Septic Arthritis

- septic arthritis is a medical emergency because it can lead to rapid joint destruction and has a 10-15% risk of mortality
- knee and hip are most commonly affected joints
- most commonly caused by hematogenous spread of bacterial infection (gram positive cocci > gram negative bacilli)
- risk factors: very young, portal of entry, recent infection
- poor prognostic factors: older age, immunocompromised, delay in treatment, previously damaged joint, joint prosthesis
- consider empiric antibiotic therapy until septic arthritis is excluded from history, physical examination and synovial fluid analysis
- see Infectious Diseases, *Gonoccocal Arthritis* ID14 and Orthopedics, *Spetic Joint* OR10

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Degenerative Arthritis: Osteoarthritis

- see Family Medicine, FM40

Definition

- progressive deterioration of articular cartilage and surrounding joint structures caused by genetic, metabolic, biochemical, and biomechanical factors with secondary components of inflammation

Classification (Based on Etiology)

- primary (idiopathic)
  - most common, unknown etiology
- secondary
  - post-traumatic or mechanical
  - post-inflammatory (e.g RA) or post-infectious
  - heritable skeletal disorders (e.g. scoliosis)
  - endocrine disorders (e.g. acromegaly, hyperparathyroidism, hypothyroidism)
  - metabolic disorders (e.g. gout, pseudogout, hemochromatosis, Wilson's disease, ochronosis)
  - neuropathic (e.g. Charcot joints)
    - atypical joint trauma due to peripheral neuropathy (e.g. diabetes mellitus, syphilis)
  - avascular necrosis (AVN)
  - other (e.g. congenital malformation)

Pathophysiology

- the process appears to be initiated by abnormalities in biomechanical forces and/or, less often, in cartilage
- elevated production of pro-inflammatory cytokines is important in OA progression
- tissue catabolism > repair
- contributing factors (mechanisms unknown): genetics, alignment (bow-legged, knock-kneed), joint deformity (hip dysplasia), joint injury (meniscal or ligament tears), obesity, environmental, mechanical loading, age and gender
- now considered to be a systemic musculoskeletal disorder rather than a focal disorder of synovial joints

Epidemiology

- most common arthropathy (accounts for ~75% of all arthritis)
- increased prevalence with increasing age (35% of 30 yr olds, 85% of 80 yr olds)

Risk Factors

- genetic predisposition, advanced age, obesity (for knee and hand OA), female, trauma

Signs and Symptoms

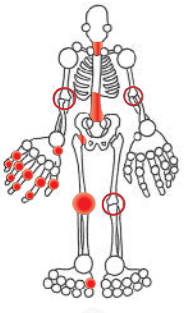
- localized to affected joints (not a systemic disease)
- pain is often insidious, gradually progressive, with intermittent flares and remissions; neuropathic pain may also be present
- fatigue, poor sleep, impact on mood (depression, anxiety)

Table 9. Signs and Symptoms of OA

Signs	Symptoms
Joint line tenderness; stress pain ± joint effusion Bony enlargement at affected joints Malalignment/deformity (angulation) Limited ROM Crepitus on passive ROM Inflammation (mild if present) Periarticular muscle atrophy	Joint pain with motion; relieved with rest Short duration of stiffness (<1/2 h) after immobility Joint instability/buckling Joint locking due to "joint mouse" (bone or cartilage fragment) Loss of function or other internal derangements (e.g. meniscal tear)

OA of MCP joints can be seen in hemochromatosis or CPPD-related disease (chondrocalcinosis)

ESR can also be elevated in anemia, end-stage renal disease, females, increased age, and obesity



- Hand (DIP, PIP, 1st CMC)
- Hip
- Knee
- 1st MTP
- L-spine (L4-L5, L5-S1)
- C-spine
- Uncommon: ankle, shoulder, elbow, MCP, rest of wrist

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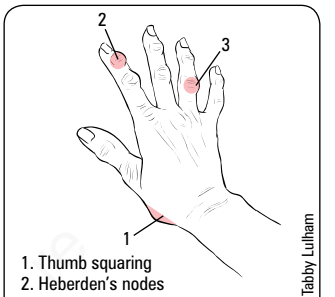
Figure 3. Common sites of joint involvement in OA



1. Thumb squaring
2. Heberden's nodes
3 Bouchard's nodes

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Figure 4. Hand findings in OA

Joint Involvement

- generalized osteoarthritis: 3+ joint groups
- asymmetric (knees usually affected bilaterally)
- hand
  - DIP (Heberden's nodes = osteophytes → enlargement of joints)
  - PIP (Bouchard's nodes)
  - CMC (usually thumb squaring)
  - 1st MCP (other MCPs are usually spared)
- hip
  - usually presents as groin pain ± dull or sharp pain in the trochanteric area, internal rotation and abduction are lost first
  - pain can radiate to the anterior thigh, but generally does not go below the knee
- knee
  - initial narrowing of one compartment, medial > lateral; seen on standing x-rays, often patellar-femoral joint involved
- foot
  - common in first MTP and midfoot
- lumbar spine
  - very common, especially L4-L5, L5-S1
  - degeneration of intervertebral discs and facet joints
  - reactive bone growth can contribute to neurological impingement (e.g. sciatica, neurogenic claudication) or spondylolisthesis (forward or backward movement of one vertebra over another)
- cervical spine
  - commonly presents with neck pain that radiates to scapula, especially in mid-lower cervical area (C5 and C6)

Differential Diagnosis of Elevated ESR
- Systemic inflammatory diseases
- Localized inflammatory diseases
- Malignancy
- Trauma
- Infection
- Tissue injury/ischemia

Investigations

- blood work
  - normal CBC and ESR, CRP
  - negative RF and ANA
- radiology: 4 hallmark findings, see sidebar
- synovial fluid: non-inflammatory (see Table 8)

The Radiographic Hallmarks of OA
- Joint space narrowing
- Subchondral sclerosis
- Subchondral cysts
- Osteophytes

Treatment

- presently no treatment alters the natural history of OA
- prevention: prevent sports injury, healthy weight management
- **non-pharmacological therapy**
  - we ght loss (minimum 5-10 lb loss) if overweight
  - physiotherapy: heat/cold, low impact exercise programs
  - occupational therapy: aids, splints, cane, walker, bracing
- **pharmacological therapy (see Table 33)**
  - 1st line- oral: acetaminophen/NSAIDs
  - treat neuropathic pain if present (anti-depressants, anti-epileptics, etc.)
  - joint injections: corticosteroid (effective for short-term treatment), hyaluronic acid (evidence of long-term benefits)
  - topical: capsaicin, NSAIDs
  - glucosamine ± chondroitin (efficacy not well supported)
- **surgical treatment**
  - joint debridement, osteotomy, total and/or partial joint replacement, fusion (see Orthopedics, OR6)

Seropositive Rheumatic Disease

- diagnosis vs. classification in rheumatology
  - diagnostic criteria are often dependent on disease progression and evolution over time, as early objective measures are often unavailable
  - classification criteria are derived from studying patients with long-term diseases and clear diagnoses in order to determine which criteria have good specificity in the early prediction of certain diagnoses
- seropositive arthropathies are characterized by the presence of a serologic marker such as positive RF or ANA
- a small subset of the vasculitides the small vessel ANCA-associated vasculitides, have a measurable serological component, but they are often considered a separate entity from seropositive disease by experts