

Musculoskeletal System

Arthritis & Related Rheumatic Problems

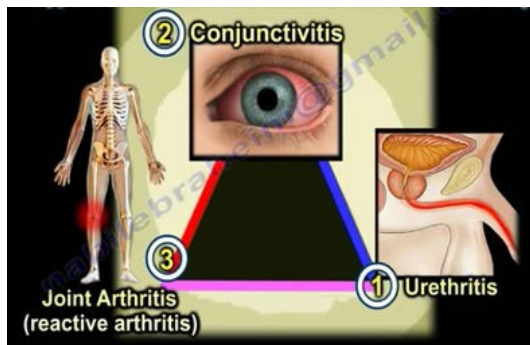
Ankylosing Spondylitis

- 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
 - **Proper positioning** → firm mattress, no pillows, avoid flexion (prolonged standing, sitting, walking), exercises- strengthening, stretching ROM
 - **Respiratory Exercises → deep breathing**
 - Resources

Reiter's Syndrome

- **Arthritis, urethritis, & conjunctivitis**
- Unknown etiology
 - Reactive arthritis triggered by GU or GI infections (chlamydia, salmonella, campylobacter)
 - 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 begin 1-3 wks after the initial infection
 - **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:
 - Asymptomatic 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^h 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
 - o Diffuse scleroderma – trunk, face, and proximal & distal extremities
 - o Limited scleroderma – limited to sites distal to the elbows & knees, can involve the face & neck
- **CREST Syndrome**
 - o Calcinosis- calcium deposits (fingers, forearms, pressure points)
 - o Raynaud's Phenomenon – intermittent vasospasm of fingertips (cold/stress)
 - o Esophageal dysmotility
 - o Sclerodactyly – tightening of skin on fingers and toes
 - o Telangiectasis – 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 O₂ 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

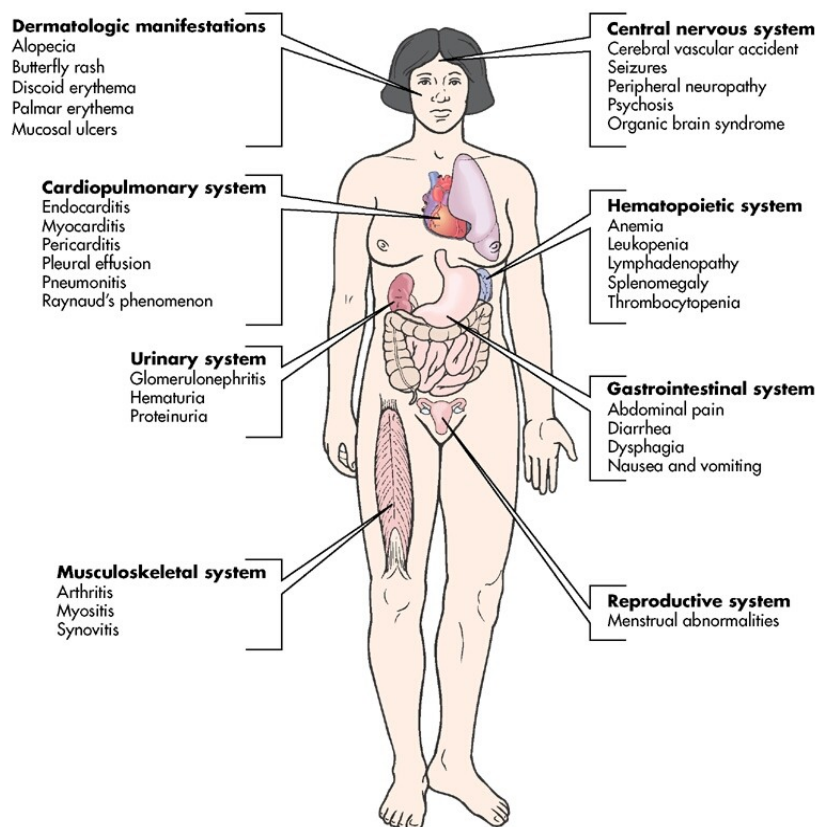
- **Prognosis**
 - Variable
- **Diagnosis**
 - H&P
 - Slightly ↑ ESR
 - Serum Creatinine- if kidneys involved
 - UA- hematuria, proteinuria
 - +ANA (antinuclear antibody)
 - Skin biopsy
 - X-rays – chest & GI- calcifications
 - Testing related to specific complaints (PFT's)
- **Treatment**
 - No specific 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**



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- **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, Cytosan, 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

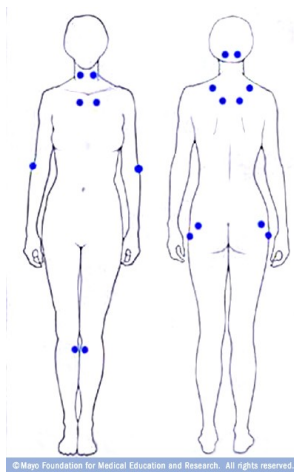
Fluctuates over time

-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.scleroderma.org
- www.lupus.org