The Swollen Joint: What to Do

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Patient history is critical

When a patient presents with acute joint swelling, properly assessing his/her history is of paramount importance (Table 1). The physical exam is also important, although to a lesser degree.

Articular swelling

Articular swelling is usually fusiform and should involve the joint in its entirety, as opposed to the more localized swelling and tenderness that can accompany tendonitis or bursitis. Periarticular problems, such as rotator cuff tendonitis, can cause more problems with certain movements related to the action of the involved anatomical structures (*i.e.*, above shoulder-level activities or full abduction is painful with a supraspinatus tendonitis).

Inflammation

Inflammation classically involves dolor, calor, rubor and tumour (pain, heat, redness and swelling) and these features can be sought in inflammatory arthropathies as well. However, rheumatoid arthritis (RA) and many other types of inflammatory arthropathies do not usually present with red joints. If the inflamed joints are red (Table 2), then the top two conditions to rule out are septic joints and crystal arthropathies (*i.e.*, gout, pseudogout or hydroxyapatite [such as, acute calcific periarthritis]).

If an inflammatory axial component is present, then one must consider the seronegative spondyloarthropathies, which includes :

- ankylosing spondylitis (AS),
- inflammatory bowel disease (IBD)-associated arthropathies,

Harry's case

Harry, 60, is a married executive who presents with a one week history of swelling and pain involving his hands, wrists, knees and feet.

History

- He woke up with the pain and states that he has never had joint problems in the past. There has been no significant response to 400 mg of ibuprofen b.i.d., p.r.n.
- He has noticed nodular lesions over his elbows, developing over the past year or so, but the functional inquiry was otherwise non-contributory
- Harry has had non-insulin-dependent diabetes mellitus of two years duration, as well as hypertension
- He is a non-smoker and has, on average, two drinks of rum/vodka per day
- There is a family history of hypertension, diabetes mellitus and coronary artery disease
- Examination confirms synovitis over the involved joints, as well as nodular lesions over the olecranon processes

Medications

Harry's medications include:

- 500 mg of metformin t.i.d., p.o.
- 50 mg of hydrochlorothiazide q.d., p.o.
- 81 mg of acetylsalicylic acid p.o. q.d.

Lab results

Harry's laboratory investigations reveal the following:

- Complete blood count: all normal other than a white blood cell (WBC) count of 10.5, with a normal differential and normal platelet
- Uric acid: 420 umol/L
- Rheumatoid factor (RF): 25 kU/L,
- Antinuclear antibodies (ANA): positive (homogeneous and speckled pattern)
- Erythrocyte sedimentation rate: 50 mm/h,
- C-reactive protein: 15 mg/L
- Liver function tests: normal
- Serum creatinine: normal

Go to page 96 to find out more.

More on Harry

Differential diagnosis

The main differential diagnosis for Harry involves inflammatory arthropathies that can present with nodular lesions. Nodular rheumatoid arthritis (RA) and tophaceous gout would be at the top of the differential diagnosis in this case.

Gout can present in a polyarticular fashion and can also present with tophi without any history of past attacks.

The incidence of false positive ANA and RF goes up with age. Aspiration of Harry's right knee revealed an inflammatory fluid with a WBC of 32,000 with 85% polymorphs. The cultures were negative, but intracellular birefringent crystals were seen, confirming the diagnosis of gout.

He had been on an inadequate dosage of ibuprofen and was treated with 75 mg of diclofenac, slow release b.i.d., with good response.

Table 1

Important historical points

- Articular/periarticular/nonarticular (extraarticular, bone, nerve)
- Monoarthritis/oligoarthritis/polyarthritis
- Noninflammatory/inflammatory
- Axial/peripheral, small/large, upper/lower extremity, symmetric/asymmetric
- Extraarticular manifestations
- Characteristics of musculoskeletal pain: OPQRST
  - O: Onset
  - P: Precipitation and ameliorating factors
  - Q: Quality
  - R: Radiation
  - S: Severity
  - T: Timing
- Functional limitations

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- psoriatic arthritis (PsA) and
- reactive arthropathies/Reiter's syndrome (ReA).

Extra-articular manifestations

It is important to get a history of any extraarticular manifestations, such as diarrhea and ocular inflammation, to help with the diagnosis. A patient who has psoriasis, often with nail changes, presenting with an acutely swollen and a red distal interphalangeal (DIP) joint may very well have PsA. A patient with seropositive disease (*i.e.*, RA or connective tissue diseases) may have:

- subcutaneous nodules,
- sicca symptoms,
- nasopharyngeal ulcers,
- serositis,
- Raynaud's phenomenon,
- vasculitic rashes and
- other systemic problems.

Seronegative arthropathies (*i.e.*, AS, IBD, PsA, ReA) can present with:

- enthesitis,
- ocular inflammation,
- mouth ulcers,
- urethritis,
- psoriasis and
- other inflammatory manifestations of the GI or genitourinary tract.

Joint distribution

The joint distribution is important. Classical RA involves a symmetrical inflammatory polyarthritis of the small, medium and large joints with relative sparing of the spine, other than the cervical spine. PsA can present in various ways, including an asymmetric oligoarthritis or monoarthritis that frequently attacks the upper extremities (*i.e.*, DIP joint, such as in Figure 1). Gout classically presents acutely, often with pain involving lower extremity joints, peaking within 24 hours, with exquisitely

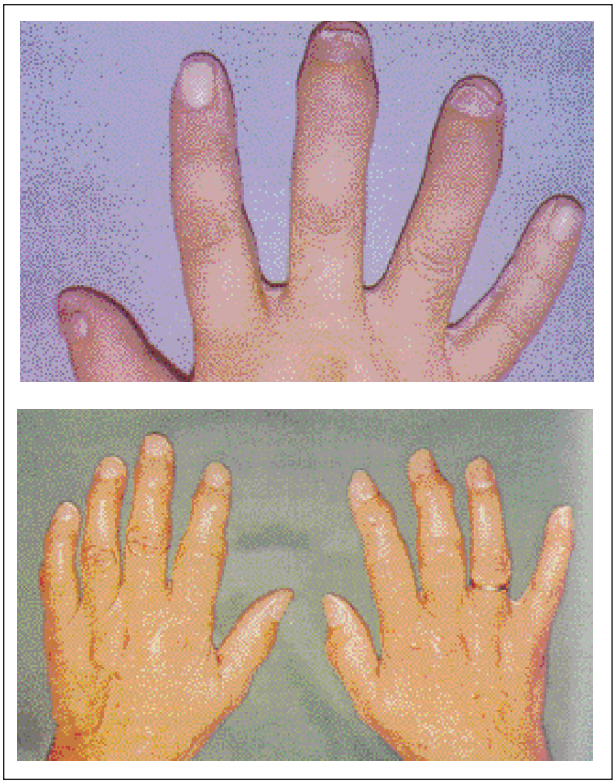

Figure 1. Rheumatic disorders affecting the distal interphalangeal (DIP) joints. Top: Psoriatic arthropathy of two DIP joints and the interphalangeal joint of the thumb, together with nail dystrophy.
Bottom: Osteoarthritis is the commonest disorder affecting this segment (Heberden's nodes).

Table 2
The red-hot joint

- Infectious: bacterial, viral, *etc.*
- Crystal-induced: gout, pseudogout, hydrozyapatite
- Psoriatic arthritis (PsA)
- Reactive arthritis
- Palindromic rheumatism
- Bacterial endocarditis
- Traumatic arthritis

Table 3
Risk factors for infectious arthritis

- Intravenous drug abuser
- High-risk sexual behaviour
- Recent pneumonia, pyelonephritis, skin infection
- Immunosuppression
- Prosthetic joint or pre-existing chronic arthropathy (*i.e.*, RA)
- Comorbid medical problems (diabetes mellitus, renal failure, chronic alcohol use, *etc.*)

tender, warm and red joints that can frequently resolve with peeling over the inflamed joint (Figure 2). Attacks of gout can classically be triggered by:

- alcohol,
- certain foods (*i.e.*, red meats, shellfish, *etc.*),
- medications (low-dose acetylsalicylic acid, diuretics, *etc.*) and
- various physical stressors (dehydration, *etc.*).

Infections must be ruled out

Infections are serious and usually treatable (Figure 3), particularly if diagnosed early. Any accessible joint fluid should be aspirated for synovioanalysis, known as the three Cs:

- culture,
- cell count,
- crystals.

Some joints (such as the hip joint) are best aspirated under fluoroscopic guidance. It is important to consider the risks for an infectious arthropathy (Table 3). Infections can present insidiously in some high-risk individuals (*i.e.*, elderly, immunocompromised). Crystal arthropathies, such as gout, can mimic or coexist with infectious arthropathies. It is also important to note that infections and crystal arthropathies can present as oligoarthropathies or polyarthropathies.

The differential diagnosis of acute monoarthritis (Table 4) and acute polyarthritis (Table 5) is similar in many respects. Viral arthropathies usually present with polyarthropathies, but rarely can present as a monoarthropathy or oligoarthropathy. Similarly, RA is classically an inflammatory polyarthropathy but can initially present as a monoarthritis or oligoarthritis (frequently becoming polyarticular within six months to 12 months of presentation).

Table 4

Acute monoarthritis

- Infections:
  - > Viral
  - > Bacteria
    - - *Staphylococcus aureus*
    - - Hemolytic strep
    - - Gram-negative
    - - *Haemophilus influenza*
    - - *Neisseria*
    - - Mycobacterial
    - - Lyme disease
- Crystals:
  - > gout
  - > pseudogout
  - > hydroxyapatite
- Traumatic:
  - > hemarthrosis
- Seronegative:
  - > PsA
  - > Reiter's syndrome (ReA), *etc.*
- Others:
  - > hemarthrosis (bleeding disorders anticoagulation, *etc.*)
  - > palindromic rheumatism
  - > RA/connective tissue diseases
  - > sarcoidosis
  - > bacterial endocartitis
  - > neoplasm
  - > avascular necrosis
  - > neuropathic joint, *etc.*

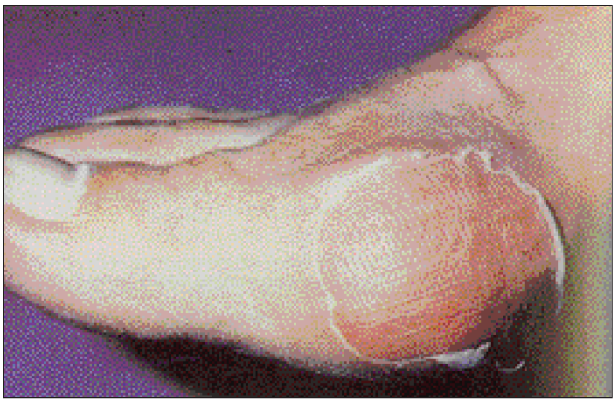

Figure 2. Acute gout. The first metatarsophalangeal joint is involved, at some time, in approximately 75% of patients. Desquamation of the skin often occurs.

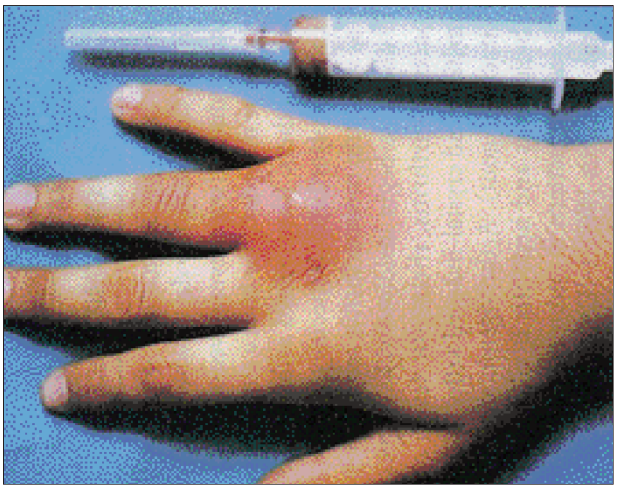

Figure 3. The red hot joint. Septic arthritis of the ring finger, metacarpophalangeal joint showing swelling and intense redness of the skin.

Laboratory and radiologic investigations

There are few specific or diagnostic tests in rheumatology and one should do a test only if the pre-test probability of a particular condition is high. The incidence of false-positive rheumatoid factor (RF) and antinuclear antibodies goes up with age. Patients with hyperuricemia may not have gout, while patients with gout attacks may be normouricemic. Synovioanalysis should be performed anytime joint fluid is accessible (Table 6). A diagnosis of septic arthritis can be confirmed if an organism can be identified on Gram staining and/or cultures. Even patients with presumed osteoarthritis can frequently present with an inflammatory arthropathy, such as gout (Figure 4) or pseudogout.

The demonstration of crystals (particularly intracellular) confirms the presence of a crystal arthropathy. Certain bloodwork can be more specific, such as the use of anti-Sm and anti-ds DNA antibodies for the diagnosis of systemic lupus erythematousus (although these tests are not very sensitive). Anticyclic citrullinated peptide antibodies are increasingly being used, as they have a higher sensitivity and specificity for rheumatoid disease than the RF. Acute phase reactants (*i.e.*, erythrocyte sedimentation rate and C-reactive

Table 5

Acute inflammatory polyarthritis

- Infections:
  - > Viral:
    - *Rubella*,
    - Hepatitis,
    - Parvovirus B19,
    - Epstein-Barr virus,
    - HIV, *etc.*
  - > Bacterial:
    - *Gonococcal*
    - *Meningococcal*
    - *Non-gonococcal*
    - Bacterial endocarditis
    - Lyme disease, *etc.*
    - Acute rheumatic fever
- Non-infectious:
  - > RA
  - > Seronegative arthropathies: ReA, PsA, *etc.*
  - > Systemic lupus erythematosus and other connective tissue diseases
  - > Crystals (*i.e.*, polyarticular gout, *etc.*)
  - > Serum sickness
  - > Sarcoid arthritis
  - > Vasiculitis, *etc.*

Table 6

Synovioanalysis

Measure	Normal	Nonin	Inflamm	Septic	Hemorr
Vol (ml)	< 3.5	Oft > 3.5	Oft > 3.5	Oft > 3.5	Us > 3.5
Clarity	Transp	Transp	T-opaq	Opaque	Bloody
Color	Clear	Yellow	Yel-opaq	Yellow	Red
Viscos	High	High	Low	Variable	Variable
WBC	< 200	200 to 2,000	2,000 to 50,000	> 50,000	200 to 2,000
PMNs%	< 25	< 25	> 50	> 75	50 to 75
Culture	Neg	Neg	Neg	Oft pos	Neg
Glucose	~ blood	~blood	Dec	Dec	~blood

Nonin: Noninflammatory
Vol: Volume
Yel: Yellow
Oft pos: Often positive
Viscos: Viscosity
Inflamm: Inflammatory
Oft: Often
Neg: Negative
Dec: Decreased
PMNs: Polymorphonuclear leukocytes
Hemorr: Hemorrhagic
Transp (T): Transparent
Us: Usually
~: Roughly the same as blood levels

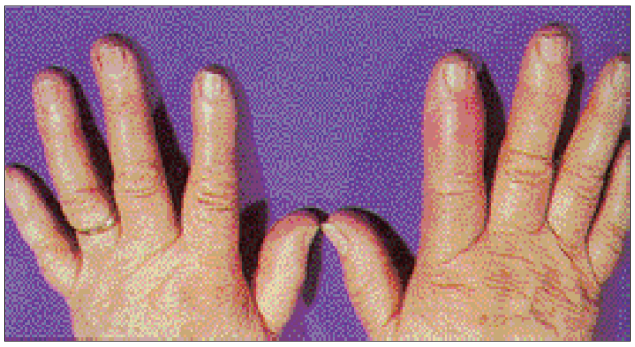

Figure 4. Gouty attack superimposed on a Heberden's node. The clinical combination may be found in elderly patients treated with diuretics as seen in the DIP joint of the right index finger in this 78-year-old woman. Note mild desquamation of the overlying skin.

protein) can help point towards a diagnosis of septic arthritis or of an inflammatory arthropathy. Other bloodwork, such as a complete blood count, liver function tests and kidney function tests, can be used to help screen for systemic manifestations of the underlying process and/or to monitor for drug toxicity.

Plain radiographs are rarely useful in an acute setting, unless there is trauma involved. With subacute disease, or in patients with acute flares, sacroiliac changes may suggest a seronegative spondyloarthropathy. Pseudogout disease frequently manifests in joints with chondrocalcinosis (*i.e.*, wrists and knees). The erosions of gout can present differently from those of rheumatoid disease, often presenting in extraarticular positions or showing unusual features (such as, the shelf sign where a spicule of bone juts out forming a shelf).

There are few specific or diagnostic tests in rheumatology and one should do a test only if the pre-test probability of a particular condition is high.

Take-home message

- The history is most important in diagnosing rheumatic conditions
- Laboratory data may be supportive, but usually cannot be used to establish a diagnosis
- Arthrocentesis should be performed, if possible, in all cases of acute joint swelling, to help rule out septic arthritis and/or crystal arthritis and also to help differentiate inflammatory from noninflammatory conditions
- Avoid using steroids, if possible, in management (unless one is sure there is no infection and the diagnosis is clear)
- Time may be needed to firmly establish a diagnosis and to help refine therapy

Management and treatment

The treatment of acute joint swelling depends on the underlying disease process. If infection is a reasonable possibility, then coverage with antibiotics is advisable, pending culture results. Steroids (oral or parenteral) should be avoided, if at all possible, unless infections have been ruled out or are unlikely. The indiscriminate use of steroids can also make diagnosis problematic. Infections can be treated using the appropriate antimicrobial agent, based on the offending organism(s) and their sensitivity profile.

Crystal arthropathies, such as gout, can be treated with full doses of nonsteroidal anti-inflammatory drugs (NSAIDs), colchicine and/or steroids. Colchicine works best when initiated within 24 hours to 48 hours of the onset of an attack (gout or pseudogout) and can be used in a dose of 0.6 mg b.i.d. to t.i.d. Many patients develop side-effects on colchicine (*i.e.*, diarrhea, vomiting, abdominal cramps) and the dose may need to be adjusted accordingly (it is also important to adjust the dose based on the renal function). Prednisone can be used to treat a crystal arthropathy, like gout, if infections have been ruled out. A dose of 25 mg to 50 mg for one day or two days may be required, tapering rapidly over seven days to 10 days. Rebound flares may require a more slow taper, or the concomitant use of other agents, such as colchicine with the prednisone.

If only one or a few joints are involved in confirmed gout cases, then intraarticular steroid injections would be very reasonable. If the crystal arthropathy involves too many joints or involves joints that are more difficult to inject (*i.e.*, small joints of the hands and feet) and oral therapy is not effective or is contraindicated, then 60 mg of triamcinolone acetonide can be quite successfully used. Intravenous steroids and subcutaneous adrenocorticotrophic hormone (SC ACTH) can also be used, although they do not provide any significant advantages over the regimens already discussed. Allopurinol should not be initiated or the dose of it changed during a gout attack, as that can prolong an attack, or make it more severe.

In cases of acute presentation of a systemic inflammatory arthropathy, such as RA or PsA, full doses of NSAIDs should be used. Disease modifying antirheumatic drugs will need to be initiated as soon as possible and the patient should be referred to a rheumatologist. Dx

Resources
1. Hochberg M, Silman AJ, Smolen JS, et al: *Rheumatology.* Third Edition. Mosby Inc, 2003, pp. 191-199.
2. Harris E, Budd R, Firestein G, et al: *Kelley's Textbook of Rheumatology.* Seventh Edition. Elsevier Saunders, 2005, pp. 483-521.