



UNDERSTANDING OF GOUT WITH A HOMOEOPATHIC APPROACH: A REVIEW ARTICLE

Dr. Dharti Patel

PG-Scholar, Department: - Organon of medicine & Homoeopathic Philosophy, Parul Institute of Homoeopathy and research hospital, Parul University, Vadodara.

Dr. Alpesh Jayswal

Associate Professor, Department: - Organon of medicine & Homoeopathic Philosophy, Parul Institute of Homoeopathy and research hospital, Parul University, Vadodara

Dr. Khyati P. Lakhani

Professor and HOD, Department: - Organon of medicine & Homoeopathic Philosophy PG, Parul Institute of Homoeopathy and research hospital, Parul University, Vadodara.

ABSTRACT

BACKGROUND: Gout is a chronic metabolic disorder caused by high uric acid (hyperuricemia), which forms painful crystal deposits in the joints—most commonly the base of the big toe. Driven by obesity, diet, and lifestyle changes, its global prevalence is rising. Standard care focuses on conventional diagnostics and treatments to manage inflammation and lower uric acid levels. Additionally, homeopathic approaches focus on holistic, individualized care based on a patient's specific symptom profile to prevent flares and improve long-term quality of life. **OBJECTIVE:** · To review the epidemiology, etiology, pathophysiology, clinical manifestations, diagnostic criteria, and conventional management of gout. · To evaluate the impact of gout on quality of life and associated risk factors. · To discuss the homoeopathic and miasmatic approach in the management of gout. **METHODS:** A comprehensive literature review was conducted across databases like Google Scholar, PubMed, NCBI, and standard homeopathic textbooks for studies published between 1991 and 2026. Information on gout's presentation, diagnosis, conventional treatments, and homeopathic management was systematically collected and analyzed.

KEYWORDS : Gout, Hyperuricemia, inflammatory arthritis, Homoeopathic individualized approach, Miasmatic approach, Quality of life.

INTRODUCTION:

Gout is the most prevalent type of inflammatory arthritis. It develops due to hyperuricaemia, a persistent increase in serum urate concentration that causes supersaturation of urate in body tissues, resulting in the accumulation and deposition of monosodium urate crystals within and surrounding the joints.^[1]

Gout is an inflammatory disorder caused by uric acid crystals from hyperuricemia (over 7.0 mg/dl in men; over 6.0 mg/dl in women). It is rare in children and premenopausal women, most commonly striking men aged 40 to 50. While normal levels rise after puberty for men (~5.2 mg/dl) and after menopause for women, only a minority with high levels develop gout. Currently, it affects 2 to 26 people per 1,000, and global cases are rising.^[2]

Gout prevalence is rising in affluent societies due to increasing rates of obesity and metabolic syndrome. The condition presents as sudden, explosive episodes of intense pain, swelling, and redness usually in the big toe which, if left untreated, can progress to chronic joint deformities and kidney complications.^{[3][4]}

DEFINITION:

Gout is the term used to describe the constellation of clinical features that result from deposition of Microcrystals of sodium urate monohydrate or uric acid from hyperuricemic body fluids.^[5]

ICD-11: According to ICD 11 Gout under FA25 – crystal arthropathies, with subtypes such as acute gout (FA25.00) and chronic gout (FA25.01).^[6]

ETIOLOGY:

Causes of increased urate production

Dietary -Purine-rich and fructose-rich foods, weight loss (fasting).

Hematological-Myeloproliferative and lymphoproliferative diseases

Other-Psoriasis, tumor lysis syndrome^[7]

Causes of reduced renal urate excretion

Drugs-Cyclosporine, thiazides, loop diuretics, aspirin (500-1000 mg/dl)

Renal-Hypertension, polycystic kidney disease, chronic renal failure of various etiologies Metabolic/endocrinological-Dehydration (often associated with surgery), lactic acidosis, ketosis, hypothyroidism

Other-Obesity

Combined mechanisms-Alcohol, shock, metabolic syndrome (obesity, hypertriglyceridemia)^[7]

PATHOPHYSIOLOGY:

Serum urate concentration is the primary driver of gout risk. Because humans lack the enzyme uricase, we cannot break uric acid down into a more soluble compound called allantoin, making it the final product of purine metabolism. The body normally maintains a pool of about 1 gram of urate, turning over 60% of it daily through diet and cellular breakdown. The kidneys filter out two-thirds of this daily waste, while the digestive tract excretes the rest. Hyperuricemia—the underlying cause of gout—occurs when blood urate levels become elevated. While genetics and environment can sometimes cause the body to overproduce uric acid, the issue is caused by the kidneys failing to excrete it properly in the vast majority of cases.^[5]

CLINICAL FEATURES:

• Acute gout:

1. **Onset:** Sudden, explosive pain that frequently strikes overnight and peaks within 24 to 48 hours.
2. **The Pain:** Severe and intolerable; even light contact from a bedsheet or sock is excruciating.
3. **Location:** 80% of first attacks affect a single joint. 50% occur at the base of the big toe (podagra). It can also strike ankles, knees, wrists, and surrounding tendons or bursae (like the elbow).^[8]

- **Intercritical gout:** The interval between acute gout attacks can vary greatly, ranging from a few days to several years. During the symptom-free period between attacks, patients usually remain asymptomatic, although monosodium urate (MSU) crystal deposition may continue to progress silently.^[8]

• Chronic gout:

1. **The Cause:** Untreated, long-term high uric acid causes solid crystal deposits (tophi) to build up in joints, tendons, and the ear helix.
2. **Worsening Factors:** Risk increases with frequent attacks, multiple affected joints, or upper limb involvement.
3. **Rapid Risk Group:** Postmenopausal women with heart or kidney failure taking long-term diuretics can develop hand and foot tophi very quickly.^[5]

RISK FACTORS: Some of the factors that increase your chances of gout include:

1. **Diet:** Purine-rich foods, beer, and high-fructose corn syrup raise uric acid levels.
2. **Obesity:** Higher weight increases uric acid production; obesity can raise risk tenfold.
3. **Genetics:** A family history of gout significantly increases personal risk.
4. **Medical Conditions:** Diabetes, heart disease, and kidney failure (which blocks uric acid clearance) raise risk.
5. **Hypertension:** High blood pressure doubles risk, and the diuretics used to treat it further elevate uric acid.^[9]

IMPACT OF GOUT ON HEALTH-RELATED QUALITY OF LIFE

- **Physical Limits:** Known as "the unwalkable disease," it causes severe pain and swelling that restricts movement and makes daily activities difficult.
- **Mental Health:** Physical disability can lead to emotional distress, including increased anxiety and depression.
- **Comorbidities:** Limited movement and recurrent flares increase the risk of other health issues like hypertension, heart disease, and diabetes.
- **Severity Factors:** Frequent attacks, multiple affected joints, and a high number of tophi (urate deposits) significantly worsen overall well-being.
- **Management:** Beyond medication, lifestyle changes are essential to prevent flares, improve mobility, and boost quality of life.^[10]

➤ Assessment of Quality of Life in Gout

- Commonly used scales include:
- SF-36 (Short Form-36 Health Survey)
- HAQ (Health Assessment Questionnaire)
- Likert Scale
- Gout Impact Scale (GIS).^[10]

INVESTIGATION:

- **Serum Uric Acid Test:** A blood test that checks for high uric acid. However, levels can be normal during a flare-up, and not everyone with high levels gets gout.
- **Synovial Fluid Analysis:** A needle extracts fluid from the joint to look directly for uric acid crystals. This is the most accurate way to confirm a diagnosis.
- **X-ray:** Primarily used to rule out other conditions. Actual joint damage from gout does not show up on an X-ray until the disease has progressed for several years.
- **Ultrasonography (Ultrasound):** Uses sound waves to spot early uric acid crystal deposits and hard accumulations (tophi) inside and around the joints.
- **Dual-energy computed tomography (DECT):** Combines advanced multi-angle X-rays to clearly map and visualize exactly where uric acid crystals are deposited inside the joints.^[11]

DIAGNOSTIC CRITERIA:

CLINICAL CRITERIA:

1. Rapid onset (often overnight) of severe pain together with redness and swelling, in 1 or both first Metatarsophalangeal (mtp) joints
2. Tophi.^[12]
3. Consider gout in people presenting with rapid onset (often overnight) of severe pain, redness or Swelling in joints other than the first mtp joints (for example, midfoot, ankle,

knee, hand, wrist, Elbow).^[12]

4. Assess the possibility of septic arthritis, calcium pyrophosphate crystal deposition and inflammatory arthritis in people presenting with a painful, red, swollen joint.^[12]
5. Consider chronic gouty arthritis in people presenting with chronic inflammatory joint pain.^[12]

TREATMENT: Conventional treatment for Gout

- NSAIDs (e.g., ibuprofen, naproxen, indomethacin, celecoxib): Relieve pain and swelling.
- Colchicine: Effectively stops flare-ups.
- Corticosteroids (e.g., prednisone): Oral pills or joint injections for fast relief.
- Production Blockers (e.g., allopurinol, febuxostat): Lower uric acid creation.
- Excretion Enhancers (e.g., probenecid): Help kidneys flush out uric acid.^[13]

HOMOEOPATHIC MANAGEMENT:

- **Lycopodium:** Chronic gout, contracted fingers/toes, worse on right side, improves with movement.
- **Nitric acid:** Sudden, sharp, splinter-like pain in toes that disappears quickly.
- **Natrum sulph:** Worse in damp, cold weather and on the left side; relieved by changing positions.
- **Colchicum:** Extreme sensitivity to touch/movement in big toe and heel; worse evening to morning.
- **Medorrhinum:** Tender heels/soles and enlarged finger joints; worse by day, better near the seaside.
- **Calcarea fluor:** Enlarged finger joints; worse during rest and weather changes, better with warmth.
- **Calcarea carb:** Swollen joints and nodules.worse from cold and washing, better from pressure and dry air.
- **Sulphur:** Stiffness and joint itching; worse standing/ resting, better in warm weather.
- **Benzoic acid:** Painful nodules, wrist swelling, deposits; worse when uncovered, better with warmth.
- **Pulsatilla:** Drawing heel pain; better in open air/warmth, worse from direct heat.
- **Coffea cruda:** Rheumatic gout linked to excessive coffee intake; relieved by warmth.
- **Rhus tox:** Hot, swollen joints; worse at night, better from continuous movement.
- **Thuja:** Heel pain, red/swollen fingertips, cracking joints; worse at night/heat, better drawing limb up.^[14]

CONCLUSION:

Gout is a common, growing inflammatory disorder that requires early intervention to protect physical function. While conventional treatments effectively manage acute flares and lower uric acid, long-term use can carry side effects. Homeopathy offers a holistic, individualized alternative based on overall symptom totality to reduce attack frequency and improve health. More clinical studies are needed to further validate homeopathy's efficacy in managing the condition.

MIASMATIC ANALYSIS FOR GOUT ^[15]		
Miasm	Core pathological Character	Key Features
PSORIC	Functional Imbalance Hypersensitivity before structural changes occur.	• Functional metabolic disturbance • Hyperacidity & digestive derangement • Intermittent joint pain
SYCOSIS (Predominant)	Excess & Infiltration The primary miasm in chronic gout due to accumulation.	• Uric acid deposition • Recurrent inflammation • Nodular swelling & tophi • Joint stiffness • Metabolic sluggishness

SYPHILITIC	Destruction & Degeneration Marked by tissue breakdown and physical deformity.	• Joint destruction • Ulceration of tophi • Chronic tissue degeneration • Chronic tissue degeneration
TUBERCULAR	Erratic Burnout Characterized by rapid shifts, wasting, and instability.	• Rapid exhaustion • Alternating inflammatory conditions • Marked weather sensitivity • Family history of chronic metabolic disease

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