The diagnosis of osteoarthritis

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Highlights / Hoogtepunte

- The underlying disease process for osteoarthritis.
- How to make a clinical diagnosis of osteoarthritis.
- To differentiate between OA and RA, also refer to the May issue's article: "Diagnosing rheumatoid arthritis."

- Die onderliggende siekteproses in osteoartritis
- Hoe om 'n kliniese diagnose te maak van osteoartritis.
- Om te onderskei tussen osteoartritis en rumatoïede artritis, kan u ook verwys na die artikel "Diagnosing rheumatoid arthritis" in die Mei-uitgawe.

SA Fam Pract 2003;45(5):24-31

In the past osteoarthritis (OA) has been considered a predominately degenerative joint disease, affecting more people than any other arthritis. However, it is now realised that the problem is not simply a degenerative cartilage disorder, but a problem of all the tissues involved in maintaining joint stability – the functional joint unit. It is characterised by progressive loss of cartilage and bony margin overgrowth. In addition, there is no doubt that inflammation and synovitis are present in a significant percentage of patients, and indeed, a more aggressive form, erosive osteoarthritis may occur, associated with significant synovitis, and may be confused with inflammatory arthritis, such as rheumatoid arthritis.

The problem is acquired as a consequence of metabolic, genetic, mechanical and other influences. The involvement of the cartilage, results in secondary effects on the joint capsule, synovium, and periosteal nerve endings. It is these processes that result in pain, with further impact by psychological stress and depression or other psychological factors that influence chronic pain.

The cartilage predominantly consists of collagen type 2 fibres linked by covalent bonds, which confer tensile strength. The matrix of the cartilage is formed by the chondrocytes which are embedded within it. The matrix consists of proteoglycans and non collagenous glycoproteins. Within the matrix is water, tightly bound to the glycoprotein macromolecules. The chondrocytes get their nutrition from the surrounding fluid. The chondrocytes communicate via the fluid within the matrix by diffusion. The macromolecules give the cartilage the capacity for reversible deformation. They are hydrophilic and allow water molecule adherence between the collagen fibrillary network. The largest glycoprotein molecule is aggrecan. This consists of glycosaminoglycan chains predominantly made of keratan sulphate (KS), N-Acetyl glucosamine-galactose sulphated dimer, and chondroitin sulplhate (CS), a dimer of N-acetyl galactosamine and glucuronic acid.

In disease there is loss of the matrix, release of cytokines including IL-1, TNF and mixed metalloproteinases as well as prostaglandins by the chondrocytes. Fibrillation of the cartilage surface and attempted repair with osteophyte formation then occurs. The adjacent synovium is frequently observed to be inflamed and symptoms of inflammation, with rest pain and stiffness, and findings of swelling, heat of joints and effusions may be present.

The incidence of OA is not an inevitable consequence of ageing, although it is more prevalent the older one gets, and will affect about 80% of citizens over the age of 65. Other risk factors include obesity, female gender, being post menopausal or of Caucasian race, prior trauma, sports injuries, overuse syndromes and most importantly, family history.

The disease may be classified in several ways, but most commonly we divide it into primary or secondary OA (**Table 1**).

The diagnosis may be either clinical or radiological. It is important to realise that there is poor correlation between symptoms and radiological appearance.

CLINICAL HISTORY AND EXAMINATION

The clinical assessment remains the most important aspect of the assessment, as the laboratory investigations and radiology may be entirely normal.

Symptomatically the disease is characterised by pain with activity throughout the day, and transient/short-lived stiffness, known as "gelling", associated with rising in the morning and after inactivity, lasting minutes rather than hours. The greater the degree of inflammation, the greater the complaint of stiffness and the presence of rest pain. Therefore the history should focus on several key questions:

- When is the pain maximal?
- How long does the stiffness last on walking?
- Is there stiffness after rest ("gelling")?
- Is there swelling?

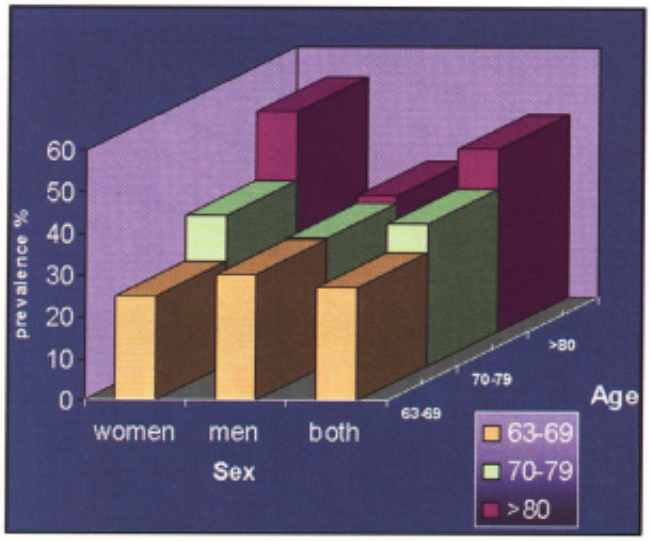

Figure 1: Prevalence of asymptomatic – radiological OA Felson DT. *(The epidemiology of osteoarthritis: results from the Framingham Osteoarthritis Study. Semin Arthritis Rheum 1990;20:(Suppl. 1):42-50.)*

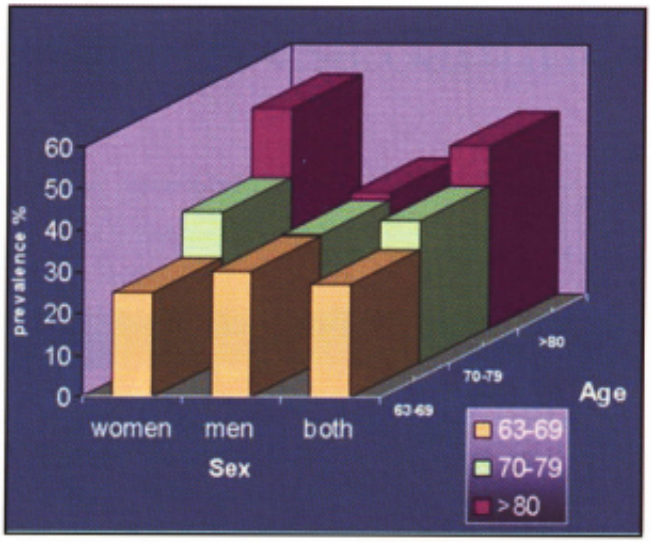

Figure 2: Prevalence of symptomatic OA Felson DT. *(The epidemiology of osteoarthritis: results from the Framingham Osteoarthritis Study. Semin Arthritis Rheum 1990;20: (Suppl. 1): 42-50.)*

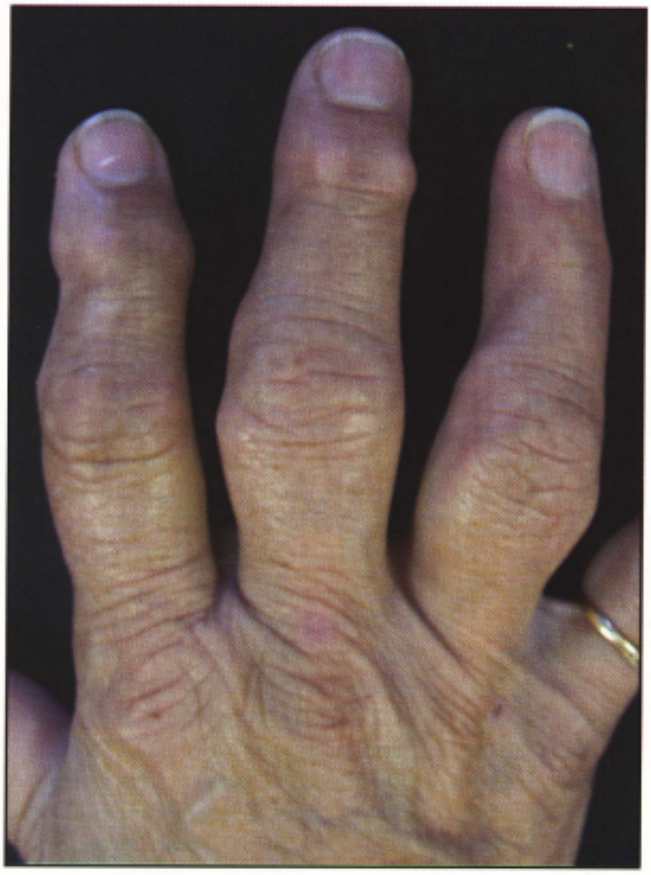

Figure 3: Finger involvement – clinical – showing bony thickening

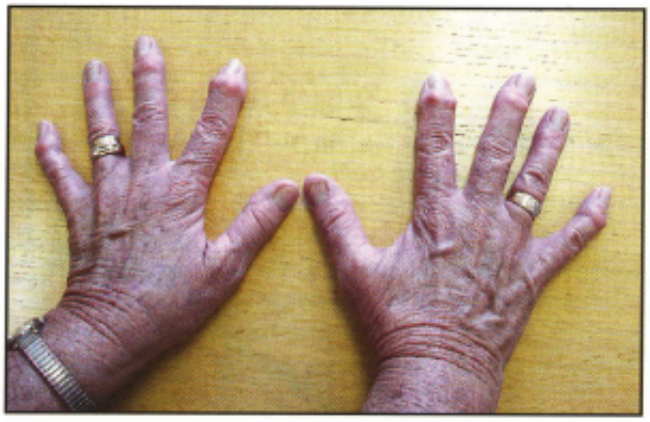

Figure 4: Hand joint involvement showing bony thickening of the DIP (Heberden nodes) and PIP joints (Bouchard nodes)

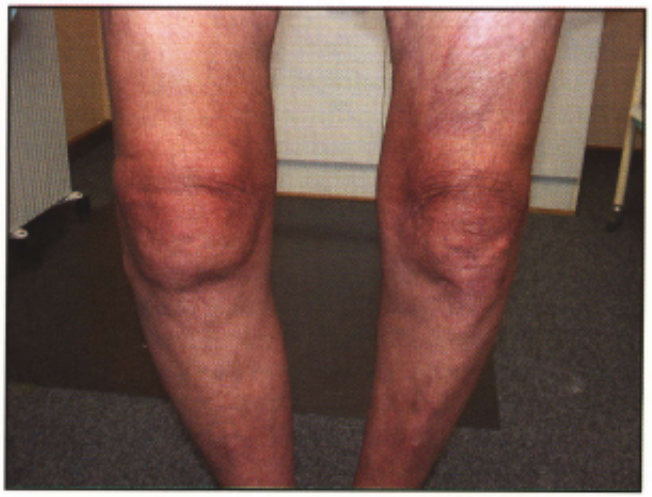

Figure 5: Large joint involvement showing varus deformity at the knee

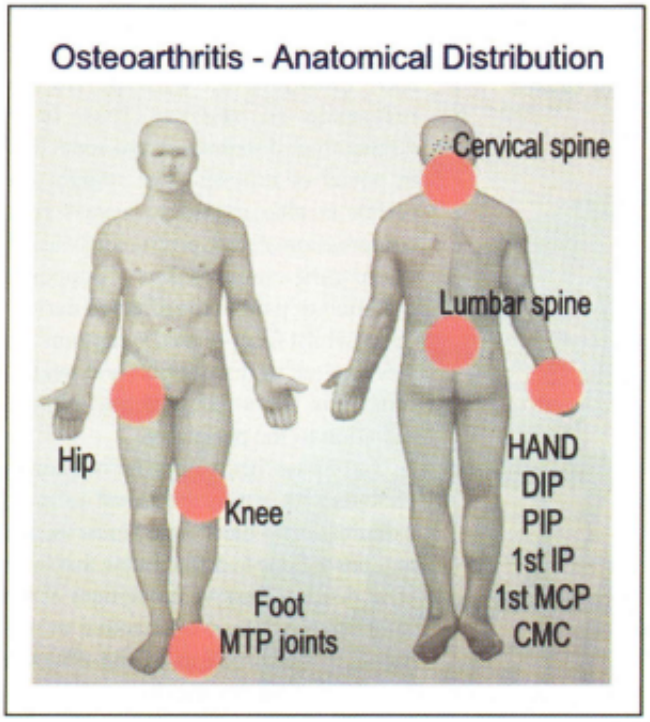

Figure 6: Anatomical distribution of joint involvement in osteoarthritis

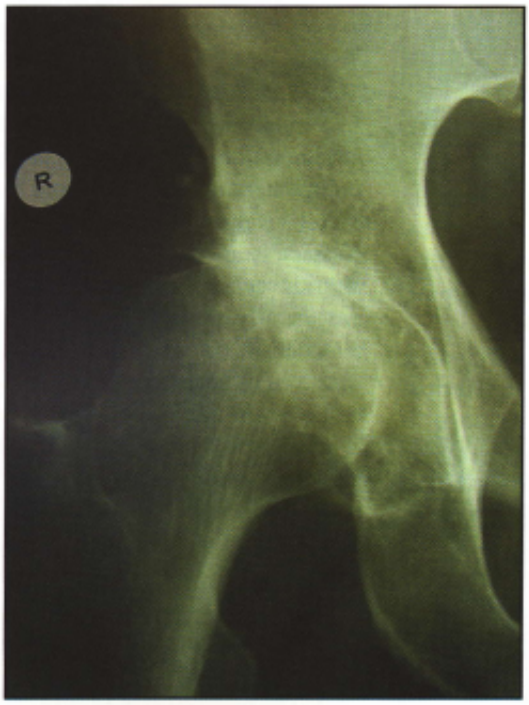

Figure 8: Osteoarthritis of the hip showing joint space narrowing at the weight bearing surface and osteophyte formation

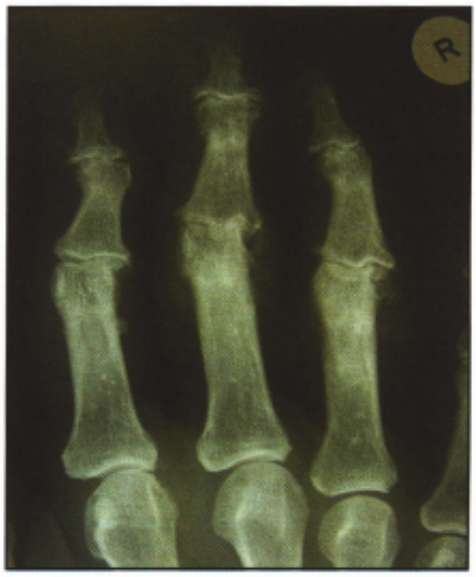

Figure 7: X-ray appearance showing joint space narrowing, sclerosis and osteophyte formation

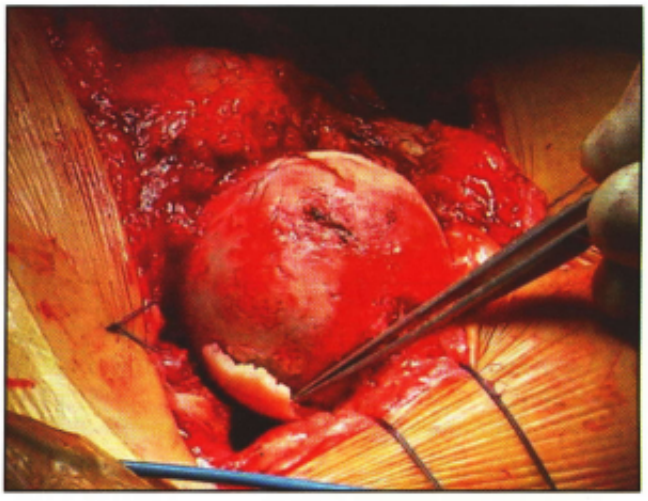

Figure 9: Appearance of the cartilage at hip surgery showing cartilage loss

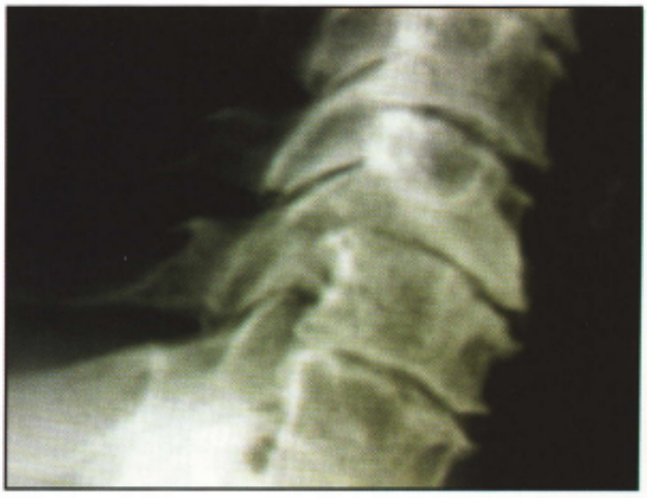

Figure 10: Neck involvement showing disc narrowing, sclerosis and osteophytes

Table 1: Classification of OA	
Primary	**Secondary: Common causes**
Genetic	Metabolic/endocrine
Erosive/progressive	Acromegaly
	Ochronosis
	Haemachromatosis
	Hyperparathyroidism
	Crystal deposition
	Trauma/surgery
	Fracture
	Post meniscectomy
	Occupational
	Sport related injury
	Inflammatory joint disease
	Sepsis
	Inflammatory arthropathy
	Anatomic/developmental
	Slipped epiphysis
	Congenital dislocation of the hip
	Perthes
	Hypermobility

- Which joints are involved?
- Are there any predisposing factors?
  - Trauma or sports injury.
  - Family history.
  - Obesity.
  - Underlying medical disease.

The joints may become tender on minor impact and become increasingly restricted in their range of movement and may have a subjective sensation of crepitus due to opposing irregular surfaces rubbing against each other.

Each joint is then examined with a particular focus on the distribution of involvement as well as the exclusion of synovitis, which is more typical of inflammatory arthritis such as RA. In OA the usual finding is the presence of bony swelling and the finding of only a comparatively low amount of soft tissue swelling. Typically the examiner finds bony thickening, reduced range of movement of the joints, crepitus and if advanced, mechanical derangement of the joint with instability. Mild synovitis may be noted. The typical joints involved, are the distal interphalyngeal (DIP) joints, the proximal interphalyngeal (PIP) joints, the joints at the base of thumb, the cervical and lumbar spine, and the hip and knee (Table 2).

In the case of the spine, as a consequence of facet joint arthritis, there is a restriction in the range of movement. Such limitation is usually assymetrical, compared to the symmetrical restriction of inflammatory

Table 2: Joint distribution of OA (adults aged 70)	
Joints	**Frequency %**
DIP	40
PIP	15
CMC	30
Knees	30-40
Hips	10
DIP: distal interphalangeal, PIP: proximal interphalangeal, CMC: Carpometacarpal	

backache. The peripheral neurological examination is done to exclude nerve root entrapment, with particular reference to sensory loss in a dermatomal distribution and reduction in power or reflexes. The straight leg reflex is also useful to assess root compression. A disc protrusion causing nerve root compression produces a radiation of pain to the affected dermatome, whilst facet-related symptoms are more vague in distribution and tend to remain in the axial skeleton without radiation to the periphery.

Soft tissue rheumatism is common in patients with osteoarthritis and a simple examination of the affected muscles and tendons is done to differentiate this from joint disease. Passive movement of the joint is pain free, whilst active movement in the direction of the affected muscle reproduces the discomfort.

Laboratory

Laboratory tests remain an unreliable diagnostic method, and if anything, are to exclude other pathologies or for the diagnosis of secondary causes. In general, standard blood tests are normal. The most useful screening tests for inflammation include the erythrocyte sedimentation rate (ESR) and the C-Reactive protein (CRP). These are classically elevated in inflammatory arthritis such as rheumatoid arthritis, but are usually normal or only slightly elevated in osteoarthritis. Secondary causes of osteoarthritis for which blood tests might help include hypothyroidism, hyperparathyroidism, acromegaly and haemochromatosis.

Aspiration of the joint fluid is unreliable but tends to have a low cell count and show less inflammatory changes, but is useful in certain clinical situations of acute flare to exclude crystal arthropathy such as pseudogout (pyrophosphate crystals), and gout (uric acid crystals) that may be coexistant clinical problems. In addition, sepsis can be investigated by aspiration and doing microscopy and cultures where clinically indicated.

Radiology

The radiological appearance provides the easiest method for diagnosis and classification of osteoarthritis (Table 3).

Table 3: Radiological classification of osteoarthritis (OA)		
Grade	**Classification**	**Description**
0	Normal	No features of OA
1	Doubtful	Minute osteophyte Doubtful significance
2	Mild	Definite osteophyte. Normal joint space
3	Moderate	Moderate joint space reduction
4	Severe	Joint space greatly reduced Subchondral sclerosis
Kellgren JH, Lawrence JS. *Atlas of standard radiographs. The epidemiology of chronic rheumatism, vol. 2.* Blackwell Scientific: Oxford, 1963.		

The typical changes are joint space narrowing, marginal osteophytes, sclerosis of subchondral bone as well as subchondral cyst formation. The pattern in large joints is typically unicompartmental or mainly weight-bearing surface involvement. In inflammatory arthritis such as RA, changes are usually bicompartmental or throughout the joint surfaces.

Arthroscopy provides a direct method to view the synovium and classification of severity is possible depending on the appearance of the cartilage (Table 4).

Table 4: Arthroscopic classification of severity of OA	
Grade	**Description**
1	Swelling and softening of cartilage. Oedema and cellular infiltrate
2	Superficial fibrillation
3	Deeper and large cartilage fibrillation
4	Visualisation of underlying subchondral bone.
Ayral X, Dougados M, Listrat *et al.* Chondroscopy: a new method for scoring chondropathy. *Semin Arthritis Rheum* 1993; **22**:289-97.	

MANAGEMENT

An assessment of function and impact on daily life is essential to determine how to manage the patient. Functionally there is enormous impact of the disease in terms of social, domestic, occupational and psychological aspects. This impact makes it imperative that ongoing research into meaningful therapies continues.

The therapeutic aim is to treat pain, maintain motor strength, range of joint movement, mobility and therefore function. Strategies include pharmacological and non-pharmacological approaches. Surgical intervention requires strict indications.

Nonpharmaceutical therapy includes weight loss, orthotics, assistive devices, braces, canes and physical therapy with exercise, aimed at muscle strengthening.

Pharmacological therapy largely consists of analgesia, and antiinflammatories. With the safety component of COXIB drugs, such as Celecoxib and Rofecoxib, the latter are becoming first line in management of pain and inflammation and are likely to replace the older conventional NSAIDs. However, a greater role is now forseen with disease modifying drugs including glucosamine sulphate, chondroitin sulphate and hyaluronan where appropriate.

The specific differences of treatment between osteoartrhritis and rheumatoid arthritis will be discussed in the next issue.❐

Please refer to the CPD questionnaire on page 61.