''बेटी बचाओ, बेटी पढ़ाओ''

JAYOTI VIDYAPEETH WOMEN'S UNIVERSITY, JAIPUR

FACULTY OF HOMOEOPATHIC SCIENCE

Teaching Methodology

Faculty Name : JV'n Dr. Ravi Jain (Asso. Professor & HOD)

Program : BHMS

Course : Practice of Medicine

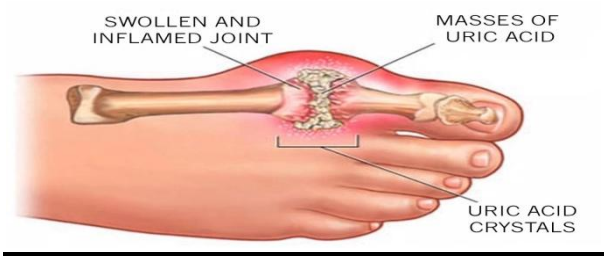
Session : Gout

Academic Day starts with –

- Greeting with saying **'Namaste'** by joining Hands together following by 2-3 Minutes Happy session, Celebrating birthday of any student of respective class and **National Anthem**

Lecture Starts with-

- **Review of previous Session-** In previous session as I had discussed about Psoriatic Arthritis.
- **Topic to be discussed today-**. In todays lecture I will start with Gout.
- **Lesson deliverance (ICT, Diagrams & Live Example)-**
  - PPT (20 Slides)
  - Diagrams



Picture of changes in Gout

- **Gout** is a form of inflammatory arthritis with high levels of **uric acid** in the blood.
- The acid forms needle-like crystals in the joint.
- It cause sudden, severe episodes of pain, tenderness, redness, warmth and swelling.
- **Hyperuricemia :** Plasma and extracellular fluids become supersaturated with uric acid.
- Normal plasma uric acid : 2.5-6 mg/dl in women, 3.4-7 mg/dl in men.
- Gout is a metabolic disorder of purine metabolism.
- Characterized by recurrent attacks of :
- Acute arthritis
- Chronic deforming arthropathy
- Formation of tophi
- Systemic complication like renal failure.

Incidence

- It affects middle aged to elderly men and postmenopausal women.
- M: F ratio is 7:1 -9:1
- Only around 5% of hyperuricemic patients develop gout.
- Most women with gouty arthritis are postmenopausal and elderly, have osteoarthritis and arterial hypertension that causes mild renal insufficiency, and usually are receiving diuretics.

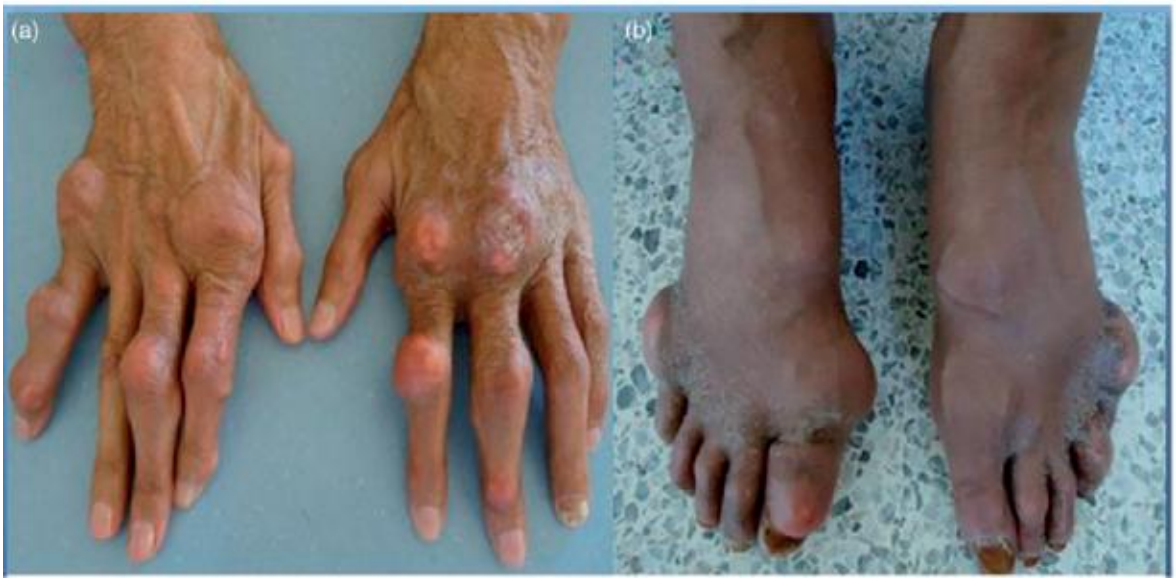
Pathogenesis

- Uric acid is the end product of purine nucleotide degradation.
- Its production is closely linked to pathways of purine metabolism, with the intracellular concentration of 5-phosphoribosyl-1-pyrophosphate (PRPP)

- Uric acid is excreted primarily by the kidney through glomerular filtration, tubular secretion, and reabsorption.
- Hyperuricemia arises with overproduction or reduced excretion of uric acid or a combination of the two.
- Arthritis is caused due to deposition of monosodium urate (MSU) crystals in the synovium.
- Polymorphonuclear leucocytes ingests crystals and release lysosomal enzymes which causes inflammation.
- Acute arthritis : Crystals are demonstratable in the synovium and articular cartilage.
- Chronic arthritis : Erosion of articular cartilage, proliferation of synovial membrane and pannus formation.- secondary OA changes occurs.
- Tophi are nodular urate deposits in and around the joints in articular cartilage.
- **Histologically**
- Monosodium urate crystals are surrounded by mononuclear cell infiltration and foreign body giant cells which leads to:
- Osteoarthritic changes,
- Ankylosis of joints,
- Tissue destruction.
- Long standing cases develops multiple renal calculi, pyelonephritis, and atherosclerosis.

Clinical Features

- Gout passes through 3 clinical stages :
- Asymtomatic hyperuricemia
- Acute gouty arthritis
- Chronic tophaceous gout.



Acute Arthritis

- Only one joint is affected initially, but polyarticular acute gout in subsequent episodes.
- First joint : The metatarsophalangeal joint of the first toe is involved.
- Inflamed Heberden's or Bouchard's nodes may be a first manifestation of gouty arthritis.
- It begins at night with dramatic pain, swelling, warmth, and tenderness.
- Attack will generally subside spontaneously after 3–10 days.
- There are recurrent episodes with intervals of varying length with no symptoms between attacks.
- **Podagra** : Painful affection of the foot occurring as a result of metatarsophalangeal arthritis.
- Later other joints tarsal joints, ankles, and knees also are also involved.
- These are associated with fever and other constitutional disturbances.

Precipitation Factors

- Dietary excess : red meat, liver, pancreas, testes, peas, etc.

- Trauma,
- Surgery
- Excessive ethanol ingestion
- Hypouricemic therapy
- Serious medical illnesses such as myocardial infarction and stroke.

Chronic Arthritis

- Recurrent attacks presents with nonsymmetric synovitis.
- It manifest only as periarticular tophaceous deposits in the absence of synovitis.
- The tophi are soft and small but later becomes hard and may reach upto 7 cm in diameter.
- The tophi may ulcerate discharging chalky material.
- Common sites of tophi : around olecranon, ankles, tendo-achilles, helix of ear and other joints.

Complication

- Renal damage : by obstruction of renal tubules by urate crystals, urate deposition in the renal parenchyma leading to renal failure. Major cause of death in gout.
- Cardiovascular system : Hypertension and ischemic heart disease.
- Gout is associated with insulin resistance.

Diagnosis

- **Synovial fluid analysis** : demonstration needle-shaped MSU crystals.
- **Serum uric acid** : elevated but normal levels do not rule out gout.
- **Urine uric acid**: excretion of >800 mg/day in the absence of drugs suggests overproduction.

- **Screening for risk factors** : urinalysis; serum creatinine, liver function tests, glucose and lipids; complete blood counts.
- If overproduction is suspected, measurement of erythrocyte hypoxanthine guanine phosphoribosyl transferase (HGPRT) and PRPP levels.
- **Joint x-rays**: demonstrate cystic changes, erosions with sclerotic margins in advanced chronic arthritis.
- If renal stones suspected, abdominal flat plate (stones often radiolucent), possibly IVP.
- Chemical analysis of renal stones.

Differential Diagnosis

- Septic arthritis
- Reactive arthritis,
- Calcium pyrophosphate dihydrate (CPPD) deposition disease,
- Rheumatoid arthritis.

Treatment

- **Acute stage** : symptomatic relief only since attacks are self-limiting and will resolve spontaneously.
- Analgesia
- NSAIDs
- Colchicine
- Intraarticular glucocorticoids
- Systemic glucocorticoids
- Uric acid–lowering agents

University Library Reference-

- Davidson's Principles and Practice of Medicine – Elsevier Publication, 23rd Edition.
- Golwalla Medicine for students, Jaypee Brothers, 25th Edition
- Harrisons Manual of medicine – MC Graw Hill, 19th Edition
- Harrisons Principles of Internal medicine – 19th Edition, McGraw-Hill Education

Online Reference

https://www.mayoclinic.org/diseases-conditions/ankylosing-spondylitis/symptoms-causes/syc-20354808#:~:text=Ankylosing%20spondylitis%20is%20an%20inflammatory,be%20difficult%20to%20breathe%20deeply.

https://my.clevelandclinic.org/health/diseases/16595-ankylosing-spondylitis-as

https://www.webmd.com/ankylosing-spondylitis/what-is-ankylosing-spondylitis

https://www.versusarthritis.org/about-arthritis/conditions/ankylosing-spondylitis/

- **Suggestions to secure good marks to answer in exam-**
  - Define Ankylosing Spondylitis. Write the etiology, pathogenesis, clinical features, of the same.
- **Questions to check understanding level of students-**
  - Enumerate various investigations commonly done for the identification of locomotor diseases.
- **Next Topic-**
  - Reactive Arthtitis its investigations and management.

Academic Day ends with-

National song' Vande Mataram'