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CLINICAL PRESENTATION OF GOUT AND MANAGEMENT

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Abstract:
Gout is a condition that arises from an excessive buildup of uric acid, resulting in the formation of urate crystals. Symptoms include acute and chronic arthritis, tophi, interstitial renal disease, and uric acid nephrolithiasis. Elevated levels of serum uric acid are often observed. Diagnosis is typically made based on criteria established by the American College of Rheumatology. Monosodium urate crystals can be found in the synovial fluid of the affected joint. Treatment options are readily available and can be administered by general practitioners. Lifestyle changes such as avoiding obesity, meats, shellfish, and alcohol while promoting low-fat meals and vegetables are recommended. Medications like non-steroidal anti-inflammatory drugs, colchicine, and corticosteroids are commonly used in treatment. This article aims to understand the clinical presentation and management of gout.
Keywords: *Gout, Arthritis, Treatment.*

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INTRODUCTION:

A serum concentration of uric acid above 7mg/dl is known as hyperuricemia, which can be caused by either an excess production or insufficient elimination of uric acid. This condition may also result from genetic mutations, with pre-menopausal women being shielded by hormones. Uric acid, the final product of purine metabolism in the body, serves no functional purpose. Unlike other animals, humans lack uricase, an enzyme that converts uric acid into a water-soluble form, leading to its buildup. Either excessive production or inadequate elimination of uric acid can result in its accumulation in the body. While hyperuricemia is considered a risk factor for gout, the link between the two is not always clear. However, in most cases, gout is caused by insufficient elimination of uric acid.[1,2] Acute and chronic arthritis, tophi, interstitial renal disease, and uric acid nephrolithiasis can manifest clinically. Elevated serum uric acid levels are often observed. Diagnosis can be made based on criteria outlined by the American College of Rheumatology. Monosodium urate crystals may be detected in the synovial fluid of the affected joint. Treatment options are readily available, and general practitioners can carry out diagnosis.

CLASSIFICATION OF HYPERURICEMIA:

Overproduction of uric acid:

Any imbalance in purine metabolism can result in excessive uric acid production. A daily intake of 600 mg in a diet free of purines is typically considered normal for a healthy adult male with regular kidney function. It is recommended to monitor uric acid levels through a 24-hour check without dietary restrictions. Levels exceeding 800mg in a 24-hour check are considered higher than normal.[3]

Under excretion of uric acid:

The kidneys are responsible for efficiently excreting uric acid. Glomeruli in the kidneys filter uric acid, which is then reabsorbed in the proximal tubule and secreted in the distal part. Tubular secretion is how maximal excretion is achieved. Most cases of hyperuricemia result from insufficient uric acid excretion.[3]

Overproduction of urate	Decreased excretion of uric acid	Acidosis	Drug ingestion	Combined mechanism
• Primary-idiopathic hyperuricemia • Hypoxanthine-guanine phosphoribosyltransferase deficiency • Phosphoribosylpyrophosphate synthetase overactivity • Hemolytic processes • Lymph proliferative disease • Myeloproliferative disease • Polycythemia vera • Psoriasis (severe) • Paget's disease Rhabdomyolysis, Exercise, alcohol, obesity,purine-rich diet	• Primary idiopathic hyperuricemia • Renal insufficiency • Polycystic kidney disease • Diabetes insipidus • Hypertension •	• Lactic acidosis • Diabetic ketoacidosis • Down Syndrome • Starvation ketosis • Berylliosis • Sarcoidosis • Lead intoxication • Hyperparathyroidism • Hypothyroidism • Toxemia of pregnancy • Bartter's syndrome	• Salicylates (less than 2g/d) • Diuretics • Alcohol • Levodopa-carbidopa (Sinemet) • Ethambutol (Myambutol) • Pyrazinamide • Nicotinic acid (niacin; Nicolar) • Cyclosporine (Sandimmune)	• Glucose-6-phosphate dehydrogenase deficiency • Fructose-1-phosphate aldolase deficiency • Alcohol • Shock

Table (1): Causes of Hyperuricemia [3]

CLINICAL MANIFESTATION

The presence of urate crystals in different areas of the body causes symptoms in gout cases. These crystals trigger the body's immune response, resulting in extensive bodily harm as inflammatory agents become active. When urate crystals accumulate in the synovial fluid of joints, an excessive immune response due to cytokines and neutrophils can cause permanent harm, destruction, and deformities in the joints.[4]

Acute gout:

Various factors like a high protein diet, binge drinking, parenteral nutrition, and prophylactic surgery can trigger the rapid onset of acute gout. This condition is characterized by a sudden surge in uric acid levels, with the attack peaking within a day or two and lasting up to a week if left untreated. The symptoms, including erythema, swelling, acute pain, and mild fever, may be mistaken for septic arthritis and cellulitis due to their similarities. Although central joints are less affected, the exact reason for this is unclear, and sometimes, a family history of gout can be identified. The intense pain associated with acute gout remains unexplained, as it is believed to be triggered by an immunological challenge to the system.[3,5-7]

Interval Gout:

Once the patient fully recovers and no longer shows symptoms, it is called interval gout. While certain doctors recommend prophylactic medicine during this time, others suggest waiting to see if any future attacks will occur. Therapy is highly recommended in cases where preexisting renal problems or tophi are present.[8]

Tophaceous Gout:

Over a prolonged period of illness, urate crystal deposits develop into nodular masses in the soft tissue known as tophi, causing scarring and deformity in the joints. This process can happen from 3 to 42 years after the first episode.[2]

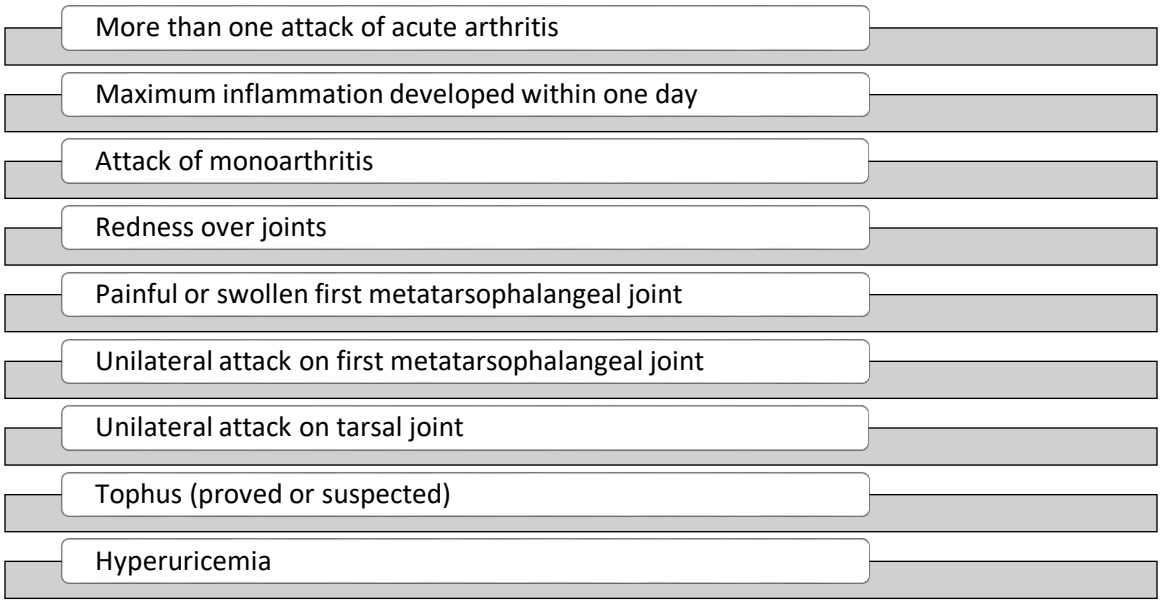
DIAGNOSIS OF GOUT:

Preliminary Criteria For The Clinical Diagnosis Of Gout According To the American College of Rheumatology:

The American College of Rheumatology has established certain preliminary criteria for the clinical diagnosis of Gout, stating that six or more of these criteria must be present to make a diagnosis [Figure 1][9]. A typical manifestation of gout involves the occurrence of monoarthritis characterized by sudden onset, often leading the individual to be abruptly awakened from sleep due to the intense pain experienced. The most frequently affected areas include the great toe, which holds significant historical relevance, and the wrists, ankles, and feet. The first metatarsophalangeal joint is commonly involved, causing considerable discomfort. For individuals already identified as being at risk, various online tools and resources are available, such as risk calculators, to assist in monitoring their condition. In older individuals, polyarticular and oligoarticular episodes may be observed, with a lower level of pain reported compared to other age groups, indicating variations in the presentation of gout across different demographics.[10]

Due to cardiac problems and ineffective heart function in gout patients, various lab tests, such as complete blood count, urine analysis, serum creatinine, serum uric acid, and blood urea nitrogen, are recommended. Radiography is not very helpful in detecting tophi, which typically appears later in the disease. Therefore, synovial fluid aspiration plays a key role in diagnosis.[11]

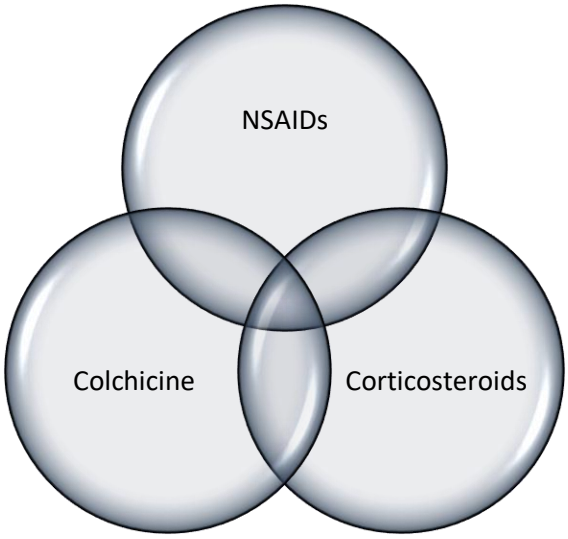
Figure [1]: American College of Rheumatology Criteria to Diagnose Gout



TREATMENT

The first treatment for acute gout typically involves NSAIDs, with Indomethacin being the preferred medication. Ketorolac could also offer similar advantages. NSAIDs can have significant negative effects on the gastrointestinal tract and should be avoided in individuals with gastrointestinal issues, kidney problems, or heart conditions. These medications are usually recommended for around a week.[12,13] Corticosteroids may be administered to patients who are experiencing difficulties with NSAIDs and other medications. After discontinuation, there may be a rebound effect, so the treatment should be continued for ten to fourteen days with the correct dosage. It can be administered orally, intramuscularly, or intra-articularly with varying dosages, such as prednisone at 40 mg for four days, followed by 20 mg for four days, and then 10 mg for four days. Colchicine has been used for a long time as a treatment, but it can lead to nausea and vomiting. It is most effective when given promptly after the onset of the disease and loses effectiveness with a longer delay. It is administered at an initial dose of 1.2 mg, followed by a 0.6 mg dose one hour later, and then a maintenance dose of 0.6 to 1.2 mg per day. Colchicine does not possess pain-relieving properties. Caution is advised for patients with kidney or liver damage. The cost-benefit ratio is high, and there is also a problem of limited availability. High doses can be toxic, which further restricts its usage.[14] Allopurinol, classified as a xanthine oxidase inhibitor, is the primary treatment for recurring gout. It can be administered to patients with cardiac and renal problems, yielding positive outcomes. The dosage should be determined based on the levels of serum uric acid.[15-17]

Figure [2]: Drug of choice for Gout



Prevention of Recurrent Gout:

Several practices are recommended to prevent gout recurrence. Overweight individuals should aim to shed weight while reducing alcohol intake is also recommended. If hyperuricemia is asymptomatic, treatment may not be necessary. Merely experiencing joint pain and hyperuricemia is insufficient to warrant

gout treatment. Deciding on prophylactic therapy involves careful consideration due to the numerous side effects associated with gout medications. The patient's involvement is crucial in treatment decision-making. Medications are typically prescribed to patients who have more than two gout attacks annually, as well as the presence of tophi and radiological signs. Patients with kidney problems taking colchicine should undergo a complete blood count and creatinine kinase levels check. [18]

CONCLUSION:

Gout can be effectively managed when applied with the right knowledge and correct treatment approach. The field of gout treatment has seen significant development thanks to the introduction of new xanthine oxidase inhibitor medications. Microbiological technology has enhanced our understanding of how the kidney handles urate, leading to further advancements. When prescribing urate-lowering drugs, it is essential also to provide prevention to minimize the risk of future flare-ups. Current treatment methods involve NSAIDs, low-dose oral corticosteroids, and colchicine. It is possible that gout medications targeted at specific locations may soon become available. Ongoing research and studies may introduce novel therapies in the future. For now, utilizing the existing treatment modalities effectively can help gout patients lead a normal life.

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