

# Lecture 8.

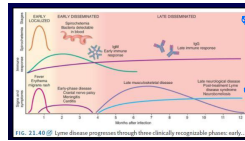
\* **Suppurative arthritis** : Suppuration means destruction of tissue forming (liquefactive necrosis, abscess, Pus ...) mainly by **Bacterial infection** and reach joint through blood stream "hematogenous spread"

- There is difference regarding which organism effect depends on age like: less than 2 years: H-influenza / older children & adult: S. aureus
- Patient who have sickle cell disease have a little bit higher incidence of Salmonella infection (G+ve bacilli) / Gonococcus young adult (think about G+ve bacilli) / Part of STD (sexual transmitted) (Neisseria meningitidis and Neisseria gonorrhoeae)
- Clinically: Sudden acute Pain + Swollen & warm Joint, most common affected Knee Joint with systemic manifestation (fever/leukocytosis/elevated ESR)
- \* Diagnosed: diagnosed depends on clinical picture, however the best way is aspirate joint and Pus coming out send it for culture and microscopic analysis
- Treatment: intravenous antibiotics & acute suppurative arthritis shouldn't be missed

## \* Lyme arthritis

Most common things you need to know

- ① it is an infectious arthritis, Part Systemic disease
- ② it has multiple Phase and its due to an immune response initially by IgM then by IgG
- ③ very rare in Jordan



it is slightly specific type of arthritis Part of systemic disease due infection cause by spirochetes (Borrelia burgdorferi) Patient will have initial generalized systemic symptoms like (fever, erythema migrans rash) after that there is some neuritis with central nerve palsy and sometimes the organism reaches the meninges and cause meningitis and reach heart cause endocarditis. Initial infection will induce primary immune response (IgM) after that when spirochetes are disseminated the (IgG) late immune response start appearing during that the MSS symptoms start appearing. Some times its self-limiting and sometimes its need treatment then after that there will be neurological deficit not knowing (neuroborreliosis) because organism is Borrelia burgdorferi.

common in our region

\* **crystal-induced arthritis**: Crystals deposited in joint cause disease → deposition of crystal trigger inflammatory reaction which will accumulating and destroying the cartilage (can be acute or chronic)

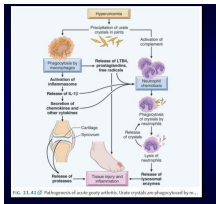
(Produce inside the body)

- Endogenous crystals: \* Mono sodium urate "MSU" (Gout) \* Calcium Pyrophosphate dehydrogenase, "CPPD" (Pseudo Gout)

① Gout: transient attacks of arthritis any joint can be involve mainly big toe, triggered by deposition of MSU crystals. (usually very Painful) Swollen toe

\* Uric acid: is a purine metabolite increase production of uric acid & decrease excretion from kidney cause hyperuricemia (major cause of Gout)

\* with hyperuricemia, risk increase with: 20-30 years of age, obesity, alcohol, genetic predisposition, drug (thiazides which are diuretic & uric acid in blood)



Reason of any cause for hyperuricemia → uric acid crystal ↑ they will get deposited in the joints → activation of multiple inflammatory cell macrophage & neutrophils → inducing a cascade of inflammatory reaction activation of inflamase → release IL-1 & IL-6 → activation of neutrophils these will also induced by phagocytosis of crystals by neutrophils causing lysis → release more inflammatory enzyme more processes lead tissue injury and destruction in the joint. major ending event presence of uric acid crystal due presence of hyperuricemia

\* treatment: life style modification (↓ amount of eating meat & liver because they rich purines), NSAIDs & Colchicine in acute gout, Xanthine oxidase inhibitor (Allopurinol) in chronic and prevention

MORPHOLOGIC CHANGES OF GOUT:	
Acute arthritis	Dense inflammation of synovium, MSU crystals in neutrophils, -ve birefringent
Chronic tophaceous arthritis	Repetitive attacks & crystals deposition in the joint; thick synovium, pannus
Tophi in various sites	Cartilage, ligaments, bursae and tendons
Gouty nephropathy	MSU crystals deposition in kidney; nephrolithiasis & pyelonephritis

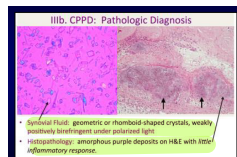


Amorphized the destroy by chronic Gout → chalky white material are calcified uric mono sodium urate clefts (MSU crystals) these are the two tophi which are equal reducing inflammatory reaction in many regions, cartilage, ligament and tendon

② Pseudogout: >50 years, incidence increase with age / idiopathic (unknown cause) may be genetic or secondary

\* CPPD crystal induced arthritis via triggering inflammatory reaction (same as MSU) / secondary are usually with Diabetes Mellitus Previous joint damage (HPT), hemochromatosis "where iron is deposited cause deposition of CPPD crystals"

\* can present in Acute, subacute, chronic forms \* treatment: supportive, no preventive measure so far (no curative Trx)



Microscopic analysis shows CPPD Calcium Pyrophosphate crystals which appear shorter than "MSU" crystals and are often rhomboidal under a Polarizing filter They are positively birefringent appearing blue when aligned parallel with slow axis of the compensator MSU are needle shaped and yellow under polarizing light.

Summary

Arthritis

- Osteoarthritis (OA, degenerative joint disease), the most common disease of joints, is a degenerative process of articular cartilage in which matrix breakdown exceeds synthesis. Inflammation is minimal and typically secondary. Local production of inflammatory cytokines may contribute to the progression of joint degeneration.
- Rheumatoid arthritis (RA) is a chronic autoimmune inflammatory disease that affects mainly small joints, but can be systemic. RA is caused by a cellular and humoral immune response against self-antigens, particularly citrullinated proteins. TNF plays a central role and antagonists against TNF are of clinical benefit.
- Seronegative spondyloarthropathies are a heterogeneous group of likely autoimmune arthritides that preferentially involve the sacroiliac and vertebral joints and are associated with HLA-B27.
- Suppurative arthritis describes direct infection of a joint space by bacterial organisms.
- Lyme disease is a systemic infection by Borrelia burgdorferi, which manifests, in part, as an infectious arthritis, possibly with an autoimmune component in chronic stages.
- Gout and pseudogout result from inflammatory responses triggered by precipitation of urate or calcium pyrophosphate, respectively.

ملاحظة  
7x8

NEGATIVE VS POSITIVE BIERFRINGENCE

Interference Colors in Gout and Pseudo-Gout Crystals

Monosodium Urate (Gout) Crystals

Calcium Pyrophosphate Dihydrate (CPPD) Crystals

Figure 6: Pseudogout Crystals

Yellow (birefringent)

Blue (birefringent)

Blue (birefringent)

Yellow (birefringent)

Monosodium Urate (Gout) Crystals

Calcium Pyrophosphate Dihydrate (CPPD) Crystals

remember that

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