Editorial

Gout or Gouty Arthritis

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Gout was known even in ancient times. The Greek physician Hippocrates referred to it some 2400 years ago. It was believed in the middle ages, that this disease was caused by “drop” of (Latin gulla “drop”) poisonous body fluid which enters the joints. The classical description of gout was written in 1660 by the English physician Thomas Sydenham, who himself suffered from it. Most forms of gout are inherited.

Gout is a type of arthritic condition which is increasing in prevalence. It is an acute gouty arthritis, which appears suddenly without warning, especially in men between the ages of 40 and 60, and in women after the age of 60. In U.S. alone about 3 to 6 million people have been reported to be affected by gout. In the third world it may even be more prevalent. Usually a single joint, especially the big toe, is the main affected part, though it may rarely affect any other joint. Many patients complain about pain of lower extremities, especially the big toe but it may later involve joints in other finger also.

Causes and Risk Factor:
Though its causes may be manifold but the prime factor has been identified to be the excess of uric acid and its salts sodium urate, which is an end product of the chemical reaction in the body and results as a breakdown of purines (nucleoprotein). Purins are an important component of the amino acid found in the food protein.

Under normal condition, uric acid crystals, may be formed in joints spaces and appear when its level becomes higher than normal, in the blood. This condition is referred to as hyperuricemia. However it may even occur when the uric acid level is normal. If the uric acid crystals are formed in the joints, they cause inflammation in the body and these irritate the surrounding tissues. The natural defense mechanism results in the symptoms of swelling, redness and pain. Though these symptoms may subside with time, the inflammation may damage the joint and may persist for a long time. It is therefore necessary to address the symptoms without delay and thus avoid its recurrences.

Mode of the formation of uric acid

The main reason for the accumulation of high uric acid level in the joints is either increased production of uric acid or slow process of removal of uric acid from the body. It is not necessary that every person with hyperuricemia will develop gout but it increases the chances of attack. The risk factor is greater in men of advanced age as compared to women.

There are two major ways in which excess of uric acid occurs. In one form of gout, the kidneys are unable to excrete sufficient amount of the normal production of uric acid. In the second form, the body produces more uric acid than normal kidney can remove. Some individuals with the second form have an inherited defect and lack certain key enzymes such as hypoxanthine-guanine phosphoribosyltransferase, which act to reduce the amount of uric acid produced by the body. Excess uric acid may also occur as secondary consequences of many conditions that disturb body chemistry such as pernicious anemia, starvation or the taking of certain drug. Gout that is caused in this manner is called as secondary gout.

Symptoms:

Gout has four major aspects: gouty arthritis, urate gouty kidney, uric acid gouty kidney and gouty-tophus. Sodium urate crystals may form deposits, called tophi, almost anywhere in the body, producing the condition known as gouty tophus. Tophi in or near joints will eventually cause foreign body, or immune reaction, the crystals stimulating the body’s white blood cells and inducing an acute inflammation. The resulting condition is known as gouty arthritis.

The typical gouty arthritis involves only one joint, usually the big toe, ankle or knee. Usually it appears early in the morning, with a sudden severe pain in the affected joint, which swells. The nearby skin turns purple and shining. The pain tends to subside by day
and return again at night, on and off, for almost a week. This process is repeated, other joints may become affected badly to some destruction of bone.

Urate gouty kidney occurs when an excess of urate in the blood causes stones to form in the kidney. Uric acid gout of kidney is caused by kidney stones largely of crystals of uric acid rather than sodium urate. Kidney stones formed in gout may cause kidney failure and death.

The effected joint become red, swollen hot and tender quickly, even on slight touch. The pain may become unbearable and may last few hours and weeks. It may start as flare and continue for long until it subsides or may start once again as a flare. High intake of red meat, organ meat, shellfish, dried bean and heavy use of alcohol or beer, may increase gout condition.

Diet and Lifestyle/Treatment:
Regulation of diet with a view to reduce uric acid, such as diet low in purine, may minimize the risk of a flare. Food especially red meat, shellfish should be avoided. Low fat dairy product, lack of alcohol, reduction of weight and exercise also help in gout condition. Control of inflammation and management by pain killer, non steroidal anti-inflammatory drugs (NSAID), colchicines, corticosteroid, naproxen, fenoprofen, ibuprofen, sulindac, piroxicam, ketoprofen, celecoxib, may be effective. Patients suffering from heart failures, liver disease, ulcers, and improper kidney cannot take NSAID. High doses of medication cause side effect such as nausea, vomiting, diarrhoea. Corticosteroid and colchicines are important classes of medication used against gout, flare up. Application of ice-bag on the effective joint helps in reducing the pain.

Quick change in uric acid level in the blood is likely to cause gout. This includes infection and use of abrupt starting or stopping of medicines known as Xanthine Oxide inhibitors, which are used to lower uric acid level by reducing the production of uric acid. (Aspirin, cyclosporin, ethambutol, levodopa, niacin, pyrazinamide, warfarin, diuretics, tacrolimus, over use of laxatives among other).

Two main classes of medication help lower uric acid level. First type of medicines is called Xanthine Oxidase inhibitor and they block the breakdown of purine into uric acid in the liver. The second class is called uricosurics, which increases the amount of elimination from the body through kidney and urine. They may however cause attack due to an abrupt change in uric acid level. This may seem to be a setback but the medication should not be discontinued. Treatment should enhance for 3-6 months.