

## REVIEW ARTICLE

# Monoarthritis in the emergency department

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Monoarthritis, defined as the inflammation of a joint, may be either acute or chronic and arise from a variety of causes. Septic arthritis is responsible for the highest rates of morbidity and mortality. The most common cause is the presence of microcrystals. A clinical history and careful physical examination are of great help in establishing the cause. Bilateral radiographs of the joints are essential. Unless contraindicated, arthrocentesis should be undertaken to establish the etiologic diagnosis. [Emergencias 2011;23:218-225]

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Monoarthritis is defined as inflammation of a single joint. Oligoarthritis refers to inflammation of two or three joints and polyarthritis is used when more than three joints are involved<sup>5</sup>. According to evolution time, the condition is also classified as acute, meaning an evolution time of less than six weeks, or chronic, referring to a longer period. The affected joint presents with classic signs of pain, redness, heat, tumefaction and functional incapacity<sup>5</sup>.

The inflammatory process is due to a number of factors affecting both the synovial membrane, with consequent thickening, and the synovial fluid, which may increase in volume or spread to other structures surrounding the joint. Because of this, a thorough physical examination is important, to differentiate between the diagnosis of arthritis and other peri-articular processes, such as tendinitis, bursitis, cellulitis, panniculitis, etc.<sup>5,10</sup>.

Inflammation of a joint as the reason for an emergency department visit is not uncommon. The joint is usually swollen, with increased volume to a greater or lesser extent, erythematous in most cases, and often with increased local temperature. Clinically, the patient reports moderate to severe pain, and functional incapacity due to limited mobilization of the joint. The pain usually has inflammatory characteristics, ie. it does not di-

sappear with rest, or mechanical characteristics, according to etiology.

It can affect any joint, and its location is a determining factor for diagnosis.

## Diagnosis

The patient's medical history should include information such as age, sex, profession and toxic habits. The physician should also investigate previous episodes, travel to exotic places, presence of gastroenteritis or previous urinary tract infection, and diseases of interest such as hyperuricemia, psoriasis, reactive arthritis, etc. The patient should be questioned about other symptoms associated with arthritis, such as fever (which suggests a septic process), hair loss, photosensitivity, skin lesions such as psoriasis, ulcers, Raynaud's syndrome, etc.. We should also record when the picture began and whether onset was acute or progressive, triggering factors, intensity, location, and the characteristics of the pain in order to differentiate between pain with inflammatory characteristics (no improvement with rest) and mechanical characteristics<sup>2</sup>.

Physical examination should include each and every joint in search of others that may also be

tender and swollen, and the physician should look for skin lesions, wounds, presence of gout tophi, oral or genital lesions, ocular changes, and the presence of subcutaneous nodes.

On inspection, the inflamed joint usually appears swollen and the skin is sometimes reddish (Figure 1).

On palpation it generally feels warmer than the contralateral joint, and pressure causes pain<sup>1,10</sup>.

It is vitally important to determine if there is fluid leakage. In the knee it is useful to test for this manually using the following maneuver: with one hand, place the thumb and index finger on either side of the infrapatellar space (Figure 2). With the other hand, press the patella lightly. In cases of joint effusion, movement of the joint fluid and patella is readily observed<sup>1,10</sup>.

Another maneuver is to manually mobilize the joint, testing for the degree of limitation, both in flexion and extension. Also, to differentiate whether there is joint inflammation, periarticular inflammation or referred pain, one must take into account that the former produces pain with both active and the passive movement, while periarticular inflammation only tends to produce pain on active mobilization. With referred pain there is rarely any limitation of movement<sup>5</sup>.

### Complementary tests

All joint inflammation should be X-rayed, and in cases of limb joints the corresponding healthy joint should also be X-rayed for comparison<sup>5</sup>.

Laboratory tests should include complete blood count with leukocyte count, ESR if possible, and biochemistry. If ESR is not available, fi-



**Figure 1.** Proximal interphalangeal monoarthritis.



**Figure 2.** Knee joint maneuver.

brinogen level as acute phase reactant provides useful information. Urine analysis is also important<sup>9</sup>.

X-ray of the inflamed joint will show an increase in soft tissue compared with the contralateral healthy joint. Generally, in initial phases this increase is often the only finding that appears, though in cases of arthrosis decompensation, gout or chondrocalcinosis (pseudo-gout) other findings may provide some guidance. There may also be other signs such as periarticular erosions, localized osteoporosis, bone tumors etc.

In the case of arthrosis, the affected joint appears with typical radiographic signs such as asymmetric compression of the joint space, subchondral sclerosis and the presence of osteophytes. Gout can be associated with the presence of gouty tophi. In the case of chondrocalcinosis there is usually calcification of the articular disc visible as a line on the knee, with calcification of triangular carpal ligament or symphysis pubis.

In all inflamed joints, arthrocentesis should be performed<sup>9,10</sup>, both for joint relief and analysis. Normal synovial fluid is clear, transparent, viscous to the touch and often noticeably warm in inflamed joints. In the case of septic arthritis, the fluid appears cloudy, yellowish or even white.

After removing the liquid to be analyzed, three samples are placed in tubes containing a few drops of heparin. The most urgent test for diagnosis and management is biochemistry with blood count, glucose, ADA, and proteins, as well as the presence of crystals and Gram test. In one sample, joint fluid glucose is compared with blood levels, decreasing depending on whether the fluid is inflammatory or septic, and reaching values close to zero in cases of tuberculosis or rheumatoid arthritis. The other two samples are used for cul-

ture and pathological anatomy study. Fluid analysis shows different characteristics, depending on whether the origin of the picture is mechanical, inflammatory or septic (Table 1)<sup>3</sup>.

Synovial fluid with mechanical characteristics suggests diseases such as osteoarthritis, osteonecrosis, post-traumatism, reflex sympathetic dystrophy, osteochondritis dissecans, amyloidosis, sarcoidosis, localized osteoporosis, pigmented villonodular synovitis or infectious arthritis in patients with human immunodeficiency virus (Figure 3).

Synovial fluid with inflammatory characteristics suggests microcrystal deposits, infection, foreign body reaction, spondylitis, rheumatoid arthritis, psoriasis, systemic lupus erythematosus, Behcet's disease, palindromic rheumatism, rheumatic fever or the presence of parasites (Figure 4)<sup>2,3</sup>.

## General measures

Faced with a monoarthritis, whatever its cause, one should always advise rest of the joint for the duration of the acute phase.

Symptomatic treatment is based on anti-inflammatory agents, analgesics and gastric protection, sometimes with steroids, but the essential thing is to treat the underlying cause of monoarthritis, as explained below. The patient must be admitted to hospital when there is general malaise attributable to monoarthritis, blood-streaked synovial fluid with coagulation disorder, positive Gram stain, more than 50,000 leucocytes/mm<sup>3</sup> in the absence of crystals, synovial fluid with inflammatory characteristics plus fever or chills, synovial fluid with inflammatory characteristics in patients with a history of risky sexual contact or suspected gonococcal infection, and radiological bone lesion suggestive of bone tumor or osteomyelitis<sup>2,8</sup>.

## Causes of monoarthritis

As mentioned, it is very important to establish the etiology of an articular inflammatory



**Figure 3.** Sero-hematic synovial fluid in mechanical arthritis.

process, especially in the case of septic arthritis due to the high morbidity and mortality associated with it. While the causes are numerous (Table 2), this article addresses the most frequent causes, among which we would emphasize microcrystals<sup>5</sup>.

### Monoarthritis due to microcrystals

#### Gouty arthritis

Gouty arthritis is inflammation of a joint secondary to uric acid precipitation leading to the presence of monosodium urate crystals.

This occurs mostly in men over 40 years of age (except in the elderly where the prevalence is higher in women<sup>7</sup>). A history of chronic alcohol abuse, hyperuricemia, hypercholesterolemia and diabetes mellitus is often noted. Location is usually in the metatarsal-phalangeal joint at the base of the big toe, known as podagra (Figure 5), although it can also appear in the heels, ankles or wrists. The inflammation is usually monoarticular, although recurrent polyarticular episodes may appear.

Triggering factors in acute gouty arthritis are varied: changes in physical activity - often associated with periods of hospitalization or bed rest, dietary excesses, changes in treatment such as

**Table 1.** Differences in synovial fluid<sup>5</sup>

	Normal	Inflammatory	Septic	Non-inflammatory
Appearance	Transparent, colorless	Opaque, translucent yellow	Opaque, yellow	Transparent, yellow
Viscosity	High	Low	Variable	High
Leucocytes	< 200/mm	5.000 a 75.000 /mm	> 50.000/mm	200 a 2.000/mm
Glucose	Normal	< 50% glycemia	< 50% glycemia	Normal
PMN (%)	< 25	> 50	> 75	< 25
Bacteria (gram)	No	No	Frequent	No

PMN: Polymorphonuclear.



**Figure 4.** Inflammatory synovial fluid, gouty arthritis (courtesy of the Spanish Society of Rheumatology image bank).

diuretics or of the treatment for hyperuricemia itself, or local trauma.

Progressive pain increases in a few hours to excruciating pain, with mixed or inflammatory characteristics, associated with fever, frequently with rapid onset and at night. The joint appears swollen, there is fluid effusion and great functional incapacity, with cutaneous redness on the surface of the joint. On resolution of the swelling, there is usually skin peeling. After successive acute episodes in patients with poor therapeutic control, skin tophi appear in locations such as the pinna or periarticular regions [gouty tophi have been reported on Heberden nodes (Figure 6)<sup>10</sup>] and several joint deformities may appear in cases of chronic gout.

Diagnosis is based on a detailed medical history, taking into account the onset of the picture and the location. Physical examination is impor-

tant to detect gouty tophi, both periarticular and the external ear. Laboratory tests should include a complete analysis with CBC, white blood cell count, biochemistry with special importance attached to levels of urea, creatinine and transaminases, the latter possibly increased in chronic alcoholism and urinalysis.

Radiology does not usually provide much diagnostic information, except in the case of patients with recurrent acute gout and tophi, and in chronic gout with typically deformed joints. Arthrocentesis shows inflammatory synovial fluid and green birefringent crystals under polarized light microscopy.

The goal of treatment for an acute attack of gouty arthritis is to relieve the pain, reduce the inflammation and prevent further acute attacks. Maximum-dose oral anti-inflammatory agents are used, together with gastric protection. Initially the recommended treatment is indomethacin 25-50 mg every 8 hours or diclofenac 50 mg every 8 hours, both being better tolerated than the classic colchicine which, at high doses, often produces gastrointestinal intolerance, so the guidelines recommend 1 mg every 8 hours. Exceptionally glucocorticoids can be used for treatment, with prednisone at doses of 30 to 50 mg a day.

Great care in dosing is important; for an acute attack, allopurinol medication for example would cause a sharp decline of uric acid on increasing urinary excretion, which in turn would mobilize deposits of uric acid, thereby worsening the patient's symptoms. Nor should one withdraw, remove or change previously scheduled treatment doses, since this may also aggravate the picture. In acute attacks of gout, NEVER change the treatment with allopurinol<sup>6</sup>.

To prevent future attacks, the patient should be advised on lifestyle modifications, with a low-purine diet, no alcohol and healthy physical exercise.

**Table 2.** Causes of monoarthritis<sup>1,10</sup>

Acute	Chronic	Other
Infectious Arthritis:	Infectious Arthritis: Mycobacteria. Fungi. Brucellosis.	Common causes: Meniscus and ligament injury. Fractures. Osteoarthritis.
Bacterial spirochetes (Lues, Lyme's disease...)	Spirochetes.	Less common causes: Aseptic osteonecrosis. Osteochondritisdissecans. Synovial chondromatosis. Haemarthrosis, Benign and malignant tumors. Villonodular synovitis.
Viruses mycobacteria fungi.	Spondyloarthropathy. Rheumatoid arthritis.	Metastasis. Reflex sympathetic dystrophy. Neuropathic arthropathy.
Microcrystal arthritis:	Juvenile chronic arthritis. Sarcoidosis. Vasculitis.	
Gout (monosodium urate).	Connective tissue disease. Microcrystal arthritis:	
Pseudogout (calcium pyrophosphate).	Foreign body synovitis. Sympathetic synovitis.	
Calcium oxalate hydroxyapatite. Lipids.		
Inflammatory rheumatic disease:		
Spondyloarthropathy (reactive arthritis, psoriatic arthritis). Rheumatoid arthritis (rare).		
Behcet Lupus. Sarcoidosis		



**Figure 5.** Podagra (courtesy of the Spanish Society of Rheumatology image bank).

### Chondrocalcinosis

Chondrocalcinosis or pseudo-gout is caused by deposits of calcium pyrophosphate crystals and calcification of the hyaline and fibrous cartilage. It usually affects people older than 50 years, especially women. Hyperparathyroidism, acromegaly and hypothyroidism are predisposing factors. There are a number of factors that may precipitate an episode of acute inflammation as in gouty arthritis: hospitalization, concomitant disease and stressful situations. Onset is usually sudden, with pain and inflammation that is not as intense as in gouty arthritis, self-limited, and predominantly affecting the knee (Figure 7). There may be fever, especially during the first few days of the picture. Diagnosis is mainly based on radiologic tests, showing calcification of the knee cartilage, the triangular carpal ligament or the anterior pubis, as well as the results of Arthrocentesis showing inflammatory synovial fluid with calcium pyrophosphate crystals. The treatment of acute chondrocalcinosis is similar to that of gout. It is essential to rest the joint and prevent local heat<sup>6,10</sup>.

### Septic arthritis

This is serious disease requiring early diagnosis and treatment. It may affect patients of any age and sex, and incidence increases with age and certain predisposing conditions. These include the presence of diseases such as diabetes mellitus and cancer, or pharmacological drugs such as immunosuppressive agents or corticosteroids. In addition, patients undergoing surgery, especially joint prosthetic surgery, and intravenous drug users (IDU) are more prone to the di-



**Figure 6.** Tophus on a Heberden node (courtesy of the Spanish Society of Rheumatology image bank).

sease<sup>5,6</sup>. Septic arthritis is caused by different germs, varying with age and certain host factors.

- Less than three months: *S. aureus*, Enterobacteria, *Streptococcus p.*
- 3 to 6 months: *S. aureus*, *H. influenzae*, *Streptococcus*, Enterobacteria.
- Sexually promiscuous adults: *gonococcus*.
- Non-promiscuous adults: *S. aureus*, *Streptococcus A*, Enterobacteria.
- Arthritis by direct inoculation (prosthesis, puncture, surgery etc.): *S. epidermidis* (40%), *S. aureus* (20%), Enterobacteria, *Pseudomonas*.
- IDU or HIV + patients: *S. aureus*.
- Prosthetic joint: *S. epidermidis*. If long term, *S. aureus*.

The affected joint appears highly inflamed, reddish surface skin and a large increase in temperature, much more so than in other types of monoarthritis<sup>4</sup>. The pain is of great intensity, preventing movement and producing early and very limiting functional incapacity. It is associated with fever, lymphadenopathy and even soft tissue abscesses and general malaise, but the absence of fever does not rule out septic arthritis. Generally affecting large joints, it may appear in any part of body including the sacroiliac. Muscle atrophy can develop if the patient is immobilized over a long period of time<sup>6</sup>.

With gonococcal arthritis, the symptoms vary slightly: there is usually fever, intense pain and inflammation as in the other forms, but skin rash appears on the limbs, vesicular or macular, and tendonitis in wrists or ankles.

Brucella arthritis<sup>6</sup> tends to mainly affect the sacroiliac and spine; it is accompanied by sweating, generalized myalgia, headache, etc. The attending



**Figure 7.** Left knee monoarthritis due to pseudo-gout in subacute phase.

physician should suspect this form of arthritis in patients in contact with animals.

Diagnosis is based on the physical examination and the above-mentioned signs, as well as observation of antalgic contracture of the muscles near the joint. The joint is very limited in terms of movement, this being one of the only signs in deep joints. Analytically, the presence of leukocytosis with left deviation is noted. In addition, whenever there is suspicion of brucella arthritis, the Rose Bengal test should be performed. Radiological alterations appear within 6-10 days after onset, showing cartilage damage with widening of the articular space and, increasingly, juxta-articular osteoporosis, subchondral erosion, space narrowing and reactive sclerosis<sup>6</sup>. But perhaps the most important diagnostic test is arthrocentesis, showing a cloudy septic fluid that requires culture and urgent biochemical analysis with Gram staining to identify the causal agent.

All cases of septic arthritis with arthrocentesis confirmation should be hospitalized<sup>6</sup>. The affected joint must be rested, and sustained flexion should be avoided. Daily arthrocentesis and joint fluid removal should be performed<sup>7</sup>.

Early treatment with parenteral antibiotics should be initiated while waiting for synovial fluid culture results. The probable microorganism responsible for the infection clearly determines the choice of antibiotic and current guidelines recommend the following<sup>6,7</sup>:

- Known Gram stain:
  - Gram positive cocci: intravenous cloxacillin 2 g every 6 to 8 hours. Alternatively, and in immunocompromised patients: vancomycin.
  - Gram negative: intravenous ceftriaxone 2 g daily.

In children under 6 years, cefuroxime 30 to 100 mg/kg/day. In immunocompromised patients, add aminoglycosides.

- Bacilli: intravenous ceftazidime 1 g every 8 hours.

- No Gram stain: empiric cloxacillin plus third-generation cephalosporin.
- Infection of prosthetic joint: ciprofloxacin and vancomycin.
- Brucella arthritis: doxycycline for 45 days associated with streptomycin for the first 2-3 weeks.

If there is no improvement after 48-72 hours the patient should undergo appropriate surgical drainage<sup>6</sup>.

#### *Arthritic decompensation*

This often appears in elderly patients with previously diagnosed osteoarthritis. Decompensation may present as episodes of joint swelling, pain and even fluid leakage. It occurs frequently in the early stages of the disease and is more common in the proximal interphalangeal and distal metacarpal joints, with pain of low intensity. There are a series of triggers such as being bedridden, certain serious illnesses or surgery, and arthritic decompensation is especially frequent in the knee (Figure 8)<sup>1</sup>. Diagnosis is primarily based on radiological images with signs of degenerative joint and increased soft tissue according to the intensity of inflammation. Arthrocentesis shows no crystals in the synovial fluid. Treatment is based on rest, arthrocentesis and anti-inflammatory agents. In case of severe osteoarthritis, surgery should be considered once the acute phase has passed.

#### *Fibrocartilage ligament lesions*

Lesions of the meniscus, intra-articular ligaments and free bodies may be the underlying cause of monoarthritis. Generally, after meniscus injury caused by abnormal movement or overload, joint fluid leakage appears associated with pain and sometimes leads to joint blockage<sup>6</sup>.

Clinically, the joint appears painful and swollen but with little or no temperature rise. Onset is acute. The patient is usually afebrile, with episodes of joint lock, particularly striking in the case of the knee.

The synovial fluid is clear and with few cells. Radiology does not usually show any alterations. Generally, when faced with this picture, the most specific test is nuclear magnetic resonance imaging<sup>6</sup>.

Treatment is based on rest and avoiding weight load on the joint. Aspiration is recommended in cases of fluid leakage, with analgesics and anti-inflammatory agents pending surgical assessment after the acute phase has subsided.

### *Reactive Arthritis*

This refers to inflammation of a load-bearing joint (ankles and knees) 2-3 weeks after suffering genitourinary or gastrointestinal infection. The microorganisms involved are *Yersinia*, *Shigella* and *Salmonella*. Also included in this category are post-streptococcal arthritis and rheumatic fever, although the latter is more usually associated with migratory oligoarthritis<sup>4,10</sup>.

Reactive arthritis appears most often in young men. It usually begins insidiously, affecting load-bearing joints, especially the ankles, but sacroiliitis or spondylitis may also be found. It is accompanied by fever, general symptoms and extraarticular manifestations such as conjunctivitis, mouth thrush, blennorrhagic keratoderma of the hands and feet, balanitis and urethritis in men. It also tends to cause enthesitis, lumbar pain with inflammatory characteristics, Achilles tendinitis, plantar fasciitis, chest pain and dactylitis<sup>1,10</sup>.

Blood tests show leukocytosis with increased acute phase reactants, nonspecific alteration of serum protein, and anemia in chronic cases.

Radiography, initially, does not show changes except for increased soft tissue, but as the picture progresses, it shows developing osteoporosis, bilateral sacroiliitis, irregular periostitis, especially in the legs, and finally spondylitis with discontinuous syndesmophytes<sup>10</sup>.

Acute phase treatment involves anti-inflammatory agents, the most effective being phenylbutazone and indomethacin. The joint should be rested and later physiotherapy is recommended. Patients with reactive arthritis should be referred to a specialist for the appropriate immunosuppressive treatment.

### *Hemarthrosis*

The etiology of hemoarthritis is usually trauma, associated with bone fractures and muscular or tendon injuries. Alternatively, it may be secondary to bleeding disorders, anticoagulant therapy, myeloproliferative processes, joint prosthesis and, rarely, pigmented villonodular synovitis. Periarticular edema, ecchymosis and erythema may be observed. Clinically, there is pain and knee swe-



**Figure 8.** Monoarthritis of the knee due to arthrosic decompensation.

lling, usually severe in cases of tension hemarthrosis. The affected joint usually remains in antalgic posture with painful and limited mobility<sup>6</sup>. Diagnosis is based on arthrocentesis revealing abundant red blood cells in the synovial fluid which appears with various shades of red. Since one of the causes of hemarthrosis is the bone fracture, radiography is required to rule out this cause.



**Figure 9.** Distal interphalangeal monoarthritis in psoriatic arthritis (courtesy of the Spanish Society of Rheumatology image bank).

Treatment is based on rest, synovial fluid extraction to relieve pain, and analgesics. There is controversy about the value of performing arthrocentesis in low-grade hemarthrosis<sup>6,10</sup>. In patients taking anticoagulants, the dose should be reduced or even suspended. In cases of coagulopathy, by treating the underlying disease the patient generally shows clinical improvement.

### *Other monoarthritis*

Some autoimmune diseases with oligoarticular or polyarticular involvement may present as monoarthritis, as occurs with ankylosing spondylitis, psoriatic arthritis (Figure 9), spondyloarthropathy, inflammatory bowel diseases (ulcerative colitis, Crohn's disease), juvenile chronic arthritis or SAPHO syndrome, which is associated with acne.

## Monoarthritis en urgencias

### Revuelta Evrard E

La monoartritis es la inflamación de una articulación, que puede ser aguda o crónica. Su etiología es múltiple, siendo la artritis séptica el cuadro con mayor morbilidad. La monoartritis por microcristales es la más frecuente. Una historia clínica y una exploración física precisa resulta de gran ayuda para establecer el diagnóstico etiológico de la enfermedad. La radiología bilateral de las articulaciones resulta fundamental. Siempre que no esté contraindicado debería realizarse una artrocentesis para el diagnóstico etiológico de una monoartritis. [Emergencias 2011;23:218-225]

**Palabras clave:** Monoartritis. Urgencias. Artrocentesis. Etiología.

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