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GOUT
The Indian Scenario





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CHAPTER 1

Introduction to Hyperuricemia and Gout

Shivam Mehra, Kamal Mehra, Nindiya Kapoor Mehra, Bharat Veer Manchanda, Arvind J Vatkar

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INTRODUCTION

Uric acid is a chemical molecule produced when the body breaks down purines. Purines are chemical substances contained in DNA and RNA that are necessary for cell function and play an important role in biological processes such as nucleotide synthesis. They have a two-ring structure and are often found in adenine and guanine with their chemical formulae of $C_5H_5N_5$ and $C_5H_5N_5O$, respectively. Purines are present in a variety of foods, with the highest concentrations found in organ meats, shellfish, and vegetables such as asparagus, cauliflower, spinach, mushrooms, and green peas.

Uric acid is an organic compound that contains carbon (C), hydrogen (H), nitrogen (N), and oxygen (O) atoms. The molecular formula is $C_5H_4N_4O_3$, and its structure consists of a fused ring system with nitrogen atoms **(Fig. 1)**.

Uric acid was first discovered in the late 18th century. Swedish scientist Carl Wilhelm Scheele discovered uric acid in 1776 while researching kidney stones. Scientists like Sir Archibald Garrod conducted more studies in the 19th century to better understand its involvement in gout and other metabolic illnesses.

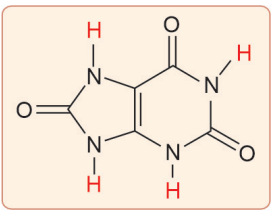

Fig. 1: Molecular structure of uric acid.

URIC ACID, HYPERURICEMIA, AND GOUT

Uric acid is the result of extrinsic pool of purines and intrinsic purine metabolism in humans and higher primates. In all other mammals, the enzyme uricase converts uric acid to allantoin that gets eliminated through urine. The metabolism and production of uric acid involve processes that depend on multiple factors regulating its production in the liver and excretion by the gut and kidneys. Uric acid predominantly exists as urate, that is, in its salt form. The rise in the urate concentration in blood leads to the formation of uric acid crystals. Since it has a low water solubility (solubility limit in blood being 6.8 mg/dL in humans), higher levels of uric acid (>6.8 mg/dL) result in the formation of monosodium urate crystals (MSU).

Higher uric acid levels or hyperuricemia is a product of more uric acid production, dysfunctional renal excretion, or a combination of both. Hyperuricemia leads to deposition of urate crystals in the joints and kidneys. Increasing evidence suggests the relationship of hyperuricemia with multiple conditions including gout, diabetes, hypertension, renal disease, metabolic syndrome, and cardiovascular disease.

Gout is a form of arthritis that is associated with hyperuricemia. It occurs when urate crystals, formed from excess uric acid, accumulate in the joints, causing inflammation, pain, and swelling. Gout typically affects the big toe but can

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also impact other joints such as the ankles, knees, elbows, wrists, and fingers.

The link between hyperuricemia and disease is the strongest for gout, a common rheumatic disease characterized by acute inflammatory arthritis. Around 8.3 million Americans are affected by gout. Although these numbers in India are not very clear. A study conducted in Bhigwan Village of India estimated a prevalence of 0.12% according to the International League of Nations Against Rheumatism, Community Oriented Program for Control of Rheumatic Diseases (ILAR COPCORD). Another study revealed that 15.8% of affected patients are <30 years of age and urban Indian population has more presence compared to rural population.

FACTORS AFFECTING URIC ACID LEVELS: GENETIC AND ENVIRONMENTAL FACTORS

The cause of high uric acid level could be genetic or due to environmental factors such as diet and health. The correlation of diet with uric acid levels has not been fully understood, however, purine-rich foods such as meat (for example, red meat, bacon, pork, and mutton) and seafood have been suggested to increase uric acid levels. Choi and coworkers conducted a study for the association of gout with purine-rich foods, protein intake, and dairy intake. During 12 years of follow-up amongst 47,120 men that had no history of gout at baseline, 730 new cases of gout were reported. The authors reported a 1.41-fold and 1.51-fold increase in the risk of gout with increased meat and seafood consumption, respectively. No increased risk was found with increase in total protein intake and purine-rich vegetables. The study also reported that the risk of gout was low with higher consumption of low-fat dairy products.

Alcohol intake particularly consumption of purine-rich alcoholic beverages like beer is associated with increased

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risk for hyperuricemia. In another study by Choi and Curhan including data from 14,809 participants (both men and women) in the Third National Health and Nutrition Examination Survey (NHANES), the authors documented that serum uric acid levels increased with increasing beer consumption followed by liquor intake (p values for trend < 0.001) whereas moderate wine drinking does not affect serum uric acid levels. Also, it has been shown that alcohol intake increases uric acid both by reducing urate excretion and increasing production.

Multiple reports have also been published on the association of cigarette smoking with uric acid levels. It is reported that there is an inverse relation between smoking and serum uric acid levels. A study conducted on 300 participants reported that plasma uric acid levels were significantly lower in smokers than in nonsmokers. However, another study on Japanese male working population showed that serum uric acid levels were more in ex-smokers followed by nonsmokers and then present smokers.Another major risk factor for hyperuricemia is sedentary lifestyle. It was shown in a study that participants who spent ≥10 h/day in sedentary behavior were more likely to have hyperuricemia than those who spent <5 h/day.

As external factors and lifestyle are risk parameters for hyperuricemia and since lifestyle choices vary amongst the different Indian communities, we sought to study the prevalence of hyperuricemia in these communities. Also, there is a dearth of large-scale data (in terms of gender, age, lifestyle, and community) on the prevalence of hyperuricemia in subjects amongst different community populations. In this book, we talk about a multicentric prospective study that was undertaken to determine the prevalence of hyperuricemia in subjects across four major communities.

Further, we also demonstrate our study that determined the association between increased uric acid levels and lifestyle of different communities.

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CAUSES OF HYPERURICEMIA AND GOUT

- *Diet*: High intake of purine-rich foods (red meat, seafood, and organ meats), alcohol, and sugary beverages
- *Genetics*: Family history of gout or hyperuricemia
- Gout, a condition characterized by elevated uric acid levels and urate crystal formation in joints, is largely influenced by genetics. Recent studies have identified several genetic variants linked to increased risk of gout, including SLC2A9, ABCG2, and SLC22A12, which regulate uric acid transport and excretion. A 2018 study highlighted the importance of genetic predisposition in gout, with certain alleles increasing the risk by affecting renal urate handling and promoting hyperuricemia. Another 2020 study highlighted the role of ABCG2 mutations in early-onset gout. Gout inheritance is complex, involving autosomal dominant and polygenic mechanisms. Families with a history of gout often show a higher prevalence, suggesting a hereditary component. Gene therapy for gout is still in its experimental stages, with researchers exploring the potential of CRISPR-Cas9 technology to correct specific genetic mutations associated with hyperuricemia.
- *Medical conditions*: Obesity, hypertension, kidney disease, diabetes, and metabolic syndrome.
- *Medications*: Certain drugs may exacerbate gout by raising uric acid levels via a variety of ways. Diuretics, such as thiazide and loop diuretics, impede kidney uric acid excretion, resulting in hyperuricemia. Low-dose aspirin reduces renal urate excretion and promotes uric acid retention. Immunosuppressants, such as cyclosporine, reduce renal urate production, which contributes to high uric acid levels. Niacin, which is used to treat cholesterol, may also raise uric acid levels by decreasing renal urate clearance. Gout may interact with drugs, necessitating dosage modifications. Understanding these interactions

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is critical for healthcare practitioners seeking to optimize treatment methods for gout patients, ensuring successful management and reducing medication-related problems.

SYMPTOMS OF GOUT

- Sudden, intense joint pain
- Swelling and redness around the affected joint
- Warmth and tenderness in the joint
- Limited range of motion in the affected joint
- Acute gout is characterized by the rapid onset of intense pain, swelling, warmth, and redness in the afflicted joint, which often wakes patients from their sleep. The first episode normally peaks within 24 hours and may linger anywhere from a few days to a couple of weeks if not addressed. Podagra, a special word for gout affecting the big toe, is the most prevalent presentation in 50% of first-time gout attacks, producing severe pain and inflammation at the metatarsophalangeal joints. Other joints affected by gout include the ankles, knees, elbows, wrists, and fingers, which become painful, erythematous, and swollen, limiting mobility and quality of life.

DIAGNOSIS OF HYPERURICEMIA AND GOUT

- *Blood test*: Measures the level of uric acid in the blood.
- *Joint fluid test*: Checks for urate crystals in the synovial fluid.
- *Imaging*: X-rays, ultrasound, or CT scans to detect joint damage or crystal deposits. Gout X-rays are frequently seen later in the disease.

Signs include tophi are soft tissue opacities caused by the deposition of urate crystal. Ultrasound would show joint effusion which refers to an increase in joint fluid. "Rat bite" lesions are defined as periarticular or marginal erosions with overhanging borders. Well-defined sclerotic borders. Chronic

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Fig. 2: Rat bite erosions seen in hand X-ray in gout.
Source: Gibson MC. Gout x ray. [online] Available from https://www.wikidoc.org/index.php/Gout_x_ray [Last accessed August, 2024].

joint space narrowing suggests cartilage deterioration. *Bone changes*—subchondral bone cysts and spurs may develop **(Fig. 2)**.

TREATMENT AND MANAGEMENT OF GOUT

- *Medications*:
  - *Nonsteroidal anti-inflammatory drugs (NSAIDs)*: To reduce inflammation and pain
  - *Colchicine*: To relieve acute gout attacks
  - *Corticosteroids*: To control severe inflammation
  - *Urate-lowering therapy*: Allopurinol or febuxostat to reduce uric acid levels.
- *Lifestyle changes*:
  - *Diet*: Reduce intake of purine-rich foods, alcohol, and sugary drinks.
  - *Hydration*: Drink plenty of water to help flush out uric acid.

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- *Weight management*: Achieve and maintain a healthy weight.
- *Exercise*: Regular physical activity to improve overall health.

- *Preventive measures*: Regular monitoring of uric acid levels and adherence to prescribed treatments to prevent recurrent gout attacks.

DIFFERENTIAL DIAGNOSIS

- *Pseudogout* (calcium pyrophosphate deposition disease) often affects bigger joints, such as the knee. Frequently has a more chronic course.

  Special tests include joint aspiration reveals calcium pyrophosphate crystals that are rhomboid-shaped and positively birefringent under polarized light **(Fig. 3)**.

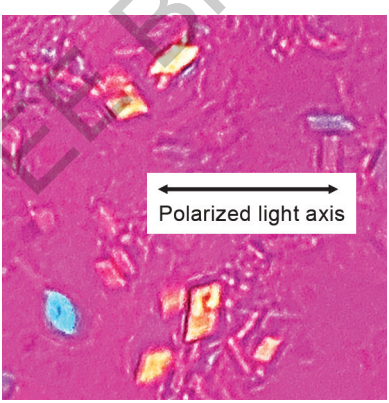

Fig. 3: Microscopy showing rhomboid shaped crystals in synovial fluid of pseudogout.

Source: Wikipedia. Calcium pyrophosphate dihydrate crystal deposition disease. [online] Available from https://en.wikipedia.org/wiki/Calcium_pyrophosphate_dihydrate_crystal_deposition_disease [Last accessed August, 2024].

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- *Septic arthritis symptoms*: Severe pain, fever, and high white blood cell count. Affects every joint, but particularly the knee.

  Special tests include joint aspiration with a positive bacterial culture and an increased white blood cell count in synovial fluid. Gram staining of synovial fluid may reveal bacterial organisms.
- *Rheumatoid arthritis* symptoms include symmetric joint involvement, prolonged morning stiffness, and fatigue.

  Special tests include rheumatoid factor (RF) and anticitrullinated protein antibody (ACPA) testing, as well as joint imaging for erosions.
- *Osteoarthritis symptoms* include joint pain and stiffness, which increase with activity and improve with rest. Typically affects weight-bearing joints such as the knees and hips.

  Tests include X-rays that reveal joint space narrowing, osteophytes, and subchondral sclerosis.
- Signs and symptoms of *psoriatic arthritis* include joint discomfort and swelling, which may be asymmetrical. Associated with psoriatic skin lesions.

  Tests include clinical diagnosis based on imaging and the presence of skin lesions. A typical "Koebner phenomenon" which elicits a new psoriatic lesion formed by a controlled scratch in 10–20 days is also diagnostic of psoriasis.

COMPLICATIONS

- *Tophi*: Deposits of urate crystals that form lumps under the skin.
- *Kidney stones*: Caused by uric acid crystals in the kidneys.
- *Joint damage*: Chronic inflammation can lead to joint deformity and damage.

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Proper management of hyperuricemia and gout involves a combination of medication, dietary modifications, and lifestyle changes to reduce symptoms and prevent complications.

CONCLUSION

Gout is a complex and multifaceted condition characterized by the accumulation of urate crystals in the joints, leading to intense pain, inflammation, and potential joint damage. As a form of inflammatory arthritis, gout's prevalence has risen in tandem with lifestyle changes, including dietary habits and increased life expectancy. Understanding the underlying pathophysiology, risk factors, and clinical presentation of gout is crucial for early diagnosis and effective management.

Throughout this chapter, we have explored the foundational aspects of gout, including genetic and environmental factors, epidemiology, and the biochemical mechanisms driving uric acid accumulation. We have also examined the clinical manifestations of gout, ranging from acute flares to chronic tophaceous gout, and the impact these have on patients' quality of life.

The introduction to gout sets the stage for a deeper exploration of its diagnostic criteria, treatment options, and preventive strategies, which are critical for improving patient outcomes. As we move forward, it is essential to keep in mind that gout is not only a medical condition but also a lifestyle-related disease, influenced by factors such as diet, alcohol consumption, and comorbidities like obesity and hypertension.

By fostering a comprehensive understanding of gout in its early stages, healthcare providers can better tailor interventions to manage the disease effectively, prevent complications, and enhance the overall well-being of those affected by this condition.

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Salient Features

- *Comprehensive coverage*: Detailed exploration of uric acid, hyperuricemia, and gout, including causes, symptoms, diagnosis, treatment, and complications
- *In-depth pathophysiology*: Explanation of the biological mechanisms of gout, focusing on urate crystals and uric acid
- *Indian context focus*: Insights into the prevalence, dietary influences, and lifestyle factors specific to the Indian population, supported by a multicentric study
- *Diet and lifestyle guidance*: Practical advice on diet, weight management, and lifestyle changes for gout prevention and management
- *Medical management*: Overview of treatment options, including medications, alternative therapies, and emerging treatments
- *Patient and healthcare provider perspectives*: Real-life case reports and guidance for personalized care in gout management
- *Research and advancements*: Discussion of current research and future directions in gout treatment
- *User-friendly format*: Accessible language with illustrations to simplify complex concepts
- *Expert contributions*: Insights from leading experts in rheumatology, orthopedics, and health, offering a well-rounded perspective

This book is designed to be a definitive guide for managing gout effectively.

Sachin Yashwant Kale is a distinguished Orthopedic Surgeon based in Navi Mumbai, currently serves as Professor and Head of Unit at DY Patil Medical College and Hospital in Nerul, Navi Mumbai, Maharashtra, since February 2017. His extensive professional experience also includes positions such as Associate Professor and Lecturer at the same institution since 2004, and Registrar at TNMC Nair Hospital, Mumbai, Maharashtra. He is an MS in Orthopedics from TNMC Nair Hospital and holds a Diploma in Orthopedics. He is a Consultant Orthopedic Surgeon affiliated with several hospitals including Apollo Hospital, Fortis Hospital, and Medicity Hospital in Navi Mumbai. As an active member of the orthopedic community, he holds prominent roles such as President of the Navi Mumbai Orthopedic Association and Executive Council Member of the Bombay Orthopaedic Society. Dr Kale has made significant contributions to medical research and education, serving as Editor-in-Charge of the Journal of Medical Thesis, Associate Editor of the Journal of Orthopaedic Case Reports, and Editor for the Journal of Clinical Orthopaedics. His specialized training includes joint arthroplasty fellowships and trauma management courses. He is recognized for his research and publications, with more than 100 papers in esteemed journals, reflecting his dedication to advancing orthopedic science and practice. His published works cover a wide range of topics, including robotic total knee replacements, thyroid levels in women with lower back pain, and innovative techniques in fracture fixation, among others. Dr Kale's achievements also include a Guinness World Record for Most Authors on a Single Peer-Reviewed Academic Paper, underlining his collaborative and impactful contributions to the medical field.

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