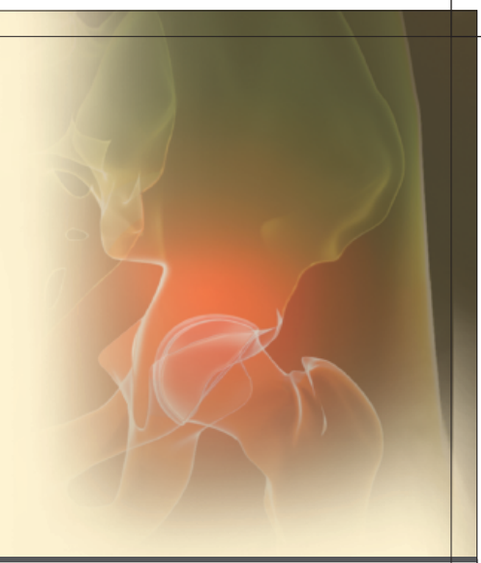
Rheuma Facts

A Quarterly Magazine

7th Issue, July 2015



Current News

Consider LDL-C and HDL-C when estimating CV risk in RA

By: NICOLA GARRETT, Rheumatology News Digital Network
MAY 2, 2015

FROM ARTHRITIS & RHEUMATOLOGY

Despite a similar relationship between lipid levels and cardiovascular disease risk in patients with or without rheumatoid arthritis, people with RA have an almost two-fold increased risk of having a major cardiovascular event, research shows.

The findings confirm that existing CV risk calculators are suboptimal for patients with RA and that improved methods of measuring risk in this population are needed, say the study authors from the Brigham and Women's Hospital in Boston. They also show that there may be potential benefit in considering both LDL-C and HDL-C levels when estimating CV risk in RA, according to Dr. Katherine P. Liao and her associates from Brigham and Women's Hospital, Boston (Arthritis & Rheumatology 2015).

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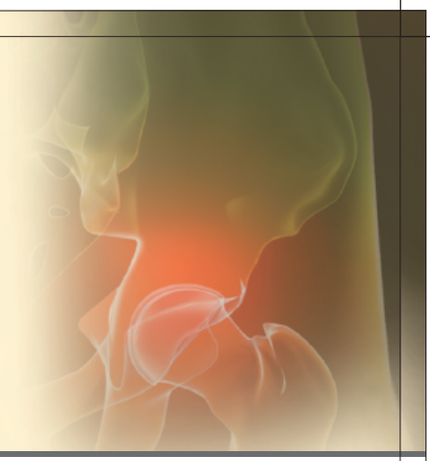

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Diagnosing an Acute Hot Swollen Joint in Adults
A Practical Approach

Compiled by:
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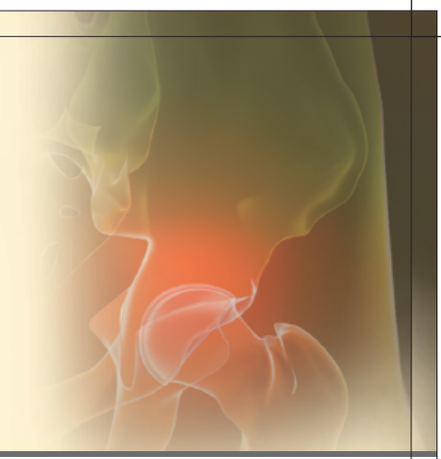


Acute monoarthritis can be the initial manifestation of many joint disorders. The first step in diagnosis is to verify that the source of pain is the joint, not the surrounding soft tissues. The most common causes of monoarthritis are crystals (i.e., gout and pseudogout), trauma, and infection. A careful history and physical examination are important because diagnostic studies frequently are only supportive. Examination of joint fluid often is essential in making a definitive diagnosis. Leukocyte counts vary widely in septic and sterile synovial fluids and should be interpreted cautiously. If the history and diagnostic studies suggest septic arthritis, aggressive treatment can prevent rapid joint destruction. When an infection is suspected, culture and Gram staining should be performed and antibiotics should be started. Light microscopy may be useful to identify gout crystals, but polarized microscopy is preferred. Blood tests alone never confirm a diagnosis, and radiographic studies are diagnostic only in selected conditions. Referral is indicated when patients have septic arthritis or when the initial evaluation does not determine the etiology.

Diagnosing Acute Monoarthritis

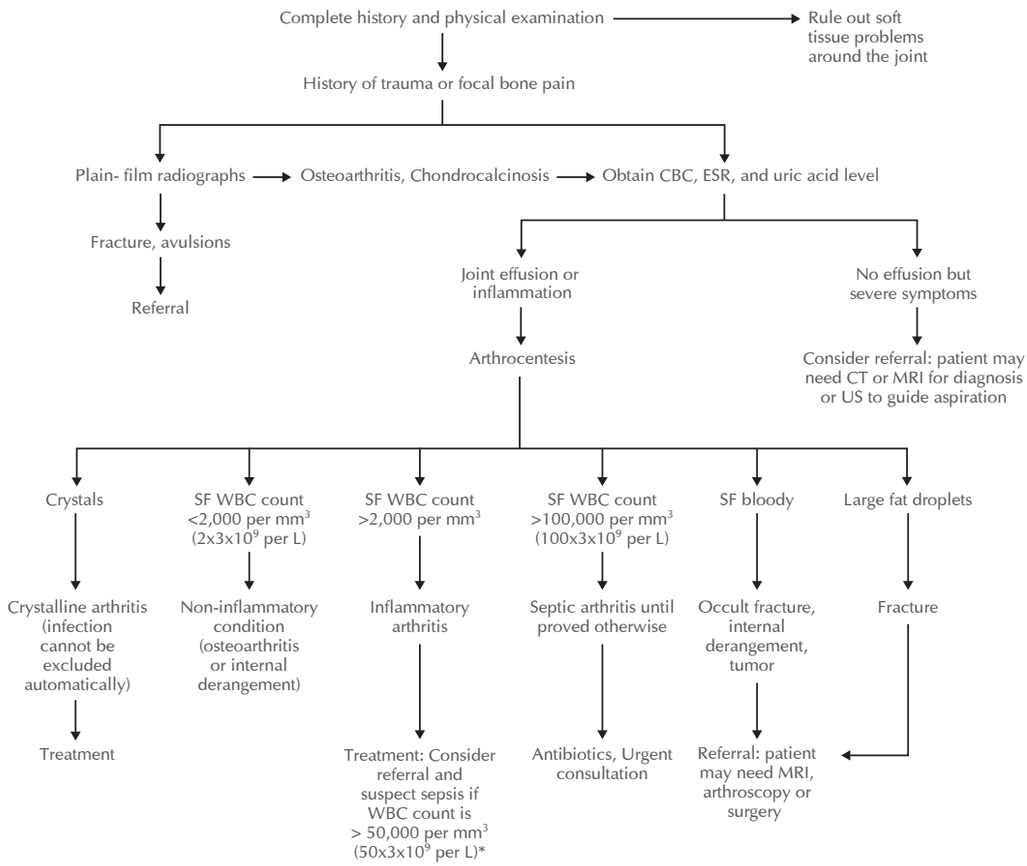
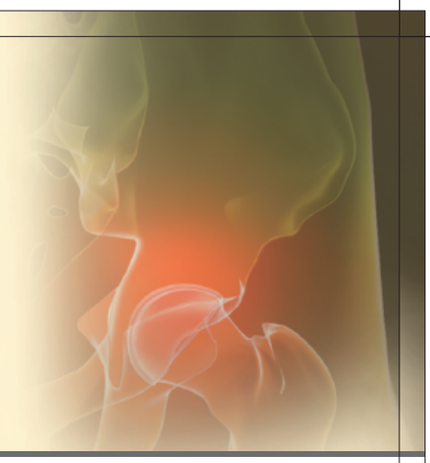


FIGURE 1.
A suggested algorithm for the evaluation of patients who present with acute monoarthritis. (CBC = complete blood count; ESR = erythrocyte sedimentation rate; CT = computed tomography; MRI = magnetic resonance imaging; US = ultrasonography; SF = synovial fluid; WBC = white blood cell count)



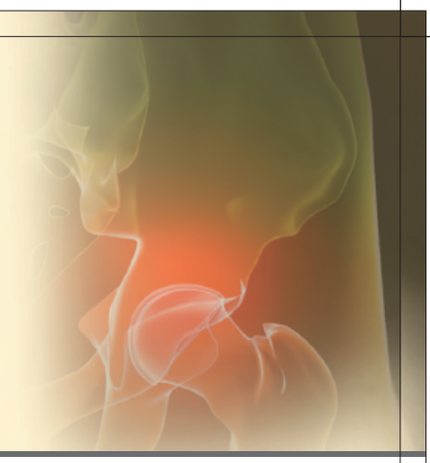
Etiology of Acute Monoarthritis

Acute monoarthritis in adults can have many causes (Table 1), but crystals, trauma, and infection are the most common. Prompt diagnosis of joint infection, which often is acquired hematogenously, is crucial because of its destructive course. The most important risk factors for septic arthritis are a prosthetic hip or knee joint, skin infection, joint surgery, rheumatoid arthritis, age greater than 80 years, and diabetes mellitus. Intravenous drug use and large-vein catheterization are predisposing factors for sepsis in unusual joints (e.g., sternoclavicular joint).

TABLE 1

Causes of Acute Monoarthritis

Common causes	Less common causes	Rare causes
Avascular necrosis of bone	Bone malignancies	Amyloidosis
Crystals	Bowel-disease–associated arthritis	Behçet's syndrome
Monosodium urate	Hemoglobinopathies	Familial Mediterranean fever
Calcium pyrophosphate dehydrate	Juvenile rheumatoid arthritis	Foreign-body synovitis
Apatite	Loose body	Hypertrophic pulmonary
Calcium oxalate	Psoriatic arthritis	Osteoarthropathy
Hemarthrosis	Rheumatoid arthritis	Intermittent hydrarthrosis
Infectious arthritis	Reactive arthritis	Pigmented villonodular synovitis
Bacteria	Sarcoidosis	Relapsing polychondritis
Fungi		Still's disease
Mycobacteria		Synovioma
Viruses		Synovial metastasis
Lyme disease		Vasculitic syndromes
Internal derangement		
Osteoarthritis		
Osteomyelitis		
Overuse		
Trauma		



Gonococcal arthritis is the most common type of non-traumatic acute mono-arthritis in young, sexually active persons in the United States. It is three to four times more common in women than in men. Non-gonococcal septic arthritis, the most destructive type, generally is mono-articular (80 percent of cases) and most often affects the knees (50 percent of cases). Staphylococcus aureus is the most common pathogen in non-gonococcal septic arthritis (60 percent in some series), but non-group-A beta-hemolytic streptococci, gram-negative bacteria, and Streptococcus pneumoniae can be present.

Anaerobic and gram-negative infections are common in immuno-compromised persons. Inflammation of a single large joint, especially the knee, may be present in Lyme disease.
Mycobacterial, fungal, and viral infections are rare. Monoarticular inflammation can be the initial manifestation of human immunodeficiency virus (HIV) infection.

Many types of crystals can trigger acute monoarthritis, but monosodium urate (which causes gout) and calcium pyrophosphate dihydrate (CPPD, which causes pseudogout) are the most common. Calcium oxalate (especially in patients who are receiving renal dialysis), apatite, and lipid crystals also elicit acute monoarthritis. Transient arthritis sometimes results from intra-articular injection of corticosteroids. Osteoarthritis may worsen suddenly and manifest as pain and effusion. Spontaneous osteonecrosis may occur in patients with risk factors such as alcoholism or chronic corticosteroid use. Aseptic loosening is often the source of pain in a prosthetic joint. Infection, commonly from a skin source, is also possible and requires urgent attention.

History

Any acute inflammatory process that develops in a single joint over the course of a few days is considered acute mono-arthritis (also defined as mono-arthritis that has been present for less than two weeks). Establishing the chronology of symptoms is important (Table 2). Rapid onset over hours to days usually indicates an infection or a crystal-induced process. Fungal or mycobacterial infections usually have an indolent and protracted course but can mimic bacterial arthritis.

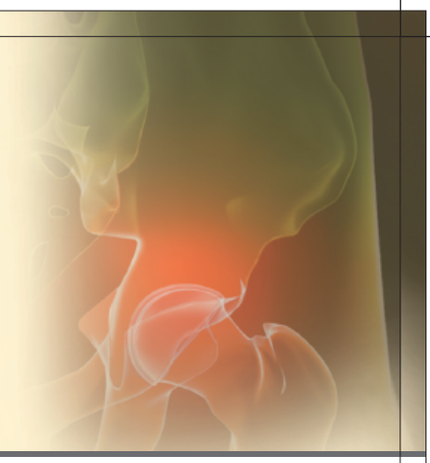


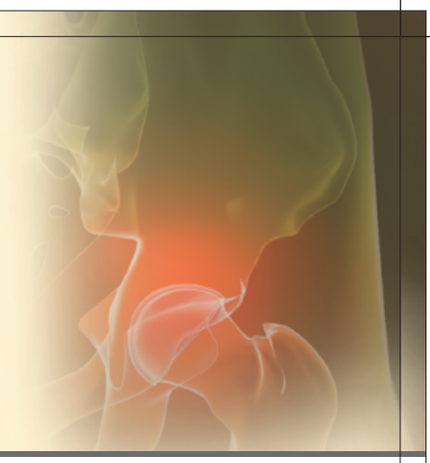
TABLE 2

Useful Diagnostic Clues in Patients Presenting with Joint Pain

CLUES FROM HISTORY AND PHYSICAL EXAMINATION	DIAGNOSES TO CONSIDER
Sudden onset of pain in seconds or minutes	Fracture, internal derangement, trauma, loose body
Onset of pain over several hours or one to two days	Infection, crystal deposition disease, other inflammatory arthritic condition
Insidious onset of pain over days to weeks	Indolent infection, osteoarthritis, infiltrative disease, tumor
Intravenous drug use, immunosuppression	Septic arthritis
Previous acute attacks in any joint, with spontaneous resolution	Crystal deposition disease, other inflammatory arthritic condition
Recent prolonged course of corticosteroid therapy	Infection, avascular necrosis
Coagulopathy, use of anticoagulants	Hemarthrosis
Urethritis, conjunctivitis, diarrhea, and rash	Reactive arthritis
Psoriatic patches or nail changes such as pitting	Psoriatic arthritis
Use of diuretics, presence of tophi, history of renal stones or alcoholic binges	Gout
Eye inflammation, low back pain	Ankylosing spondylitis
Young adulthood, migratory polyarthralgias, inflammation of the tendon sheaths of hands and feet, dermatitis	Gonococcal arthritis
Hilar adenopathy, erythema nodosum	Sarcoidosis

Fractures and ligamentous or meniscal tears resulting from trauma can present as mild to moderate monoarticular swelling. The pain characteristically worsens with movement and improves with rest. There may be no history of trauma in patients with fractures secondary to osteoporosis. Penetrating injuries, such as those from thorns, can cause acute synovitis, with symptoms sometimes occurring months after the injury.

Patients might note concurrent or pre-existent involvement of other joints. Sequential monoarthritis in several joints is characteristic



of gonococcal arthritis or rheumatic fever. Monoarthritis occasionally is the first presenting symptom of an inflammatory polyarthritis such as psoriatic arthritis but is an unusual initial symptom of rheumatoid arthritis. When the history reveals longstanding symptoms in a joint, exacerbations of pre-existing disease (e.g., worsening of osteoarthritis with excessive use) should be differentiated from a superimposed infection. In patients with rheumatoid arthritis, pain in one joint out of proportion to pain in other joints always suggests infection.

Sexual history and history of illegal drug use, alcohol use, travel, and tick bites should be ascertained. Reactive arthritis sometimes can develop after a gastrointestinal or sexually transmitted disease. Certain occupations, such as farming and mining, frequently are associated with overuse injures and osteoarthritis.

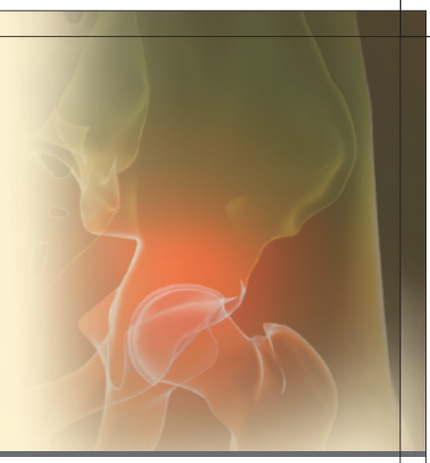
Pseudogout affecting the wrists and knees is most common among elderly persons. Disseminated gonococcal infection, reactive arthritis, and ankylosing spondylitis affect young adults. Gout, which occurs more often in men, affects the first metatarsophalangeal joint, ankle, mid-foot, or knee; accompanying fever, redness, and pain can mimic infection. Minor trauma can precipitate gout or introduce infection through a break in the skin.

Physical Examination

When a patient complains of joint pain, the first step is to determine whether the source of the pain is the joint or a periarticular soft tissue structure such as a bursa or tendon. It is not uncommon to find that "hip pain" actually is the result of trochanteric bursitis. Asking the patient to point to the exact site may be helpful. Unlike with true joint inflammation, redness or swelling generally is not present with periarticular pain. However, a patient with inflammation of certain bursae (e.g., prepatellar bursitis, olecranon bursitis) may present with redness or swelling that mimics joint inflammation.

True intra-articular problems cause restriction of active and passive range of motion, whereas periarticular problems restrict active range of motion more than passive range of motion. Maximum pain at the limit of joint motion (i.e., stress pain) is characteristic of true arthritis. In tendonitis or bursitis, joint movements against resistance elicit pain. For example, elbow pain resulting from septic arthritis occurs with active and passive motion in any direction. In contrast, elbow pain resulting from lateral epicondylitis (i.e., "tennis elbow") worsens with resisted active extension or supination of the wrist. Specific maneuvers can be diagnostic for other conditions, such as medial epicondylitis; bicipital and rotator cuff tendonitis; trochanteric bursitis; and patellar, prepatellar, and anserine bursitis.

Joint effusion may not be readily visible. In the knee joint, the **"bulge sign"** can signal a small effusion. The medial or lateral compartment is stroked, and the fluid moves through the suprapatellar area into the opposite compartment, resulting in a visible bulge. To detect effusion in the elbow joint, the triangular recess (area between lateral epicondyle, olecranon process, and radial head) in the lateral aspect should be palpated. To detect



effusion in the ankle, the joint should be palpated anteriorly. Maneuvers for examining other joints are reviewed elsewhere.

Joint pain may be referred from internal organs (e.g., shoulder pain in a patient with angina). Referred pain should be suspected in patients with a normal joint examination.

The general physical examination: May provide other diagnostic clues (Table 2) or reveal involvement of other joints. Fever and tachycardia may signal infection, but they are not reliable indicators, especially in immuno-compromised patients and patients who are taking corticosteroids or antibiotics. Patients with gonococcal infection may have a rash, pustules, or hemorrhagic bullae. Patients with longstanding gout may have tophi (i.e., firm subcutaneous deposits of urate) over the olecranon prominence, first metatarsal joints, or pinnae. Patients with reactive arthritis may have inflamed eyes. A new cardiac murmur and splinter hemorrhages in the nail folds suggest endocarditis.

Diagnostic Studies

Arthrocentesis is required in most patients with monoarthritis and is mandatory if infection is suspected. In some instances, obtaining as little as one or two drops of synovial fluid can be useful for culture and crystal analysis.

Superimposed cellulitis is a relative contraindication to arthrocentesis. The procedure can be performed safely in patients who are taking warfarin (Coumadin). An experienced physician should perform arthrocentesis in these patients and use the smallest possible needle size.

Removal of as much synovial fluid as possible offers symptomatic relief and helps to control infection. If the fluid is loculated, aspiration of large amounts of fluid will be difficult; massaging the joint may help increase the amount of fluid aspirated. If infection is suspected, intravenous antibiotics should be administered before culture results become available. If needle drainage is ineffective, urgent arthroscopic or surgical drainage is indicated.

Until infection has been ruled out, corticosteroids should not be injected into a joint.

If even the smallest suspicion of infection exists, synovial fluid should be sent for a white blood cell (WBC) count with differential (specifically, the percentage of polymorphonuclear neutrophilic leukocytes), crystal analysis, Gram staining, and culture.

Sterile tubes should be used for culture. If examinations are delayed, a tube with ethylenediaminetettraacetic acid should be used for anticoagulation, because anticoagulants (e.g., oxalate, lithium heparin) used in other tubes can confound crystal analysis. Synovial fluid cultures are more likely to be positive in patients with non-gonococcal arthritis (90 percent) than in those with gonococcal arthritis (less than 50 percent).

Synovial fluid may be categorized as noninflammatory, inflammatory, or hemorrhagic, depending on the appearance and cell counts (Table 3). Normal synovial fluid is colorless and transparent. Noninflammatory synovial fluid may be colorless or yellow and transparent enough to read through, whereas inflammatory synovial fluid is not transparent.

TABLE 3

Categorization of Synovial Fluid, with Associated Conditions

NONINFLAMMATORY: < 2,000 WBC PER MM^3(2X 10^9 PER L)	INFLAMMATORY: < 2,000 WBC PER MM^3
Osteoarthritis	Septic arthritis (usually > 50,000-100,000)
Trauma	Crystal-induced monoarthritis (e.g., gout, pseudogout)
Avascular necrosis	Rheumatoid arthritis
Charcot's arthropathy	Spondyloarthropathy
Hemochromatosis	Systemic lupus erythematosus
Pigmented villonodular synovitis	Juvenile rheumatoid arthritis, Lyme disease, other crystalline arthritides

WBC = white blood cell.
*—Synovial fluid analysis in patients with septic arthritis often shows more than 90 percent polymorphonuclear neutrophilic leukocytes.

If a polarized microscope is not available, a tentative diagnosis can be made if needle-shaped monosodium urate crystals are identified using an ordinary light microscope (Figure 2). CPPD crystals are smaller rods, squares, or rhomboids and are difficult to identify with light microscopy. The finding of crystals within leukocytes is virtually diagnostic of crystal-induced arthropathy but does not rule out a superimposed infection (Table 4).

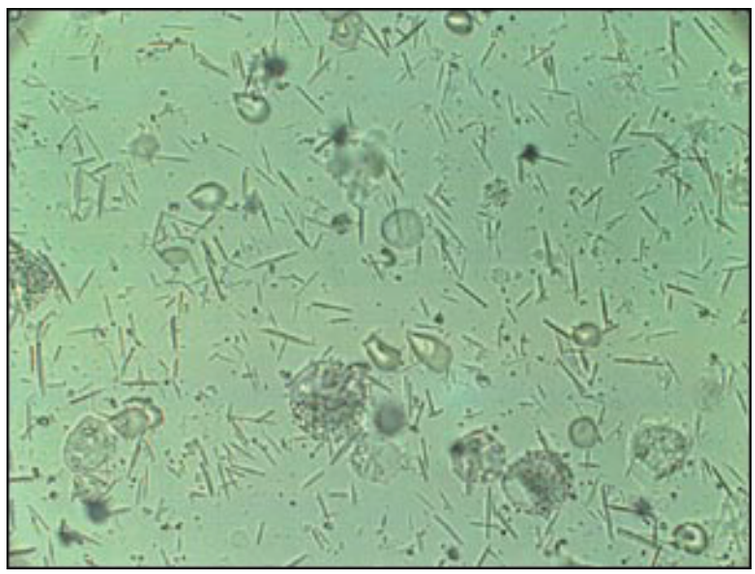


FIGURE 2.
Needle-shaped monosodium crystals seen by light microscopy of synovial fluid in a patient with gout.

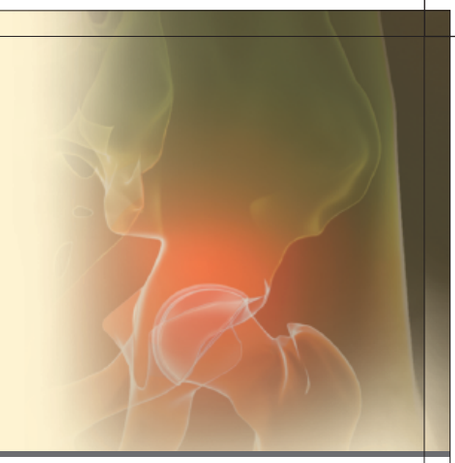


TABLE 4

Common Errors in Diagnosing Acute Monoarthritis

ERROR	REALITY
The problem is in the joint, because the patient complains of "joint pain."	The soft tissues around the joint can be the source of the pain (e.g., olecranon bursitis of the elbow, prepatellar bursitis of the knee).
Crystal-proven diagnosis of gout or pseudogout rules out infection.	Crystals can be present in a septic joint.
The presence of fever is useful in distinguishing infectious causes from other causes.	Fever may be absent in patients with infectious monoarthritis but can be a presenting feature in acute attacks of gout or pseudogout. Fever may occur for other reasons in certain patients (e.g., the immuno-compromised).
A normal serum uric acid level makes gout a less likely diagnosis.	Serum uric acid levels often are lowered in patients with acute gout. Conversely, there may be unrelated hyperuricemia in patients with other conditions.
Gram staining and culture of synovial fluid are sufficient to exclude infection.	Cultures of blood, urine, or another primary site of infection (e.g., abscess) must be obtained and repeated as necessary if infection is strongly suspected clinically. Culture results may be negative in early infection.

The complete blood cell count may show leukocytosis in some patients with infection. An erythrocyte sedimentation rate may distinguish inflammatory arthritis from noninflammatory arthritis, but this test is nonspecific and may be overused. Tests for HIV and Lyme disease antibodies may be obtained if appropriate, but serologies usually are not helpful in identifying the cause of acute monoarthritis. Indiscriminately ordering tests such as rheumatoid factor and antinuclear antibodies can result in confusion, because false-positive results are common.

Blood cultures should be obtained in patients with suspected septic arthritis. Cultures are positive in about 50 percent of non-gonococcal infections but are rarely positive (about 10 percent) in gonococcal infection. Pharyngeal, urethral, cervical, and rectal swabs are necessary if gonococcal infection is suspected.

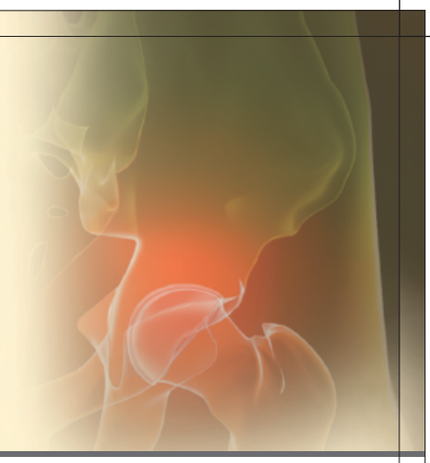
IMAGING:

X-RAYS

Although plain-film radiographs often show only soft tissue swelling, they are indicated in patients with a history of trauma or patients who have had symptoms for several weeks. Occasionally, unsuspected bony lesions, such as osteomyelitis or malignancy, may be detected. The presence of chondrocalcinosis could support but not confirm CPPD arthritis.

Ultrasound: Ultrasound scanning is useful to detect an effusion particularly in the hip.

Radionuclide scanning: Can detect infection



in deep-seated joints.

Magnetic resonance imaging: Is superior in detecting ischemic necrosis, occult fractures, and meniscal and ligamentous injuries.

Other diagnostic procedures, such as synovial biopsy or arthroscopy, may be useful to rule out deposition diseases (e.g., hemochromatosis, atypical infections) and intra-articular tumours.

Indications for Referral

CIRCUMSTANCE	REASONS FOR REFERRAL
Failed arthrocentesis or joints that are difficult to aspirate, such as hips and sacroiliac joints	Need for computed tomography or ultrasound-guided arthrocentesis
Septic arthritis	Urgent consultation, hospitalization for intravenous antibiotics, joint drainage, débridement; infectious disease consultation for atypical infections
Suspected inflammatory polyarthritis or recurrent monoarthritis unresponsive to treatment	Rheumatologist evaluation and management
Undiagnosed chronic monoarthritis	Need for closed synovial biopsy or arthroscopy

The Gold-Standard for Predicting Fracture Risk FRAX

Compiled by:
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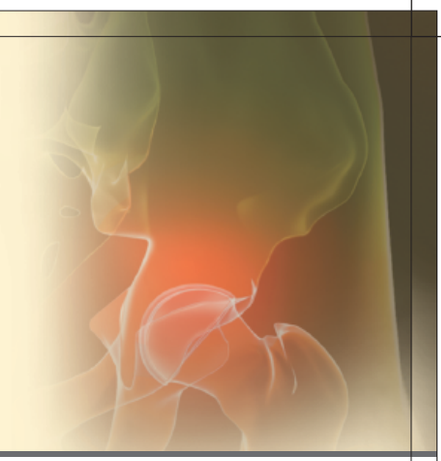


A Tool to Access Fracture Risk in the Absence of BMD

The WHO FRAX tool has become the gold standard model for assessing osteoporotic fracture risk, with or without BMD measurements. It can estimate 10-year fracture risk even in the absence of BMD.

Just type fracture risk assessment tool FRAX on google search from your computers or smart phones to download. It will ask you basic information about the patient and will give the result immediately.

Some of the common questions that arise in relation to the tool and offers advice on using FRAX in day-to-day practice.

1. How do I use FRAX if I don't have access to BMD tests?

BMD is a very good predictor of fracture and low BMD is associated with high fracture risk. The problem is that even people who have BMD above the classic WHO T-score threshold of -2.5SD have a huge burden of fracture.

FRAX is not dependent on BMD. You can assess the patient on the basis of clinical factors alone and get a very good idea of whether the person is at low or high risk of future fracture. The optimal use of FRAX is with concurrent use of BMD but where BMD resources are limited, BMD testing should be targeted at patients what lie closest to the intervention threshold.

If you have BMD, use it. If you don't, use FRAX. If you have limited access to BMD, use a combination of both i.e. use FRAX initially to assess fracture risk and if the patient's FRAX score lies close to the intervention threshold, refer them for BMD to assist you in making a decision on whether or not to initiate treatment.

2. Should I use lumbar spine or femoral neck BMD?

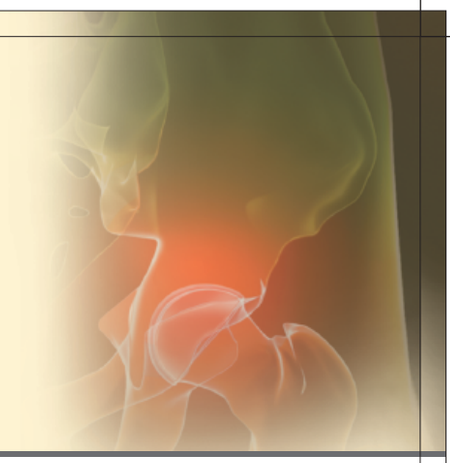
The tool has been designed to estimate risk based on femoral neck T-scores. FRAX will overestimate fracture probability where lumbar spine BMD is much higher than femoral neck BMD. As a rule of thumb, the final score should be increased or decreased by 10% for each rounded T-score SD difference between lumbar spine and femoral neck BMD measurements. If the lumbar spine T-score is 1SD higher than the femoral neck T-score, decrease the FRAX score by 10%, and vice verse if the femoral neck is 1SD higher than the lumbar spine.

3. At what FRAX score should I initiate treatment?

The threshold at which to initiate treatment is very much dependent on the individual country. Many countries have now incorporated FRAX into national osteoporosis guidelines and specify thresholds for initiation of therapy.

The evidence shows that osteoporosis therapies have the greatest benefit in patients who are at highest risk of fracture. Generally, a FRAX score of 20 would be an appropriate point to consider prescribing medication. But no assessment tool over-rides clinical judgement. It comes down to the individual clinician and the individual patient. If you have a patient in front of you who has a FRAX score of 20%, you must decide if that is an acceptable level of risk for that patient, taking into account any comorbidities, the possible down side of treatment and the cost of treatment.

Similarly, if you have a patient who has a FRAX score below the intervention threshold but who you know is a frequent faller, that puts them above the intervention threshold clinically and



you should initiate therapy.

4. My country isn't listed. What should I do?

FRAX has been designed to estimate risk according to country-specific data on fracture risk and mortality. When using the tool, it is important to select the correct country. New countries are being added all the time as adequate data becomes available. If your country is not available, choose a surrogate country based on the likelihood that it is representative of your country in terms of life expectancy and fracture incidence.

5. Does FRAX apply to men as well as women?

Yes. The risk factors work similarly in men and women in different countries in terms of relative risk. However, absolute risk will vary since, at any given age, the absolute risk of fracture and absolute risk of death varies. In addition, risk factors have variable importance depending on age (e.g. a family history), or on the presence or absence of other risk factors. For example, low BMI is much less of a risk factor when BMD is taken into account.

6. What age groups does the tool apply to?

FRAX can be used to estimate the 10-year probability of fracture from 40 years of age upwards. If you enter an age below 40, the tool will calculate the probability of fracture for that patient at 40. You should use your own clinical judgement to interpret this risk based on the patient's other risk factors.

7. Why doesn't FRAX take account of the number of previous fractures?

Different factors were included in the original FRAX model based on the availability of strong, global, evidence to support their impact on the probability of future fracture. Where such evidence was not available to support the role of a specific factor, it was omitted from the calculation. Evidence was not available from all cohorts to support the inclusion of a numerical value for former fractures. Clinical judgement is important here and a higher than average number of fractures should, in practice, encourage the initiation of further tests or treatment where appropriate.

8. Why is the dose of glucocorticoid not included?

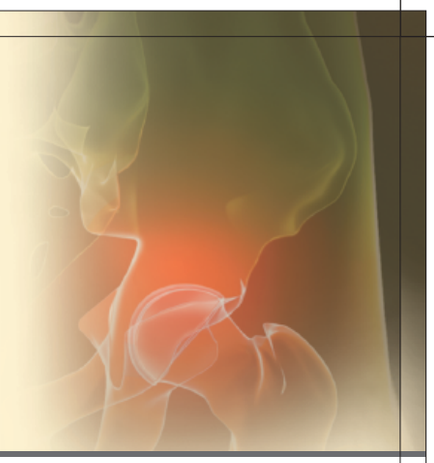
Again, when the original FRAX model was designed, there was insufficient data available across all cohorts on the impact of glucocorticoid dose on future fracture risk. However, Kanis and colleagues have documented a lower risk with lower steroid doses and a higher risk with higher doses. Further study is required on this issue but it has been suggested that for patients on low-dose glucocorticoids, the FRAX score should be adjusted downwards by 35% and for those on high doses, the score should be adjusted upwards by 20%.

9. Is ethnicity important?

Ethnicity has a marked effect on fracture risk. This is already reflected in the US model where epidemiological information on fracture and mortality rates are available within the Asian, Black, Caucasian and Hispanic communities. When similar evidence is available in different regions, models for the same and other ethnic groups will be developed for other parts of the world.

10. How often should I re-FRAX patients?

FRAX is a living tool. It is constantly evolving as new evidence comes to light. At the moment, we are re-evaluating people who have undergone a FRAX assessment to determine the most appropriate interval at which people

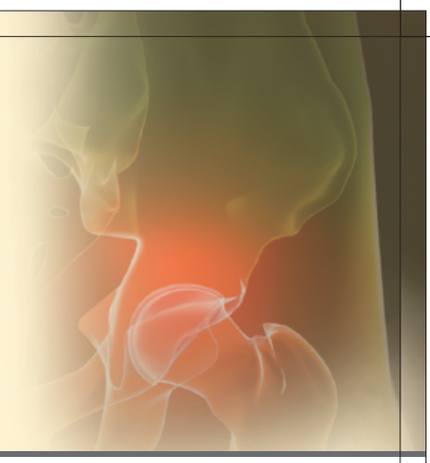


should be reassessed, but at this point, I would recommend that patients be reassessed on a five-yearly basis.

Risk factors

For the clinical risk factors a yes or no response is asked for. If the field is left blank, then a "no" response is assumed. The risk factors used are the following:

Age	The model accepts ages between 40 and 90 years. If ages below or above are entered, the programme will compute probabilities at 40 and 90 year, respectively.
Sex	Male or female. Enter as appropriate.
Weight	Enter as appropriate.
Height	Enter as appropriate.
Previous Fracture	A previous fracture denotes more accurately a previous fracture in adult life occurring spontaneously, or a fracture arising from trauma which, in a healthy individual, would not have resulted in a fracture. Enter yes or no (see also notes on risk factors).
Parent Fractured Hip	This enquires for a history of hip fracture in the patient's mother or father. Enter yes or no.
Current Smoking	Enter yes or no depending on whether the patient currently smokes tobacco (see also notes on risk factors).
Glucocorticoids	Enter yes if the patient is currently exposed to oral glucocorticoids or has been exposed to oral glucocorticoids for more than 3 months at a dose of prednisolone of 5mg daily or more (or equivalent doses of other glucocorticoids) (see also notes on risk factors).
Rheumatoid Arthritis	Enter yes where the patient has a confirmed diagnosis of rheumatoid arthritis. Otherwise enter no (see also notes on risk factors).
Secondary Osteoporosis	Enter yes if the patient has a disorder strongly associated with osteoporosis. These include type I (insulin dependent) diabetes, osteogenesis imperfecta in adults, untreated long-standing hyperthyroidism, hypogonadism or premature menopause (<45 years), chronic malnutrition, or malabsorption and chronic liver disease.
Alcohol 3 or more units/day	Enter yes if the patient takes 3 or more units of alcohol daily. A unit of alcohol varies slightly in different countries from 8-10g of alcohol. This is equivalent to a standard glass of beer (285ml), a single measure of spirits (30ml), a medium-sized glass of wine (120ml), or 1 measure of an aperitif (60ml) (see also notes on risk factors).
Bone Mineral Density (BMD)	(BMD) Please select the make of DXA scanning equipment used and then enter the actual femoral neck BMD (in g/cm^2). Alternatively, enter the T-score based on the NHANES III female reference data. In patients without a BMD test, the field should be left blank (see also notes on risk factors) (provided by Oregon Osteoporosis Center).



Notes on Risk Factors

Previous Fracture

A special situation pertains to a prior history of vertebral fracture. A fracture detected as a radiographic observation alone (a morphometric vertebral fracture) counts as a previous fracture. A prior clinical vertebral fracture or a hip fracture is an especially strong risk factor. The probability of fracture computed may therefore be underestimated. Fracture probability is also underestimated with multiple fractures.

Smoking, Alcohol, Glucocorticoids

These risk factors appear to have a dose-dependent effect, i.e. the higher the exposure, the greater the risk. This is not taken into account and the computations assume average exposure. Clinical judgment should be used for low or high exposures.

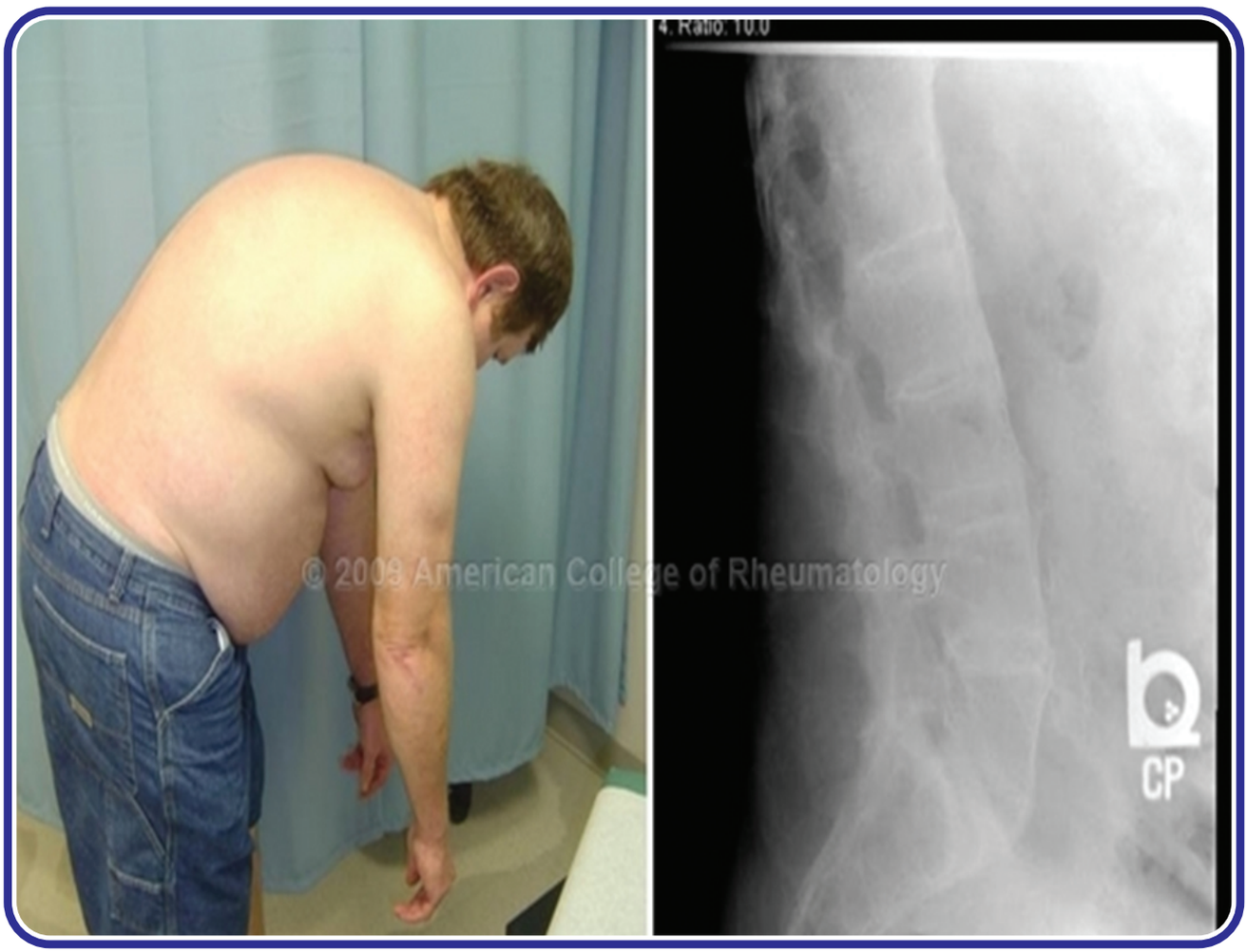
Rheumatoid Arthritis (RA)

RA is a risk factor for fracture. However, osteoarthritis is, if anything, protective. For this reason reliance should not be placed on a patient's report of 'arthritis' unless there is clinical or laboratory evidence to support the diagnosis.

Bone Mineral Density (BMD)

The site and reference technology is DXA at the femoral neck. T-scores are based on the NHANES reference values for women aged 20-29 years. The same absolute values are used in men.

Photo Quiz



Question

What is your diagnosis for the above given picture ?

Answer of last quiz

- Pseudofracture
- Osteomalacia