GOUT

Gout is a syndrome caused by an inflammatory response to the formation of monosodium urate monohydrate crystals that develop secondary to elevated serum uric acid levels (hyperuricemia). Acute and chronic forms are recognized. Hyperuricemia may be due to environmental and/or genetic factors.

Clinical features

Gout most commonly affects middle-aged males, but it also affects women with the same risk factors. It is an acute and usually relapsing self-limiting arthritis. A chronic form associated with collections of crystals - tophus formation occurs, and bone and joint destruction can occur. It especially involves the first metatarsophalangeal joints (the big toe), but it can also affect the heels, ankles, knees, fingers, and hands. Gout has been called the 'disease of kings' or of the 'wealthy'. The term 'gout', is derived from Latin 'gutter' (a drop) which reflects the notion that gout resulted from a local instillation of malevolent or bad humor. These concepts are well illustrated in paintings of persons afflicted with gout. It is also seen as a "disease of plenty" - which is an incorrect generalization. It is commonly associated with:

- Obesity - being too fat (Check your BMI)
- Heavy alcohol intake
- High blood pressure
- Kidney impairment
- Water medication (diuretic) use.

Hyperuricemia has been defined as a serum or plasma urate concentration greater than 7.0mg/dl (0.42mmol/l) in males, and 6.0mg/dl (0.36 mmol/l) in females.

It is important to recognize the clear distinction between hyperuricemia and gout. Hyperuricemia is clearly a risk factor for the development of gout, the risk increasing with a higher urate concentration. However, many years of hyperuricemia have usually elapsed before the development of an acute gouty arthritis attack, and in some hyperuricemic people gout may never develop.

Acute gout is characterized by rapid onset of pain and swelling with associated redness of the affected joint. The pain may be excruciating – even the lightest touch may be unbearable!

The lower limbs are involved more often than upper limbs. In over half of the initial attacks, the onset is in the first metatarsophalangeal (MTP) joint, the big toe joint. Over time this joint is affected in some 90% of patients with gout, although almost any joint can be affected. Mild attacks may resolve within one to two days. More severe attacks exhibit rapidly increasing pain reaching its peak usually within a few hours, remaining at this level for some one to three days, then slowly remitting, such that the attack has often subsided within seven to ten days. It may take several weeks before very severe attacks settle completely.

Several factors have been recognized as precipitants of acute attacks of gout. These include: Acute illness, Trauma, Surgery, Alcohol (especially beer and wines), and Drugs that either increase or decrease the plasma urate concentration, especially diuretics.

Patients who develop chronic gout usually are those whose hyperuricemia is not controlled by dietary changes or medication. Tophi appearing within the first two years of gout are extremely rare and patients have usually suffered from gout for at least 10 years before tophi develop. The tophi are collections of crystals that form deposits in soft tissue, joint, bone and tendons. They cause erosion and destruction of the bone and joint, and cause damage that may lead to crippling. Such crippling is I feel a tragedy, as this situation is completely controllable, and preventable - to be crippled from gout in today's modern world of medicine is "completely unnecessary, and tragic".

Therapy for Gout is directed towards:

- Management of the pain of the acute attack.
- Prevention of further attacks and thus the complications of chronic gout.

It is important when considering therapy for gout to distinguish between therapy for reducing inflammation and that for managing hyperuricemia. The decision to introduce drugs to normalize hyperuricemia depends in part on 1. The number of previous attacks of acute gout, 2. The degree of hyperuricemia, 3. The presence or abscence of reversible factors, and 4. The presence of tophi.

Therapy is first aimed at completely controlling the acute attack, and then if indicated, controlling the uric acid level at the source or by increasing it's removal from the body. Introduction of these second line drugs must never be during the actual acute attack, as they will potentially aggravate the acute episode and prolong it. Urate lowering drugs will not normally be used after just a single attack of gout, but should be considered after the second or third attacks. Furthermore, they should virtually always be used for tophaceous gout. The patient needs to understand that **the decision to commence anti-**

hyperuricemic therapy usually implies life-long treatment and they need to be committed to this plan.

Both increases and decreases in plasma urate concentrations may precipitate or prolong an attack of gout. Therefore, therapy aimed at reducing urate concentrations should be delayed until after the complete resolution of all signs of inflammation. It must be noted that one of the commonest cause of difficulty in controlling an attack, is the simultaneous administration or withdrawal of drugs that alter the plasma urate concentration.

Acute Gout

Colchicine and Nonsteroidal anti-inflammatory drugs (NSAIDs) are effective in the treatment of acute gout and are much superior to aspirin. In addition, NSAIDs are superior to colchicine in terms of speed of onset of action. Thus, despite having been used for centuries, colchicine is usually reserved for patients in whom NSAIDs are contraindicated.

Intra-articular administration of Corticosteroids is a particularly effective means of terminating an attack of gout. Resolution is typically complete within 12–24 hours. This form of treatment is of particular value in some patients with renal impairment and other conditions where the use of full doses of other drugs may be relatively contraindicated.

Recurrent Gout

Despite the use of effective prophylaxis, only correction of the hyperuricemia can alter the underlying tendency to gout. Drugs to correct hyperuricemia act either by promoting the kidney's excretion of urate (uricosuric agents) or by decreasing urate production - by inhibiting xanthine oxidase (allopurinol)
It cannot be stressed too much that antihyperuricemic drugs should not be commenced until an attack of gout has settled completely. Frequently I use prophylactic doses of colchicine or NSAID's to minimize the risk of inducing an attack of acute gout, while starting these second line or preventative medications.

Tophaceous Gout

The principle of treatment of tophi is to lower the plasma urate concentration to such a degree, as to allow urate to be resorbed from the surface of the tophi. The uric acid is slowly excreted by the kidney, and production is lower than excretion, resulting in net loss of uric acid. During this phase, it is useful to add citrosoda 10ml twice a day to the medication regimen to help prevent deposition of crystal in the kidney, and to help prevent kidney stone development.

The long term goal is to maintain the urate concentration within the middle of the "normal" serum level range. This requires the long-term use of urate-lowering drugs over many years. Colchicine and most NSAIDs, while controlling acute attacks, will not prevent the formation of tophi and may, by preventing the inflammatory response, actually increase the development of tophi unless hyperuricemia is controlled at the same time.

General Guidelines for Gout Prevention

Take your prescribed medication regularly. Be careful not to injure the joint as the resulting inflammation can easily trigger an acute attack! Avoid purine-rich foods. The higher the purine content, the more uric acid will be produced in the body. If you are overweight, you must work to achieve your ideal weight through slow, controlled weight loss (maximum 500 g per week). Rapid or sudden fasting is not recommended as this can raise uric acid levels and aggravate gout. Avoid heavy, rich meals with high fat and protein content. **Alcohol should be avoided, particularly wine since it interferes with uric acid excretion.**

Diet Guidelines

Forbidden - Very high purine content
Herring, meat extracts, mussels, sardines, yeast (brewer's and baker's).
Alcohol - Alcohol contains no purine but interferes with uric acid excretion.

Avoid - High purine content - eat not more than one item once a week.
Anchovies, bacon, chicken soup, Beef, lamb leg or chops, pheasant, salmon, sausage, trout, turkey, veal, venison, lobster, and crab.

Be careful - Moderate purine content - eat not more than one item 4 times a week.
Asparagus, bass, bouillon, brains, cauliflower, chicken, duck, halibut, ham, kidney beans, lentils, lima beans, liverwurst, mushrooms, oysters, peas, pork, rabbit, roe, shad, spinach, tongue, tripe, tuna, wholegrain cereals and bread.

Acceptable - Low or no purine content - eat as often as desired.
Beverages -tea, coffee, cocoa, chocolate, carbonated soft drinks, fruit juices, sugar, sweets.
Vegetables: (except those under BE CAREFUL)
Vegetable and cream soups (no meat stock)
Butter, fats of all kinds (in moderation)
Bread (except wholegrain)
Cereals (except wholegrain)
Cheese : all kinds (in moderation)
Eggs
Fruit

Milk - buttermilk, condensed, malted
Nuts - all kinds, peanut butter

Fluids - Drink plenty of water (2-3 liters per day) to help flush uric acid through the kidneys. Please note - if you have cardiac or kidney disease, your doctor may actually require fluid restriction - and you should consult him regarding fluid intake allowance as this may need to be carefully balanced!