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Hyperuricemia and Gout - Old Disease with New Challenges

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Abstract: ***Gout is one of the oldest diseases of mankind. In recent time the incidence of gout and hyperuricemia has been increased due to faulty life style and bad food habits. Gout is a disorder of purine metabolism. Hyperuricemia alone is one of the most common risk factor for development of gout. Clinically gout is characterized by pain and swelling of joints, especially 1st metatarsal joint. Conventional system uses analgesics and urate lowering drugs, but they usually produce adverse drug reactions. This article is about basic knowledge of hyperuricemia and gout.***

Keywords: Gout, Hyperuricemia, Serum uric acid

1. Introduction

Gout is one of the oldest diseases of mankind. Gout is a common disorder of purine metabolism [1]. It is a disease caused by deposition of monosodium urate (MSU) crystals in tissue such as cartilage, synovial membrane, bone and tissue [2]. This deposition occurs when serum uric acid levels exceed the saturation point of MSU crystal formation, a condition called Hyperuricemia [3].

Hyperuricemia is a condition characterized by abnormally elevated levels of serum urate (sUA), while gout is an inflammatory response to MSU crystals formed secondary to hyperuricemi [4]-[5].

Hyperuricemia has been defined as a serum or plasma urate concentration greater than 7.0 mg/dl in males and 6.0mg/dl in females [5].

2. Epidemiology

2.1 Prevalence and Incidence

The prevalence of gout and hyperuricemia has increased considerably over the last 25 years. This increase may be due to an aging population, changes in lifestyle and diet (e.g. higher intake of fructose-containing soft drinks and alcoholic beverages, such as beer), and an increasing prevalence of co morbidities associated with hyperuricemia and gout, such as obesity, hypertension, widespread use of thiazide diuretics, and chronic kidney disease (CKD) [6].

The prevalence of hyperuricemia (HU) varies with age and gender. In the previous literature, SUA levels were reported to increase with advancing age

The general prevalence of gout is 1–4% of the general population. In western countries, it occurs in 3–6% in men and 1–2% in women. In some countries, prevalence may increase up to 10%. Prevalence rises up to 10% in men and 6% in women more than 80 years old. Annual incidence of gout is 2.68 per 1000 persons.

2.1. Classification/ causes of hyperuricemia

Hyperuricemia can be classified into two types [5]:

Primary or idiopathic hyperuricemia: The hyperuricemia is considered primary when it exists in the absence of coexisting diseases or drugs that alter uric acid production or excretion. It usually last lifelong.

Secondary hyperuricemia: This refers to excessive urate production (overproducer) or diminished renal clearance (under excretion) occurring as a consequence of another disease, drug, dietary product, or toxin

3. Risk factors for development of gout

We can classify risk factors of gout into two broad categories- non modifiable risk factors and modifiable risk factors.

3.1 Non- modifiable risk factors

Age- The incidence of gout increases as the age advances. Signs and symptoms are more prominent in the old individuals. In Indian population maximum incidence of gout is seen after the age of 30 [7].

Sex- Gout is often stated as male disease. Incidence of gout is more in case of males as compare to female, before the age of sixty five. It may be due to uricosuric effect of estrogen decreased after menopause [8].

3.2 Modifiable risk factors

Hyperuricemia- It is one of the most important and modifiable risk factors for the development of gout.

Dietary factors
Dietary factors also play an important role in development of gout. Dietary consumption of meat and sea food has been associated with increased risk of development of gout.
In some studies it is seen that dairy products have some protective function in gout. Alcohol consumption especially consumption of beer is associated with trigger attack of gout.

Diuretic use

The uses of diuretics for certain medical conditions like hypertension, renal diseases and cardiac failure are associated with an increase risk of Hyperuricemia and gout. The exact mechanism is not clear but diuretic induced hyperuricemia occurs via inhibition of renal urate excretion.

4. Clinical Features

Gout can occur into four phases-

4.1 Asymptomatic Hyperuricemia

In some individuals hyperuricemia may be an accidental finding and they may never lead to gout. It is observed that in some cases, even during acute attack of gout, serum uric acid levels remains in normal limit. It may be due to release of ACTH (Adrenocorticotrophic Hormone) in response of stress and due to its uricosuric effects. But, asymptomatic hyperuricemia may be lead to hypertension and some other vascular diseases [9].

4.2 Acute gout or acute gouty arthritis

In most of the cases acute gout is presented as single distal joint involvement. The classical feature of acute gout attack is involvement of the 1st metatarsophalangeal joint (MTP), which is known as "Podegra". Gout is one of the most common causes of monoarthritis. A typical attack of gout can be easily remembered with Sydenham's description of acute gouty arthritis. The attack is acute, it starts in the night, the joints and swollen tissue are swollen, hot, red, shiny and extremely painful [5]-[10].

There may be chill and mild fever. Pain of acute attack is usually unbearable. Patient cannot walk properly even cannot wear a socks or allow someone to touch the joint. Underlying skin is so much sensitive that even patient cannot tolerate covering with bed sheet. Severe pain, often describe 'worst pain ever'.

There are certain factors which can precipitate acute attack. Some of them are infection, trauma, surgery, dieting, and even hypouricemic therapy. The exact mechanism is not clear but it may be due to lactic acidosis in infection, ketoacidosis in fasting, increased tissue breakdown in trauma and surgery. Due to sudden use of urate lowering agents there may be release of more crystals from the edges of tophi. This attack is usually self-limiting and completely resolved within 10-15 days. Acute attack may also manifest as bursitis, tenosynovitis or cellulitis [5].

4.3 Intercrtical periods

In between acute attack there may be some periods of remission in which patient remain asymptomatic. The second attack may occur next year or after many years. As the pathology advances, the frequency of attacks and sites of joints involvement increases with time. In later stage if untreated, there may be joint erosion or chronic pain. Everything depends upon the level of serum uric acid [5].

4.4 Chronic tophaceous gout

Development of chronic tophaceous gout is usually a late feature. It takes approximately 10 years for the development of tophaceous gout, but in patients of renal failure they may develop within one year. Large crystals of MSUM may produce irregular firm nodules which are known as 'tophi'. The usual site of appearance of tophi is extensor surface of fingers, hands, forearm, elbows, Achilles tendons and sometimes the helix of year.

5. Investigations and diagnosis

Gout is one of the most common causes of inflammatory arthritis, yet it is easily misdiagnosed. European league Against Rheumatism (EULAR), in 2006, produced its first evidence based recommendation for diagnosis of gout. The gold standard for the diagnosis of gout is presence of MSU crystal by polarized light microcopy of the synovial fluid or tophaceous material. These crystals are needle-shaped and generally 5µm - 20 µm. In acute attack the synovial fluid may appear as yellow, cloudy and non-viscous. There may be high number of white blood cells, predominantly neutrophils.

6. Management

Gout being the most common inflammatory arthritis is poorly managed. Several organizations have published guidelines for the diagnosis and management of gouty arthritis, including EULAR, ACR and British society for Rheumatology. The overall management of gout has three phases- controlling acute flares, serum uric acid level <6mg/dl and prevent occurrence of gout attack [11]-[14].

Hyperuricemia have a definite role in development of gout and the principle management of gout is reduction in serum urate levels to less than <6mg/dl, which is below the saturation point of MSU (6.8 mg/dL), to inhibit formation of new crystals and to promote dissolution of existing crystals. Further keeping sUA< 6 mg/dL can also helps in reduction of inflammation and tophus formation.

A number of studies found that patients experienced fewer flares and better therapeutic outcomes when their sUA levels remained at <6 mg/dL.

In a study conducted by Miki Kakutani- Hatayama it was observed that management of certain factors, which increase the risk of development of hyperuricemia and gout, can maintain the serum uric acid level in normal limit. Common factors are obesity, excessive alcohol intake, food rich in purine (red meat, pulses), dehydration, consumption of soft drinks etc [15].

To overcome obesity, weight reduction can be recommended. There are certain foods, which can be consumed in large quantities like skimmed milk, yogurt low in fat, soybeans etc. Food items which contain purine in high quantities should be avoided like red meat, high fructose corn syrup, kidney, sea food like shellfish etc. There are many prospective studies on dietary recommendations. Subjects who consume more seafood, meat and more purine

rich food have greater chance to develop hyperuricemia and gout [16].

Alcoholic drinks especially beer should be avoided. There should be complete abstinence from alcoholic drinks, minimum three days in a week [17].

Other important life style changes include maintaining hydration levels with water intake more than 2 litres /day. In an another study it was observed that vitamin c supplementation has a significant role in reduction of serum uric acid level as compared to placebo[18].

6.1 Auxiliary measures

1) Topical Ice Application in additions to pharmacologic therapy is an important pain relieving measure.
2) Hot and cold compressions- Alternating hot compress for three minute with a cold one for thirty seconds provide pain relief and increase circulation.
3) Massaging of foot gently by pressing the area between bottom of big toe and ball of foot is another effective home remedy.

7. Discussion

Gout and hyperuricemia, although are not life threatening but can cause considerable morbidity and affects quality of life of an individual. Also hyperuricemia alone is most important risk factor for other clinical conditions like caradio-vascualar, renal and other vascular diseases. Early diagnosis and management alone can prevent further progression of disease.

Reduction and maintenance of serum uric acid levels to 6.0 mg/dL, a range below the limit of solubility of urate in serum, is usually the treatment goal in the management of hyperuricemia. Urate-lowering pharmacotherapy is a keystone in the management of hyperuricemia. For prevention of gout attacks, diet less in protein can play a considerable role.

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