Journal of Medical and Pharmaceutical Innovation
ISSN : 2347 - 8136

Review Article

Hyperuricaemia and It's Homeopathic Management

Dr. Nowmani Shaikh* and Dr. Asmita Vinod Kumar Pandey
Guru Mishri Homoeopathic Medical College and PG Institute, Shelgaon, Jalna, India (MS)

ABSTRACT
Gout is a inflammatory response due to crystal deposition caused by formation of monosodium urate crystals in joints and other tissues. It is a common inflammatory arthritis that has increased in prevalence in recent decades. Gout normally results from the interaction of genetic, constitutional and environmental risk factors. It is more common in men and strongly age related. A major determinant is the degree of elevation of uric acid levels above the saturation point for urate crystal formation, principally caused by inefficient renal urate excretion. Local joint tissue factors may influence the topography and exten crystal deposition. Recent studies have provided information on dietary risk factors for gout: higher intakes of red meat, fructose and beer are independently associated with increased risk, whereas higher intakes of coffee, low-fat dairy products and vitamin C are associated with lower risk.

Keywords: Gout, Hyperuricaemia, uric acid concentration, Homeopathic approach

Address for Correspondence:
Dr. Nowmani Shaikh
Department of Pediatrics
Guru Mishri Homoeopathic Medical College and PG Institute,
Shelgaon, Jalna, India (MS)
Conflict of Interest: None Declared!

(Received 10 September 2023; Accepted 17 September 2023; Published 22 September 2023) ISSN: 2347-8136 ©2023 JMPI

INTRODUCTION

Gout is an inflammatory response to the MSUM crystals formed secondary to hyperuricaemia. The major clinical manifestations are acute synovitis, chronic erosive and deforming arthritis, tophi, nephrolithiasis, interstitial nephritis and hypertension The epidemiology of hyperuricaemia is different from that of gout. Mean uric acid (urate) concentrations are age and sex-related. Prepubertally, in males the mean concentration is around 3.5 mg/Dl, with a steep rise to 5.2 mg/dl at puberty. In females the rise is Appreciated only after menopause (up to 4.7 mg/dl). Hyperuricaemia Has been defined as a serum or plasma urate concentration greater Than 7.0 mg/dl in males and 6.0 mg/dl in females. The prevalence of hyperuricaemia varies amongst communities. Only about a tenth of patients of hyperuricaemia exhibit gout. The incidence of gout varies in populations from 0.2 to 3.5 per 1000, with an overall prevalence of 2.0 to 26 per 1000. Gout is rare in children and premenopausal females. The peak age of onset in males is between 40-50 yrs.

Risk factors

Serum uric acid concentration is the single most important determinant of the risk of developing gout. With normal renal function, its blood concentration depends mainly on the breakdown of nuclear proteins and dietary purine load (Fig. 1). Provocative factors include diuretics. Alcohol, dietary excesses, surgery, trauma, sepsis, stress, starvation and dehydration.

Classification

Hyperuricaemia and gout may be classified as primary or secondary. Increased production of uric acid as a cause of gout is seen in only 10-15 per cent of subjects (overproducers) and in 80-90 per cent, uric acid excretion is impaired.

ASSOCIATED DISORDERS Obesity, hypertension and hyperlipidaemia are important associated disorders. A negative association exists between gout and rheumatoid Arthritis.

Causes of hyperuricaemia

Primary hyperuricaemia Increased uric acid production Idiopathic (10%)

Specific enzyme defects

Hypoxanthine – guanine phosphoribosyltransferase (HGPRT)

Deficiency. Increased PP-ribose-P-synthetase (PRPP)activity Decreased uric acid excretion. Idiopathic (90%)

Secondary hyperuricaemia Increased uric acid production:

Increased purine synthesis de novo-specific enzyme defects

Complete HGPRT deficiency (Lesch-Nyhan syndrome) Glucose 6-phosphatase deficiency (type I) Increased turnover of preformed purines

Myeloproliferative disorders – polycythaemia vera, granulocytic

Leukaemias. Lymphoproliferative disorders – lymphomas, myelomas Others

Macroglobulinaemia (Waldenstrom's)

Carcinomatosis

Chronic haemolytic anaemia Gaucher's disease

Exfoliative psoriasis

Decreased uric acid excretion: Renal-chronic renal disease, nephrogenic diabetes insipidus

Increased levels of organic acids – exercise, starvation, ketoacidosis, alcohol. Drugs-diuretics, low-dose aspirin, pyrazinamide, cyclosporine, Ethambutol

Other medical disorders – hyperparathyroidism, myxoedema, Down's syndrome, lead nephropathy, sarcoidosis

Clinical features Hyperuricaemia and gout can occur in four phases.

1.Asymptomatic Hyperuricaemia

Hyperuricaemia may be an incidental finding and may never lead to gout. Conversely, during acute gouty arthritis serum uric acid levels may not be elevated. Acute severe overproduction of urate, as may occur with cytotoxic chemotherapy, is associated with a high risk of acute renal failure. Asymptomatic hyperuricaemia needs close observation but no active treatment.

2. *Acute Gouty Arthritis*

The big toe (first metatarsophalangeal joint) is the classic site for urate gout. One-third of patients may get their first attack at another site such as the in-step of the foot, ankle, knee or hand joints. Sydenham's classic description lists the important clinical characteristics used to diagnose a typical attack. The attack is acute, it starts in the night. The joint and surrounding tissues are swollen, hot, red, shiny and extremely painful. There is a mild fever with chills. Left untreated.The attack will start to improve in a week or two. The skin over the joint may subsequently desquamate.Atypical manifestations include tenosynovitis, bursitis, cellulitis, or mild pain and discomfort without swelling, lasting a day or two. Acute gout in one joint may provoke migratory attacks affecting other joints over subsequent days (cluster attacks). Pauci or polyarticular gout attacks are more common in women, especially with diuretic use.

3.Intercritical Gout

After the first attack of gout the second episode may never occur or occur after several years. However, in most, the next few episodes occur within one year. The frequency of attacks and number of sites involved gradually increase with time.

4.*Chronic Gout (Tophaceous Gout)*

Development of chronic tophaceous gout depends on uncontrolled hyperuricaemia of long duration. Many patients have usually suffered From gout for at least 10 years before tophi developed. But tophi or chronic polyarthritis may occur as early as 3 year or as late as 40 years after the first acute attack. Tophi appear as firm, nodular or fusiform swellings. In inflamed tophi the overlying skin may be erythematous. In ulcerated tophi, white chalky material-the urate crystals- may exude. The common locations of tophi are the great toe, feet, hand joints and olecranon (see Colour Atlas), Toph of of

the car, though classic is uncommon The gout in elderly patients especially women may Present differently compared to typical gout occur in middle aged men (Table

HYPERURICAEMIC KIDNEY

After gouty arthritis, renal disease is the most frequet Complication of hyperuricaemia. Urate nephropathy

Attributed to the deposition of MSUM crystals in there interstitial tissue. It is related to the formation of s acid crystals in the collecting tubules, pelvis or uret This can manifest as acute uric acid nephropathy oracid calculi. Calcium oxalate renal stones occur more commonly in patients with hyperuricaemia or gout than in normo-uricaemic subjects. Renal failure accounts for 10-25 per cent of deaths of gouty patients. Co-existent hypertension may be more important than urate in the pathogenesis of renal failure.

It should be remembered that hyperuricaemia may be seen secondary to chronic renal failure without gout.

INVESTIGATIONS The synovial fluid is turbid with low viscosity and greatly elevated Neutrophils. The joint fluid should also be examined under polarised light. Urate crystals are needle shaped, generaly 5-25 um in length, intracellular or extracellular in location and negatively birefringent Urate crystals can be demonstrated in synovial fluid and gouty tophi Demonstration of urate crystals is the gold standard for the diagnosis of gout.

Hyperuricaemia is confirmed if two or more fasting serum uric acid levels are elevated. Overproducer status can be determined by more than 700 mg 24 hour urinary urate on a normal diet, or by a single urine sample with uric acid/creatinine ratio (normal <0.5). Assessment of renal functions, lipid profile and acute phase reactants such as ESR Help in the management. Radiographs may be normal in early disease. In patients with Recurrent attacks or with chronic gout, X-rays show punched-out erosions with over hanging edges, mostly paraarticular, asymmetrical And eccentric.

AUXILIARY MEASURES

- Cut down on foods containing oxalate, such as spinach, rhubarb, beets, nuts, chocolate, black tea, wheat bran, strawberries, and beans.

Include foods rich in magnesium and low in calcium, such as barley, bran, corn, rye, oats, soy, brown rice, avocado, banana, and potato.

Restrict purines in your diet. Foods with a high purine content include beef, goose, organ meats, sweetbreads, mussels, anchovies, herring, mackerel, and yeast. Foods with a moderate amount of purines include meats, poultry, fish, and shellfish not listed above. Spinach, asparagus, beans, lentils, mushrooms, and dried peas also contain moderate amounts of purines.

Reduce or eliminate trans fatty acids, found in commercially-baked foods, such as cookies, crackers, cakes, French fries, onion rings, donuts, processed foods, and margarine.

Avoid alcohol because Alcohol is thought to increase the risk of gout due to the metabolism of ethanol to acetyl CoA leads to adenine nucleotide degradation, resulting in increased formation of adenosine monophosphate, a precursor of uric acid. Alcohol also raises the lactic acid level in blood, which inhibits uric acid excretion.

Drink 6 to 8 glasses of filtered daily to help flush uric acid from the body. Dehydration often triggers a gout attack.

HOMEOPATHIC APPROACH

Benzoic acid- Joints crack on motion. Tearing with stitches. Pain in tendo Achillis. Rheumatic gout; nodes very painful. Gouty deposits. Ganglion; swelling of the wrist. Pain and swelling in knees. Bunion of great toe. Tearing pain in great toe.

Berberis vulgaris – Old gouty constitution.Rheumatic paralytic pain in shoulders, arms, hands and fingers, legs and feet. Neuralgia under finger-nails, with swelling of finger- joints. Stitching between metatarsal bones as from a nail when standing. Pain in balls of feet on stepping.

Urtica urens- pain in acute gout deltoid; pain in ankles, wrists.

Colchicum- pain Tearing in limbs during warm weather, stinging during cold. Pins and needles in hands and wrists, fingertips. Limbs, lame, weak, tingling. Pain worse in evening and warm weather. Joints stiff and

feverish; shifting rheumatism; pains worse at night. Inflammation of great toe, gout in heel, cannot bear to have it touched or moved. Tingling in the finger nails. .

Ledum-.Gouty pains shoot all through the foot and limb, and in joints, but especially small joints. Swollen, hot, pale. Throbbing in right shoulder. Pressure in shoulder, worse motion. Cracking in joints; worse, warmth of bed. Gouty nodosities. Ball of great to swollen (Bothrops). Rheumatism begins in lower limbs and ascends (Kalmia opposite). Ankles swollen. Soles painful, can hardly step on them (Ant c; Lyc).

Rhododendrone- Joints swollen. Gouty inflammation of great toe-joint. Rheumatic tearing in all limbs, especially right side; worse, at rest and in stormy weather. Stiffness of neck. Pain in shoulders, arms, wrists; worse when at rest. Pains in bones in spots, and reappear by change of weather.

REFERENCES

1. API, textbook of medicine,12 th edition.
2. William Boericke, Pocket Manual of Homoeopathic Materia Medica, 9th edition.
3. Dr. SR Phatak, Materia Medica of Homoeopathic Medicines,
4. Roddy E, Zhang W, Doherty M. The changing epidemiology of gout, Nat Clin Pract Rheumatol, 2007, vol. 3 (pg. 443-9).
5. Wallace KL, Riedel AA, Joseph-Ridge N, Wortmann R. Increasing prevalence of gout and hyperuricemia over 10 years among older adults in a managed care population, J Rheumatol, 2004, vol. 31 (pg. 1582-7).
6. Lawrence RC, Helmick CG, Arnett FC, et al. Estimates of the prevalence of arthritis and selected musculoskeletal disorders in the United States, Arthritis Rheum, 1998, vol. 41 (pg. 778-99).
7. Helmick CG, Felson DT, Lawrence RC, et al. Est the prevalence of arthritis and other rheumatic conditions in the United States. Part I, Arthritis Rheum, 2008, vol. 58 (pg. 15-25).
8. Lawrence RC, Felson DT, Helmick CG, et al. Estimates of the prevalence of arthritis and other rheumatic Conditions in the United States. Part II Arthritic conditions.

