Hyperuricemia

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It is most commonly defined by serum uric acid concentrations greater than 7.0 mg/dL in men or greater than 6.0 mg/dL in women.

Hyperuricemia can occur by two main mechanisms: Increased production (overproducers) , decreased excretion (underexcretors) or a combination of both

Consequences of hyperuricaemia

The hyperuricemia can lead to the deposition of mono sodium urate crystals in the joints, and an inflammatory response to the crystals, causing first acute and then progressing to chronic gouty arthritis.

Nodular masses of monosodium urate crystals (tophi) may be deposited in the soft tissues, resulting in chronic tophaceous gout

●Urate crystals may also appear as kidney stones and lead to painful obstruction of the urinary tract.

Gout is classified as primary or secondary:

Primary gout

It is associated with "essential "hyperuricemia,in more than 99% of cases, the cause is uncertain, but the condition is probably due to a combination of :

(1) Metabolic overproduction of purines (25% of patients have increased amidophosphoribosyl transferase activity).

(2) Decreased renal excretion (80% of patients show decreased renal secretion of uric acid).

(3) Increased dietary intake.

Secondary gout

- In rapidly growing malignant tissue, e.g. leukemia, lymphomas.
- In psoriasis, when turnover of skin cells is increased.
- Tumor lysis syndrome.

Reduced excretion of urate
- Chronic renal failure
- Prolong metabolic acidosis
- Drugs,e.g. thiazide diuretics , low dose salicylate

Diagnosis

The definitive diagnosis of gout requires aspiration and examination of synovial fluid from an affected joint (or material from a tophus) using polarized light microscopy to confirm the presence of needle-shaped monosodium urate crystals.

Treatment

- Reducing dietary purine intake, e.g red meats.
- An acute attack of gout can be treated with a non- steroidal anti-inflammatory drug (**NSAID**) or sometimes steroids.
- **Colchicine**, which has an anti-inflammatory effect and inhibits neutrophil activation, can be used in acute gouty arthritis .
- Reducing urate production by using drugs that inhibit xanthine oxidase activity, such as **allopurinol** which is structurally similar to hypoxanthine, so acting as a competitive inhibitor of the enzyme.
- Increasing the renal excretion of urate by using drugs, such as **probenecid.**