Musculoskeletal System
Arthritis & Related Rheumatic Problems

Ankylosing Spondylitis

- Also known as Rheumatoid Spondylitis
- Chronic, progressive inflammatory disease of the spine & sacroiliac joint
- More in men than women – usually < 40 yrs old
- Unknown etiology
- Genetic predisposition – HLA-B27
- Inflammatory process triggers the disease

- **Pathophysiology**
  - Spondylitis = inflammation of the spine
  - Ankylose = fuse
  - Inflammation causes bones of the spine to grow together or fuse
  - Inflammation begins at the sacroiliac joint & destroys the articular cartilage
  - Cartilage replaced with new bone growth
  - Inflammation progresses up the spine = fusion of the entire spine

- **Clinical Manifestations**
  - Initially = low back pain, hip, knee, or shoulder pain & swelling, mild fever, ↓ appetite, fatigue
  - Pain - worse at night & in the AM, improves with mild activity
  - Over time, back pain ↓ & back motion becomes restricted
  - Fusion of the sacroiliac joints & spine up through the cervical spine may occur over 10-20 yrs
  - Kyphosis develops = bent over compensating hip-flexion contractures
  - Knees flex when they attempt to move their head into an upright position

  - Systemic Signs & Symptoms:
- Peripheral arthritis of shoulders, hips, knees
- Iritis = ocular inflammation; uveitis = intraocular inflammation- most common nonskeletal symptom
- Aortic valve regurgitation
- Pulmonary fibrosis

- **Diagnosis**
  - HLA - B27
  - (human leukocyte antigen)
  - X-Ray → syndesmophytes that bridge the vertebrae –"bamboo" appearance of spine
  - **New bone growth**
  - ↑ ESR, alk.phos, CPK, CK
  - MRI – early cartilage abnormality

- **Treatment**
  - **Goal: maintain maximal skeletal mobility**
  - Exercises & proper posture
- **Prone lying 3-4 x day for 15-30 min**
  - Rest
  - Heat
  - Hydrotherapy – pain and facilitate spinal expansion

– Anti-inflammatory analgesics
• Salicylates, NSAIDS, corticosteroid injections
– DMARDs (Methotrexate) – joint involvement, not so much spinal & BRMs (infliximab/ Remicade)- inhibits action of TNF (which promotes inflammation)
– Surgery
• Spinal osteotomy – for extreme flexion deformities
• THR for hip ankylosis

• **Prevention of Flexion Deformities**
– Keep HOB flat to **prevent flexion deformities**
– **Encourage prone position at least 2x day**
– Position flat on back with affected joints in position of extension

• **Nursing Management**
  • Teaching → disease and treatment
  • Pain Management
  • **<u>Proper positioning</u>** → firm mattress, no pillows, avoid flexion (prolonged standing, sitting, walking), exercises- strengthening, stretching ROM
  • **Respiratory Exercises → deep breathing**
  • Resources

Reiter's Syndrome

• **<u>Arthritis, urethritis, & conjunctivitis</u>**
• Unknown etiology
  • Reactive arthritis triggered by GU or GI infections (chlamydia, salmonella, campylo-bacter)
  • Usually affects **men**
  • 85% with a +HLA-B27 (genetic marker)
• Mucocutaneous lesions (painless superficial ulcerations)
• Achilles tendonitis or plantar fasciitis
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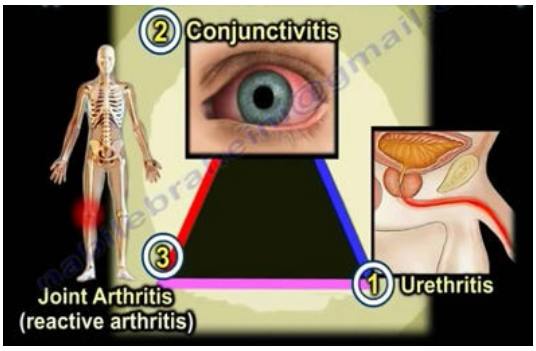


• **Diagnosis**
  • Few lab abnormalities; usually dx with S/Sx

• **S/S:**
  • **Urethritis –usually 1st S/Sx**
  • Asymmetrical arthritis – weight bearing joints
  • Arthralgia's usually <u>begin 1-3 wks after the initial infection</u>
  • **Conjunctivitis**
  • Fever, anorexia, wt. loss, Achilles tendonitis

- **Treatment**
- **Symptomatic:**
- Arthritis (NSAIDs)
  - Ophthalmic steroids- conjunctivitis
- Urethritis (antibiotics)
- Chlamydia (doxycycline & treat partner)
- NSAIDs – joint inflammation or DMARDs ie: methotrexate and sulfasalazine

-Physical Therapy

- **Prognosis: 2-16 weeks**
  - Usually with complete remission & full joint movement; ½ with recurring attacks
  - Others with chronic course → continued synovitis & x-ray changes resembling AS, may = complete disability

Septic Arthritis

- *Bacterial arthritis*
- Invasion of synovial membranes with microorganisms: staph & strept

- **Predisposing Factors:**
  - Joint trauma or surgery
  - Intra-articular injections
  - RA

- **Pathophysiology**
  - Synovial tissues become inflamed as a response to the bacterial invasion
  - Joint cavity becomes inflamed & + pus in the synovial membrane & fluid
  - If it progresses >abscesses form in synovium & bone, and destroy the cartilage
  - Ankylosis of the joint

- **S/Sx**
  - pain, swelling, & tenderness
  - Fever, shaking with chills

- **Diagnosis**
  - Arthrocentesis – joint aspiration
    - Presence of organisms, ↓ glucose content
  - ↑ WBC, sed rate, & C-reactive protein
  - Blood cx's before abx
  - X-ray = bone changes late in the process if not treated

- **Treatment:**
  - Prompt diagnosis & treatment - joint destruction
  - Antibiotics – responds within 2 weeks or may take as long as 4-6 weeks
  - Rest or immobilization of joint - ↓ pain
  - Open surgical drainage & irrigation
  - Active ROM after infection subsides

Gout

- **Metabolic Disorder**
  - Men > Women; Peaks at age 50; Genetic tendency
  - Result of **prolonged hyperuricemia**
  - Problems with **synthesizing purines or poor renal excretion of uric acid**

- **Classifications of Gout:**
  - Primary = hereditary *defect of purine metabolism*
    - ↑ production & ↓ excretion of uric acid
  - Secondary = *related to another disorder or a result of meds* known to inhibit uric acid excretion or ↑ rate of cell death (chemo)
    - Obesity, DM, hypertension, atherosclerosis, malignant disease, ETOH
- **Pathophysiology**
  - ↑ serum uric acid
  - Urate crystals form in synovial tissue causing severe inflammation

3 Clinical Stages of Gout:
-Aysmptomatic hyperuricemia
-acute gouty arthritis
-chronic or tophaceous gout

- ASA & Diuretics precipitate an attack
- **S/Sx**
- **Acute S/Sx**: extreme pain (lessen the pain!), swelling, erythema of involved joints, low fever
- Inflammation of great toe (podagra) – usually the 1st joint affected (1st MTP)
- Other joints affected include ankle, heel, & knee
- Usually rapid onset – subsides in 2-10 days
- **Chronic gout** = multiple joint involvement with deposits of sodium urate crystals (**tophi**)
- Usually seen in the synovium, vertebrae, along tendons, in the skin, and cartilage

- **Complications**
  - Chronic inflammation > joint deformity
  - Cartilage destruction > OA
  - Kidney stone formation from ↑ uric acid excretion
  - Renal disease
- Infection & urate deposits causing obstruction

- **Diagnosis**
  - H&P and S/Sx
  - ↑ serum uric acid
  - Normal or ↑ 24° urine for uric acid
  - Aspirate joint – urate crystals in synovial fluid; helps r/o septic arthritis and pseudo-gout (calcium phosphate crystals)
  - Assess kidney function

- **Treatment**
- Acute Attack:
- Anti-inflammatory agents
- Xanthine Oxidase Inhibitor = Colchicine – **large dosage given until side effects occur (N,V,D) or pain subsides (12 hours)**
- NSAIDS (indomethacin/indocin, butazolidine); not aspirin
  - Corticosteroids – do not stop abruptly
- Joint rest
- Prevent future attacks:
- Maintenance dose of colchicine
- Wt. reduction if necessary
- Avoid ETOH & high purine foods
- Meds to ↓ serum urate concentration

Foods High in Purines

High amounts of purines

- Sardines, Herring, Mussels, Liver, Kidney, Venison, Meat soups

Moderate amounts of purines

- Chicken, Salmon, Crab, Veal, Bacon, Pork, Ham, Beef

- **Treatment**
  - Prevent uric acid kidney stones & other associated conditions
- *↑triglycerides & hypertension*
  - **Meds to enhance uric acid excretion (uricosuric)**
    - Can cause renal damage; stay well hydrated

- probenecid/Benemid- ↑ dose until uric acid is WNL → cannot take ASA with this s/fx: D,N/V, cramping, hair loss

- sulfinpyrazone- cannot tolerate benemid
  s/fx- nausea, heartburn

  - **Meds to decrease uric acid formation (xanthine oxidase inhibitors)**

- allopurinol/Zyloprim– encourage drinking extra fluids

  - febuxostat/Uloric for hyperuricemia

  - Additional: Losartan promotes urate diuresis for HTN

- **Nursing Management**
  - Teaching
  - Comfort measures – rest until pain ↓; bed cradle
  - Prevent complications: kidney stones

Systemic Sclerosis (Scleroderma)
Scleroderma

- Disorder of the connective tissue characterized by fibrotic, degenerative, & inflammatory changes in the skin, blood vessels, synovium, skeletal muscle, & internal organs
  - **Skin Thickening and tightening**

- **Incidence:**
  - ↑ in women, childbearing years
  - More in African American women
  - 30-50 yr olds

- **Pathophysiology**
  - Unclear cause (environmental, occupational)
  - *Overproduction of collagen*

  - Cell changes occur → platelet aggregation → **fibrosis, ↓ elasticity, stenosis, & occlusion of vessels**

- Classification for systemic sclerosis
  - Diffuse scleroderma – trunk, face, and proximal & distal extremities
  - Limited scleroderma – limited to sites distal to the elbows & knees, can involve the face & neck

- **CREST Syndrome**
  - **C**alcinosis- calcium deposits (fingers, forearms, pressure points)
  - **R**aynaud's Phenomenon – intermittent vasospasm of fingertips (cold/stress)
  - **E**sophageal dysmotility
  - **S**clerodactyly – tightening of skin on fingers and toes
  - **T**elangiectasis – spider-like hemangiomas (hands/forearms/palms/face/lips)

- **S/Sx**
  - Raynaud's Phenomenon (paroxysmal vasospasm of digits)
- ↓ blood flow to fingers & toes on exposure to cold (**white** of blanching phase)
- Followed by cyanosis as Hgb releases O_2 to the tissues (**blue** phase)
- Then erythema or rewarming (**red** phase) – N/T
- *may precede the systemic disease*
  - Symmetrical painless swelling or thickening of the skin of the fingers & hands → progresses to the trunk (scleroderma)
- Taut, shiny, thick skin
  - Esophageal hypomotility (heartburn)
  - GI Complaints – abd. distension, diarrhea or constipation
  - Lungs → pleural thickening & pulmonary fibrosis: lung disease= main cause of death now
  - Cardiovascular → pericarditis, pericardial effusion, cardiac dysrhythmias, myocardial fibrosis
  - Renal disease → malignant HTN > irreversible renal insufficiency; ACE inhibitors &

transplants

- **Prognosis**
  - Variable
- **Diagnosis**
  - H&P
  - Slightly ↑ ESR
  - Serum Creatinine- if kidneys involved
  - UA- hematuria, proteinuria
  - +ANA (antinuclear antibody)
  - Skin biopsy
  - X-rays – chest & GI- calcifications
  - Testing related to specific complaints (PFT's)

- **Treatment**
  - No specific treatment
  - Prevent or treat secondary complications
  - Anti-inflammatory meds, penicillamine (thins the skin), colchicine (↓ collagen production), steroids, immunosuppressant's
  - Antacids / PPI's
  - Vasodilator & antihypertensive meds (captopril/capoten)
    CCB & ARBs – Raynaud's; ACE - renal
  - Physical therapy

Systemic Lupus Erythematosus (SLE)
Lupus

- Chronic, progressive, inflammatory, multi-system disease of connective tissue
- Often involves the skin, joints, serous membranes, kidney, hematological system, & neurological

- **Incidence:**
  - ↑ women, 3x more in African American women
  - Often affects adolescents & young adults
- **Etiology**
  - Unknown
  - Possible Causes:
- Autoimmune response causes immune complexes containing antibodies to be deposited in tissue = tissue damage

- Viral Infection

- Some Meds → procainamide & quinidine, some seizure meds; months to years after continuous therapy

- May cause lupus-like syndrome (within a few hours to days after starting)

- Genetic Predisposition- HLA

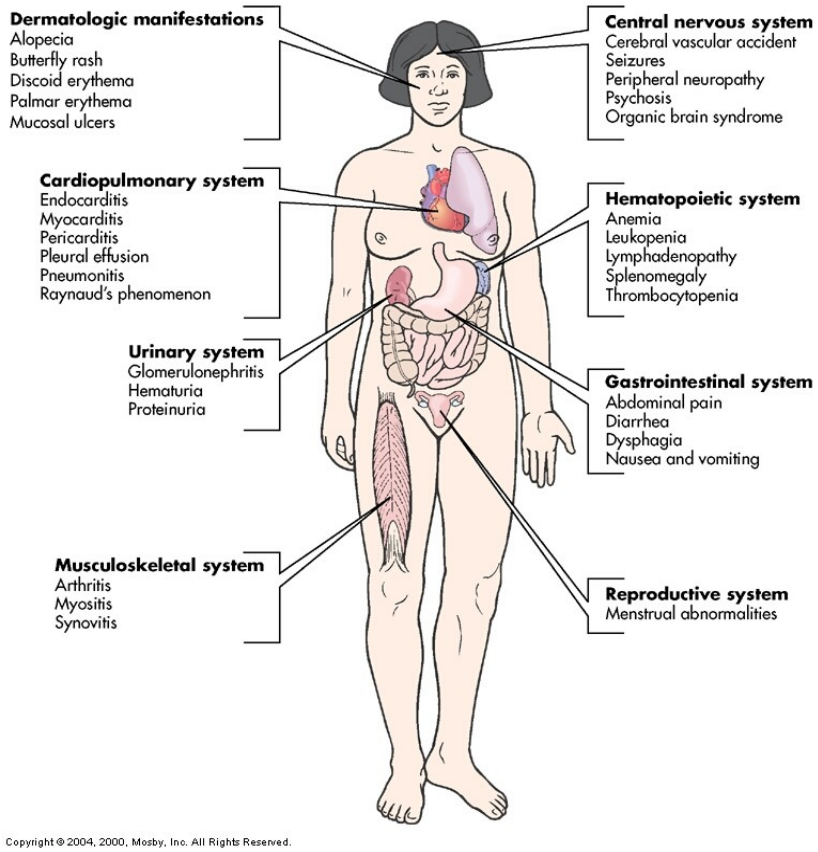
  - Hormones
    - Onset and exacerbation after onset of menarche, birth control, during/after pregnancy, worse in postpartum

  - Environmental Factors- sun

- **Pathologic Manifestations of Lupus**
  - An autoimmune process
  - Antinuclear antibodies (ANA) affect DNA in cell resulting in the formation of immune complexes in serum & organs.
  - Immune complexes > inflammation & damage to organs or cause vasculitis which deprives the organs of arterial blood and oxygen.

  - Synovial involvement
  - Severe vasculitis with necrosis of the walls of the small arteries
  - Renal Involvement – renal failure
  - Lymph node necrosis
  - Development of small white spots in the retina (cytoid bodies)
  - Lesions of the nervous system

- **S/Sx:**
  - Variable
- Mild to rapidly progressive
  - No characteristic pattern; No predictable system
  - Alternating **periods of remission & exacerbation**
  - Initial S/Sx is often arthritis → precedes the onset of multisystem disease by many years
  - Fever, wt. loss, excessive fatigue may precede an exacerbation
  - Classic butterfly rash – across the bridge of the nose & cheeks in 40% of patients
- anywhere on the body → mostly seen on the face and chest
  - Sunlight (UV radiation) can cause a severe skin reaction
  - Oral ulcers
  - Alopecia
  - Arthritis with morning stiffness
  - Pericarditis, atherosclerosis, pleurisy
  - Hematuria, proteinuria, casts in urine, ↑ creatinine level
  - Seizures, Organic Brain Syndrome (disorientation, memory, psychiatric symptoms)
  - **Infection – especially pneumonia**



- **Diagnosis**
  - H & P
  - Labs: ↑ ESR, anemia, ↓ WBC & plt count, +ANA, anti-double-stranded DNA, anti-Smith (Sm) – specific for SLE
  - EKG or CXR → pericarditis or pleural effusion
  - Urine → casts, protein, RBCs
  - SLE is diagnosed primarily on a distinct criteria: ***patient history, physical examination, and laboratory findings***

- **Criteria for Diagnosis of SLE**
  - Malar rash: butterfly rash
  - Photosensitivity: rash after sun exposure
  - Arthritis: non-erosive, involving 2 or more joints
  - Renal Disorder: proteinuria, casts
  - Antinuclear antibodies: 97% have + ANA
  - Discoid rash: red, scaly patches, plugged hair follicles on face and other sun exposed areas
  - Oral Ulcers
  - Serositis: pleuritis or pericarditis
  - Neurologic disorders: seizures, psychosis
  - Hematologic disorder: anemia, leukopenia, thrombocytopenia
  - Immunologic disorder: + LE prep

- **Medical Management**
  - Rest
  - Exercise to maintain mobility
  - Avoidance of sun

  - Meds
    - Anti-inflammatory analgesics → control arthritic pain

    - Antimalarial meds – extensive rash (hydroxychloroquine/Plaquenil)- eye exam

    - Corticosteroids – severe neurologic & renal involvement- taper

    - Ointments – skin rash, topical steroids

    - Cytotoxic meds if others fail (Imuran, Cytoxan, methotrexate)

  - Renal Dialysis & Transplant

- **Nursing Care**
  - Teaching – disease, monitor temperature, skin care, treatment, and follow-up
  - Emotional support
  - Home management
  - ↓ precipitating factors to exacerbations
    - Fatigue, sun exposure, stress, infection
  - Pregnancy may cause an exacerbation of S/Sx
  - Lupus Foundation – www.lupus.org

Polymyositis & Dermatomyositis

Polymyositis

- Inflammatory disease involving voluntary (striated) muscles
- Increased in women, 45-65yrs but can occur at any age
- Men & women ages 30-60
- Unknown etiology
- Degeneration of muscle fibers – necrosis of muscle fibers- blood vessels supplying the muscles become inflamed- interstitial fibrosis

- **S/Sx**
  - Exacerbations & remissions
  - Proximal muscles affected first (pelvis & shoulder)
  - Muscle weakness – no pain
  - Contractures
  - **Dermatomyositis** – dusky red rash on face, neck, shoulders, anterior chest, upper back, & arms (skin involvement with muscle weakness)
    - Gottron's papules: scaly, smooth, or raised rash on knuckles & sides of hands

- **Diagnosis**
  - H&P

- EMG (electromyography)
- Muscle biopsy (necrosis, degeneration, regeneration, fibrosis)
- Increased muscle enzymes (CPK) reflects muscle damage

- **Treatment**
  - Rest
  - Corticosteroids (topical for rash)
  - Immunosuppressant's
  - Physical Therapy

Sjogren's Syndrome

- Autoimmune response
- Inflammation & dysfunction of the exocrine glands (salivary & lacrimal glands)
- Women >40 yrs of age, but at any age
- Genetic
- Environmental

- **S/Sx**
  - Decreased tearing – keratoconjunctivitis sicca
- Burning, photosensitivity
  - Dry mouth - xerostomia
- Fissures, dysphagia, dental caries
  - Dry nasal & respiratory passages
- Cough
  - Enlarged parotid glands
  - Hoshimotos' & Grave's Disease
  - Lymph nodes, bone marrow, visceral organs

- **Diagnosis**
  - Histology (salivary and lacrimal glands)
  - + ANA & RF
  - Anemia, leukopenia, inc ESR
  - Confirm dx with ophthalmological exam (Schirmer's Test), salivary flow rates, salivary gland biopsy

- **Treatment**
  - Symptomatic
  - Artificial tears, increase fluids, dental hygiene, humidification
  - Restatis (cyclosporine- eye drops)

Fibromyalgia Syndrome (FMS)

- Chronic central pain syndrome marked by widespread, nonarticular musculoskeletal pain and fatigue with multiple tender points

-Occurrence
Previously healthy, young, & middle-aged women

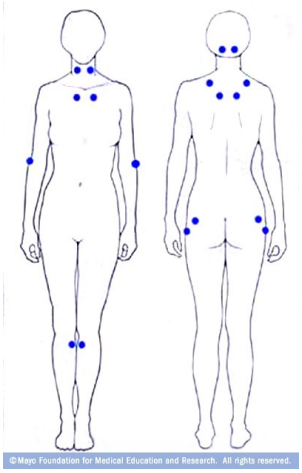
-Etiology
- Familial tendency
- Infectious trigger- recent illness or trauma may trigger in susceptible people
- Dysfunction in hypothalamic-pituitary-adrenal axis (changes can affect mood physical and mental health)
- Alteration in CNS

-Signs & Symptoms:
Malaise, fatigue, cognitive dysfunction, headache, sleep disturbances, depression, anxiety, fever, generalized musculoskeletal pain- widespread burning

-Course of Disease
Variable intensity of symptoms
Fluctuates over time

-Diagnosis
- No definitive lab tests or joint or muscle exams
- Diagnosis of exclusion
  -if two criteria met: 1) pain in 11/18 tender points on palpation, 2) history of widespread pain is noted for at least 3 months

-Treatment
- Symptomatic
- Antidepressants- duloxetine /Cymbalta
- Heat, massage, regular stretching, biofeedback, stress management, relaxation training
- Widespread pain- pregabalin/lyrica
- Education- rest can help with the pain and aching and tenderness
- Consistent support is needed
- Stress management – trigger for pain



Resources for Arthritic Conditions

- www.arthritis.org
- www.sceroderma.org
- www.lupus.org