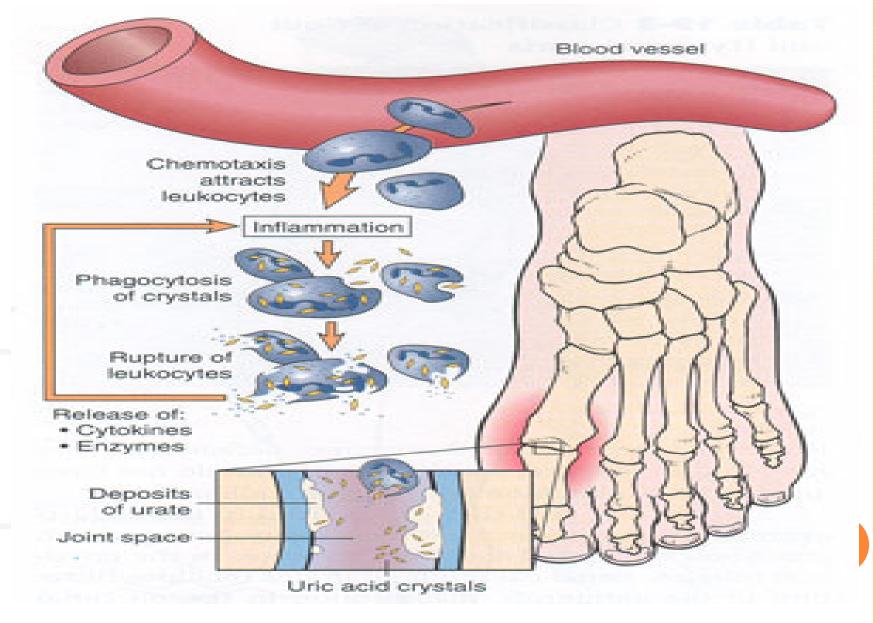
GOUTY ARTHRITIS

INTRODUCTION

- Uric acid is a waste of purine metabolism
- Excreated by kidney
- Gout occur as a result of
 - overproduction of uric acid
 - decreace excreation of uric acid.
- Gout is an attack of uric acid deposits in joints
- Usually found in distal joints like joints of feet and legs

WHAT IS GOUTY ARTHRITIS



- It is due to accumulation of Uric acid crystals in synovial fluid with a surrounding area of inflammation leading to acute arthiritis.
- At 30 °C, the solubility of lowered. Therefore, uric acid isdeposited in cooler aeas of body. This is called a *tophus* and is often described as an arthritic "great toe".
- Increased excretion of uric acid may cause deposition of uric acid crystals in the urinary tract leading to calculi or stone formation with damage to kidney

Cause

PRIMARY HYPERURICEMIA

- 5-phosphoribosyl amido transferase
- Abnormal PRPP synthase
- Deficiency of enzymes of salvage pathway
- Glucose-6-phosphatase deficiency
- Glutathione reductase variant

SECONDARY HYPERURICAEMIA

Increased production of uric acid

- Rapidly growing tumours
 - leukaemia, lymphomas, polycythemias
- Treatment of large malignant tumour
 - Post Radiation & chemotherapy
- Increased damage to tissue as in trauma
- Raised rate of catabolism in starvation

Reduced excretion

- Renal failure
- Lactic acidosis
- Ketoacidosis due to interference with tubular secretion
- Treatment with thiazide diuretics which inhibit tubular secretion of uric acid

CLINICAL FEATURES

- Precipitated by high purine and high intake of alcohol.
- Alcohol leads to accumulation of lactic acid.
- Affects the first metatarsophalangeal joints.
- The joints are extremely painful.
- synovial fluid will bifringent urate crystals.
- In chronic cases, uric acid may get deposited around the joints causing swelling (**tophi**)
- Swelling of Joint (Tophi)
 - Stiffness
 - Warm and red
 - Possible fever
- In chronic cases ,deposition of urate crystal in renal medulla leads to urolithiasis and renal damage.

Associate disease

Renal lithiasis
Uric acid nephropathy
Urate nephropathy



Inflamed tophaceous gout Three inflamed tophi over the proximal interphalangeal joints in a patient with chronic tophaceous gout. Several of the lesions ruptured spontaneously over the next three days, exuding a pasty material composed of urate crystals and inflammatory cells but no organisms. The inflammation largely subsided over one week after the administration of a nonsteroidal antiinflammatory drug. Courtesy of Michael A Becker, MD.

http://www.uptodate.com



THE FOUR STAGES OF GOUT

Asymptomatic
Acute
Intercritical
Chronic

ASYMPTOMATIC

- A- meaning without indicates that there are no symptoms associated
- Patient will be unaware of what is happening
- Gout can only be determined with the help of a physician

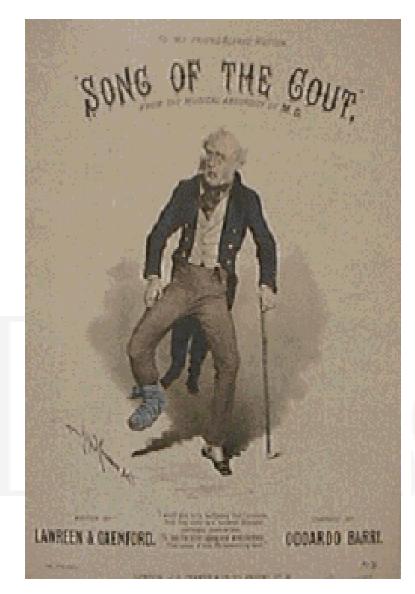
ACUTE

- Severe and sudden onset
- Involve one or a few joints
- Frequently starts nocturnally
- Joint is warm, red and tender

INTERCRITICAL

• More concentration of uric acid crystals

CHRONIC



- Continuous or persistent over a long period of time
- More bone and joint damage
- Treatment required
- Not resolved easily

DIAGNOSIS

Based on history and physical

Confirmed by arthrocentesis

Urate crystals: needle-shaped, either free floating or within neutrophils & macrophages.

Uric acid level non specific.

Normal value : 4 - 6 mg%

30% may show normal level

Urine collection:

• <800 mg underexcertor (<600 purine-free diet)</p>



• X-ray

Acute
 Soft tissue swelling

Chronic

- chronic tophaceous gouty arthritis
- Bony erosions are noted throughout the carpal bones
- Sclerosis and joint-space narrowing in the first metatarsophalangeal joint, as well as in the fourth interphalangeal joint .

DIFFERENTIAL DIAGNOSIS

- Septic arthritis
 - Tuberculosis, Syphilis, Gonococcal
- Osteoarthritis
- Traumatic arthritis
- Rheumatoid arthritis
- Acute Rheumatic fever
- Psoriatic arthritis
- Due to Haemoglobinopathies
 - Sickle cell crisis
- Blood disorder
 - Leukemia
- Connective tissue disorder
 - SLE

TREATMENT

- 1. Reduce dietary purine intake
- 2. Restriction of alcohol
- 3. Increase water intake

4. Colchicine

- Reduces pain, swelling, and inflammation
- 5. Other NSAIDS & Corticosteroid
 - to reduce inflammation
- 6. Probenecid
 - Reduces the reabsorption of uric acid from kidney tubules.

7. Allopurinol

- 1. Reduce urate production.
- 2. Allopurinol is competitive inhibitor of Xanthine oxidase thereby decreases the formation of uric acid.
- 3. Xanthine oxidase converts allopurinol to alloxanthine which is more effective inhibitor of Xanthine oxidase

"DISEASE OF KINGS"

Rich foods have a higher concentration of Purine . This could cause major problems for a person afflicted with gout.

ORGAN MEATS
SEAFOOD
LENTILS
PEAS
YEAST
BEER

