Gout: Not Crystl Clear

Madhu Chandra
Advisor in Rheumatology, In Charge, Ortho- rheumatology Clinic, KIMS, Hubli

Email: lorealmadho@yahoo.com

INTRODUCTION

Gout is a common disorder of uric acid metabolism. It has been called as disease of kings. The term GOUT derived from Latin word gutter (a drop). It is important to know the clear distinction between hyerurecemia and out hyperurecemia is risk factor for the development of gout. Important recent biological advances have given us a clear picture of gouty inflammation which is triggered by MSU crystals. Acute gouty arthritis is self-limiting by nature, but patient agony is more. Multiple factor contributes for turning on and turning off the acute gout. Early definitive diagnosis by crystal demonstration in synovial fluid. And definitive treatments by NSAIDS, corticosteroids, colchicines and allopurinol have improved the prognosis of gout.

Gout is a common disorder of uric acid metabolism that can lead to deposition of monosodium urate crystals in soft tissues, joints and kidney. If untreated, joint destruction and renal damage.
Gout is definitively diagnosed by demonstration of urate crystals in aspirated fluid.

Frequency
India- approximately 2% of population has gout.

Age peak age of onset of gout in men is in fourth to sixth decade of life.In women, peak age of onset is in sixty to eighth decade of life, because uric acid level increases after menopause. Men have four times increased chances than women.

Sex-
Male: female ration ranges from 7:1 to 9:1.gout is rare in premanopausal women, because estrogenic hormones have uricosuric actions.

Below 65 years, men: women ratio is 4:1
Above 65 years, men: women ration is 3:1

Associated conditions-gout and hyperuricemia are associated with obesity, hypertension, diabetes, hyperlipidimia and insulin resistance. Patho physiology of gout- uric acid is a weak acid, presents mainly as urate.in extra cellular fluid sodium is more; hence urate mainly presents as monosodium urate crystals. A concentration more than 7mg at 37 degree Celsius, urate crystal formation and crystallises increases and get deposited in and around the joints.

How MSU crystal induce inflammation-
A gout attack is triggered by a release of naked crystals or a precipitation of crystal in a synovium near the joint space. These crystals are engulfed by neutrophils and macrophges.there is release of inflammatory chemical substances like IL, TNF, and lysosomal enzymes, leads to joint destruction

Hyperurecemia is defined as a serum uric acid level more than 7mg/dl, as measured by the automated enzymatic method.

Clinical Presentation gout has four clinical presentations.
1. Asympathomatic urecemia.
2. Acute gouty arthritis.
3. Intercritical gout.
4. Chronic tophous gout.

Classification of Gout:
1. Primary gout-inborn error of uric acid metabolism.
2. Secondary gout-the cases that develop in the course of another disease.

Asymptomatic Hyperurecemia-here serum uric acid level is higher, but patient do not have any arthritis symptoms, renal stones and tophus formation.

Acute gouty arthritis:
1. 1.90% cases first attack usually a 1 st metatarsophalengel joint of great toe.
2. Attack start with severe onset on pain in the great toe usually night time.
3. Severe pain, patient cannot tolerate even touch of bed sheet.
4. Sometimes fever and malaise.
5. Joints are red, hot, swollen and very tender.

If untreated, resolve in 1-2 weeks.

Gout can present as polyarthrits in 10% of cases.
Commonly seen in elderly women, renal impairment and those taking thiazide diuretics.

Definitive diagnosis by:
1. Aspiration of joint and demonstration of MSU crystals.
2. Clinical triad- Môno arthritis.

Hyperurecemia
Response to colchicines.

Chronic tophus gout:
Classical features are
1. Attacks are more polyarticular.
2. Less inflammation.
3. Upper limb joints are involved.
4. Attacks occur more frequently.
5. Formation of tophi.

Tophi- it is a collection of urate crystals.
Common site of occurrence-helix of ear, fingers, toes, olecranon burse.

Lab studies:
1. Synovial fluid aspiration-even 1ml is enough to demonstrate urate crystals.
2. Classical appearance of urate crystals-needle shaped, negative birefringent, and intercellular in nature.

Serum uric acid level:
Not a reliable.
5-8% normal population have elevated uric acid.
5-20% 0f hyperurecemic develop gout.corelate with clinical symptoms.

24 hours –urine uric acid:
Normal: 600-800mg/24hrs.
More than 800mg of uric acid is called overproducers of uric acid.
Less than 600mg of uric acid are called underexcreators.
More than 1100mg/24, patients require renal monitoring. Because of renal stones and renal nephropathy.

Other test: Blood sugar
Lipid profile
Liver function
Kidney function
Treatment: Acute gouty arthritis

Cap.indomethacin 50mg every 6-8 hourly.
Tab. naproxen 500mg every 12houry.
Tab.etorocoxib, 120mg.

Tab.colchicine 0.5mg every hourly until pain subsides.
Maximum dosage of colchicines is 6mg/24hours.

Role of Corticosteroids in Acute Gout:
1. Very effective in 12-24 hours.
2. Combine with NSAIDS faster the recovery.
3. Chances of rebound flares.

Dosage:
Inj.ACTH 40IU im
Inj.trimcinolone 60mg
Inj.methyl prednisolone 160mg

Tab.predinisolone-40-60mg, for 1-3 days, then tapers the dosage in 2 weeks.

Treatment of symptomatic hyperurecemia:

Life long therapy with antihyperurecemic drugs is needed, in following conditions.
1. More than 3 acute attacks.
2. renal stones
3. Tophous gout.
4. X-ray changes.

Allopurinol:
It is a drug of choice in overproduces and underexcreators.

Dosage-300mg morning dose.
Max-800mg.

Indications:
1. Uric acid excretion more than 11oomg/24hrs.
2. Urate nephropathy.
3. Primary gout.
4. Tumor lyses syndrome patients.

Adverse effects-nause, headache, dyspepsia, diarrheoa, and allopurinol hypersensitive reactions.

Colchicines Prophylaxis:
Is needed in all patient whose is taking antihyperurecemic drugs, to prevent acute flares.
Dosage-0.5mg BD, for 6 months.

Uricosuric drugs-theses promote urate excretion by kidney.
1. Probenecid.
2. Sulphinpyrazone.

Criteria for uricosuric drugs:
1. Less than 60 year's patients.
2. Normal renal function.
3. Underexcretors-less than 600mg/24, uric acid excretion.
4. No renal calculi.

Gout and Diet:
Avoid: Alcohol, red meat, sea food.
Peas, palak.and cauliflower.

Coffee, dairy products, vitamin c associated with decreased risk of gout.

However, many dietary modifications are under study.

Newer Drugs in Gout:
1. Recombinant Uri case
2. Oxipurinol
3. Benzbromarone-uricosuric drug
4. Febuxostat-xanthine oxidase
5. Losartan
6. Fenofibrate

CONCLUSION
- Hyperurecemia and gout are not synonymous.
- Hyperurecemia is strongly associated with insulin resistance and metabolic syndrome.
- Definitive diagnosis by crystal demonstration in synovial fluid.
- Don't treat hyperurecemia in all.
- Allopurinol is very effective in overproducers and under producers.

- Life long therapy with allopurinol.
- Cautious use colchicines.
- Dietary modification is still puzzle.

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