. Osteoarthritis of the hip is uncommon in non-white populations.
15. **Geographical variation.** Several very disabling polyarticular forms of osteoarthritis occur with high frequency in remote parts of the world and are almost unknown in the West. Mycotoxins or trace element abnormalities have been suggested as causal factors.

Major risk factors in individuals

16. **Analytical studies** of osteoarthritis have identified further major risk or predisposing factors for individuals. These fall into two groups:
 - 16.1. **Group A:** Factors affecting **constitutional susceptibility** to osteoarthritis.
 - 16.2. **Group B:** Local factors - causing abnormal joint loading or stress at specific joint sites.

Group A factors - constitutional

17. **Obesity.** This is strongly associated with osteoarthritis of the knee but not the hip. How obesity links to osteoarthritis is unknown. The association of obesity with osteoarthritis of the distal interphalangeal joint of the hand suggests that metabolic rather than mechanical factors are critical.
18. **Genetics.** Some osteoarthritis variants are strongly familial eg Heberden's nodes of the fingers and primary generalised osteoarthritis. This was first described in the 1950s and is characterised by involvement of distal finger joints, toes, knees and spine. Recent evidence from twin studies supports the notion that family history is important in all types of osteoarthritis. The correlation of osteoarthritis was consistently higher in identical compared with non-identical twins. As yet the nature of the genetic influence in osteoarthritis is speculative. It may involve a structural defect, alterations in bone or cartilage metabolism or a genetic influence on a known risk factor.
19. **Sex hormones.** Animal studies suggest that sex hormones play a role in osteoarthritis. Certain clinical subtypes of osteoarthritis are more common in women. Osteoarthritis frequently has its onset around the menopause and may be associated with hysterectomy.

20. **Bone density.** There is a negative association between osteoporosis and osteoarthritis at certain sites particularly at the hip. Similarly in osteopetrosis and acromegaly, a high incidence of premature osteoarthritis is observed. These observations suggest a role for bone density.

Group B factors - local

21. The most common local factors causing osteoarthritis in individuals are joint injury and stress.
22. **Major direct injury** includes fractures affecting joint surfaces, major fractures distant from the joint but altering its mechanics, soft tissue injuries within the joint - meniscal tears and complete or partial tear of ligaments. The evidence shows that osteoarthritis is not the inevitable result of direct injury. Rather osteoarthritis results in predisposed individuals from the effects of direct injury combined with the factors discussed above to produce osteoarthritis. Follow up studies have not shown an association between chondromalacia patellae and subsequent patello-femoral osteoarthritis.
23. **Joint stress factors.** Most commonly osteoarthritis occurs in a joint where sufficient stress has been applied over a period resulting in damage to the cartilage. This situation may arise where there is:
- 23.1. **Bone deformity** eg in relation to articular fracture, acetabular dysplasia, slipped epiphysis and Perthes' disease
 - 23.2. **Malalignment of a joint** eg genu valgum
 - 23.3. **Internal derangement of joints**
 - 23.4. **Compression of opposing articular surfaces**
 - 23.5. **Prolonged joint immobilisation**
24. There is no evidence that short-term repetitive trauma is linked to osteoarthritis in the absence of a single major injury. Repetitive trauma should be differentiated from repeated specific joint injuries (eg repeated tears of a ligament) where each injury is associated with clinical signs and symptoms and temporary disablement. A number of occupational and sporting associations with osteoarthritis have been described. It is thought that the link arises through prolonged periods of repetitive trauma.
25. An increased incidence of osteoarthritis has been described in miners (knees and spine), cotton workers (distal interphalangeal joints) and pneumatic drillers (elbows), ballet dancers (ankles), prize fighters (metacarpophalangeal joints) and baseball pitchers (elbows).
26. Individuals who participate in sports at a highly competitive level, especially with abnormal or injured joints have a greater risk of developing osteoarthritis. Ordinary use of normal joints and low impact activities associated with regular exercise and recreational pursuits (including jogging and running) are not associated with an increased incidence of osteoarthritis.

27. There are other relatively uncommon **joint stresses** pre-dating the development of osteoarthritis. These include **local chemical stresses**. Osteoarthritis may be seen in the following situations.
- 27.1. **Metabolic** - for example urates in gout or pigments in alcaptonuria or haemochromatosis. The mechanism is thought to be that these substances are deposited in cartilage, altering its susceptibility to stress.
- 27.2. **Hormonal** - for example acromegaly and diabetes mellitus which both show abnormal chondrocyte metabolism which predisposes to osteoarthritis.
- 27.3. **Therapeutic drugs** - alkylating agents may damage chondrocytes and high dose of corticosteroids may depress cartilage activity. This occurs if steroids are injected intra-articularly or when they are administered systemically in an immunosuppressive dose for a prolonged period of time.
28. **Repeated synovial** haemorrhage in a joint for example in diseases characterised by clotting defects can lead to osteoarthritis.
29. **Significant cold** injury can lead to osteoarthritis and similarly **avascular necrosis** as a result of deep sea diving, accidents and decompression sickness. Both of these stresses may result in osteoarthritis many years after the injury.
30. Osteoarthritis can follow **infection in a joint** and may also arise in a joint affected by an **inflammatory arthritis** such as rheumatoid arthritis.
31. **Neurological conditions** such as syringomyelia and tabes dorsalis may lead to a disruptive type of osteoarthritis eg Charcot's joint, which typically affects the knees. A combination of factors is responsible.
32. Osteoarthritis is not caused by adverse climate, damp clothes or sleeping on the ground. There is evidence that low barometric pressure may worsen the symptoms of established clinical disease. There is also evidence that stiffness occurs with rest but loosens with activity. Depression may increase the self perception of symptoms and disablement. However these factors do not cause or affect the course of the underlying pathological process.

CONCLUSION

33. **Osteoarthritis** is a common chronic condition which affects the synovial joints. Clinically it may be primary or secondary. Its causes and pathogenesis are not completely understood. External factors alone do not cause it and, in general, constitutional and familial factors are important in all cases. Several aetiological factors have been identified and are discussed above.

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