Diagnosis of Osteoarthritis
Guidelines and Current Pitfalls

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Summary

Diagnostic and classification guidelines for osteoarthritis based on clinical and radiographic signs for the most frequently involved joints (knee, hip and hand) have been developed by the American College of Rheumatology. Excellent, new ‘high tech’ biochemical and imaging methods and guidelines for clinical evaluation of the condition and its therapy have recently been developed and validated. Accordingly, we have consistent and reliable methods for assessing the established condition, but they provide little help in making an early and correct diagnosis for the different forms of osteoarthritis.

Both the diagnostic process and the differential diagnosis consist of careful history taking and detailed analysis of the complaints (especially pain), with proper physical examination including a search for the source of pain and tenderness. Not all deformities and pain in and around the joints are signs and symptoms of osteoarthritis, even if the patient is of ‘osteoarthritis age’ and the joint shows ‘osteoarthritis signs’ on the x-ray. On the other hand, even if the underlying disease is osteoarthritis, the symptoms and signs may be due to disorders secondary to the basic disease (e.g. enthesopathy or tendinopathy) and the patient’s complaints can be helped more easily by physiotherapy and local injections. The course of the disease and close follow-up yield diagnostic clues in cases where the cross-sectional diagnostic measures fail to provide them. Analysis of serum, joint fluid and x-ray films can be of diagnostic value, and other imaging methods (ultrasound, radioisotope scanning, computerised tomography, magnetic resonance imaging, arthroscopy) are also useful tools.

Differential diagnosis is not only of theoretical value; misdiagnosis of osteoarthritis leads to either omitted or unnecessary treatment, and causes psychological stress to the patient.

Osteoarthritis is the most common and most ancient articular disease of mankind.[1,2] Earlier and recent data concerning the prevalence[3,4] and incidence[5,6] of the condition prove that osteoarthritis affects most of the community. It is an important cause of long term health problems and one of the most frequently reported reasons for long term disability in the population.[7] Osteoarthritis is also very common in the animal world and can be traced back in phylogensis as far as the reptiles.[8]

It is now widely accepted that osteoarthritis represents a heterogeneous group of overlapping pathological conditions causing damage to the articular cartilage and leading to clinically and radiographically recognised impairment of the joints and disability and handicap of the patient.[9,10] This ‘failure of the joints’ is analogous to cardiac or renal failure. The insufficiency of the joint can be due either to abnormal loading of normal cartilage, or to normal loading of abnormally weak
cartilage.\textsuperscript{[11,12]} Although osteoarthritis is a disease with increasing incidence and prevalence in those aged over fifty years,\textsuperscript{[15,61]} this is not due simply to ‘wear and tear’, or aging of the cartilage, which is clearly a different process.\textsuperscript{[13]}

The classification and diagnosis of osteoarthritis is often confused in everyday practice because of different forms of the disease, and heterogeneity in localisation, presenting symptoms and final outcome.\textsuperscript{[9,14-16]}

1. Classification of Osteoarthritis

As a clinical entity, osteoarthritis can be divided into primary and secondary forms (table 1).\textsuperscript{[14,15]}

The cause of primary osteoarthritis is still not clearly understood. Genetic\textsuperscript{[17,18]} and hormonal factors\textsuperscript{[19]} play a very important role, such as in the development of Heberden’s nodes and primary generalised osteoarthritis. It is widely disputed whether the primary event is thickening of the cartilage and sclerosis of the subchondral bone, damaging the shock-absorption ability of the joint, or the escape of cartilage from hormonal and metabolic control, causing initial hyperplasia in the early stage of primary osteoarthritis and later an increased breakdown of cartilage.\textsuperscript{[11]} Subtle, unrecognisable anatomical abnormalities of articular surfaces or subchondral bone may also play a role.\textsuperscript{[20]}

Secondary osteoarthritis may be due to any inflammatory joint disease, overuse of the joint, traumatic lesions, metabolic processes weakening the cartilage, and congenital or acquired anatomical deformities.\textsuperscript{[21]}

In the diagnostic work-up, the pattern, distribution and some specific features should be noted.\textsuperscript{[9,16]} Both primary and secondary osteoarthritis include mono-, oligo- and polyarticular forms. The most frequently affected joints are the knee, hip, distal interphalangeal joints and the thumb base of the hand, and the cervical and lumbar apophyseal joints of the spine.

Different localisations and subsets of osteoarthritis are susceptible to different risk factors.\textsuperscript{[22]}

1.1 Osteoarthritis of the Knee Joint

Osteoarthritis can affect the 3 main compartments of the knee joint differently: the most frequently involved compartment is the medial tibiofemoral (75%), the second is the patellofemoral (50%), while lone lateral tibiofemoral osteoarthritis is relatively rare (25%).\textsuperscript{[10,23]} The most frequent combination is the coexistence of medial tibiofemoral and patellofemoral osteoarthritis.\textsuperscript{[10]} The cause may be evolutionary; tibiofemoral osteoarthritis was far less prevalent in ancient skeletons than hip or patellofemoral disease.\textsuperscript{[24]} Not surprisingly, the different compartments are susceptible to different risk factors: obesity, knee injury and meniscectomy for tibiofemoral osteoarthritis, and post-traumatic states, subluxation of the patella and valgus deformity of the knee for patellofemoral osteoarthritis.\textsuperscript{[25,26]} The different compartments of the knee should be examined and evaluated separately.\textsuperscript{[27]}

1.2 Osteoarthritis of the Hip Joint

Osteoarthritis of the hip joint also has specific distribution sites;\textsuperscript{[28]} the superolateral subset is the commonest (60%), probably due to the frequently
found acetabular dysplasia. Medial pole hip disease is less common (25%) and can cause protrusioacetabuli. The concentric type is rare (15%), but this form is more strongly linked with the generalised forms of the condition (mostly with Heberden’s nodes) than the other 2 forms.\textsuperscript{29}

There are differences in the effects of the various risk factors on knee osteoarthritis compared with hip osteoarthritis: in the latter, there is a weaker association with obesity, hip injury and Heberden’s nodes, but a marked association with occupational activity, such as farming, where the odds ratio for hip osteoarthritis for farmers is between 7 and 10.\textsuperscript{30-32}

1.3 Osteoarthritis of the Hand Joints

Osteoarthritis of the hand joints has 2 characteristic joint distributions. The distal interphalangeal joints (Heberden’s nodes) are affected in about 70% of cases, the first metacarpophalangeal joints (thumb-base) in 60%, and all other joints of the hand in less than 30%.\textsuperscript{9,33} Interphalangeal joint osteoarthritis is strongly associated with obesity and medial tibiofemoral osteoarthritis.\textsuperscript{34} Erosive osteoarthritis is a specific subset where the inflammatory process causes not only joint space narrowing, subchondral sclerosis, osteophytes and cyst formation, but also erosions in the distal interphalangeal joints.\textsuperscript{35}

1.4 Classification Criteria

The classification is an ‘aetiopathogenesis-directed’ process that provides rational and useful group categories (including frequent and rare forms of the condition), but is of little help for the clinicians’ everyday problems associated with individual features and variations of osteoarthritis.\textsuperscript{9,36} Thus, classification criteria would never be the same as diagnostic criteria, as the latter is a ‘symptom-directed’ process. Osteoarthritis as a clinical entity can be recognised only if it is associated with clinical symptoms and signs, such as pain, stiffness and restriction of movements. This is reflected in the classification criteria of knee,\textsuperscript{37} hand\textsuperscript{38} and hip osteoarthritis\textsuperscript{39} identified by the American College of Rheumatology. A recently modified version\textsuperscript{15} of these classification criteria is presented in table II.

2. Diagnosis of Osteoarthritis

Diagnosis includes not only the designation of a disease but also knowledge of the patients and their condition. In the case of osteoarthritis, this means that full diagnosis also includes the subsets of the disease, defining localisation, radiological and clinical staging, secondary consequences such as inflammation, joint loosening, intra-articular loose bodies, tendinous or ligamentous tear and/or enthesopathy, secondary bursitis, joint contracture, periarticular muscle spasm, entrapment neuropa-thy (e.g. the entrapment of digital nerves in osteoarthritis of metatarsophalangeal joints, or of the ulnar nerve in osteoarthritis of the elbow) or radiculopathy (uncovertebral or facet joint osteoarthritis of the cervical spine), and assessment of the functional capacity of the patient.

The above recommendations represent the classical ‘clinical’ diagnosis of osteoarthritis. In order to look at the problem as a whole, we should consider both 1) the specific need for defining osteoarthritis for large epidemiological studies,\textsuperscript{40} including case definitions for the knee, hip and hand osteoarthritis; and 2) the level of the general practitioner, who needs simple, but valid and reliable diagnostic criteria in order to provide the proper therapy.\textsuperscript{41,42}

Recent comparative community-based studies have found that the best diagnostic tool for recognising osteoarthritis for epidemiological studies is simple x-ray imaging and its careful and comparative analysis. The most valuable diagnostic x-ray sign is different in each joint site. The most sensitive and specific indicator of osteoarthritis of the knee is the presence or absence of a definite osteophyte,\textsuperscript{43} which should be accompanied by joint space narrowing in hip osteoarthritis\textsuperscript{44} and sclerosis of the subchondral bone in hand osteoarthritis.\textsuperscript{45} Surprisingly, clinical symptoms and signs do not play a determining role in the epidemiological diagnosis of osteoarthritis.\textsuperscript{40,43-45}
2.1 Radiological Assessment

Radiological assessment of osteoarthritis, both for individual and epidemiological studies, has always required a radiological reference standard. The Kellgren and Lawrence grading scale\[46\] has been used for over 30 years, but recent population studies have demonstrated its limitations.\[47,48\] These comprise a lack of clarity in interpretation, its reliance on the presence of osteophytes, and neglect of the importance of other specific features of the different joint sites, such as careful and reproducible positioning techniques. There is a growing consensus that an updated osteoarthritis reference standard is needed as a conventional global score, but particular features of different joint sites should also be emphasised.\[48,49\]

However, pathologically or radiologically detectable osteoarthritis is not always associated with clinical symptoms.\[3\] Indeed, these previous observations have been confirmed by recent studies.\[50,51\] Even in the most advanced radiographic grade about 40% of radiologically detectable osteoarthritis remained silent, and this included not only relatively mild cases. This fact indicates either that the consequences of pathological osteoarthritis can be compensated for by the organism, or that the pathological process of the

<table>
<thead>
<tr>
<th>Clinical</th>
<th>Clinical, laboratory and radiographic</th>
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<tbody>
<tr>
<td><strong>Knee</strong></td>
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<tr>
<td>1. Knee pain (on most days of previous month)</td>
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<tr>
<td>2. Crepitus</td>
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<td>3. Morning stiffness &lt; 30 min</td>
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<td>4. Age &gt; 38 years</td>
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<td>5. Bony enlargement</td>
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<td>Criteria fulfilled if: 1.2.3 &amp; 4</td>
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<tr>
<td>1.2 &amp; 5</td>
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<td>1.4 &amp; 5</td>
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<td><strong>Hip</strong></td>
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<td>1. Hip pain (on most days of previous month)</td>
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<td>2. Internal rotation &lt; 15°</td>
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<td>3. ESR &lt; 45 mm/h</td>
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<td>4. Hip flexion &lt; 115°</td>
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<td>5. Internal rotation &gt; 15°</td>
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<td>6. Morning stiffness &lt; 60 min</td>
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<td>7. Age &gt; 50 years</td>
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<td>8. Pain on internal rotation</td>
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<td>Criteria fulfilled if: 1.2 &amp; 3</td>
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<td>1.2 &amp; 4</td>
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<td>1.5,6,7 &amp; 8</td>
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<tr>
<td><strong>Hand</strong></td>
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<tr>
<td>1. Hand pain, aching or stiffness (on most days of previous month)</td>
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<tr>
<td>2. Hard tissue enlargement &gt; 2 of 10 hand joints[a]</td>
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<td>3. Metacarpophalangeal swelling &lt; 2 joints</td>
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<td>4. Bony enlargement &gt; 2 or more distal interphalangeal joints</td>
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<td>5. Deformity &gt; 1 of 10 hand joints[a]</td>
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<tr>
<td>Criteria fulfilled if: 1.2,3 &amp; 4</td>
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<td>1.2,3 &amp; 5</td>
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\[a\] Second and third distal interphalangeal, second and third proximal interphalangeal, and first carpometacarpal in both hands.

Abbreviation: ESR = erythrocyte sedimentation rate.
cartilage or other joint structures does not lead to clinical symptoms. The other end of the scale, the clinically positive but radiologically negative cases, represent a quickly progressing subgroup. Consequently, these patients would be an important potential target group for osteoarthritis prevention programmes.\[52,53\]

2.2 Diagnostic Criteria

General practitioners, who work among the everyday problems of patients, require clear indicators regarding the diagnostic criteria for osteoarthritis. In particular, they need to know what to include in the diagnosis and what to exclude.\[9\]

Inclusion criteria comprise
- the symptoms (such as pain of mechanical type)
- the signs (such as clinical signs of deformity or radiological signs of joint space width loss).

Exclusion criteria include
- all clinical conditions and/or radiological signs coexisting with or mimicking osteoarthritis.

The diagnostic and differential diagnostic processes consist of analysis of clinical symptoms and the results of laboratory and imaging methods, as well as outcome measures.

3. Pitfalls in the Diagnosis of Osteoarthritis

Misinterpretation of clinical symptoms and signs can be a pitfall in the diagnosing of osteoarthritis; therefore, careful analysis of these is of paramount importance (table III). Proper diagnosis and differential diagnosis are not only of theoretical value; misdiagnosis of osteoarthritis leads either to omitted or unnecessary treatment, and causes psychological stress to the patient.\[41,42\]

3.1 Analysis of Clinical Symptoms

3.1.1 Analysis and Differential Diagnosis of Pain

Pain is the chief symptom of osteoarthritis. The characteristic pain of osteoarthritis is of mechanical type, increasing with movement and joint loading and decreasing and ceasing at rest.\[54,55\] The pain is worst when starting movement after rest (in the morning and after prolonged sitting or resting); however, it then eases, but later increases after extended periods of walking or movement, because of overload. In the inflammatory stage of osteoarthritis, pain at rest also occurs. Nocturnal pain is most characteristic of inflammatory pain, e.g. in hip joint osteoarthritis, and is usually associated with hip joint effusion detectable by ultrasound.\[56\]

It should be noted that pain may also arise from various secondary lesions, such as enthesopathy, periarticular muscle spasm or bursitis. Interpreting this kind of pain as being caused by osteoarthritis itself is certainly an error and a pitfall, because pain of this type can be successfully managed by physiotherapy and local injections at the lesion site.

Misinterpretations in the evaluation of pain in established osteoarthritis can occur when
1. The source of pain is not osteoarthritis but arthritis of other origin, mechanical injury, or neurological or soft tissue disorders, occurring independently of osteoarthritis.
2. The pain is caused by osteoarthritis but not at the joint suspected, e.g. referred knee pain from hip osteoarthritis or hip pain caused by L4 radiculitis due to osteoarthritis of the apophyseal joints of the lumbar spine. The localisation of the dominating pain and the characteristic neuropathic pain can help in the differential diagnosis.
3. The real source of pain is secondary soft tissue alteration caused by the osteoarthritic process itself, such as ligamentitis, bursitis, enthesopathy, etc. around the osteoarthritic joint, due, for example, to valgus or varus deformity of the knee.

3.1.2 Analysis and Differential Diagnosis of Deformity

Deformity in osteoarthritis originates from the gradual destruction of the articular structures, such as the cartilage, synovium and subchondral bone. It may cause characteristic diagnostic signs and features, including stiffness after rest, swelling of the bony margins of the joints, limitation in movement, and cracking and crepitus of the joint on movement. Deformities caused by diseases other than osteoarthritis show very similar clinical signs and symptoms and thus represent possible causes
Table III. The most important causes of misdiagnosis of osteoarthritis (OA)

**Misinterpretation of pain**
The source of pain is **not** osteoarthritis, but
- Arthritis of other origin
- Pathological changes of the adjacent bone (tumour, osteomyelitis, metabolic bone diseases, etc.)
- Mechanical injuries, pathological fractures
- Referred pain of neuritis, neuropathy or radiculopathy (e.g. L4 radiculopathy may cause pain in the knee or in the greater trochanter, etc.)
- Other neurological disorders causing stiffness of joints (parkinsonism, central motor neuron damage, etc.)
- Soft tissue disorders, independent from OA (e.g. pes anserinus bursitis of the knee, de Quervain tendinitis, etc.)

The source of pain **is** osteoarthritis, but not at the joint suspected
- OA of the hip, pain localised in the knee
- OA of the cervical apophyseal joints (C4-5) causing pain in the shoulder
- OA of the lumbar apophyseal joints causing pain in the hip, knee or ankle
- OA of the acromioclavicular joint causing pain in the glenohumeral joint

The source of pain is caused by secondary soft tissue alterations of osteoarthritis
- Ligamentitis (especially of the knee)
- Enthesopathy, tendinopathy, due e.g. to joint contracture
- Bursitis (i.e. semimembranous bursitis, Baker cyst)

**Misinterpretation of deformity**
- Pseudohypertrophic osteoarthropathy,
- Psoriatic arthritis (distal type)
- Flexion contracture of the joints
- Mucopolysaccharidoses
- Neurogenic arthropathies
- Calcium pyrophosphate dihydrate crystal deposition disease
- Genu varum and valgum deformity of origin other than OA

**Misinterpretation of x-ray films**
- Arthritis with previous OA changes
- Initial stage of osteoarthritis; x-ray signs may be missing
- Diffuse idiopathic skeletal hyperostosis syndrome
- Flexion contracture can cause a virtual loss in joint space width
- Neurogenic and metabolic arthropathies

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for the misdiagnosis of osteoarthritis (table III). These types of deformities include the following:

- **Hypertrophic osteoarthropathy**, which might be mistaken for generalised osteoarthritis affecting the fingers. Digital clubbing, nail alterations, and subperiosteal new bone formation on the x-ray are differentiating features.

- Distal type of psoriatic arthritis involving distal interphalangeal joints. Psoriatic lesions of the nails and skin may differentiate this disorder.

- Flexion contracture of the joints, especially of the fingers, knees, and hips in middle-aged or elderly people might suggest osteoarthritis. Common causes of flexion contracture of the fingers include Dupuytren’s contracture, posttraumatic and neurological conditions, and diabetic finger contractures. In the knee, flexion contracture can be caused by joint-locking (loose bodies) and, in the case of the hip joint, by aseptic necrosis of the femoral head.

- Mucopolysaccharidoses often cause flexion contracture of the fingers and other peripheral joints, with joint deformity and even pain, especially in the Hurler and Hunter’s syndromes. These deformities usually develop in early youth.

- Neurogenic arthropathy (Charcot joints) may mimic osteoarthritis, especially in the knee and hip. The bizarre and luxuriant ossifications around the joint, as well as the laxity of the joints, are more severe than is usual in osteoarthritis. The relatively mild pain and the underlying neurological condition are common distinguishing features.
In the case of calcium pyrophosphate dihydrate (CPPD) crystal deposition disease, the connection between osteoarthritis and this disease is not clear. Recurring CPPD arthritis may cause secondary osteoarthritis, especially in the knee, but crystals might be free in the joint cavity because of the osteoarticular process. Chondrocalcinosis on the x-ray and the detection of CPPD crystals in the joint fluid provide clues for diagnosis.

Genu varum and valgum can be a consequence of knee joint osteoarthritis but may also be due to congenital deformity, previous trauma, rickets, underlying osteomalacia or Paget's disease.

4. How to Avoid Diagnostic Pitfalls

Diagnostic pitfalls can be avoided by careful history taking, physical examination of the patient and the use of valid imaging methods.

Outcome measures are also useful diagnostic tools in differentiating the various primary and secondary forms of osteoarthritis and changes during the disease process.

4.1 Careful Patient History

The key to obtaining a careful patient history involves questioning the patient about the following:

- The incidence of osteoarthritis in the family, especially in suspected cases of generalised osteoarthritis.

- The time of occurrence of initial symptoms and signs. Secondary osteoarthritis due to trauma, overuse, joint laxity, joint dysplasia, osteochondromatosis, osteochondritis dissecans, metabolic diseases, etc., may occur at a younger age. Joint deformities of mucopolysaccharidosis, rickets, etc., that mimic osteoarthritis occur also at a young age.

- The circumstances in which first symptoms and signs appeared. Were they connected to trauma, however minor, which may have caused tendinous, ligamentous, meniscal or muscular tears?

- Previous infection, which might have caused low- or high-grade joint infection or post-infectious arthritis. Note that tuberculous arthritis of the knee, hip or shoulder often occurs in osteoarthritic joints in the elderly.

- Underlying diseases such as diabetes, hypothyroidism, haemochromatosis, etc. that might cause secondary osteoarthritis.

- The pattern of pain and stiffness, in order to determine whether it is really characteristic of osteoarthritis.

4.2 Physical Examination

4.2.1 Elicitation of the Pain

At physical examination, one of the most important tasks of the examiner is to elicit the pain the patient is complaining of and to determine its source, i.e. do active and passive movements of the joint elicit pain?

In many cases, careful physical examination of the patient may avoid most of the diagnostic pitfalls. The pain felt in the knee often arises from the hip or the L4 nerve root or femoral nerve, and can be elicited by movements of the hip or lumbar spine. Similarly, pain felt in the shoulder may originate from the cervical spine and can be provoked by cervical movements, or pain felt at the greater trochanter may arise from the lumbar spine. Pain in the fingers or toes may originate from nerve (tunnel syndromes) or nerve root irritations, and can be provoked by special tests for nerve or nerve root involvement.

Pain in the patellofemoral joint can be provoked by pressing the patella against the femoral joint surface. Examination of the knee includes tests that may provoke ligamentous, meniscal, or tendo-periosteal pain, and pain arising from bursae.

Tender points should be identified. Very often, not only is the characteristic joint tenderness present, but tenderness of the enthesis may also exist. In cases of enthesopathy mistaken for osteoarthritis, only these spots (e.g. the pes anserinus area in the case of knee osteoarthritis) are tender.
4.2.2 Evaluation of Swelling and Deformity
The second important part of the physical examination is evaluation of swelling and deformity. ‘Hard swelling’ or ‘hard nodes’ around the joint are characteristic of osteoarthritis, especially around the distal interphalangeal (DIP) joints (Heberden’s nodes) and proximal interphalangeal (PIP) joints (Bouchard’s nodes), and the medial and lateral tibiofemoral compartments of the knee joint. The nodes are palpable much earlier than they can be recognised on x-rays, although at this stage they are visible on bone scintigrams as hot spots.[76]

Soft tissue swelling may also be a feature of osteoarthritis, in addition to, or without the presence of, hard nodes. Soft tissue swelling and palpable joint effusion is often present in PIP and DIP joints and especially in the knee, where swelling and effusion might be the presenting sign of osteoarthritis. If there is no radiographic evidence of osteoarthritis, and the synovial effusion is non-inflammatory, arthroscopy may be indicated to rule out meniscal tear, the presence of loose bodies, etc.[77]

Palpation of crepitus and other audible and palpable phenomena may occasionally be of importance. Fine crepitation felt throughout the whole range of movement is mostly of capsular origin and may also appear in young healthy persons and is not of diagnostic importance. Coarse cracking felt with movement may be due to severe damage of the cartilage, and the phenomenon is caused by the movement of uneven surfaces moving on each other. One or two cracks felt over the knee can be a sign of a loose body or meniscus tear, while around the hip this is usually caused by the iliotibial ligament jumping over the greater trochanter.[68]

4.2.3 Examination of Joint Function
Examination of joint function is also an important part of the physical examination and may have diagnostic value. For example, in osteoarthritis of the hip, initially, internal rotation and abduction are restricted, followed by restriction of adduction, hyperextension and external rotation. If extension is restricted first, it raises the suspicion of other disorders, psoas abscess or iliopsoas bursitis.[78] Similarly, extension contracture of the knee (inability of flexion) is characteristic of spastic paresis, joint locking due to a loose body, or contracture developed during immobilisation, but certainly not osteoarthritis. A flexed position of the DIP joint suggests osteoarthritis or psoriatic arthritis. When free passive movement of DIP joints is proven, the flexed position is probably due to rupture of the extensor tendon.

Examining abnormal passive movements due to joint laxity is of great importance. These abnormalities are common, especially in the knee and finger joints of osteoarthritis patients, and are due to laxity of the capsule and ligaments. This situation usually causes a bigger problem for the patient than restriction of movement.[41]

4.3 Analysis of Laboratory Findings
Laboratory findings do not play a very important role in the diagnosis of osteoarthritis.[37-39,79] Sedimentation rate is usually normal, or somewhat elevated in the inflammatory stage, not exceeding 30 to 35 mm/h.[80] Joint fluid is noninflammatory: its colour is clear, viscosity high, mucin clot is firm and cell number is under 2000/mm³. In some cases synovial fluid volume may be as high as 40 to 50ml, but even in these cases the ratio of polymorphonuclear leucocytes remains below 25%. [80] In very destructive forms, even bloody effusion can occur. In some cases, CPPD or hydroxyapatite crystals may be detected in the effusion.[81,82]

Other laboratory investigations may provide assistance in diagnosing underlying metabolic diasease such as hypothyroidism, acromegaly, ochronosis, mucopolysaccharidoses, haemochromatosis, diabetes, etc.[21] Rheumatoid factor may be of value in the rare case where there is a need for differentiating rheumatoid arthritis and generalised osteoarthritis; the distribution of articular involvement in the two diseases is generally different. Differential diagnostic problems may arise in the uncommon circumstance when rheumatoid arthritis begins in a patient with existing generalised osteoarthritis, or erosive osteoarthritis evolves into rheumatoid arthritis.[80]
4.3.1 Biochemical Markers

The development of sensitive and specific biochemical markers of osteoarthritis raises the prospect of a tool for early diagnosis, as well as the possibility of objective monitoring of both the natural course of the disease and the effect of therapy. [83-85] Markers under investigation include cartilage components, bone-derived components and indicators of synovial activity detectable in serum, synovial fluid and urine. Unfortunately, none of these have yet been validated in robust community studies. [84,85]

The cartilage-derived biochemical markers include keratan sulfate epitopes such as KS-5D4, KS-ANP91 and KS-2D3, which indicate catabolic activity and occur primarily after joint trauma. Different types of chondroitin sulfate epitopes, CS-3B3, CS-7D4 and CS-846, as well as type II collagen propeptide-C and cartilage oligomeric matrix protein, seem to be sensitive markers of cartilage anabolic activity, indicating cartilage repair. [86]

Bone-derived markers indicate activity of the subchondral region in osteoarthritis. Elevated levels of the urinary collagen crosslink pyridinoline and deoxypyridinoline indicate catabolic action, [87] while the elevated levels of osteocalcin and bone-specific alkaline phosphatase may be signs of increased anabolic-type repair processes in the subchondral bone. [88] In a recent study, elevated synovial fluid osteocalcin levels were associated with the severity of late-phase technetium bone scan. [89] Markers of synovial proliferation include type III collagen propeptides [90] and serum hyaluronic acid; the latter seems to be a predictor of disease progression in osteoarthritis of the knee. [91]

4.4 Imaging Methods

4.4.1 Radiography

Bidirectional (usually anteroposterior and lateral) x-ray films are required for the knees, but in the case of hands and hips, anteroposterior films are usually sufficient. [92] For weight-bearing joints, x-ray films should always be taken in a weight-bearing position. [93] Careful and reproducible positioning is essential for imaging both the tibiobemoral and the patellofemoral compartments of the knee. [92,94,95] Higher sensitivity, specificity, and reproducibility of the x-ray pictures can be achieved by novel x-ray imaging techniques, such as digital image analysis [96] and micro-focal radiography. [97]

In everyday practice, the familiar findings of osteoarthritis are still used, including narrowing of joint space, sclerosis of subchondral bone and formation of subchondral cysts and osteophytes.

It should also be noted that the flexion position of the joint may spuriously narrow the joint space, resulting in a misinterpretation in radiographic diagnosis of osteoarthritis (table III).

Occasionally, even when palpable enlargement of the articulation is present, radiography may not show pathological alteration. In this situation, scintigrams performed by bone-seeking radioisotopes (99mTc-phosphonate) may provide diagnostic clues, showing accumulation of the nuclide around DIP joints. [98]

4.4.2 Ultrasound

Ultrasound is theoretically useful in measuring the width of cartilage, but this procedure has not yet been standardised. Ultrasound is largely used for detection of effusion in the hip joint, [56] for Bakers cyst, and for diagnosing ligamentous and/or tendinous tears. [99]

4.4.3 Magnetic Resonance Imaging

Theoretically, magnetic resonance imaging (MRI) techniques should provide a tool for measuring the width of articular cartilage. However, the technique is not yet fully developed. [100] The advantage is that different tissues of the joint can be imaged by different sequences. MRI can detect loose bodies and meniscal tears in the knee joint and, in the hip, it can provide very early differential diagnosis of avascular necrosis of the femoral head mimicking the initial stage of osteoarthritis. [101] It can also detect swelling and cyst formation of the apophyseal joints of the spine, which sometimes cause not only back pain but also root irritation or compression. [100]
4.4.4 Arthroscopy

Arthroscopy is a useful and sensitive tool for assessment of almost all structures of the osteoarthritic joint and provides a direct, magnified view of the 6 articular cartilage surfaces of the knee. In addition, it can directly image the actual state of cruciate ligaments, plicae, and synovium. The use of needle arthroscopes is a quick and safe means for diagnosis and follow-up of the condition, and the destruction of the cartilage can be graded during the chondrosopic process. The only disadvantage of arthroscopy is that it is still an invasive method. At present, it cannot be replaced by any other imaging method, since it is the only way of directly visualising the articular structures.

4.5 Outcome Measures

Outcome measures can help us to evaluate the functional status of the patients, and they provide an objective tool for assessing the severity of impairment, functional disability and handicap at personal and community levels. In addition, these measures provide us with valuable information for differentiating surgical and nonsurgical therapy for osteoarthritis. All well known general outcome measures, such as the Health Assessment Questionnaire (HAQ), and the recently developed MOS 36-Item Short Form Health Survey (SF-36) can be used for this purpose.

Two specific outcome measures for osteoarthritis have been developed: the Lequesne algofunctional index and the Western Ontario and McMaster Osteoarthritis Index (WOMAC). Index for assessing the severity of osteoarthritis of the knee and hip. Validation studies have proven the high sensitivity and specificity of the latter in clinimetric studies.

5. Conclusions

Diagnosing osteoarthritis is not as easy and simple as it seems at first glance. Even when the presence of osteoarthritis is evident, the pain described by the patient may arise from lesions secondary to the disease, such as enthesopathy, bursitis, muscle spasm, or inflammation, which may be successfully treated by physiotherapy or local injections. Careful analysis of the presenting symptoms and signs is mandatory.

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