USANA Technical Bulletin

Gout

Description

- Gout, also known as gouty arthritis, results from monosodium urate deposits that cause red, swollen, and acutely painful joints. Gout may affect any joint but mostly affects those in the feet, especially the great toe, ankle and midfoot.[1]

Causes

- Painful gouty arthritis is caused by uric acid crystal deposits in joint tissue. This happens when uric acid levels in the body are abnormally high.
- The underlying cause of primary gout is not clearly understood but in many patients the disease results from decreased renal excretion of uric acid. In a few patients gout is linked to a genetic defect in purine metabolism that causes an overproduction of uric acid.[1]
- Gout and hyperuricemia can be promoted by obesity, acute weight gain, alcohol intake, high blood pressure, abnormal kidney function, and certain drugs.

Types

- Primary gout typically occurs in men over thirty and postmenopausal women who take diuretics. Secondary gout develops during the course of another illness (i.e. obesity, diabetes mellitus, hypertension, renal disease and others) or following treatment with drugs (i.e. hydrochlorothianzide).[1]

At Risk

- The tendency to develop gout and elevated blood uric acid level (hyperuricemia) is often inherited.

Prevention and Management

- Gouty arthritis attacks can be precipitated by dehydration, injury, fever, heavy eating, heavy drinking of alcohol, and recent surgery. Because obesity may be a factor in secondary gout, weight loss is often suggested.
- Almost all people with gout have too much uric acid in their blood, a condition called hyperuricemia. However, there are many people who have hyperuricemia but not gout.
- It is important to drink plenty of fluids.

- Adjunct therapy attempts to reduce the consumption of alcohol and purine-rich foods such as anchovies, liver, sardines, kidneys, sweet-breads, lentils and other beans.
- An experimental study suggested that vitamin C lowers uric acid levels in serum by increasing renal excretion.[2]
- Zinc levels may be depressed during disease activity.[3]

Sources of Additional Information

- http://www.medicinenet.com/MAINMENU/encyclop/ARTICLE/Art_G/gout.htm
- http://www.ohsu.edu/cliniweb/C5/C5.799.414.html
- http://weber.u.washington.edu/~dboone/key/subjects/arthritis/gzzzzzzz1_2.html

Abstracts

Emmerson BT. The management of gout. N Engl J Med 1996 Feb 15;334(7):445-51.

We now have sufficient knowledge to be able to identify the factors contributing to hyperuricemia in most patients with gout. Some of these factors, such as obesity, a high-purine diet, regular alcohol consumption, and diuretic therapy, may be correctable. In patients with persistent hyperuricemia, regular medication should lower the serum urate concentration to an optimal level. The continuing challenge is to educate patients about correctable factors and the importance of regular medication and ensure their compliance so that attacks of gout do not recur.

References

[1] Diseases. Springhouse (PA):Springhouse Publishing; 1993. p 759.
[2] Stein HB et al. Ascorbic acid-induced uricosuria: A consequence of megavitamin therapy. Ann Intern Med 1976;84(4):385-8.
[3] Mataroan Perez L et al. [Zinc in arthrosis and microcrystalline arthritis]. Rev Clin Esp 1991;189(2):60-2.