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# Gout

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Illustrated by Bol's Eye Comics.

## Introduction

Gout is a syndrome caused by an inflammatory response creating an over production of uric acid increasing uric acid levels in the blood and body fluids. Gout is defined as a disorder resulting from tissue deposition of MSU crystals (in joints, bursae, bone, and certain other soft tissues, such as ligaments, tendons and occasionally, skin) and/or crystallization of uric acid within the renal collecting system (tubules and renal pelvis) that typically occurs in acid urine" (Terkeltaub & Edwards, 2013, p. 20). Gout is among the oldest diseases affecting approximately 3.9% of adults in America (Terkeltaub & Edwards, 2013, p. xv). Historically, Gout was the "disease of kings." An early century caricature depicts gout in affluent middle-aged men that over indulge in alcohol and food. Today, the population stereotypes the average patient with impressions of self-limiting behavior such as drinking and diet (Terkeltaub & Edwards, 2013, p. xv). Gout prevalence and incidence are on the rise. Gout pathophysiology is complex and not easily understood affecting the patients' quality of life. Gout treatment is associated with treatment failure and noncompliance. Consequently, "Most patients with gout are treated in general practice and never enter the domain of the rheumatologist, and general practitioners (GPs) are unlikely to read guidelines that are published in rheumatology journals or present on rheumatology websites" (Perry & Madhok, 2010, p. 2233). Increasing knowledge and understanding of gout, clinicians can significantly influence the quality of life through treatment options for patients and practitioners.

## Signs and Symptoms

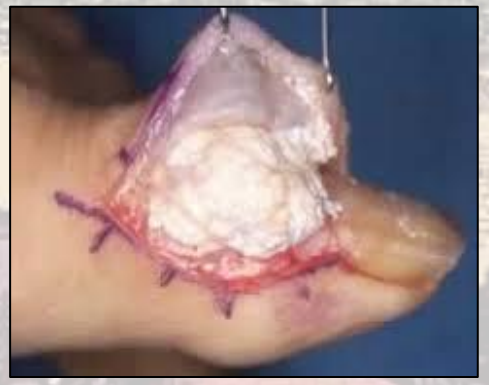
- Signs and symptoms of gout start with an elevation in serum uric acid levels greater than 6.8 mg/dl.
- Four clinical manifestations are seen in gout: asymptomatic hyperuricemia, acute gout, intercritical gout, and chronic advanced gout.
- Symptoms occur when elevated uric acid in the bloodstream creates urate crystals triggering a gout flare.
- A flare is an acute onset within 24 hours with extremely painful mono or oligoarthritis, which often results in sick leave.
- Higher uric acid levels predict more flares and development of tophi. Tophi are an accumulation of monosodium urate crystals that can form swollen areas in the joints, cartilage, bones, and skin nodules.
- Hyperuricemia can be non-symptomatic but typically precedes gout.

## Significance of Pathophysiology

**Asymptomatic Hyperuricemia**
Hyperuricemia can be non-symptomatic but typically precedes gout. Asymptomatic gout is associated with lower uric acid levels. Asymptomatic gout is the elevation of uric acid in the blood with no clinical symptoms. Silent tissue deposits of urate may be present. Gout affects the lower extremities often at night and may present as an itchy feeling.

**Acute Intermittent Gout**
Acute gout attacks are usually sudden, nocturnal and triggered by alcohol intake, dehydration, joint injury recent surgery, and fever. Acute gout treatments priority is to relieve the acute symptoms by reducing inflammation and reducing serum urate. Medications include NSAIDS, oral colchicine, and corticosteroids.
The classic presentation is podagra, 90% of patient have this inflammation of the first metatarsophalangeal joint of the foot. Lower extremity joints are most common for initial gout attacks. Acute gouty arthritis results from increased uric acid levels that cause monosodium urate crystals (MSU) to deposit in the joints. MSU attract leukocytes to the synovial space where the MSU crystals get phagocytized and destructive enzymes cause further inflammation and tissue damage (Hardy, 2011, p. 14).
Patients may have systemic symptoms such as low grade fever, chills, and malaise. Laboratory data may reveal elevated sedimentations rate and leukocytosis.
Intercritical gout is when acute gouty arthritis returns to an asymptomatic phase.

**Advanced Gout**
Untreated gout will progress to advanced gout. Intercritical periods disappear and patient develops chronic arthritis due to tophi causing synovial inflammation making joints painful and swollen. Chronic tophaceous gout is a cycle of attacks that over time, attacks will occur more frequently, affect more joints and last longer. "Some 60% of patients will have a second attack in the first year, and 78% will have a second attack within two years" (Hardy, 2011, p. 14)



Clinical example excision of gouty tophi

## Underlying Pathophysiology

Symptoms occur when elevated uric acid in the bloodstream creates urate crystals that deposit in the joints or tendons causing inflammation. Inflammatory cytokines and neutrophils trigger by the immune response from crystal deposits in the joints. "MSU crystals deposit into tophi typically in cool, poorly vascularized peripheral connective tissue" (Terkeltaub & Edwards, 2013, p. 43) Uric acid is the byproduct of purine catabolism generated from the activity of xanthine oxidase (Zychowicz, Pope, & Graser, 2010, p. 624). Purines are chemicals found in the human body obtained through the diet. "Evolutionary loss of functional uricase, the enzyme that converts uric acid to the more soluble allantoin in most other mammals, causes hyperuricemia risks in humans" (Zychowicz et al., 2010, p. 624). Hyperuricemia occurs from an overproduction of uric acid (10%) or urate under-excretion (90%) by the kidneys. Inherited mutations in renal urate transporters, acquired kidney disease, hypertension, insulin resistance (metabolic syndrome), high alcohol intake, lead toxicity, medications are contributing factors to inefficient renal excretion of uric acid (Terkeltaub & Edwards, 2013, table 1.3). Genetic disorders, disorders of increased cell turnover, and diet result in overproduction of uric acid (Terkeltaub & Edwards, 2013, table 1.3). The clinical presentation of gout is monoarticular pain and swelling in the lower extremities especially the great toe. Patients complain of symptoms more often at night with a duration of 3 to 10 days before seeking treatment (Hardy, 2011, p. 14). Treatment goals are to reduce gout flare, protect against future attacks, and lower serum urate.

## Implications for Nursing Care

- **Patients with gout show reduced health related quality of life exhibit reduced social functioning due to pain, limited activity, and immobility. Quick treatment of acute gout flares and prevention of subsequent attacks is essential, "subjects perception of their overall gout related pain and functioning were highly correlated with HRQOL and disability" (Smith, Bracken, & Smith, 2011, p. 1114).**
- Improving outcomes in the management of gout necessitates the nurse to become knowledgeable of the stages of gout including treatment goals and treatment complications. Nursing strategies includes detecting risk factors, identifying predisposing factors, and recognizing symptoms. Nurses educate patients on acute attacks, preventing attacks, dietary modification, and general considerations of gout through all four stages. Gout is on the rise and affects approximately 3 to 5 million people in the United States (Hardy, 2011, p. 14). Due to the sporadic nature of gout, identifying risk factors is essential to improving outcomes.
- Risk factors include advanced age, male gender, postmenopausal female gender, drugs (diuretics, low-dose aspirin, cyclosporine), hypertension, high alcohol intake, high body mass index, diet high in meat and seafood, and genetic influences (Vannucchi, 2012). With only "approximately 20% of patients will make lifestyle changes" (Hardy, 2011, p. 19), increasing the need for nurses to education about joint destruction during the intercritical periods is critical. Advance practice nurses identify that gout may be a sign of other comorbidities such as diabetes mellitus, hypertension, renal manifestations, obesity, metabolic syndrome, heart failure, hyperlipidemia, and cardiovascular disease.
- Rheumatologists are specialist trained in the acute and chronic management of gout. Hardy (2011) states that studies show that gout is a common cause for inpatient rheumatologist referrals, and primary care physicians refer only 3% of patients to a rheumatologist (p. 19). The American College of Rheumatology established treatment guidelines to set a standard for the treatment of gout. Crittenden & Pillinger (2013) identify gout treatment guidelines including dietary considerations, treatment of acute gout attacks, lowering urate in established gout patient, preventative treatment, and seeking expert guidance. Non-pharmacological treatment of gout includes applying ice and resting the joints.
- Pharmacologic treatment includes non-steroidal anti-inflammatory drugs, urate lower drugs, glucocorticoids, intra-articular steroid with careful consideration to coexisting illness and severity of symptoms. Advanced practice nurses educate and monitor the gout patient for adverse side effects associated with gout treatment. Key strategies focus on mediation compliance, potential side effects, and contraindications.

## Conclusion

Gout tends to be undertreated and on the rise. Poor patient understanding, physician failure to develop targeted treatment plans, and failure of therapies are reasons for inadequate gout treatments. Nurses play a significant role in improving outcomes for the identification and management of gout through education, collaboration, and compliance. Gout patients add significantly to national health care costs in gout patients with more frequent attack. Acute gout symptoms encourage patients to seek treatment due to pain. Intercritical periods reduce medication compliance and dietary management resulting in increased risk of tophi development and acute gouty attacks. Routine follow up is essential to achieve treatment goals. Nurses sensitize the patient on the lifelong compliance of urate-lowering medications, dietary restrictions, and the importance of maintaining a healthy weight through diet and exercise. Nurses are essential to improve the patients' quality of life but also improving utilization of health care resources.

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