DEPARTMENT OF PATHOLOGY AND MICROBIOLOGY

GOUT

- Is a heterogenous group disease characterized by hyperuricemia.
- Caused by a disorder of purine metabolism
- Resulting in deposition of urate crystals in synovium
- Recurrent acute arthritis progressing to chronic deforming arthritis,formation of tophi,
- Systemic complication like renal failure.

Causes

Primary or genetic gout

- primary overproduction
- under excretion of uric acid

Secondary gout –

- hyperuricemia ,
- overproduction or
- Defective excretion of uric acid

Pathogenesis

Depostition of Monosodium urate crystals in the synovium. ⟹ Polymorphonuclear leucocyte ingest the crystals. ⟶ Release lysosomal enzymes cause inflammation ⟶ Crystal seen in synovium, articular cartilage

PATHOLOGY

- Erosion of articular cartilage, proliferation of synovial membrane.
- pannus formation, cystic erosion of bones 2^{nd} ary osteoarthritic changes
- **TOPHI**:- nodular urate deposits found in & around the joints & in the articular cartilage.

3 clinical stages

asymptomatic hyperuricemia
acute gouty arthritis
chronic tophaceous gout.

PSEUDO GOUT

- Calcium pyrophosphate crystal deposition disease / CPPD
- Calcium pyrophosphate dihydrate crystals may be deposited in joint tissues and this lead to acute gout like attack.
- Familial or due to metabolic disorders , Men -50 yrs Both sexes
- Seen in synovial fluid, Deposits in hyaline cartilage ,fibrocartilage.
- Menisci of knee, articular disc of distal radioulnar joints, acetabulum, symphysis pubis & annulus fibrous of lumbar & dorsal intervertebral discs.
- **C/F** - silent
- Recurrent attack of subacute inflammation, progressive degeneration of joints.
- Gross – chalky white deposits
- Micro – rhomboidal crystals.

OSTEOARTHROSIS

- Osteoarthritis Degenerative joint disease
- Elderly people, WEAR & TEAR OF joint

Aetiology

- Hereditary, Over use of joint, Aging, 2nd ary osteoarthritis- obesity, Hypermobility , Orthopedic deformities, Endocrine disorders- diabetes mellitus, acromegaly, hyperparathyroidism

Pathogenesis

Mechanical stress → distrupt the collagen fiber network of articular cartilage. ⟶ Chonrocyte function & number changes. ⟶ Lubricanting mechanism impaired. Normal surface of matrix –lost. ⟶ fibrillation of the cartilage fibers, CLEFT formation. ⟶ Synovial fluid access to the deeper layer of the cartilage.

Pathology

- Destruction of load bearing cartilage. Cartilage – **calcification** . Exposed weight bearing bone undergoes eburnation. **osteophytes** from the margin of articular bone Subchondral bone may fracture to form cysts. Crystal liberates to synovial cavity. Synovial inflammation & effusion occurs.
- **c/f** Knee ,cervical lumbar spine,hip,shoulder & distal interphalangeal joints.—affected.
- Heberden's nodes- Pain,stiffness <sitting posture. < walking downstairs,slopes.
- Quadriceps atrophy Transient effusion occurs. Osteophytes may be palpated around the joint.

Crepitus develop