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Comparative Study of Dietary, Genetic and Lifestyle Factors in Gouty Arthritis Patients in East Coastal Region of South India

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Abstract

Gouty arthritis is a metabolic disorder marked by elevated uric acid levels and the deposition of urate crystals, often in the first metatarsophalangeal joint. Diet significantly impacts uric acid metabolism, with vegetarian and non-vegetarian diets potentially affecting uric acid levels differently. Additionally, genetic variants and lifestyle factors play important roles in influencing gout severity and associated complications. A cross-sectional study aimed to compare the dietary, genetic and other lifestyle factors that influence gouty arthritis patients. About 82 gouty arthritis patients were included. Serum uric acid levels, genetic variants, and demographic, and clinical data were collected and analyzed. Among non-vegetarian patients, 85% exhibited significantly elevated serum uric acid levels (>10 mg/dL), in contrast to only 15% of vegetarian patients. Over 70% of non-vegetarian individuals had identifiable genetic variants. Furthermore, non-vegetarian patients experienced a higher frequency of acute gout attacks, greater pain severity, and increased tophi formation. The non-vegetarian diet is associated with higher uric acid levels and greater disease severity in patients with gouty arthritis and the Vegetarian diet is beneficial for patients with gouty arthritis, as it is associated with lower serum uric acid levels, fewer acute gout attacks, and reduced medication requirements.

Keywords: *Dietary and Lifestyle Factors, Genetic Variation, Gouty Arthritis, Serum Uric Acid Level.*

Introduction

Gouty arthritis is a common metabolic disorder affecting primarily middle-aged and elderly men, as well as postmenopausal women. This condition is marked by the accumulation of monosodium urate crystals in joints and tissues, causing recurrent acute inflammation [1]. The main cause of gout is hyperuricemia, enabling crystal formation. Uric acid, a byproduct of purine metabolism, is influenced by factors such as genetics, kidney function, and dietary habits [2].

Among the Indian population, 0.5-1.0% of adults could be affected with gouty arthritis. The predominantly male population has a higher prevalence rate with the age of above 40 years. The urban population has having high prevalence or flare-ups of gouty arthritis, which is low in rural populations [3].

Dietary habit of gouty arthritis patients has a significant influence on uric acid metabolism. Purine foods rich, like red meats, seafood, and alcoholic drinks, shall increase uric acid levels and also increase the risk of gout flares. In opposite to that, a low-purine diet especially plant-based foods shall help in the regulation of serum uric acid levels. Hence, Hippocrates, the Father of Medicine has mentioned gout as the "disease of kings". The potential benefits of vegetarian diets in the

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management of gout have been increasingly explored, which gives the association of gout with the intake of low purine-rich animal proteins and high antioxidants and fibre [4].

The role of gene variation which influences the uric acid metabolism, kidney physiology and inflammatory response is also important in gouty arthritis. Research studies in the Indian population, reveal that there is an increased risk among patients with these genetic variants of hyperuricemia and gout. The major genes like SLC2A9 (Glucose Transporter 9) and ABCG2 (ATP-binding cassette subfamily G member 2) were associated firmly with gout arthritis [5]. The gene SLC2A9 shows an increased affinity for uric acid. For efficient urate ion reabsorption, this gene plays a major role in kidney proximal tubules. Any variations in SLC2A9 may result in the efficiency of urate reabsorption, which leads to an increased level of uric acid levels and leads to increased chances for the deposition of urate crystals in the joints. This takes the responsibility for the primary cause of gout flares. In this way, SLC2A9 plays a critical role in the management of gouty arthritis, since it regulates the transport of uric acid in the kidneys. The gene ABCG2 helps in the excretion of uric acid through the renal and intestinal routes. Especially, the Q141K variant (glutamine-to-lysine substitution at position 141) reduces the functions of ABCG2 significantly, leading to an impairment in the excretion of uric acid, and the risk of hyperuricemia for carriers has been increased in two folds [6,7]. This gene in gouty arthritis has the potential as both an important marker for the diagnosis and also in the management as a personalized therapeutic target.

The level of serum uric acid is used as a critical biomarker for diagnosis since it acts as a primary contributor to the onset of the disease and its progression. It is a metabolic waste of purine circulated in the bloodstream to the kidney for excretion shall lead to hyperuricemia, when there is an excess production or not excreted properly from the blood [8]. The level of uric acid has significantly elevated in gouty arthritis patients beyond its threshold. This condition leads to the deposition of monosodium urate crystals in joints and surrounding tissues [9]. This acts as a triggering factor for intense inflammatory responses, causing the painful flare-ups characteristic of gout.

In the normal population, uric acid levels range between 3.5 and 7.2 mg/dL in adults, an increase in this level beyond this range is associated with an increased risk of gout attacks [10]. In gouty arthritis patients, various factors influence the increased uric acid levels, such as genetic predispositions, dietary habits, renal function, and comorbid conditions like obesity, hypertension, and metabolic syndrome [11,12]. Hence, screening and managing the level of serum uric acid levels through dietary adjustments, lifestyle changes, and medications are central strategies in preventing gout flares and controlling disease severity.

In gouty arthritis patients, along with genetics and health, several other factors like lifestyle choices including diet, alcohol intake, body weight, physical activity, and hydration also have a significant role in the production and excretion of uric acid [13,14]. Unrevealing the impact of various dietary patterns on uric acid levels shall provide valuable insights for generating guidelines in the dietary pattern for gouty arthritis patients [15]. Along with regular screening and medication, the modification in lifestyle factors will be very effective in the control of gouty arthritis and its flare-ups [16]. However, studies comparing the effects of vegetarian and non-vegetarian on serum uric acid levels and the severity of patients with gouty arthritis are in demand. Hence, this study aimed to investigate gouty arthritic changes among individuals with vegetarian and non-vegetarian by analyzing biochemical markers, genetic variations and lifestyle influences.

Materials and Methods

A cross-sectional study was conducted in the Department of Biochemistry at VMMCH, Karaikal, to investigate the relationship between diet, genetics, and lifestyle factors in patients with gouty arthritis. Following Institutional Ethical Clearance (VMMC/BIOCHEM/2023/Nov/18), the study took place over six months, from January to June 2024 and involved 82 patients diagnosed with gouty arthritis. The Performa used a standard questionnaire and blood samples from patients were collected after getting informed consent. Data collection was performed through structured interviews and a review of medical records, which included demographic, clinical, and lifestyle information. Laboratory investigation assessed uric acid levels using the serum, while EDTA blood samples were used to isolate whole DNA for genetic analysis to identify polymorphisms in the SLC2A9 and ABCG2 genes.

SNP Genotyping

EDTA blood sample was collected for genomic DNA extraction, using a DNeasy blood and tissue kit (Qiagen, Hilden, Germany) according to the manufacturer's protocols. DNA samples were quantified and purity was checked on Nanodrop (Nabi, Microdigital). A260/230 and A260/A280 values greater than 1.8 were confirmed to ensure the integrity of double-stranded DNA. The PCR-RFLP (polymerase chain reaction-restriction fragment length polymorphism) was done to identify the affected genes. Veritti (Thermocycler) was employed to study the genotype SNPs of ABCG2 (rs72552713 and rs12505410) and SLC22A12 (rs11231825 and rs7932775) as per the standardized protocol given by Nguyen Thuy Duong et al [17]. Primers were designed using Primer3 software (Table No: 01) PCR products were digested with restriction enzymes RsaI, for rs72552713, NsiI for rs12505410, BclI for rs11231825 and Eco130I for rs7932775. The digested PCR products were subjected to electrophoresis using 2.5% agarose gel prepared with 1X TAE buffer (Mupid EX U).

Statistical Analysis

Statistical analysis was performed using SPSS software to compare uric acid levels and gout severity between the two dietary groups. Regression models were also employed to adjust for confounding factors like age, alcohol consumption and BMI. Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables were presented as frequencies and percentages. Independent t-tests were used to compare continuous variables between the two groups, while chi-square tests were used for categorical variables. A p-value of <0.05 was considered statistically significant.

Results

A total of 82 patients who were confirmed with gouty arthritis were included in this study. Most of the patients fall under female with 48 participants and male with 34 participants. The results of various study variables were recorded and analyzed as follows:

Table 1. Primers Details for Target Genes and its SNP [17]

Gene Target	SNP Number	Forward	Reverse	Amplicon Length
ABCG2	rs72552713	AGCTGCAAGGAAAGATCCAA	GGGTAAGTGCTTTGGCTGAT	166 bps
	rs12505410	CCCTTGGCACCTTAAATGAA	ATAGGTGGCTGGCCCTATTT	308 bps

SLC22A12	rs11231825	CCCTAGAGGTCACCAGACCA	ACTGGGCCATGGGCTTCTGATC	168 bps
	rs7932775	GCCTGAAAGTCAGGGACAAG	ACCACACAAGAGGGAGATGC	325 bps

Note: Table 1 shows the specific primers used for the amplification of ABCG2 and SLC22A12 genes, particularly their corresponding SNP number and amplicon length.

Analyzing the demographic and clinical characteristics of gout attacks in vegetarian versus non-vegetarian patients can shed light on the impact of dietary habits on the severity, frequency, and duration of symptoms in gouty arthritis. Although genetic factors primarily influence susceptibility to gout, lifestyle choices—particularly diet—can alter uric acid levels and potentially affect clinical presentations. Below is a comparison of typical clinical observations during gout attacks in vegetarian and non-vegetarian patients:

Table 2. Demographic and Clinical Characteristics of Study Participants

Characteristics	**Vegetarian (n=27)**		**Non-Vegetarian (n=55)**		**t test-value**	**P Value**
	Male	**Female**	**Male**	**Female**		
No of Patients	08	19	26	29		
Age (years)	55.3 ± 10.4	53.3 ± 7.4	49.1 ± 9.8	51.3 ± 8.2	0.404	0.690*
BMI (kg/m²)	24.8 ± 3.1	21.4 ± 2.2	26.2 ± 3.5	25.7 ± 3.8	0.151	0.88
Duration of gout (years)	6.8 ± 1.9	6.5 ± 2.6	8.6 ± 1.5	7.9 ± 2.8	0.220	0.826
Severity of Pain (Mean VAS score)	6±1	5±1	8±1	7±1	2.2	0.031
No of Joints involved (Mode)	Oligoarticular	Oligoarticular	Polyarticular	Polyarticular	-	-
Frequency of Gout Attacks per year (Mode)	3-4	2-3	6-7	5-6	-	-
Tophi Development (Mode)	Moderate	Moderate	Severe	Severe	-	-

Note: Table 2 gives a comparative description of the basic demographic details and basic gouty arthritis complications among the study population with both the vegetarian and Non-Vegetarian diets. Non-vegetarian patients especially males were more susceptible to this gouty arthritis and its complications.

Table 3. Analysis of Genetic Factors Involved in Gouty Arthritis

Gene SNP	**Type**	**Vegetarian (n=27)**	**Non-Vegetarian (n=55)**	**OR**	**95% CI**	***P-value***
rs72552713 Gene frequency						
ABCG2	CC	26 (96.%)	40 (82%)	1.00	1.2137 to 78.3271	0.032
	CT	1 (3.7%)	15 (18%)	9.75		
	TT	Absent in population				
Allele frequency						
	C	53 (98.1%)	95 (86.4%)	1.00	1.0752 to 65.1340	0.042
	T	1 (1.9%)	15 (13.6%)	8.37		
rs12505410	Gene frequency					
ABCG2	TT	11 (40.7%)	18 (32.72%)	1.00		
	TG	13 (48.1%)	25 (45.45%)	1.18	0.4298 to 3.2135	0.75
	GG	3 (11.2%)	12 (21.81%)	2.44	0.5616 to 10.6402	0.233
Allele frequency						
	T	35 (64.8%)	61 (55.45%)	1.00		
	G	19 (35.2%)	49 (44.55%)	1.48	0.7547 to 2.9013	0.254
rs11231825 Gene frequency						
SLC2A9	TT	15 (55.5 %)	34 (61.8%)	1.00		
	TC	9 (33.3%)	20 (36.3%)	0.9804	0.3628 to 2.6490	0.9689
	CC	3 (11.2%)	1 (1.8%)	0.147	0.0141 to 1.5318	0.1089
Allele frequency						
	T	39 (72.2%)	88 (80%)	1.00	0.3049 to 1.3856	0.2646
	C	15 (27.8)	22 (20%)	0.650		
rs7932775 Gene frequency						
SLC2A9	TT	13 (48.1 %)	28 (50.9%)	1.00		
	TC	8 (29.6%)	21 (38.2%)	1.019	0.4183 to 2.9641	0.684
	CC	6 (22.3%)	6 (10.9%)	0.2047	0.2615 to 0.9986	0.261
Allele frequency						
	T	34 (62.9%)	79 (70.5%)	1.00	0.6187 to 0.9865	0.1728
	C	20 (37.1)	33 (29.5%)	0.650		

Note: Table 3 shows the prevalence of mutated alleles of both ABCG2 and SLC22A12 genes among the vegetarian and non-vegetarian diet patients. The frequency of alleles with polymorphism was found in the non-vegetarian diet patients comparatively. The ABCG2 gene with rs12505410 polymorphism was predominantly found in the study population.

Table 4. Mean Serum Uric Acid Levels in Gouty Arthritis Patients

Levels of Serum Uric Acid	Vegetarian (n=27)		Non-Vegetarian (n=55)		Total no of Patients
	Male	Female	Male	Female	
Mildly Elevated (Male<8 mg/dl; Female <7mg/dl)	2 (5.9%)	2 (4.2%)	6 (17.6%)	5 (10.4%)	15 (18.3%)
Moderately Elevated (8.1 -10 mg/dl)	5 (14.7%)	12 (25.0%)	10 (29.4%)	11 (22.9%)	38 (46.3%)
Severely Elevated (>10 mg/dl)	1 (2.9%)	5 (10.4%)	10 (29.4%)	13 (27.1%)	29 (35.4%)
Total no of Patients	8 (23.5%)	19 (39.6%)	26 (76.5%)	29 (60.4%)	82 (100%)
Chi-Square test value		2.085	P value	0.353	

Note: Table 4 shows the level of uric acid among the vegetarian and non-vegetarian diet patients. The non-vegetarian diet patients especially females had higher levels of serum uric acid when compared to vegetarian patients.

Discussion

The gouty arthritis patients following a non-vegetarian diet in this study tend to experience approximately six gout flare-ups per year, particularly among those who regularly consume high-purine foods. For instance, non-vegetarians may have a 30% higher frequency of attacks compared to vegetarians. This finding aligns with the research conducted by Gao X, Zhang Y et al.[18]. Although vegetarian patients with high-risk genetic variants can still experience gout, they may report fewer and less frequent attacks, likely due to the lower purine content found in plant-based diets.

Similarly, the patients with a higher BMI, averaging 25-26 ± 3, were more prevalent among non-vegetarian participants. This aligns with an increased risk of gout due to elevated serum uric acid levels. According to Dai Y et al., most gouty arthritis patients with elevated serum uric acid levels also had higher BMI. Consequently, non-vegetarian patients exhibited a greater prevalence of metabolic syndrome components, such as hypertension and insulin resistance, further increasing their risk for frequent gout attacks [19]. In contrast, vegetarians had a lower average BMI of 21-24 ± 2 and experienced less metabolic comorbidity, which may contribute to a reduced risk of gout, even in the presence of high-risk genetic factors.

Non-vegetarians in our study reported higher pain levels on a visual analogue scale (VAS), with mean scores ranging from 7 to 8 out of 10, and exhibited a greater incidence of severe visible tophi development, especially in patients with prolonged hyperuricemia. In contrast, vegetarians reported milder pain, with a mean score of 5 to 6 on the VAS, and showed moderate tophi development which aligns with Sharma S et al. [20]. The presence of tophi correlated with certain genetic variants, but it was more pronounced among non-vegetarians who consistently consumed purine-rich foods.

Approximately 52% of the non-vegetarian study population reported higher alcohol consumption, particularly beer, which is likely to worsen uric acid levels and increase the frequency and severity of gout flares. In contrast, only 20% of vegetarian patients were found to have lower alcohol intake, which may contribute to their reduced frequency of gout attacks, even among those with high-risk genetic variants. This observation aligns with

findings from studies by Teng GG et al. and Zhang Y et al. [21, 22]. Additionally, regular physical activity and adequate hydration reported by vegetarians may aid in urate excretion, potentially mitigating the risk of gout flares despite genetic predisposition.

The results of the PCR testing in this study were organized for analysis. The study population was in Hardy-Weinberg equilibrium. A significant difference in CT genotype between vegetarian and non-vegetarian patients is observed when compared with the CC genotype (p= 0.032). The mutation is significantly detected in Gouty arthritic patients who consume a non-vegetarian diet, with Odd's ratio, of 9.75. Also, the T allele is associated more with non - vegetarian patients (p=0.042). In contrast, no significant difference is found in ABCG2 (rs12505410) TG and GG genotypes of patients consuming vegetarian and non-vegetarian diets. SLC2A9 (rs11231825) also gives Odd's ratio of less than 1, showing no significant difference between vegetarian and non-vegetarian patients in both genotype and allele frequency.

There is a higher frequency of the ABCG2 rs72552713 genetic variant, with more T alleles associated with Non-Vegetarian patients, with 18% of CT genotype in Non-non-vegetarian patients, when compared to 1% in vegetarian patients. Similar findings regarding genetic variant predisposition in gouty arthritis patients were reported by Kumar P et al. [23]. Although 44% of vegetarians in the study also carried these variants, their lifestyle choices may help mitigate some risks associated with these genetic predispositions. Genetic analysis revealed that patients with specific polymorphisms had higher serum urate levels, regardless of diet. However, non-vegetarians with these variants exhibited significantly higher urate levels and more frequent gout attacks than vegetarians, suggesting that diet plays a role in modulating genetic predisposition. Our study suggests that the genetic variants in ABCG2 are critical factors influencing serum urate levels and, consequently, the likelihood of developing gout. Identifying these genetic markers could facilitate early detection, and personalized treatment, and inform future therapeutic strategies targeting uric acid transport pathways.

On analyzing the serum uric acid levels among the study population, 46.3% were identified as moderately elevated in the range of 8.1 to 10 mg/dl. Among this category, the non-vegetarian population was found to be predominant with 52.3%. Comparing the severely elevated serum uric acid level (>10mg/dl) category with 35.4% of the study population, the non-vegetarians were found to be high with 85%, but it was very low in vegetarians with 15% prevalence. This difference is likely attributed to the high consumption of purine-rich foods (such as red meat, organ meats, and seafood) and alcohol, which increase urate levels and heighten the risk of gout. These findings align with the results of Takahashi K et al., which highlighted how dietary patterns among gout patients, particularly the high intake of purine-rich foods, correlate with elevated uric acid levels [24]. In non-vegetarians with genetic variants that promote high uric acid production, these levels may be especially pronounced, indicating an interaction between genetics and dietary habits. Although vegetarian patients generally had lower mean serum uric acid levels, some who consumed high-purine vegetables or fructose-rich foods could still display moderate urate levels. This suggests that vegetarian diets may help to mitigate the effects of certain high-risk genetic variants on urate levels.

Conclusion

The combination of lifestyle factors and genetic predisposition may increase the risk of gouty arthritis. Non-vegetarians tend to have

higher serum urate levels, more frequent and severe gout attacks, and a higher prevalence of tophi formation compared to vegetarians. Hence, this study suggests that a vegetarian diet may be beneficial for patients with gouty arthritis, as it is associated with lower serum uric acid levels, fewer acute gout attacks, and reduced medication requirements. These findings highlight the potential role of dietary modifications in the management of gout. Hence, adopting a vegetarian diet and lifestyle modification could be a useful adjunct therapy for patients with gouty arthritis, potentially reducing the need for pharmacological interventions. It is also suggested that clinicians develop personalized management strategies by modifying lifestyle factors like lower purine intake, physical activity, and less alcohol consumption. Improving patient education on lifestyle modifications, and a better understanding of gout's relationship with comorbid conditions may also help the patients to avoid flare-ups with gouty attacks. However, detailed studies focusing on Indian genetics and lifestyle factors about gout are still needed.

Limitations

The generalizability of the study findings has been limited by the smaller sample size of eighty-two patients included in this study. More research work on the Indian population-based region is very much essential to validate these results. Since this is an observational study, it could not establish causality. Additionally, investigating the integration of dietary modifications with other biomarkers and clinical parameters could provide a more detailed picture of the prognosis and therapeutic strategy of gouty arthritis patients.

Recommendations for Future Research

Future research should focus on longitudinal studies to establish causality and explore the mechanisms through which vegetarian diets influence uric acid metabolism.

Conflict of Interest

The authors declare that there are no conflicts of interest related to this study.

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