



Chapter 106 (9th Ed) – Arthritis

Episode Overview

- The ER approach to arthritis: the time course, consider the number of joints involved (monoarticular vs polyarticular), the distribution of joint involvement (large versus small joints and symmetrical versus asymmetrical joint involvement),.
- The possibility of septic arthritis should be considered in all patients who present with acute monoarticular arthritis.
- There is no combination of examination findings or blood tests that places septic arthritis below the threshold for performance of arthrocentesis in adult patients presenting with a new, hot, swollen, painful joint. Synovial fluid analysis is necessary to for risk stratification of septic arthritis, and delays in treatment worsen outcomes.
- The presence of crystals in synovial fluid or a negative Gram's stain result does not completely eliminate the possibility of septic arthritis.
- Bacterial arthritis can coexist with gout or pseudogout, and the result of Gram's stain is positive in only 50% to 80% of cases of septic arthritis.
- Many other common arthritides, such as gout, rheumatoid arthritis, and osteoarthritis, can be managed with NSAIDs as a first-line therapy.

Core Questions:

1. List a ddx (4 for each) for monoarticular, polyarticular symmetrical, and polyarticular asymmetrical arthritis (table)
2. What are systemic signs of arthritis diseases? List extraarticular manifestations of RA (table)
3. What are two presentations of gonococcal arthritis?
4. What are common radiologic findings in arthritis? (table)
5. List 2 indications, 2 contraindications, 2 complications of arthrocentesis
6. Describe arthrocentesis techniques for common joints. (box)
7. Describe typical synovial fluid findings by arthritis type: Describe the testing of synovial fluid and how you would interpret the results. (table)
 - Non-inflammatory
 - Inflammatory
 - Gout vs. Pseudogout
 - Septic
 - Hemorrhagic
8. How is gout managed? List 4 therapies for acute gout.
9. What are the the risk factors for gout?
10. List the microbiology of bacterial septic arthritis related to patient groups (table)
 - Neonates - infants
 - Children
 - Adolescents - adults
 - Older adults
 - Sickle cell anemia
 - Injection drug users



11. Describe treatment for septic arthritis.
12. Name six viral arthritides (table)

Wisecracks:

1. What is the normal WBC count in a joint aspiration?
2. List 6 RFs for septic arthritis
3. What is Calcium Pyrophosphate Dihydrate Deposition Disease (Pseudogout)?
4. List 5 characteristics of Seronegative spondyloarthropathies
5. List 5 pathogens responsible for reactive arthritis (aka Reiter's Syndrome)
6. Describe the Jones Criteria
7. What is erythema marginatum?
8. What to do with the anticoagulated patient with a swollen joint?
9. Differentiate between articular vs. periarticular inflammation?

Rosens In Perspective:

What are the types of joints?

“As opposed to synarthrotic suture joints of the skull and amphiarthrotic fibrocartilage unions like the pubic symphysis, the joints of concern in **acute arthritis are the synovial or diarthrotic (moving) joints.**”

These synovial joints are composed of two ends of subchondral bone covered by articular cartilage, surrounded by a capsule that is lined with a thin synovial membrane and supported by ligaments, tendons, and muscle (Fig. 106.1A).

Articular cartilage is an **avascular, aneural tissue** composed of a matrix of collagen fibers and proteoglycans synthesized by chondrocytes. The properties of articular cartilage allow tremendous load bearing. Together with the viscous lubricating synovial fluid, an ultrafiltrate of blood supplemented with hyaluronic acid and low-molecular-weight proteins, the cartilage that articulates joint movement is nearly frictionless.”

The big emergency this episode will cover again and again is our approach to the potentially septic joint.

Septic arthritis is most commonly instigated by hematogenous seeding of a diarthrotic joint. The bacteria can then proliferate in the joint, before causing a severe inflammatory reaction. Contiguous spread can also occur through direct trauma.

[1] List a differential diagnosis (4 for each) for monoarticular, polyarticular symmetrical, and polyarticular asymmetrical arthritis.

If the site of the patient's pain is articular, classifying whether the arthritis is monoarticular (eg, septic arthritis or gout) or polyarticular may aid in diagnosis. Polyarticular arthritis may be symmetrical (eg, rheumatoid or drug-induced) or asymmetrical (eg, rubella, acute rheumatic fever [ARF], Lyme disease, or gonococcal



arthritis). In addition, it may also be migratory (eg, gonococcal or rubella), subsiding in one area before presenting in another, or additive, remaining in the first joint and progressing to additional joints.

TABLE 106.1
Differential Diagnosis of Arthritis in the Emergency Department, Based on Typical Distributions

| MONOARTICULAR | POLYARTICULAR: SYMMETRICAL | POLYARTICULAR: ASSYMMETRICAL |
|---------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------|
| Septic arthritis Gout CPPD/pseudogout Osteoarthritis Trauma, hemarthrosis | Rheumatoid arthritis flare Psoriatic arthritis Polymyalgia rheumatica Enteric arthritis Ankylosing spondylitis Hep B/C induced arthritis | Gonococcal arthritis Lyme arthritis ARF Reactive arthritis Viral arthritides |

ARF, Acute rheumatic fever;
CPPD, calcium pyrophosphate dihydrate deposition Disease.



[2] What are systemic signs of arthritis? List extraarticular manifestations of RA.

TABLE 106.2
 Systemic Signs of Arthritis Diseases*

| Organ System | Findings | Diseases |
|--------------|-----------------------------|-------------------------------------------------------|
| Airway | Airway Obstruction | RA, relapsing polychondritis |
| Cardiac | Pericarditis | RA, ARF |
| | Murmurs | ARF, relapsing polychondritis, ankylosing spondylitis |
| Eyes | Iritis, uveitis | Spondyloarthropathies |
| | Conjunctivitis | Reactive arthritis |
| GI | IBD | Spondyloarthropathies |
| | Dysentery | Reactive Arthritis |
| Genitalia | Lesions, Urethral Discharge | Reactive arthritis, gonococemia |
| Hematologic | Aplastic anemia | Parvovirus |
| Neurologic | Cauda equina syndrome | Ankylosing spondylitis |
| | Cervical spine instability | Ankylosing spondylitis, RA, OA |
| Oral mucosa | Ulcerations | Reactive arthritis |
| Pulmonary | Pleuritis, nodules | RA |
| Renal | Renal crisis, ARF | Scleroderma |
| Skin | Plaques on elbows, knees | Psoriasis |
| | Sclerodactyly, calcinosis | Scleroderma |
| | Erythema chronicum migrans | Lyme disease |
| | Tophi | Gout |
| | Erythema Marginatum | Rheumatic fever |
| | Subcutaneous nodules | RA |

*Excludes rheumatic vasculitis diseases.

ARF, Acute rheumatic fever; IBD, inflammatory bowel disease; RA, Rheumatoid Arthritis

extraarticular manifestations of RA

- Osteoporosis, atlantoaxial subluxation
- Muscle weakness, synovitis, myositis, myopathies
- Sarcopenia, obesity
- Rheumatoid nodules**
- ocular/oral dryness (sjogren’s syndrome)*
- Episcleritis, scleritis, uveitis
- Interstitial fibrosis*, pneumonias, pulmonary hemorrhage
- Pericarditis, myocarditis, CAD
- Mesenteric vasculitis
- Glomerulonephritis
- Carpal tunnel syndrome; mononeuritis multiplex
- Anemia*



[3] What are two presentations of gonococcal arthritis?

1. Mono-oligoarticular arthritis
2. True disseminated gonococcal infection (sometimes termed arthritis-dermatitis syndrome)
 - bacteremia, diffuse migratory arthralgias, characteristic skin lesions, and tenosynovitis

Cervical, urethral, rectal, and pharyngeal cultures are positive in up to 75% of cases, so all mucosal orifices of the patient (and partner, if possible) should be cultured appropriately.

In disseminated gonococcal infection, the skin lesions often contain the gram-negative diplococcus.



[4] What are common radiologic findings in arthritis? (table)

TABLE 106.3: Common Radiologic Findings in Arthritis

| Arthritis | Findings |
|----------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Acute arthritis (gout, pseudogout, septic arthritis) | Soft tissue swelling |
| Late septic arthritis (need at least 8 to 10 days for changes to be seen) | Subchondral bone destruction Periosteal new bone Loss of joint space Osteoporosis Late joint space narrowing |
| Late pseudogout (knee, hip; radiocarpal, midcarpal, all MCP joints) | Linear calcification in cartilage Asymmetrical joint space narrowing MCP “hook spurs” in HHC Osteophyte formation Subchondral cyst formation Lack of osteoporosis |
| Degenerative arthritis (acromioclavicular, first carpometacarpal, first MTP, DIP joints; knee, hip, cervical spine, lumbosacral spine) | Asymmetrical joint space narrowing Sclerosis of juxta-articular bone Bone spurs and cysts—adjacent to Severe cartilage degeneration No osteoporosis |
| Tuberculous arthritis (knee, hip, shoulder) | Soft tissue swelling Marked demineralization Bone rarefaction Little reactive sclerosis Late bone destruction Joint space preserved |



| | |
|-----------------------------------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Late rheumatoid arthritis (wrist, MCP, PIP, MTP, first IP joints; foot, atlantoaxial joint, glenohumeral joint) | Symmetrical joint space narrowing Osteoporosis of periarticular bone Marginal erosions (no overhanging margins as in gout) Little reactive bone formation DIP, Distal interphalangeal; HHC, hereditary hemochromatosis; IP, interphalangeal; MCP, metacarpophalangeal; MTP, metatarsophalangeal; PIP, proximal interphalangeal. |
|-----------------------------------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|

Anyone with an acutely problematic joint(s) should have a normal x ray. If the x-ray is abnormal, the disease has likely been present for longer than 1 week.

[5] List 2 indications, 2 contraindications, 2 complications of arthrocentesis

This is the only way to definitively make the dx of septic arthritis or a crystal arthropathy!

Indications:

- Obtain joint fluid for analysis
- To drain tense hemarthrosis (trauma or hemophilia)
- Assess for blood - determining whether a laceration communicates with the joint space
- Intra-articular injection of analgesics and anti-inflammatories in the setting of acute/chronic arthritis

These are “relative” CI

- Overlying cellulitis where a joint would be aspirated
- Coagulopathy (elevated INR) or severe bleeding disorder
- Prosthetic joint

The primary complications of arthrocentesis are:

- bleeding or
- infection in the joint space,
- reaction to anesthetic agents, and
- long-term corticosteroid-related complications.



[6] Describe arthrocentesis techniques for common joints. (box)

Box 106.1 (9th Ed): Arthrocentesis Techniques for Common Joints

Wrist: Radiocarpal Joint (Dorsal Approach)

1. Identify landmarks by palpating the Lister tubercle (distal end of dorsal radius)
2. Palpate the extensor pollicis longus tendon, which passes over the radial side of the Lister tubercle (best palpated while the wrist is in extension). You will insert the needle on the ulnar side of the extensor pollicis longus tendon, just distal to the Lister tubercle.
3. Lay the wrist on a cushion so that it is flexed 20 to 30 degrees.
4. Apply traction from the fingers and mild ulnar deviation, and insert a 22-gauge needle dorsally.

Elbow: Radiohumeral Joint (Lateral Approach)

1. Identify landmarks by extending the elbow and then palpating the depression between the lateral epicondyle of the humerus and the head of the radius.
2. Keeping your finger on the radial head, flex the patient's elbow, pronate the forearm, and lay the palm on a flat surface.
3. Insert a 20-gauge needle just distal to the lateral epicondyle, directed medially.

Shoulder: Glenohumeral Joint (Posterior Approach)

1. Lay the patient's arm, internally rotated, across the waist.
2. Identify the posterolateral corner of the acromion.
3. Insert a 20-gauge needle 2 to 3 cm inferior to this point, directed anteriorly and medially (and slightly superiorly) toward the coracoid process.

Hip: Acetabulofemoral Joint (Lateral Approach)

1. Lay the patient supine and internally rotate the affected leg.
2. Palpate the greater trochanter.
3. Insert a 3.5-inch 18-gauge needle superiorly to the trochanter, horizontal and parallel to the stretcher. If the femoral neck is encountered, withdraw 2 mm to 4 mm and redirect slightly cephalad until synovium is aspirated.

Knee: Patellofemoral Joint (Medial Approach)

1. Flex the knee 15 to 20 degrees (often achieved with a rolled towel under the knee). The foot should be perpendicular to the floor.
2. Palpate the anteromedial patellar edge at the patellar midpoint or superior portion.
3. Insert an 18-gauge needle 1 cm medial to this point, directed toward the posterior surface of the patella.

Ankle: Tibiotalar Joint (Anteromedial Approach)

1. With the patient supine, have the patient plantarflex the foot.



2. Identify the anterior tibial tendon.
3. Insert a 3.5-inch 20- or 22-gauge needle medial to this tendon in the depression at the anterior edge of the medial malleolus.

Metatarsophalangeal Joint (Dorsomedial Approach)

1. Identify the distal metatarsal head and the proximal base of the first phalanx.
2. Identify the extensor tendon by asking the patient to extend the great toe.
3. While the patient is supine, flex the toe 15 to 20 degrees, and then apply traction.
4. Insert a 22-gauge needle dorsally just medial to the extensor tendon.

[7] Describe typical synovial fluid findings by arthritis type: Describe the testing of synovial fluid and how you would interpret the results. (table)

“no guidelines or studies exist supporting the use of blood testing (ESR, CRP, WBC, uric acid) as a general screen of acute undifferentiated arthritis in the ED.” - so we can't really use these tests to exclude specific causes of an acutely swollen joint. Therefore, aspiration is a must!

Table 106.4: Typical Synovial Fluid Findings by Arthritis Type

| | NON-INFLAMMATORY | INFLAMMATORY | SEPTIC | HEMORRHAGIC |
|-----------------------------------------|-----------------------------------------------------------------------------|--------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------|----------------------------------|
| Color | Clear/yellow | Yellow/white | Cloudy/opaque | Opaque, may contain fat droplets |
| Viscosity | Thick, stringy | Variable | Thin, watery | Variable |
| Synovial white blood cells | 200 to 2000/mm ³ <25,000/mm ³ +LR for SA = 0.32 | 2000 to 50,000/mm ³ <50,000/mm ³ +LR for SA = 0.42 | >25,000/mm ³ +LR for SA = 2.9 >50,000/mm ³ +LR for SA = 7.7 >100,000/mm ³ +LR for SA = 28 | <2000/mm ³ |
| Synovial polymorphonuclear cells | Variable | Variable | >90% +LR for SA = 2.7 | <25% |
| Gram stain | Negative | Negative | 29% to 65% positive | Negative |
| Leading diagnosis | Osteoarthritis | Gout, reactive arthritis | Bacterial arthritis | Trauma, hemophilia |



Important points for emphasis: (to be covered in the Wisecracks section)

- A positive Gram stain is diagnostic, but a negative result for bacteria does not rule out septic arthritis;
- Significant overlap exists between inflammatory and septic causes of acute arthritis, with very high fluid cell count or polymorphonuclear (PMN) cell pleocytosis suggesting infection, but more modest cell counts failing to exclude it.
- High cell counts ($>50,000/\text{mm}^3$) can occur in rheumatoid arthritis, gout, and pseudogout. Most of the cells in both septic and severe inflammatory arthritis are PMN cells.
- Monosodium urate crystals are needle shaped and strongly negatively birefringent (yellow when parallel to the compensator and blue when perpendicular), ranging in size from 2 to 10 μm . Calcium pyrophosphate crystals, in contrast, are polymorphic, rhomboid, and positively (although weakly) birefringent.
- Remember that septic arthritis CAN coexist with a crystal arthropathy!
- A synovial WBC count of more than 1100/ mm^3 or a pleocytosis of greater than 64% PMN cells is sensitive and specific for infection in the setting of prosthetic joints.
- Don't forget to think about periarticular causes of a irritated joint!
- lactate dehydrogenase (LDH) levels above 250 U/L are sensitive for septic arthritis, and LDH levels below this threshold seem to exclude septic arthritis, according to one study. Low synovial glucose and high protein concentrations are neither sensitive nor specific for septic arthritis.

So, let's walk through this process again: see figure 106-2

- Do that thorough hx and physical exam
 - If you think its articular
 - Get x rays
- If you think there are signs of inflammation
 - Consider any relative contraindications to arthrocentesis
 - If there are, think about drawing cultures from other sources!
- Do the arthrocentesis
 - If it's frank blood - figure out why
 - If it's bone marrow content - find the fracture!!
 - If there are <2000 WBCs think about
 - OA, internal cartilage/meniscus derangement, surrounding soft tissue problems, or a viral cause.
- If >2000 WBCs/75% PMNs
 - Figure out which type of inflammatory arthritis it is!



[8] How is gout managed? List 4 therapies for acute gout.

Without treatment, the attack is **self-limited**, peaking during 24 to 48 hours and lasting about a week.

Acute Treatment: ED pharmacologic mainstays for acute gout are **nonsteroidal antiinflammatory drugs** (NSAIDs; including cyclooxygenase 2–selective agents), **corticosteroids** (including ACTH), and **colchicine**.

| | | |
|---------------------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| NSAIDS | <ul style="list-style-type: none"> Ibuprofen Naproxen 500 mg BID Indomethacin 50 mg TID | <ul style="list-style-type: none"> Little evidence to say that one type of NSAID is superior to another Treat 24 hrs post symptoms relief (usually 2-6 days) |
| Colchicine (impedes the inflammatory response to crystals in fluid) | <ul style="list-style-type: none"> 1.2 mg PO, then 0.6 mg PO one hour later Then 0.6 mg po TID | Colchicine is contraindicated in patients with hematologic, renal, and hepatic insufficiency; its low therapeutic index makes it readily lethal in overdose. |
| Steroids | <p>Intra-articular injection (rule out SA first!)</p> <ul style="list-style-type: none"> Triamcinolone acetonide <ul style="list-style-type: none"> 20 to 40 mg for the knee 5 to 10 mg for smaller joints is Systemic steroids, such as oral prednisone (40 mg/day for 3 to 5 days, with or without a taper), | A Cochrane review of three trials of various steroids against other agents failed to demonstrate evidence of efficacy for systemic steroids. |
| Adrenocorticotrophic Hormone (Corticotropin). | 25–40 IU ACTH subcutaneously | <p>not commonly used (or studied) because of its expense and lack of general availability, synthetic ACTH is a desirable alternative to the preceding agents because of its rapid onset of action and decreased toxicity in older patients.</p> <p>American College of Rheumatology (ACR) Guidelines for gout management now endorse 25–40 IU ACTH subcutaneously as an appropriate alternative for nil per os (NPO) patients.</p> |
| Others | <ul style="list-style-type: none"> Rest, ICE, elevation Narcotics local/regional anesthetic blocks | |



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****Caution is exercised with use of these agents because hypertension, diabetes, and renal and vascular disease are prevalent in this cohort of patients.****

Patients for whom NSAIDs or colchicine is contraindicated can be treated with intra-articular injections of triamcinolone or ACTH (corticotropin).

Systemic steroids, although commonly used, have a weaker evidence base, but one trial suggests that prednisolone 35 mg/day is comparable to naproxen 500 mg twice a day during the course of 4 days.

Long Term Prophylaxis

New guidelines for gout recommended that prophylactic agents (such as, allopurinol), or newer agents (such as, febuxostat and probenecid) should neither be stopped nor initiated during an acute attack.

[9] What are the risk factors for gout?

Risk factors include:

- chronic obesity,
- hypertension,
- diabetes,
- thiazide diuretics
- cyclosporine use,
- lead or radiocontrast exposure.
- Purine-rich diets (meat; seafood, especially anchovies and shellfish; beer; and legumes) predispose at-risk individuals to attacks;
- high-fructose corn syrup and soft drinks are also implicated.

High dairy and coffee consumption have been shown to decrease risk.

And, don't bother drawing that serum uric acid! Not all patients with elevated uric acid levels or even joint crystals have acute attacks, and many patients with an acute gouty arthritis have a normal uric acid level.



[10] List the microbiology of bacterial septic arthritis related to patient groups.

Table 106.5: Microbiology of Bacterial Septic Arthritis Related to Patient

| PATIENTS | ORGANISMS |
|------------------------------|--------------------------------------------------------------------|
| Neonates and infants | <i>Staphylococcus aureus</i> , group B streptococcus, GNR bacteria |
| Children | <i>Haemophilus influenzae</i> , <i>S. aureus</i> |
| Adolescents and young adults | <i>Neisseria gonorrhoeae</i> , <i>Chlamydia trachomatis</i> |
| Older adults | <i>S. aureus</i> , <i>Streptococcus</i> , GNR bacteria |
| Sickle cell anemia | <i>Salmonella</i> |
| Injection drug abusers | <i>Pseudomonas</i> , <i>S. aureus</i> , GNR bacteria |

GNR = Gram Negative Rod

Acute nongonococcal septic arthritis in adults is caused most often by gram-positive organisms (75% to 90%),

Septic arthritis can occur simultaneously with other forms of arthritis, particularly rheumatoid arthritis and gout. The diagnosis of infectious arthritis in a patient with known crystal arthritis can be challenging because acute flare-ups of gout or pseudogout can cause fever, and crystals can precipitate in an infected joint. For this reason, arthrocentesis for Gram stain and culture should be considered in select cases.

[11] Describe treatment for septic arthritis.

Once the diagnosis is made, hospitalization is indicated for administration of intravenous (IV) antibiotics and needle, arthroscopic, or open drainage of the affected joint.

There are no randomized controlled trials of antibiotic regimens in septic arthritis. Antibiotic selection is initially based on Gram stain results and then adjusted on the basis of final culture results and sensitivities.

For gram-positive organisms, the initial drug of choice is vancomycin 30 mg/kg daily in two divided doses, as MRSA is frequently causative.

For gram-negative bacilli, use a third-generation cephalosporin, such as ceftriaxone 2 g IV once daily, cefotaxime 2 g IV three times a day, or ceftazidime with gentamicin (especially if *Pseudomonas* infection is suspected).

Although no trials of antibiotic duration have been reported, antibiotic therapy is generally continued parenterally for 2 to 4 weeks, depending on the response, and followed with 2 to 6 weeks of oral antibiotic therapy.



[12] Name six viral arthritides (table)

- Hepatitis B (mimic RA presentation, but followed by jaundice)
- Hepatitis C (mimics rheumatoid arthritis)
- HIV (usually monoarticular)
- Parvovirus B19 (cause of fifth disease in children)
- Rubella virus; Rubella vaccine virus (look for maculopapular rash and lymph nodes)
- Alphaviruses (Ross River, Chikungunya) - all mosquito borne

Wisecracks:

[1] What is the normal WBC count in a joint aspiration?

200-2000 WBC/mm³

Joint aspiration is the only way to definitively investigate a swollen joint.

Systematic reviews show insufficient evidence for a normal CRP level, ESR, WBC count, or procalcitonin level to take septic arthritis below the threshold for further evaluation in adults. Serum blood cultures reveal the causative organism less than half of the time.

Similarly, a WBC > 10k and ESR > 30 only minimally increase the likelihood of septic arthritis.

Important points for emphasis:

- A positive Gram stain is diagnostic, but a negative result for bacteria does not rule out septic arthritis;
- Significant overlap exists between inflammatory and septic causes of acute arthritis, with very high fluid cell count or polymorphonuclear (PMN) cell pleocytosis suggesting infection, but more modest cell counts failing to exclude it.
- High cell counts (>50,000/mm³) can occur in rheumatoid arthritis, gout, and pseudogout. Most of the cells in both septic and severe inflammatory arthritis are PMN cells.
- Monosodium urate crystals are needle shaped and strongly negatively birefringent (yellow when parallel to the compensator and blue when perpendicular), ranging in size from 2 to 10 μ m. Calcium pyrophosphate crystals, in contrast, are polymorphic, rhomboid, and positively (although weakly) birefringent.
- Remember that septic arthritis CAN coexist with a crystal arthropathy!
- A synovial WBC count of more than 1100/mm³ or a pleocytosis of greater than 64% PMN cells is sensitive and specific for infection in the setting of prosthetic joints.
- Don't forget to think about periarticular causes of an irritated joint!
- Lactate dehydrogenase (LDH) levels above 250 U/L are sensitive for septic arthritis, and LDH levels below this threshold seem to exclude septic arthritis,



according to one study. Low synovial glucose and high protein concentrations are neither sensitive nor specific for septic arthritis.

[2] List 6 RFs for septic arthritis

- Bimodal age distribution peaks for young children and adults older than 55 years old
- age over 80,
- low socioeconomic status,
- injection drug abuse (in which joint infections typically involve the axial skeleton but can involve extremities),
- alcoholism, diabetes, skin infections,
- advanced human immunodeficiency virus (HIV) infection or
- other immunocompromised states,
- Chronic arthritis (particularly rheumatoid, crystalline, and degenerative osteoarthritis),
- recent intra-articular corticosteroid injections or prosthetic implants.

[3] What is Calcium Pyrophosphate Dihydrate Deposition Disease (Pseudogout)

When calcium complex crystals form across articular surfaces. CPPD is manifested on radiographs as chondrocalcinosis.

When precipitating crystals trigger an inflammatory synovitis, it is termed pseudogout.

[4] List 5 characteristics of Seronegative spondyloarthropathies

- sacroiliac involvement, (chronic low back pain)
- peripheral inflammatory arthropathy (oligoarthritis)
- absence of rheumatoid factor,
- pathologic changes around the enthesis (ligamentous and tendinous insertion into bone) - heel enthesitis, dactylitis
- A genetic component related to the HLA-B27 marker (50-90% of these patients have a positive marker)
 - however, a positive HLA-B27 by itself is not diagnostic of SpA, since a significant proportion of subjects in the general population are also positive

The most important of these chronic polyarthritic inflammatory diseases are:

- ankylosing spondylitis,
- reactive arthritis,
- the arthropathy of inflammatory bowel disease (enteropathic arthritis),
- Psoriatic arthritis.



[5] List 5 pathogens responsible for reactive arthritis (aka Reiter's Syndrome)

Reactive arthritis is generally a disease of patients from 20 to 40 years old, in whom arthritis develops 2 to 6 weeks after an episode of urethritis, cervicitis, or dysentery. The syndrome is predominantly polyarticular, asymmetrical, and often additive.

- **Chlamydia trachomatis**

Other GI tract bugs:

- Salmonella
- Shigella
- Yersinia
- Campylobacter

[6] Describe the Jones Criteria

This is what we use to diagnose acute rheumatic fever (ARF). ARF is a systemic disease triggered by a complex hyperimmune response in the weeks after group A streptococcal pharyngitis.

Host cellular and humoral response to group A streptococcal infection attacks joint, cardiac, and other tissue, in part through molecular mimicry mechanisms.

The incidence of ARF has dramatically declined in recent decades, in part due to transformation of group A streptococcal strains, improvements in hygiene, and widespread antibiotic use; in the United States, incidence is estimated at 2 to 14 per 100,000. - Rosen's.

The Jones Criteria:

- **Lab evidence of prior group A strep infection****
- **Major (need 2 or more)**
 - Polyarthritis
 - Carditis
 - Chorea
 - Erythema marginatum
 - Subcutaneous nodules
- **Minor (need 1 major and 2 minor)**
 - Arthralgia
 - Fever
 - Elevated ESR/CRP
 - Long PR on ECG



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ARF arthritis responds so dramatically to salicylate or steroid therapy that the diagnosis can become clouded.

**Throat cultures are negative in 75% of patients with systemic manifestations of ARF. Antibody titers to streptolysin O and anti-DNase B may demonstrate antecedent group A streptococcal infection. Synovial aspirate is inflammatory in nature and sterile, with a widely variable synovial WBC count, no crystals, and a negative culture.

[7] What is erythema marginatum?

See Figure 106.6.

This is the pathognomonic rash found in acute rheumatic fever.

An “evanescent, pink or faintly red, nonpruritic rash involving the trunk and sometimes the limbs but not the face...The lesion extends centrifugally, with return of the skin in the center to a normal appearance. The outer edge of the lesion is sharp; the inner edge is diffuse. The lesion is also known as "erythema annulare" since the margin of the lesion is usually continuous, making a ring. Individual lesions may appear, disappear, and reappear in a matter of hours. A hot bath or shower may make them more evident.” - UpToDate

[8] What to do with the anticoagulated patient with a swollen joint?

Uptodate and a literature on Pubmed support the safety of joint aspiration on therapeutic levels of DOAC's and Warfarin. The risk of causing major hemorrhage is low (<0.2%).

So, we can't really use anticoagulation as an excuse not to tap a joint, especially if they may have a septic joint!

The only change suggested is using a 22 gauge needle!

A recent retrospective review of >1000 pts. Taking DOACs: There were no bleeding complications in any of the 1050 procedures. (avg. age 75 yrs, concomitant ASA and clopidogrel use as well!). 87% had 5 day follow-up.

Link:<https://www.ncbi.nlm.nih.gov/pubmed/?term=joint+aspiration+direct+oral+anticoagulants>

You perform arthrocentesis of a knee joint because of concern for septic arthritis. Which of the following findings from joint fluid aspiration increase the likelihood of septic arthritis?

- A. Synovial lactate dehydrogenase (LDH) of 300 U/L
- B. Synovial lactate of 3.0 mmol/L
- C. Synovial whole blood cell (WBC) count of 12,000 cells/mm³
- D. Viscous aspirate



Answer: A. Synovial LDH levels above 250 U/L are sensitive for septic arthritis, whereas lower levels seem to exclude the diagnosis. Similarly, synovial lactate above 5.6 mmol/L is associated with higher likelihood for septic arthritis. Higher synovial WBC counts are directly proportional to the likelihood of a septic joint, but 12,000 cells/mm³ is still on the low end of the spectrum.

[9] Differentiate between articular vs. periarticular inflammation?

True arthritis produces generalized joint pain, warmth, swelling, and tenderness. Often pain at REST. Discomfort increases with both passive and active motion of the joint because the inflamed synovium is exquisitely sensitive to stretching, and because all parts of the joint are involved in the inflammatory process.

By contrast, periarticular inflammation (bursitis, tendinitis, or localized cellulitis) tends to be more focal, weight bearing and passive ROM are tolerated.