The Role of Risk Factors in the Early Manifestation of Gout in Young Women

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ABSTRACT

Introduction: Gout is an inflammatory form of arthritis caused by the deposition of monosodium urate crystals in tissues and joints due to hyperuricemia. Although it is more common in middle-aged men, the increasing incidence in young women with genetic predisposition and certain lifestyle factors has become a clinical concern. Early identification and management of risk factors are crucial to prevent disease progression.

Case Illustration: A 33-year-old woman with a family history of gout and dyslipidaemia presented with intermittent joint pain in the lower extremities exacerbated by consumption of high-purine foods. The patient was diagnosed with hyperuricaemia in 2017 but did not undergo regular follow-up. Use of allopurinol and corticosteroids was effective in alleviating symptoms, but the complaints persisted.

Discussion: This case illustrates the significant role of hereditary risk factors, high-purine dietary patterns, and inadequate medical supervision in the onset of early gout. Pathophysiologically, gout begins with chronic hyperuricemia, which then triggers an inflammatory process through uric acid crystallisation. Genetic factors such as the T allele at SNP rs2231142 of the ABCG2 gene are known to impair uric acid excretion through the intestines, increasing the risk of hyperuricemia and early-onset gout. Epidemiological evidence shows a 3.64-fold increased risk of gout in individuals with high genetic risk and unhealthy lifestyles.

Conclusion: The manifestation of gout in young women is the result of a complex interaction between genetic predisposition, environmental factors, and lifestyle. Early detection of risk

factors and lifestyle-based interventions particularly a low-purine diet and monitoring of uric acid levels are key approaches in the prevention and management of gout in the productive age population.

Keywords: early gout, hyperuricemia, young women, ABCG2, genetics, risk factors, purine diet

Introduction

Gout is the most common type of inflammatory arthritis in adults, associated with hyperuricemia and chronic accumulation of monosodium urate crystals. The prevalence of gout is increasing worldwide. The characteristic symptoms of gout are severe pain and swelling around the joints, typically affecting the lower extremities such as the knees, hips, and areas around the toes.[1]

Gout is more commonly seen in middle-aged men, but it can also occur in young women, especially if there are underlying risk factors. A diet high in purines, a family history of gout or dyslipidaemia, inadequate monitoring of uric acid levels, and genetic factors are the primary causes of gout in younger individuals. The importance of early identification and management of these risk factors is crucial in preventing disease progression.[1]

This case report discusses a 33-year-old woman with a family history of gout and a diet high in purines, who exhibited gout manifestations at a young age. The focus of this report is to evaluate the role of risk factors in the development of early-onset gout, as well as the importance of early detection of gout.

Case Illustration

A 33-year-old housewife has been experiencing joint pain in her legs and hips for the past two years, characterised by intermittent, throbbing pain. The pain worsens during strenuous activities and after consuming large amounts of processed foods made from beans and red meat. The symptoms improve when treated with allopurinol and corticosteroids.

In the patient's medical history, she underwent an examination in 2017, which revealed a diagnosis of hyperuricemia and gout. The therapeutic measures administered to the patient were allopurinol and corticosteroids. According to the patient's statement, she has never undergone routine follow-ups or uric acid level tests. No lumps were found on the patient's legs.

In the RPD: There is a history of a fall involving the patient's hip after childbirth, causing pain with rare onset. The hip pain was never examined. In the Family Medical History (RPK), it is known that the patient's father and mother have gout and dyslipidaemia. In the Social History (Rsos), it is known that the patient enjoys consuming processed peanut products. The patient's husband does not have hyperuricaemia or gout. Medication History (RPO): The patient has previously taken allopurinol, simvastatin, and sodium diclofenac. The patient's last uric acid level was measured in 2018, with a value of 7 mg/dl.

The patient has significant risk factors, including a family history of gout, as well as poor lifestyle habits such as a preference for processed peanut products and infrequent routine check-ups. Hyperuricaemia is suspected to be the underlying cause of gout in the patient.

Discussion

Gout is a type of inflammatory arthritis caused by the accumulation of monosodium urate crystals in the joints and body tissues due to high levels of uric acid in the blood. Gout develops when the body produces too much uric acid or the kidneys are unable to excrete it efficiently, causing urate crystals to deposit in the joints and trigger an inflammatory reaction. The global prevalence of gout ranges from 1–4% worldwide, with an incidence rate of 0.1–0.3%. Gout is more common in men than women, with a ratio of 3:1. In Asia, the prevalence of gout varies significantly among countries.[1,2]

A 2017 meta-analysis of 30 studies published between 2000 and 2016 found a combined prevalence of gout in the adult population in China of 1.1%, with prevalence increasing slightly from 1.0% in 2000–2005 to 1.3% in 2010–2016. In South Korea, a study analysing data from the national health claims database for specialised care found that the prevalence of gout increased from 0.35% in 2007 to 0.76% in 2015 across the entire population, and is projected to rise to 1.66% by 2025. In Indonesia itself, based on data from the 2018 Riskerdas survey, the prevalence based on healthcare professional diagnosis reached 7.3%, and based on symptoms reached 24.7%. In South Kalimantan, the prevalence was 9.5%, in West Sumatra 12.7%, and in East Java 26.7%. Gout affects 1–2% of the population, most commonly between the ages of 30 and 40, and is twenty times more common in men than in women. Compared to women, men have a higher risk of developing uric acid metabolism disorders [1,2] .

Pathogenesis The development of gout is divided into several stages, the first being the hyperuricaemia stage, which is the initial basis for the onset of gout. Hyperuricaemia is an increase in uric acid levels in the blood (>6 mg/dl), typically caused by excessive uric acid

production, purine metabolism disorders, and reduced uric acid excretion by the kidneys and intestines. The excess uric acid leads to the formation and deposition of monosodium urate crystals, which attach to the joints. This crystallisation can be triggered by conditions such as low temperature, alkaline pH, and high sodium concentration within the body. Once the crystals form, macrophages activate inflammatory hormones, causing an acute gout attack characterised by severe pain, swelling, and redness. This inflammation is typically temporary because neutrophils that appear during inflammation help neutralise and stop inflammation through cytokine degradation. This inflammatory cycle repeats itself and reduces the patient's quality of life.

The clinical presentation of gout consists of several stages: asymptomatic, acute, intercritical, and chronic.

• Asymptomatic stage

At this stage, there are no specific symptoms, and hyperuricaemia and elevated uric acid levels do not occur alongside tophi, uric acid stones in the urinary tract, or other arthritis symptoms. Enzyme abnormalities may be present from birth. Men experience it after puberty, while women experience it after menopause.

• Acute stage

This stage is characterised by severe and sudden joint pain accompanied by heat and redness. Most acute attacks occur in the big toe and typically happen in the middle of the night or early morning. Attacks occur without warning and peak within a day; after ten days, the pain usually subsides, and uric acid levels are not elevated.

• Intercritical stage

Between two acute attacks: Not everyone who experiences the first attack will have subsequent attacks; most people experience the second attack between six and twelve months, but some may experience it after five to ten years. This depends on the individual. Recurrent attacks are typically longer, more severe, and involve multiple joints.

• Chronic stage

The formation of tophi, which typically appears eleven years after the first attack, marks this stage. This stage occurs when the disease is neglected. Attacks typically occur four to five times a year at this point, often causing prolonged pain, even continuously, accompanied by swelling and stiffness in the joints. Tofus formation is influenced by local factors, blood uric acid levels, and kidney function. Tofus contains uric acid between 10 and 11 mg/dl, but some

exceed 11 mg/dl. Large tofus, resembling cartilage, tendons, fatty tissue, and others, can cause deformities such as protrusions and stiffness in the joints.[4]

The clinical manifestations commonly found in gout patients are severe, sudden joint pain attacks accompanied by swelling, redness, and heat in the joints, most often affecting the big toe joint. In addition to the big toe joint, other joints such as the ankle, knee, wrist, and elbow can also be affected. Furthermore, persistent joint pain, swelling, and the formation of tophi may occur in patients with chronic gout. Gout can also cause limited mobility and joint deformities if left untreated for an extended period of time. [4,5]

The diagnosis of gout in this case is based on the patient's medical history, previous uric acid test results, and the clinical presentation of gout, where the patient's medical history reveals that they occasionally experience a dull pain in the hips and feet (specifically in the joints around the big toe/metatarsophalangeal joint). Although rare, gout can also occur in young women due to poor lifestyle and dietary habits, as well as genetic factors from parents, which are significant contributors to gout onset at a young age. According to a recent study conducted by Hyunjung Kim et al., supported by The Korean Academy of Medical Sciences in Korea in January 2025, there is a gene in humans that can increase the risk of gout, one of which is the ABCG2 gene, specifically the T allele at SNP rs2231142. [5]

This gene influences the risk of gout by increasing the likelihood of developing gout before the age of 35 through a mechanism that affects the progression from hyperuricaemia to gout by causing dysfunction of the ABCG2 transporter, thereby reducing its ability to excrete uric acid through the digestive tract, particularly in the intestines. ABCG2 functions as a urate transporter that excretes uric acid into the intestinal lumen, thereby helping to reduce serum uric acid levels. When ABCG2 function is impaired, uric acid excretion through the intestines decreases, leading to elevated serum uric acid levels/hyperuricemia. As a result, this increases the risk of monosodium urate crystal formation in the joints, triggering inflammation and the progressive of gout. [5-7]

Additionally, it is known that individuals with a high genetic risk for gout who also have a high-purine diet and engage in little physical activity have a 3.64-fold higher risk of gout (odds ratio 3.64) compared to those with a low genetic risk and a healthy lifestyle. This aligns with studies conducted in Vietnam and Europe regarding genetic factors that increase the risk of gout. Both studies also mention that the ABCG2 gene influences the onset of gout, causing it to appear at a younger age. [5-7]

Therefore, it is necessary to intervene in the patient's lifestyle, such as a low-purine diet. This diet is based on the principle of providing food that is appropriate for the patient's needs and condition. The diet is adjusted according to the patient's level of hyperuricaemia, medical condition, and ability to excrete excess uric acid. The goal of the diet is to achieve and maintain optimal nutritional status and reduce uric acid levels in the blood. Additionally, the amount of calories consumed is adjusted based on age, gender, height, weight, and activity level, as these factors influence energy requirements.9 If overweight, energy intake should be gradually reduced by 500 to 1,000 calories, or 10 to 15% of caloric needs. Furthermore, it must be ensured that there is no energy deficiency or body weight below normal before starting a low-purine diet. The presence of ketone bodies can cause an increase in uric acid, but they can also reduce the excretion of uric acid through urine. Dietary recommendations based on the Indonesian Rheumatology Association are as follows:[10.]

Table 1. Dietary Recommendations Based on the Indonesian Rheumatology Association.

Mandatory diet (foods to avoid)	Reduced diet (foods should be reduced)	Recommended diet (foods should be consumed)
Food with high purin(such as processed offal, liver, kidney)	• Beef, lamb, pork • Seafoods with high purin	Low or non-fat dairy product
Soda, corn syrup, and high fructose sweetened foods	• Jus from sweet fruits • Sugar, sugar sweetened beverages and foods • Kitchen salt	Vegetable (except spinach and water spinach)
• Excessive alcohol consumption (more than twice a day for men and more than once a day for women) • Alcohol consumption while gout present or uncontrolled gout		

Here is a list of foods high in purines :

Table 2. List of Foods with High Purines.

No	Food	Purin Level
1	Theobromine(chocolate caffeine)	2300
2	Goat spleen	773
3	Beef liver	554
4	sardines	480
5	wood ear mushroom	448
6	Beef spleen	444
7	Melinjo leaf	434
8	Cow lung	339
9	Spinach and water spinach	290
10	Cow kidney	269
11	Beef heart	256
12	Chicken liver	243
13	Sheep/goat heart	241
14	anchovy	234
15	shrimp	239

Recommended Guidelines for the Diagnosis and management of gout,2020[10]

Conclusions

Early manifestations of gout in young women can occur because of complex interactions between genetic, environmental, and lifestyle factors. Family history of gout and dyslipidaemia, consumption of high-purine foods, and lack of monitoring of uric acid levels are also major contributors to the development of gout. Additionally, genetic factors such as the T allele at the SNP rs2231142 of the ABCG2 gene further strengthen the predisposition to hyperuricaemia and the development of gout at a young age. Therefore, a comprehensive preventive approach, including patient education, lifestyle modifications, and early detection and intervention of gout risk factors in young adults, is crucial in reducing the incidence and progression of gout in the productive age population.

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