Clinical manifestations and diagnosis of osteoarthritis

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INTRODUCTION — Osteoarthritis (OA) is the commonest form of arthritis and possesses marked variability of disease expression. Although most patients present with joint pain and functional limitations [1], the age of disease onset, sequence of joint involvement, and disease progression varies from person to person. OA ranges from an asymptomatic, incidental finding on clinical and/or radiographic examination to a rapidly progressive disabling disorder eventually culminating in "joint failure."

The clinical features and approach to the diagnosis of OA will be reviewed here. The pathogenesis, epidemiology, and treatment of OA are discussed separately. (See "Pathogenesis of osteoarthritis" and "Risk factors for and possible causes of osteoarthritis".)

CLINICAL MANIFESTATIONS — The primary symptoms of osteoarthritis (OA) are joint pain, stiffness, and locomotor restriction. Symptoms usually present in just one or a few joints in a middle-aged or older person. Other manifestations in patients with OA include sequelae such as muscle weakness and poor balance [2] and comorbidities like fibromyalgia [3-6].

Symptoms and signs — The following symptoms and signs may be observed in patients with OA:

- **Pain** – Pain in OA is worse with joint use (usage-related pain) and relieved by rest. It is often the most frequent symptom and generally progresses through three stages [7]:
  - Stage 1 – Predictable, sharp pain usually brought on by a mechanical insult that eventually limits high-impact activities with relatively modest effect on function.
  - Stage 2 – Pain becomes more constant and starts to affect daily activities. There may be unpredictable episodes of stiffness.
  - Stage 3 – Constant dull/aching pain punctuated by episodes of often unpredictable, intense, exhausting pain which results in severe limitations in function.

However, not all patients go through such distinct stages, and pain progression may be arrested at any stage.

Pain is generally worse in the late afternoon and early evening but can also be worse in the morning soon after waking up [8]. There may also be night pain in severe OA which can interfere with sleep. In some people, the pain has a burning (neuropathic) quality, is widespread around the joint, and is associated with paresthesia [7]; such features also suggest comorbid fibromyalgia. Painful periarticular soft-tissue lesions may coexist, especially with large-joint OA [9]. Periarticular soft-tissue lesions cause localized pain away from the joint line, whereas OA-related pain more commonly is most severe over the joint line, except for proximal joints like the hip or the shoulder that may have the maximal pain distal to the originating joint.
• **Tenderness** – Joint-line tenderness suggests articular pathology, while tenderness away from the joint line suggests periarticular soft-tissue pathology.

• **Limitation of motion** – Reduced range of motion (equal for both active and passive movement) mainly results from marginal osteophytes and capsular thickening, but synovial hyperplasia and effusion may also contribute.

• **Bony swelling** – Bony swelling reflects remodeling of the bone and cartilage on either side of the joint and marginal osteophytes and may be evident in small (eg, finger interphalangeal, first metatarsophalangeal [MTP]) and large (eg, knee) joints.

• **Joint deformity** – Deformity is a sign of advanced joint damage.

• **Instability** – Giving way or buckling is a common symptom in knee OA. Occasionally people may stumble and fall, but usually it is a feeling of apprehension and lack of confidence to weight-bear rather than literally "giving way." It is predominantly a sign of muscle weakness with subsequent altered patellar tracking (with lateral patellar subluxation) but may also associate with true joint instability. Similar symptoms are frequently reported by patients with thumb-base OA.

**Joint distribution** — Many of the characteristic clinical manifestations of OA are related to the involvement of particular joints. OA can be categorized into localized or generalized forms of the disease.

**Single- or multiple-joint osteoarthritis** — OA has a predilection for the knees, hips, interphalangeal joints, first carpometacarpal (CMC) joints, first MTP joints and apophyseal (facet) joints of the lower cervical and lower lumbar spine (*figure 1*) [10].

OA less commonly affects the elbow, wrist, shoulder (ie, the glenohumeral joint), and ankle. When the elbow, shoulder (especially the acromioclavicular joint), and metacarpophalangeal (MCP) joints (eg, the "Missouri metacarpal syndrome") are affected, occupations that involve overuse of the upper limbs should be suspected. The symptoms at these joints are similar to those of OA at other joints (*table 1*); however, joint involvement is more often unilateral.

**Generalized osteoarthritis** — Generalized OA implies a polyarticular subset of OA typically involving the distal interphalangeal (DIP) joints, thumb bases (first CMC joints and trapeziocapsuloid joints) (*figure 2*), first MTP joints, lower cervical and lumbar facet joints, knees, and hips [11]. It is characterized by slow accumulation of multiple joint involvement over several years. Symptoms usually commence in the hands around middle age and subsequently affect the knees and other joints over the next few decades. The clinical marker for generalized OA is the presence of multiple Heberden's nodes, which are posterolateral hard swellings of the DIP joints (*picture 1*) [11]. Heberden's nodes are often accompanied by less well-defined posterolateral swellings of the proximal interphalangeal (PIP) joints referred to as Bouchard's nodes (*picture 2*). Generalized OA may occur in the absence of nodes, so called non-nodal generalized OA, which is more common in men (compared with nodal generalized OA, which is more common in women) [12,13]. There is no universal definition for the number of joints affected before someone can be classified as having generalized OA, but guidance from the American College of Rheumatology (ACR) and the European League of Rheumatology (EULAR) suggests that generalized OA is present if there is OA at the spinal or hand joints, respectively, and in at least two other joint regions [14,15].

**Imaging** — The diagnosis of OA is a clinical one based on characteristic signs and symptoms described above. When the diagnosis is unclear or important alternative diagnoses need to be considered, several imaging modalities can be used to assess the presence and severity of OA. Our approach to imaging for OA is generally consistent with guidelines developed by professional organizations [16].

• **Radiography** – Conventional radiography is the most widely used imaging modality in OA and allows for detection of characteristic features of OA including marginal osteophytes, joint space narrowing, subchondral sclerosis, and cysts [17,18]. Radiographs can also be used to measure joint space narrowing, which is sometimes used as a surrogate measure of cartilage loss. However, radiographic changes in OA are insensitive,
particularly with early disease, and often correlate poorly with symptoms [19-21]. Also, radiographic OA is a common incidental finding in older people.

- **Magnetic resonance imaging** – Magnetic resonance imaging (MRI) is not necessary for most patients with symptoms suggestive of OA and/or typical radiographic features. However, MRI can identify OA at earlier stages of disease before radiographic changes become apparent. These changes include cartilage defects and bone marrow lesions. MRI can also be used to assess pathology in other structures of the joint not visualized by radiography such as effusions, synovium, and ligaments.

- **Ultrasonography** – Ultrasonography is another imaging modality that can identify OA-associated structural changes and is useful for detecting synovial inflammation, effusion, and osteophytosis. Limitations of ultrasound include that it is operator-dependent and cannot be used to assess deeper articular structures and subchondral bone.

**Synovial fluid** — Synovial fluid from OA joints is usually noninflammatory or mildly inflammatory with less than 2000 white blood cells/mm³, predominantly mononuclear cells. Inflammatory effusion in OA may occur in the presence of calcium pyrophosphate crystals (see ‘Osteoarthritis with calcium pyrophosphate deposition’ below). A detailed discussion on synovial fluid analysis is presented elsewhere. (See "Synovial fluid analysis".)

**Osteoarthritis with calcium pyrophosphate deposition** — Calcium pyrophosphate crystals may be present in as many as 30 to 60 percent of unselected OA patients [22]. Most patients with OA and calcium pyrophosphate deposition (CPPD) are older than 60 years, and common target sites are the knees, radiocarpal joints, second and third MCP joints, shoulder, and elbow joints [23,24]. The presence of CPPD may modify OA symptoms at that site, especially with longer early morning stiffness and more signs of synovitis. The symptoms may be acute with marked pain, swelling, and tenderness, at its worst within 6 to 24 hours, typically lasting from a few days to one to two weeks; intermittent; or low-grade and persistent (chronic inflammatory CPPD disease). Joint swelling, warmth, and tenderness may be more common and more pronounced than in OA without CPPD (picture 3). Joint effusions are common and may be hemorrhagic or turbid on aspiration. Large joint effusions may leak into the surrounding soft tissues and lead to localized pain, swelling, and extensive bruising, especially at the shoulder and knee. Symptoms are mostly restricted to one or a few joints at a time, but polyarticular involvement can occur, especially involving knees, wrists, and MCP joints, that superficially may mimic RA.

Although studies give conflicting results, it is likely that OA with CPPD is not any more rapidly progressive than OA alone [25-27], and patients with end-stage knee OA with CPPD do not have any more difficulty with activities than those with end-stage knee OA alone [27]. However, a few patients with CPPD do appear to develop rapidly progressive destructive arthropathy at knees, shoulders, or hips. (See "Clinical manifestations and diagnosis of calcium pyrophosphate crystal deposition (CPPD) disease", section on 'Osteoarthritis with CPPD'.)

**CHARACTERISTICS OF SPECIFIC JOINT INVOLVEMENT** — Many of the characteristic clinical manifestations of osteoarthritis (OA) are related to the involvement of particular joints. As described above, OA has a predilection for the hand, knee, hip, and spine, and less commonly affects the shoulder, elbow, wrist, and ankle.

**Hand** — Symptoms are usually bilateral, and joint involvement is usually approximately symmetrical [15,28]. Typical symptoms affect just one or a few joints at a time [15]. Symptoms can be intermittent and target characteristic sites, i.e., distal interphalangeal (DIP) joints, thumb bases, proximal interphalangeal (PIP) joints, and second and third metacarpophalangeal (MCP) joints, in descending order of frequency [15]. Individuals without pain may still report an "aching" or stiffness in the hands [28]. Symptoms may worsen in approximately half of patients over the next six years, and the predictors of adverse clinical outcomes include a high level of baseline functional impairment and a greater number of painful joints, with no correlation between clinical symptoms and radiographic progression [29].

**Nodal osteoarthritis** — Heberden’s and/or Bouchard’s nodes plus underlying interphalangeal OA constitute nodal OA [15]. Affected people are frequently women, often with a remarkable familial predisposition. Symptoms usually start in middle age, typically around menopause, with a stuttering onset of pain, tenderness, and stiffness of
one or a few finger interphalangeal joints. At the start, there may be intermittent warmth and soft-tissue swelling, but over a period of a few years the involved interphalangeal joints usually become less painful, and signs of inflammation subside, leaving behind firm-hard bony swellings on the posterolateral aspect of the interphalangeal joints, termed Heberden's and Bouchard's nodes (picture 1 and picture 2). Heberden's nodes sometimes coalesce to form a single dorsal bar (picture 1). Over a period of several years, new interphalangeal joints go through the same process in a "mono-arthritis multiplex" fashion. In addition to nodes, affected interphalangeal joints may show restriction in movement and lateral deviation (radial or ulnar, with most deviations pointing towards the middle finger). Lateral deviation of interphalangeal joints, without instability, is a characteristic feature of nodal OA (picture 2). Nodes most frequently occur at the index and middle fingers [28]. Fully evolved nodes usually are not painful but may be a cosmetic problem.

Thumb-base OA generally affects older postmenopausal women. Individuals with thumb-base OA (OA of the first carpometacarpal and/or scaphotrapeziotrapezoid [STT] joint) have localized deep thenar, radial wrist, or thumb-base pain, exacerbated on joint use. There may also be distal radiation into the thumb and, to a lesser extent, proximally. Activities that involve pinching (opposition of the thumb to a finger) are generally most painful. A subjective grinding sensation on movement may be present. There may be radial subluxation and adduction at the thumb base, giving it a swollen "squared" appearance (picture 4) [15,28]. Localized tenderness may be present and passive thumb circumduction may be painful. Unlike interphalangeal joint OA, thumb-base OA sometimes associates with persistent symptoms and functional impairment (occasionally requiring surgery). Thumb-base OA may also occur on its own without nodal interphalangeal OA.

At the MCP joints, OA mainly targets the second and third MCP joints, most often causing bony enlargement without signs of synovitis. As described above, relatively isolated MCP joint OA sometimes occurs in older men who have had physically demanding occupations ("Missouri metacarpal syndrome") [30]. (See 'Single- or multiple-joint osteoarthritis' above.)

**Erosive osteoarthritis** — Erosive OA is an uncommon and particularly aggressive subset of hand OA. It presents with a subacute or insidious onset of pain, stiffness, soft-tissue swelling, and sometimes paresthesiae affecting multiple interphalangeal joints (ie, synchronous polyarticular onset) [15,31]. Compared with nodal hand OA, pain, tenderness, and inflammation (warmth, soft-tissue swelling, sometimes erythema) are more marked and prolonged [31,32], and erosive OA is not associated with generalized OA (OA). Erosive OA targets just interphalangeal joints (the DIP joints more frequently than PIP joints) and usually spares the thumb bases and MCP joints (picture 5) [15,32]. Lateral instability at the interphalangeal joints is an occasional but characteristic finding (picture 6), and spontaneous interphalangeal joint fusion may also occur (joint fusion is not a feature of OA). Rarely, there may be an opera glass deformity [32], and Heberden's and/or Bouchard's nodes may coexist [33].

Erosive OA has worse outcome in terms of symptom persistence and functional impairment than non-erosive hand OA [16]. Although erosive OA as a clinical entity is rare, radiographic subchondral erosions are present in one or a few joints in up to 8.5 percent of patients with symptomatic hand OA [34].

**Knee** — The knee is an important target site for OA and worldwide is the commonest single cause of lower-limb disability in adults over age 50. Knee OA is usually bilateral, although one side may be more severely affected. The patellofemoral joint and/or the medial tibiofemoral joint are most commonly affected, and isolated lateral tibiofemoral joint OA is relatively rare.

Pain location may indicate the affected knee compartment. Pain may be anteromedial or more generalized on the medial side in medial-compartment tibiofemoral joint OA or anterior in patellofemoral joint OA [35]. Pain from patellofemoral joint OA is exacerbated by prolonged sitting, standing up from a low chair, and climbing stairs or inclines (coming down often being more painful than going up). More widespread anterior knee pain with distal radiation suggests moderate to severe knee OA [36], and persistent pain at night that interrupts sleep or rest occurs in advanced OA [37]. Knee OA usually does not cause posterior knee pain unless there is a complicating popliteal
(Baker’s) cyst. The patients also report a feeling of “giving way” (especially with patellofemoral joint OA and/or quadriceps weakness) and instability, both of which can associate with falls [37].

Examination reveals typical findings (Table 1), and possibly deformities (eg, fixed flexion and/or varus, less commonly valgus), quadriceps weakness and wasting, and hip abductor and other muscle weakness may be present (Picture 7) [37-39]. Knee effusion is common, though usually mild or modest, and increases in prevalence with the severity of knee OA [40].

**Hip** — Hip OA presents with pain, aching, stiffness, and restricted movement. Pain due to hip OA is usually felt deep in the anterior groin but may involve the anteromedial or upper lateral thigh and occasionally the buttocks. Distal radiation is not uncommon, and some individuals present with distal thigh and/or knee pain without any proximal symptoms. However, unlike pain originating from the knee, such hip-referred pain is usually more generalized and may be improved by rubbing. Pain is exacerbated particularly by rising from a seated position and during the initial phases of ambulation [41]. Unlike knee OA, hip OA is frequently unilateral [42]. Both active and passive hip movements are equally painful [41]. Internal rotation with the hip flexed is frequently the earliest, and most affected movement (Picture 8) [41]. The typical end-stage deformity of hip OA is external rotation, adduction, and fixed flexion (Picture 9). Wasting of thigh muscles, a positive Trendelenburg test (Figure 3), and shortening of the affected extremity may also be present [41].

In some patients, especially older adult women, hip OA can be rapidly progressive, with a subacute onset of joint pain which progresses to joint destruction and instability in just a few months. Such rapidly progressive destructive arthropathy has been associated with basic calcium phosphate (mainly hydroxyapatite) crystals and is termed apatite associated destructive arthropathy (ADA) [43-45]. Shoulders (“Milwaukee shoulder”) [46] and knees are other target sites. Muscle wasting, deformities, and moderate to large joint effusions with "noninflammatory" (viscous, occasionally hemorrhagic, low cell count) synovial fluid are common in rapidly progressive large-joint OA.

**Facet joint** — Facet joint OA usually coexists with intervertebral disc degeneration, often loosely termed “spondylosis.” It is difficult to isolate symptoms specifically to facet joint OA. However, lumbar facet joint OA leads to localized lumbar pain, which may radiate unilaterally or bilaterally to the buttocks, groin, and thighs, typically ending above the knees [47]. Symptoms are worse in the morning and during periods of activity and are increased by stress, exercise, lumbar spine extension, rotary motions, and when standing or sitting [47]. Similarly, cervical facet joint OA may present with ipsilateral neck pain, which does not radiate beyond the shoulder and is aggravated by neck rotation or lateral flexion [48]. The clinical manifestations of neck and back pain are discussed in detail separately. (See “Evaluation of low back pain in adults” and “Acute lumbosacral radiculopathy: Pathophysiology, clinical features, and diagnosis” and “Evaluation of the patient with neck pain and cervical spine disorders” and “Evaluation of the patient with neck pain and cervical spine disorders”, section on ‘Cervical spondylosis’.)

**First metatarsophalangeal joint** — First metatarsophalangeal (MTP) joint OA is usually bilateral, and when symptomatic leads to localized big-toe pain on standing and during ambulation (especially during the “toe-off” stage of gait). Bony enlargement of the first MTP joint is a common finding. Hallux valgus deformity (when the distal end of big toe points towards the midline of the foot), hallux rigidus (or restricted flexion, and extension at the first MTP joint), and cross-over toes are common deformities. Bony enlargement at the first MTP joint and hallux valgus frequently lead to the development of a complicating bursa with additional fibrous tissue reaction on the medial aspect of the first MTP joint (“bunion”). This may get inflamed, for example by rubbing against any footwear.

Apart from the first MTP joint, OA also commonly targets the talonavicular joint in the midfoot (aggravated by pes planus) and also the subtalar and tibiotalar joints in the hindfoot.

**Differential Diagnosis** — The differential diagnosis for osteoarthritis depends largely on the location of the affected site as well as the presence of absence of additional systemic symptoms. We present several alternative diagnoses that may be considered in the appropriate clinical context. However, most disorders can usually be easily distinguished from osteoarthritis (OA).
**Rheumatoid arthritis** – OA in the middle-aged or older adult patient is most commonly confused with rheumatoid arthritis (RA) when it involves the hand joints. However, the different patterns of clinical involvement will usually lead to the correct diagnosis (table 2) see "Diagnosis and differential diagnosis of rheumatoid arthritis". The following are examples:

- Nodal OA of the hands typically affects the distal interphalangeal (DIP) joints and is frequently associated with the highly characteristic Heberden’s nodes (picture 1). By contrast, RA typically affects the metacarpophalangeal (MCP) and proximal interphalangeal (PIP) joints, and Heberden's nodes are absent. The carpometacarpal (CMC) joint of the thumb is typically involved in OA, rather than the PIP joint in RA. Swelling of the joints is hard and bony in OA; by comparison, soft, warm, and tender joint swelling is typical of RA.

- Stiffness of the joint is a very common feature of RA, but it is a relatively rare feature of OA. Furthermore, the stiffness of RA is characteristically worse after resting the joint (eg, morning stiffness), while the stiffness of OA (if present) is typically worse after any effort and is often described as evening stiffness.

- OA is characterized radiographically by narrowing of the joint space due to cartilage loss and osteophytes due to bone remodeling, but not periarticular erosions (image 1). Sea-gull (central subchondral) erosions are present in joints with erosive OA (image 2). However, many patients with longstanding RA may develop secondary OA.

- OA is classically associated with the absence of rheumatoid factor (RF) and antibodies to cyclic citrullinated peptide (CCP). OA is associated with normal levels of acute phase reactants. However, RF may be present, usually in low titer, consistent with the patient's (older) age. In addition, the erythrocyte sedimentation rate (ESR) and serum C-reactive protein (CRP) concentration may be somewhat elevated, usually secondary to an associated disease.

**Psoriatic arthritis** – Psoriatic arthritis targets the DIP joints of the hands, which can be observed in hand OA. However, unlike hand OA, psoriatic arthritis may target just one finger, often as dactylitis, and characteristic nail changes are usually present. (See "Clinical manifestations and diagnosis of psoriatic arthritis".)

**Crystalline arthritis** – Crystalline arthritis (gout and pseudogout) can become chronic and even assume a polyarticular distribution involving the fingers (tophaceous gout), wrists, knees, and other large joints. The diagnosis is established by the finding of urate or calcium pyrophosphate crystals, respectively, in synovial fluids. The presence of tophi on physical examination, and the characteristic appearance of punched out juxta-articular gouty erosions are also useful in distinguishing OA from gout (image 3). Calcium pyrophosphate crystal deposition (CPPD) disease can be diagnosed if there is radiographic articular chondrocalcinosis (image 4). (See "Clinical manifestations and diagnosis of gout" and "Clinical manifestations and diagnosis of calcium pyrophosphate crystal deposition (CPPD) disease".)

**Hemochromatosis** – The arthropathy of iron overload may be confused with hand OA. However, unlike hand OA, hemochromatosis targets the MCP joints and wrists, and it predominates in men. The characteristic radiologic findings of hemochromatosis are squared-off bone ends and hook-like osteophytes in the MCP joints, particularly of the second and third MCP joints (image 5). Chondrocalcinosis may also be present. (See "Arthritis and bone disease associated with hereditary hemochromatosis".)

**Infectious arthritis** – OA of a single joint is usually associated with mild symptoms and a noninflammatory synovial fluid (white blood cell count <2000 cells/mm³) but can also present as an acutely painful synovitis that may mimic infection [49]. The diagnosis of infectious arthritis is established by culturing the pathogen from the synovial fluid or from the blood. Patients with septic arthritis may or may not appear toxic on examination, depending upon the stage of their infection, the presence of medications that can mask infection (eg, glucocorticoids), and other clinical variables. Peripheral blood leukocytosis with a left shift is common but not invariably present. (See "Septic arthritis in adults".)
• **Other soft-tissue abnormalities** – Other soft-tissue abnormalities around a single joint may mimic OA. As an example, pain from hip OA must be distinguished from labral impingement and/or tear, avascular necrosis of the femoral head, and developmental hip dysplasia (anterior groin pain); greater trochanter pain syndrome (trochanteric bursitis or tendinitis, enthesis of gluteus medius, lateral thigh pain); and lumbar radiculopathy, sacroiliac joint dysfunction, and hip extensor or rotator muscle strain (posterior pelvic pain) [50]. (See "Evaluation of the adult with hip pain".)

**DIAGNOSIS** — Osteoarthritis (OA) may be diagnosed without the use of radiography and/or laboratory investigations in the presence of typical symptoms and signs in the at-risk age group [1,19,20].

**Clinical diagnosis** — Peripheral joint OA may be diagnosed confidently on clinical grounds alone if the following are present:

- Persistent usage-related joint pain in one or few joints
- Age ≥45 years
- Morning stiffness ≤30 minutes [1]

The presence of other clinical features of OA add to the diagnostic certainty (table 1) [1]. This approach to a clinical diagnosis is supported by the fact that radiographically assessed structural changes may be present in the absence of symptoms and vice versa [19,20]. (See 'Characteristics of specific joint involvement' above and 'Imaging' above.)

**When to consider additional testing** — Appropriate imaging and laboratory investigations should be carried out in:

- Younger individuals with joint symptoms/signs of OA
- Presence of atypical symptoms and signs such as an unusual site of involvement, symptoms and signs of joint inflammation, marked rest and/or night pain, and rapidly progressive pain
- Presence of weight loss or constitutional symptoms
- Those with knee pain and true "locking," which suggests additional mechanical derangement

Additional laboratory testing may include an erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP). Inflammatory markers are normal in OA and may be useful in excluding other diagnoses. In patients with hand arthralgias and a mix of inflammatory and mechanical joint symptoms, rheumatoid factor (RF) and anti-cyclic citrullinated peptide (CCP) antibodies may be checked to evaluate for possible of rheumatoid arthritis (RA). (See "Diagnosis and differential diagnosis of rheumatoid arthritis".)

Radiographic examination may be used to support a diagnosis of OA but is not a routine test to consider as a means to explain clinical symptoms. Patients with a robust diagnosis of OA on clinical grounds may have normal plain radiographs, and vice versa. Also, knee pain on most days of a month can precede radiographic changes of OA by several years [51]. Radiographic examination may have a role in defining the prognosis of patients with symptomatic OA [52]. However, patients should be examined carefully to exclude any other cause of joint pain, such as periarticular soft-tissue lesions (see "Overview of soft tissue rheumatic disorders"). When then there is still diagnostic uncertainty regarding the cause of joint pain, advanced imaging with magnetic resonance imaging (MRI) or ultrasonography may also be helpful. (See 'Imaging' above.)

Synovial fluid examination is not routinely required to support a diagnosis of OA. (See 'Synovial fluid' above.)

**CLASSIFICATION CRITERIA** — Osteoarthritis (OA) can be classified according to the joints affected, age of onset, radiographic appearance ("hypertrophic" versus "atrophic"), presumed etiology (eg, "secondary posttraumatic" OA), and rate of progression. Several classification systems have been proposed, each with its own strengths and weaknesses.
Common problems with classification criteria include:

- Frequent overlap, with lack of clear separation between "subsets." As an example, subchondral changes of "erosive OA" are not uncommon in just a few interphalangeal joints in nodal OA.

- Common coexistence of separate subsets, or evolution from one form of arthritis to another within an individual (eg, a patient with nodal-generalized OA may subsequently develop calcium pyrophosphate crystal deposition [CPPD] at the knees, and rapidly progressive "atrophic" glenohumeral OA)

The American College of Rheumatology (ACR) classification of OA is the most widely used classification system [14] but has several limitations, including artificial separation of nodal and erosive (non-nodal) OA as two distinct subsets of hand OA; and inclusion of intervertebral disc degeneration and Forestier's disease as a subset of spinal OA, even though OA is pathologically confined to synovial joints. The guideline development group has recognized that these distinctions are arbitrary and have yet to be validated [14,23].

SOCIETY GUIDELINE LINKS — Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Osteoarthritis".)

SUMMARY

- The primary symptoms of osteoarthritis (OA) are joint pain, stiffness, and locomotor restriction. They usually present in just one or a few joints in a middle-aged or older person. (See 'Clinical manifestations' above.)

- Many of the characteristic clinical manifestations of OA are related to the involvement of particular joints. OA has a predilection for the knees, hips, interphalangeal joints, first carpometacarpal (CMC) joints, first metatarsophalangeal (MTP) joints, and apophyseal (facet) joints of the lower cervical and lower lumbar spine [10] (figure 1). (See 'Characteristics of specific joint involvement' above and 'Single- or multiple-joint osteoarthritis' above.)

- Generalized OA implies a polyarticular subset of OA involving the distal interphalangeal (DIP) joints, thumb bases (first CMC joints and trapeziocaphoid joints), first MTP joints, lower cervical and lumbar facet joints, knees, and hips. (See 'Generalized osteoarthritis' above.)

- Peripheral joint OA may be diagnosed confidently on clinical grounds alone if the following are present (see 'Clinical diagnosis' above):
  - Persistent usage-related joint pain in one or few joints
  - Age ≥45 years
  - Morning stiffness ≤30 minutes

- Appropriate imaging and laboratory investigations are reserved for patients presenting with atypical symptoms and signs such an unusual site of involvement, symptoms and signs of joint inflammation, marked rest and/or night pain, and rapidly progressive pain. (See 'When to consider additional testing' above.)

- The differential diagnosis for OA depends largely on the location of the affected site as well as the presence of absence of additional systemic symptoms. The differential diagnosis includes rheumatoid arthritis (RA), psoriatic arthritis, crystalline arthritis (gout or pseudogout), hemochromatosis, infectious arthritis, and other soft-tissue abnormalities. (See 'Differential diagnosis' above.)

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REFERENCES


Topic 105723 Version 9.0
Joints affected in osteoarthritis

Target symptomatic joints for OA.

OA: osteoarthritis.


Graphic 105815 Version 1.0
### Principal manifestations of osteoarthritis

#### Patient characteristics

<table>
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<tr>
<th>Characteristics</th>
<th>Detail</th>
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<td>Age of onset</td>
<td>&gt;40 years*</td>
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#### Symptoms

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<th>Symptom</th>
<th>Detail</th>
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| Pain    | Affects one or a few joints at a time  
|         | Insidious onset - slow progression over years  
|         | Variable intensity  
|         | May be intermittent  
|         | Increased by joint use and relieved by rest  
|         | Night pain in severe osteoarthritis  |
| Stiffness| Short-lived (<30 minutes) and early morning- or inactivity-related |
| Swelling| Some (eg, nodal osteoarthritis) patients present with swelling and/or deformity |
| Constitutional symptoms | Absent |

#### Physical exam findings

<table>
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<th>Examination</th>
<th>Detail</th>
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| Appearance  | Swelling (bony overgrowth ± fluid/synovial hypertrophy)  
|             | Attitude  
|             | Deformity  
|             | Muscle wasting (global - all muscles acting over the joint)  |
| Palpation   | Absence of warmth  
|             | Swelling (effusion if present is usually small and cool)  
|             | Joint line tenderness  
|             | Periarticular tenderness (especially knee, hip)  |
| Range of motion | Crepitus (knee, thumb bases)  
|                | Reduced range of movement  
|                | Weak local muscles  |

OA: osteoarthritis.
* Major joint injury and certain rare conditions may predispose to OA before the age of 40 years.

Adapted from: OARSI Primer (http://primer.oarsi.org).

Graphic 108692 Version 1.0
Bones of the hand and wrist

Phalanges
Distal

Middle

Proximal

Metacarpophalangeal joints

Metacarpals

Carpals
Trapezium
Trapezoid
Hamate
Capitate
Pisiform
Triquetral
Scaphoid
Hook of hamate
Lunate

Radius
Ulna

Palmar view of the wrist and hand bones.


Graphic 51990 Version 3.0
Heberden's nodes

Heberden's nodes, appearing as discrete postero-lateral swellings (index finger) or as a dorsal bar (middle finger) over the DIP joints.

DIP: distal interphalangeal.


Graphic 105816 Version 1.0
Heberden's nodes (thumb, middle, ring, and little finger DIP joints), Bouchard's nodes (index finger PIP joint), and lateral radial/ulnar deviation (index PIP joint, ring DIP joint) in the left hand of a person with nodal OA.

DIP: distal interphalangeal; PIP: proximal interphalangeal; OA: osteoarthritis.


Graphic 105817 Version 2.0
Knee effusion

Knee effusions are usually not marked in OA. This person had a large effusion expanding the suprapatellar pouch, with positive balloon sign (fluctuance) and patellar tap, and OA with CPPD.

OA: osteoarthritis; CPPD: calcium pyrophosphate crystal deposition.


Graphic 105825 Version 2.0
**Thumb-base osteoarthritis**

Thumb-base OA: prominence and "squaring" of the thumb base, due to osteophyte formation and subluxation at the first CMC joint.

OA: osteoarthritis; CMC: carpometacarpal.


Graphic 105818 Version 1.0
Erosive osteoarthritis of the hand

Erosive hand OA with marked radial deviation and fixed flexion deformity in the left middle PIP joint, radial deviation with restriction in the index PIP joint, and bony swelling of both fingers. Note the absence of Heberden's nodes.

OA: osteoarthritis; PIP: proximal interphalangeal.


Graphic 105819 Version 1.0
Erosive osteoarthritis

Marked radial/ulnar instability in a patient with erosive OA. Such instability does not usually occur with common hand OA.

OA: osteoarthritis.


Graphic 107133 Version 1.0
Patient with unilateral knee osteoarthritis

Unilateral knee OA: swollen left knee with varus and fixed flexion deformity in a 63-year-old man with a prior history of knee trauma. On palpation there was marked crepitus, restricted flexion, bony swelling, and a small effusion. The cruciates were intact, but there was minor varus/valgus instability on stress testing.

OA: osteoarthritis.


Graphic 105821 Version 1.0
Findings on hip examination in patient with osteoarthritis

Patient with right hip OA, showing painful restriction with internal rotation in flexion. This is the “tight-pack” position for the hip (when the capsule is at its tightest) and is the first movement to be affected.

OA: osteoarthritis.


Graphic 105824 Version 2.0
Findings in patient with right hip osteoarthritis

Patient with right hip OA, showing fixed flexion and external rotation deformity.

OA: osteoarthritis.


Graphic 105823 Version 1.0
Trendelenburg test

Normally, the pelvis stays level when a patient stands on one leg. When standing on the affected leg, the pelvis tilts downward toward the unaffected side (as pictured in the abnormal patient above) because of gluteal muscle weakness on the affected side (right side in abnormal patient above).

Graphic 67453 Version 3.0
Clinical distinction between rheumatoid arthritis and osteoarthritis

<table>
<thead>
<tr>
<th>Feature</th>
<th>Rheumatoid arthritis</th>
<th>Osteoarthritis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary joints affected</td>
<td>Metacarpophalangeal</td>
<td>Distal interphalangeal</td>
</tr>
<tr>
<td></td>
<td>Proximal interphalangeal</td>
<td>Carpometacarpal</td>
</tr>
<tr>
<td>Heberden’s nodes</td>
<td>Absent</td>
<td>Frequently present</td>
</tr>
<tr>
<td>Joint characteristics</td>
<td>Soft, warm, and tender</td>
<td>Hard and bony</td>
</tr>
<tr>
<td>Stiffness</td>
<td>Worse after resting (e.g., morning stiffness)</td>
<td>If present, worse after effort, may be described as evening stiffness</td>
</tr>
<tr>
<td>Laboratory findings</td>
<td>Positive rheumatoid factor</td>
<td>Rheumatoid factor-negative</td>
</tr>
<tr>
<td></td>
<td>Positive anti-CCP antibody</td>
<td>Anti-CCP antibody-negative</td>
</tr>
<tr>
<td></td>
<td>Elevated ESR and CRP</td>
<td>Normal ESR and CRP</td>
</tr>
</tbody>
</table>

CCP: cyclic citrullinated peptide; ESR: erythrocyte sedimentation rate; CRP: C-reactive protein.

Graphic 57005 Version 5.0
Osteoarthritis of the distal interphalangeal (DIP) joints

This plain film demonstrates complete loss of the articular cartilage at all four DIP joints, large osteophytes, and ankylosis of the DIP joint of the middle finger.

Courtesy of Bruce C Anderson, MD.

Graphic 55802 Version 2.0
Erosive hand osteoarthritis

Plain radiograph of the hand demonstrating features consistent with erosive osteoarthritis (OA) of the hand. There is a central subchondral erosion and pseudo-widening at the proximal interphalangeal (PIP) joint of the fourth finger, and bony proliferation at the diaphysis. The distal interphalangeal (DIP) joint of the fifth finger has a central erosion. The DIPs of the fifth and index fingers also show subluxation, disorganization, and radial deviation. There is marked narrowing and subluxation of the first carpometacarpal (CMC) joint, which is sometimes a key feature of erosive hand OA.

Courtesy of Michael Doherty, MD, and Abhishek Abhishek, MD.

Graphic 109003 Version 2.0
Gouty arthritis

Plain radiograph of the hand demonstrating features consistent with gout. There are juxta-articular punched-out erosions and intraosseous cystic lesions, most prominent at the index proximal interphalangeal (PIP) joint and distal ulna. There is cartilage loss at the little finger PIP joint and soft tissue swelling due to a subcutaneous tophus at the index finger PIP joint. Vascular calcification of forearm vessels is also evident.

*Image courtesy of Dr. Ian Gaywood, Consultant Rheumatologist, Nottingham University Hospitals NHS Trust.*

Graphic 109001 Version 1.0
Osteoarthritis and CPPD

Plain radiograph of the hand demonstrating features of osteoarthritis (OA) and calcium pyrophosphate deposition (CPPD). There is cartilage loss, osteophytosis, and bony remodeling at all finger distal interphalangeal (DIP) joints and radial deviation of middle, ring, and little finger DIP joints; joint space narrowing, osteophytosis, and cysts at the second metacarpophalangeal (MCP) joint; and thumb-base OA (mainly narrowing and sclerosis of first carpometacarpal [CMC] and scapho-trapezioide joints). There is also scapho-lunate dissociation (which is reported to associate with CPPD) and chondrocalcinosis in the triangular cartilage.

Courtesy of Michael Doherty, MD, and Abhishek Abhishek, MD.

Graphic 109002 Version 2.0
Hand arthritis in hemochromatosis

Serial radiographs, five years apart, of the metacarpophalangeal joints in a patient with hereditary hemochromatosis. The second film shows loss of joint space at the metacarpophalangeal articulations, cyst formation, and a hook-like osteophyte on the radial aspect of the metacarpal head of the middle finger.

_Courtesy of John S Axford, BSc, MD, FRCP._

Graphic 78740 Version 2.0
Contributor Disclosures

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