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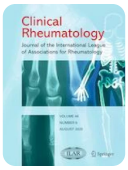

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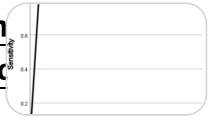

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ORIGINAL ARTICLE

Association of the *ABCG2* Q141K variant with gout in Kinh Vietnamese: a cross-sectional study

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Abstract

Background Gout is a form of microcrystalline arthritis caused by chronic hyperuricemia, leading to monosodium urate crystal deposition. The *ABCG2* gene, particularly the Q141K polymorphism, is a significant genetic factor influencing gout susceptibility and the therapeutic response to allopurinol. However, the association of Q141K with gout in the Vietnamese population remains undefined. This study investigates the relationship between the *ABCG2* Q141K polymorphism and gout susceptibility among Kinh Vietnamese individuals.

Materials and Methods This cross-sectional study includes 468 participants, comprising 234 gout patients and 234 controls. The basic clinical and paraclinical characteristics of all the participants were collected. Genomic DNA was extracted from peripheral blood samples and genotyped for the *ABCG2* Q141K polymorphism using real-time PCR. The association of *ABCG2* Q141K with gout and clinical characteristics was analyzed.

Results The *ABCG2* Q141K polymorphism is significantly associated with gout in dominant, recessive, and additive genetic models. Specifically, the A allele was identified as a risk factor, observed in 46.8% of gout patients compared to 25% of healthy controls.

Conclusion The *ABCG2* Q141K polymorphism significantly increases gout susceptibility among the Kinh Vietnamese population. The high frequency of the A allele in Vietnamese gout patients highlights the potential utility of genetic screening for appropriate preventive strategies.

Key Points
- *The data regarding the contributions of genetic factors in gout of Vietnamese population remain insufficient.*
- *This study showed that the ABCG2 Q141K polymorphism significantly increases gout susceptibility among the Kinh Vietnamese population.*

Keywords *ABCG2* · Gout · Q141K polymorphism · Vietnam

Introduction

Gout is a form of microcrystalline arthritis that results from increased levels of uric acid in the blood. It is characterized by recurring episodes of acute arthritis and the deposition of sodium urate crystals in tissues [1]. Epidemiological studies from numerous countries show that the prevalence and incidence of gout are increasing, and it is estimated that about 41 million people globally suffer from gout, with 7.4 million cases per year [2]. It is also a common form of arthritis that affects about 4% of the population in the United States, 2%–3% in the United Kingdom, and 0.9%–2.5% in Europe [3–5]. The rate increases with age to 9.3% among American adults over the age of 60 [3]. In Vietnam, according to the Community Orientated Program for the Control of

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Rheumatic Diseases (COPCORD), the incidence of gout is about 0.14% in Hanoi, the capital city of Vietnam [6]. The disease is more prevalent in men than women (5.2% and 2.7%, respectively) [3].

Although therapies for gout have been developed, the burden of the disease remains substantial, and its management is still not optimal. Comorbidities that exacerbate the burden of gout and are associated with an increased risk of mortality include hypertension (75%), chronic kidney disease (CKD) (70%), obesity (53%), and cardiovascular disease (CVD) (10% to 14%) [7]. Food contributes only ~ 1% to serum urate change, while genetic polymorphisms are estimated to contribute 23.9% [8]. A study of 419,060 participants of European origin also concluded that diet plays a relatively small role in determining serum urate and hyperuricemia based on population-attributable fractions, while body mass index (BMI) and genetic polymorphisms make a much larger contribution [9].

In 2008, Dehghan et al. conducted a genome-wide association study (GWAS) of uric acid levels and identified an association between *ABCG2* and both hyperuricemia and gout [10]. ABCG2 protein regulates serum uric acid through important physiological roles in urate excretion in both renal and extrarenal areas. ABCG2 dysfunction leads to decreased urate excretion, promoting hyperuricemia and gout [10, 11]. Many reports show several *ABCG2* variants are associated with hyperuricemia and gout, as well as the response to treatment with allopurinol [12, 13]. Among investigated variants, Q141K (rs2231142) is consistently associated with an increased risk of gout [14, 15]. This variant is relatively prevalent in Asia, making its impact on the development of gout substantial [16–18]. However, there is little data on *ABCG2* Q141K and the development of gout in Kinh Vietnamese, who number 80 million people among Vietnam's population. Therefore, this study aims to assess the association of *ABCG2* Q141K and gout in Kinh Vietnamese. These results may help improve the understanding of how genetics affect different populations with gout.

Materials and methods

Participant recruitment

This cross-sectional study was approved by the Ethical Committee of Military Hospital 175 (Approval number 3952/GCN-HĐĐĐ) and conducted from January 2023 to December 2024 at Military Hospital 175, Ho Chi Minh City, Vietnam. A total of 468 subjects participated in this study, consisting of 234 male gout patients and 234 male controls, randomly selected from rheumatology outpatient clinics and annual health check-up programs, respectively. All the participants self-reported that they were Kinh Vietnamese. A structured questionnaire was designed to collect data on anthropometric measurements, clinical data, and comorbidities. Gout patients were diagnosed by certified rheumatologists based on the ACR/EULAR 2015 criteria [19].

Anthropometric measurements and clinical data were systematically recorded. Age was calculated from date of birth to the date of participation in the study. Height was measured while standing barefoot, and weight was measured using a standard calibrated scale. Participants' smoking and alcohol consumption habits were determined using the WHO STEPS Instrument for non-communicable disease risk factor surveillance [20]. Hypertension was defined as patients either currently taking antihypertensive medication or who were newly diagnosed according to the 2020 International Society of Hypertension Global Hypertension Practice Guidelines criteria [21]. Type 2 diabetes mellitus was defined as patients either currently taking any anti-diabetic medication or who were newly diagnosed following the American Diabetes Association diagnosis criteria [22].

Each subject participating in the research study underwent blood sampling for the analysis of biochemical parameters, including urea, creatinine, glucose, aspartate transaminase (AST), alanine aminotransferase (ALT), total cholesterol (CHO), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), uric acid (UA), and C-reactive protein (CRP). These biochemical tests were performed using the endpoint and kinetic spectrophotometric method on the Beckman Coulter AU680 analyzer.

ABCG2 Q141K (rs2231142) genotyping

Peripheral blood samples (2 mL) were collected from participants into EDTA anticoagulant tubes, and genomic DNA was extracted from whole blood using the GeneJET™ Whole Blood Genomic DNA Purification Mini Kit (Thermo Fisher Scientific, Waltham, MA, USA) following the manufacturer's instructions. All DNA samples collected in the study were diluted to a final concentration of 5 ng/µL.

The real-time PCR reaction was prepared using 12.5 µL of TaqMan® Genotyping Master Mix (Thermo Fisher Scientific), 1.25 µL of 20× primers C__15854163_70 (Thermo Fisher Scientific), 2 µL of diluted DNA, and H_2O to reach the final volume of 25 µL. The real-time PCR assay was performed using the QuantStudio™ 5 Real-Time PCR System (Thermo Fisher Scientific) with the following thermal cycling conditions: 60 °C for 30 s and 95 °C for 10 min, followed by 50 cycles of 95 °C for 15 s and 60 °C for 1 min 30 s, and a final step at 60 °C for 30 s. Each PCR run included a negative control to check for contamination and a positive control (previously genotyped by Sanger sequencing) to prevent false-negative results.

Genotype determination of rs2231142 was performed using TaqMan™ MGB probes. The VIC-labeled probe was designed to hybridize specifically to the region carrying the C allele, while the FAM-labeled probe hybridized to the region carrying the A allele. If only VIC fluorescence was detected, the genotype was classified as homozygous CC. If only FAM fluorescence was detected, the genotype was classified as homozygous AA. If both fluorescence signals were detected, the genotype was classified as heterozygous AC. A subset of DNA samples, initially genotyped using the TaqMan probe method, was randomly selected for direct sequencing validation. The target region was amplified by PCR and sequenced using appropriately designed primers. The protocol for direct sequencing was described previously [23–25].

Statistical analysis

Baseline characteristics were summarized for all study participants using mean and standard deviation (SD) or median and interquartile range for continuous variables, with counts and percentages for categorical variables. To compare the differences between individual groups, a two-sample t-test or Wilcoxon rank sum test was applied. The association of rs2231142 and gout was assessed by dominant, recessive, co-dominant, and allele models using Chi-square tests. Odds ratios (OR) and 95% confidence intervals (95% CI) were used to express the association between Q141K and gout. Univariate and multivariate logistic regression were used to determine the clinical/paraclinical factors associated with gout. The results were considered significant when the p-value was lower than or equal to 0.05. All the statistical analyses were performed using SPSS Statistics for Windows version 20.0 (IBM Corporation, Armonk, NY, USA).

Results

The baseline characteristics of the participants are described in Table 1. The subjects' median age was 45, median BMI was 23.9, and all were male. Gout patients have significantly higher BMI and creatinine, CHO, TG, LDL-C, UA, and CRP levels than the controls. Gout patients also have higher rates of comorbidities, such as type 2 diabetes mellitus and hypertension. The distribution of the *ABCG2* Q141K genotype and allele is significantly different between the gout and control groups. Genetic association analysis shows that Q141K is statistically associated with gout in co-dominant, dominant, recessive, and allele models (Table 2). Interestingly, the AA genotype shows an *OR* = 6.85 (3.68–13.50) compared to the CC genotype for gout development.

Univariate logistic regression shows that BMI, creatinine, CHO, TG, LDL-C, CRP, alcohol use, and *ABCG2* Q141K are statistically associated with gout in the studied population (Table 3). After adjusting for confounding factors, including BMI, creatinine, TG, LDL-C, HDL-C, CRP, and alcohol use, by multivariate logistic regression, the AA genotype remains a significant independent marker for increased gout risk in recessive and co-dominant multivariate models with OR = 3.273 and 4.281, respectively (Table 4).

Discussion

The traditional risk factors for gout include a purine-rich diet, smoking, alcohol consumption, and comorbidities such as obesity, hypertension, and diabetes mellitus [26–29]. Our study aligns with previous research, demonstrating strong associations between these clinical factors and gout. Gout patients in this study had higher BMI values and consumed alcohol, and a significant proportion of gout patients had type 2 diabetes mellitus or hypertension. These results emphasize the colossal burden of gout and its non-communicable comorbidities.

The *ABCG2* Q141K variant results in the substitution of glutamine by lysine at position 141 of the ABCG2 protein. The Q141K variant is prevalent in different populations, with an estimated frequency ranging from 15 to 30% among ethnic groups [12, 30]. The *ABCG2* Q141K variant is an important factor influencing susceptibility to gout in numerous populations, such as Japanese, Korean, and Han Chinese [14, 17, 18, 30]. However, the impact of the Q141K variant in the Vietnamese population has not previously been extensively studied, underscoring the importance of understanding genetic predispositions that are specific to this population. In fact, the Vietnamese population shows a distinct genetic association with complex non-communicable disorders such as cardiovascular diseases and type 2 diabetes mellitus [31–35].

ABCG2 is a key uric acid transporter and has been extensively studied for its role in serum uric acid levels and gout pathogenesis. Recent genome-wide association studies (GWAS) have identified several genes associated with elevated uric acid levels and gout [10, 36, 37]. In 2016, Matsuo et al. conducted a GWAS study on gout patients and controls with normal levels of UA, identifying several genes linked to gout [37]. Among these, the Q141K polymorphism has received significant attention due to its impact on the function of ABCG2, which affects drug metabolism and uric acid homeostasis [16]. Our study identified a significant association between the Q141K polymorphism and gout in the dominant, recessive, and co-dominant genetic models. Additionally, allelic analysis of Q141K confirmed a significant association with gout, where the A allele is linked to an increased risk of gout, with a frequency of 46.8% in gout cases compared to 25% in controls.

Table 1 Baseline characteristics of the participants

Characteristics	Total $N=468$	Gout $N=234$	Control $N=234$	P-value
Age (years) Median (Q1-Q3)	45 (37–52)	44 (35–54)	46 (38–52)	0.97
Height (cm) Median (Q1-Q3)	167 (163–170)	165 (162–170)	168 (165–170)	<0.001
Weight (kg) Median (Q1-Q3)	66 (61–72)	68 (63–75)	64 (60–69)	<0.001
BMI (kg/m^2) Median (Q1-Q3)	23.9 (22.5–25.6)	24.7 (23.4–26.7)	23.0 (21.4–24.2)	**<0.001**
Ure (mmol/L) Median (Q1-Q3)	5.1 (4.4–6.0)	5.0 (4.3–6.0)	5.1 (4.4–6.0)	0.34
Glucose (mmol/L) Median (Q1-Q3)	4.7 (4.3–5.2)	5.2 (4.6- 6.0)	4.5 (4.3–4.8)	**<0.001**
Creatinine (μmol/L) Median (Q1-Q3)	94.6 (87.2–103.1)	98.2 (89.3–109.0)	92.4 (85.2–98.0)	**<0.001**
AST (U/L) Median (Q1-Q3)	26.0 (21.8–32.3)	27.8 (22.1–36.8)	25.3 (21.4–29.7)	**<0.001**
ALT (U/L) Median (Q1-Q3)	26.8 (19.3–38.9)	33.7 (23.9–55.2)	22.7 (16.8–29.0)	**<0.001**
CHO (mmol/L) Median (Q1-Q3)	5.0 (4.5- 5.7)	5.3 (4.4–6.2)	4.9 (4.5–5.2)	**<0.001**
TG (mmol/L) Median (Q1-Q3)	1.6 (1.1 −2.5)	2.3 (1.7–3.8)	1.2 (0.9–1.5)	**<0.001**
HDL-C (mmol/L) Median (Q1-Q3)	1.2 (1.0–1.4)	1.1 (1.0–1.2)	1.2 (1.1–1.4)	**<0.001**
LDL-C (mmol/L) Median (Q1-Q3)	3.1 (2.7–3.6)	3.4 (2.7–3.9)	3.0 (2.7–3.3)	**<0.001**
UA (mg/dL) Median (Q1-Q3)	6.8 (5.8–8.6)	8.6 (7.4–9.8)	6.0 (5.3–6.6)	**<0.001**
CRP (mg/dL) Median (Q1-Q3)	1.4 (0.6–3.9)	3.5 (1.6–10.9)	0.7 (0.4–1.3)	**<0.001**
Hypertension *N* (%)	85 (18.2)	85 (36.3)	0 (0.0)	**<0.001**
Type 2 diabetes mellitus N (%)	41 (8.8)	41 (17.5)	0 (0.0)	**<0.001**
Smokers *N* (%)	111 (23.7)	63 (26.9)	48 (20.5)	0.13
Alcohol use *N* (%)	174 (37.2)	103 (44.0)	71 (30.3)	**0.003**
***ABCG2* Q141K genotype** *N* (%)				
A/A	68 (14.6)	53 (22.6)	15 (6.4)	**<0.001**
A/C	200 (42.7)	113 (48.3)	87 (37.2)	
C/C	200 (42.7)	68 (29.1)	132 (56.4)	
A	336 (35.9)	219 (46.8)	117 (25.0)	**<0.001**
C	600 (64.1)	249 (53.2)	351 (75.0)	

ALT alanine aminotransferase, *AST* aspartate aminotransferase, *BMI* body mass index, *CHO* total cholesterol, *CRP* C-reactive protein, *HDL-C* high-density lipoprotein cholesterol, *LDL-C* low-density lipoprotein cholesterol, *TG* triglyceride, *UA* uric acid

Our findings are consistent with those from the meta-analysis by Lukkunaprasit et al. who reported that the AA genotype of Q141K confers a higher risk of gout compared to the CC genotype ($OR=4.53$, 95% CI: 4.10–5.00), while the AC genotype compared to CC has an OR of 2.10 (95% CI: 1.95–2.26) [38]. Similarly, in Caucasian populations, rs2231142 is associated with an elevated risk of gout, with a pooled OR of 3.24 (95% CI: 2.39–4.41) for AA vs. CC, and 1.64 (95% CI: 1.47–1.82) for AC vs. CC [38]. Similarly, Chen et al. showed a significant association between Q141K and gout ($OR=4.34$), hyperuricemia ($OR=3.37$), and hyperuricemia vs. controls ($OR=2.15$), all with a p-value < 0.001 [36].

Table 2 The association between *ABCG2* Q141K and gout

	N (%)		OR (95% CI)	*P*-value
	Gout *N* = 234	Control *N* = 234		
Dominant model				
AA + AC	166 (70.9)	102 (43.6)	3.16 (2.16–4.65) 1.0	**< 0.001**
CC	68 (29.1)	132 (56.4)		
Recessive model				
AA	53 (22.6)	15 (6.4)	4.28 (2.39–8.09) 1.0	**< 0.001**
AC + CC	181 (77.4)	219 (93.6)		
Co-dominant model				
AA	53 (22.6)	15 (6.4)	6.85 (3.68–13.50) 2.52 (1.69–3.79) 1.0	**< 0.001**
AC	113 (48.3)	87 (37.2)		**< 0.001**
CC	68 (29.1)	132 (56.4)		
Allele model				
A	219 (46.8)	117 (25.0)	2.59 (1.95–3.47)	**< 0.001**
C	249 (53.2)	351 (75.0)		

Table 3 Univariate analysis of factors associated with gout

Factors	OR (95% CI)	*P*-value
BMI	1.535 (1.387–1.700)	< 0.001
Creatinine	1.051 (1.033–1.068)	< 0.001
CHO	1.611 (1.327–1.957)	< 0.001
TG	5.189 (3.636–7.405)	< 0.001
HDL-C	0.089 (0.039–0.201)	< 0.001
LDL-C	2.012 (1.514–2.674)	< 0.001
CRP	2.084 (1.736–2.503)	< 0.001
Alcohol use	1.805 (1.235–2.639)	0.002
ABCG2 Q141K		
CC genotype	1	< 0.001
AC genotype	2.521 (1.682–3.779)	< 0.001
AA genotype	6.859 (3.604–13.504)	

BMI body mass index, *CHO* total cholesterol, *CRP* C-reactive protein, *HDL-C* high-density lipoprotein cholesterol, *LDL-C* low-density lipoprotein cholesterol, *TG* triglyceride

Table 4 Multivariate analysis of factors associated with gout in different models

Model 1: Dominant	**Factor**	**OR (95% CI)**	***P*-value**
	BMI	1.425 (1.233–1.645)	< 0.001
	Alcohol use	2.168 (1.158–4.058)	0.016
	CRP	1.671 (1.372–1.985)	< 0.001
	LDL-C	1.036 (0.659–1.603)	0.878
	HDL-C	1.008 (0.284–3.574)	0.990
	TG	3.327 (2.189–5.508)	< 0.001
	Creatinine	1.061 (1.032–1.092)	< 0.001
	CC genotype	0.451 (0.246–0.825)	0.010
Model 2: Recessive	**Factor**	**OR (95% CI)**	***P*-value**
	BMI	1.423 (1.231–1.645)	< 0.001
	Alcohol use	2.007 (1.072–3.756)	0.029
	CRP	1.626 (1.351–1.956)	< 0.001
	LDL-C	1.040 (0.658–1.646)	0.865
	HDL-C	1.014 (0.281–3.654)	0.984
	TG	3.469 (2.270–5.302)	< 0.001
	Creatinine	1.061 (1.031–1.091)	< 0.001
	AA genotype	3.273 (1.324–8.093)	0.010
Model 3: Co-dominant	**Factor**	**OR (95% CI)**	***P*-value**
	BMI	1.424 (1.231–1.648)	< 0.001
	Alcohol use	2.061 (1.094–3.883)	0.025
	CRP	1.625 (1.349–1.956)	< 0.001
	LDL-C	1.032 (0.654–1.631)	0.891
	HDL-C	1.068 (0.295–3.866)	0.920
	TG	3.465 (2.253–5.330)	< 0.001
	Creatinine	1.060 (1.031–1.091)	< 0.001
	CA genotype	1.810 (0.945–3.464)	0.073
	AA genotype	4.281 (1.642–11.163)	0.003

BMI body mass index, *CRP* C-reactive protein, *HDL-C* high-density lipoprotein cholesterol, *LDL-C* low-density lipoprotein cholesterol, *TG* triglyceride

Univariate regression analysis revealed that elevated levels of creatinine, CHO, TG, LDL-C, and CRP are associated with an increased risk of developing gout. These findings are consistent with previous studies emphasizing the impact of impaired renal function and dyslipidemia on the pathogenesis of gout [39–41]. Renal dysfunction, reflected in decreased eGFR, results in reduced uric acid excretion, thereby contributing to the development of gout [42]. Conversely, higher eGFR and HDL-C levels were found to be protective factors, reducing the risk of gout through improved renal uric acid clearance and anti-inflammatory effects [42, 43]. Lipid metabolism disorders, characterized by low HDL-C levels, high TG, and high LDL-C levels, promote inflammation and may also contribute to gout [41, 44, 45].

Multivariate analysis identified creatinine, TG, LDL-C, and CRP as independent risk factors associated with gout. Furthermore, the analysis highlighted a significant association between Q141K polymorphism and gout susceptibility. The AA genotype exhibits a stronger correlation with gout predisposition than the AC and CC genotypes. This finding is in line with previous research indicating that mutations impairing ABCG2 function can lead to increased serum uric acid levels, thereby elevating gout risk [16].

Our study has certain limitations, notably the inclusion of only male participants, which may limit the generalizability of the findings to women or other demographic groups. Future studies should address these limitations and explore

gene-environment interactions across more diverse populations to improve prevention and treatment strategies.

Conclusion

Our study confirms the significant role of *ABCG2* Q141K polymorphism and clinical factors associated with gout risk in the Vietnamese population. Identifying these associated factors lays the foundation for improved strategies for clinical management and gout prevention, highlighting the need for a comprehensive approach that integrates patient screening for both metabolic and genetic factors. Understanding the genetic contribution to gout also helps to improve therapeutic strategies using an individualized approach.

Author Contributions TM, KPN, MD, and KDN designed the research study. KPN recruited the participants for the study. LL performed the genotyping. KPN, TM, and MD analyzed the data. KPN, TM, and MD wrote the manuscript. All the authors have read and approved the manuscript.

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Data Availability Data supporting the findings of this study are available from the corresponding author [TM] on request.

Declarations

Ethical approval This study was approved by the Ethical Committee of Military Hospital 175 (Approval number 3952/GCN-HĐĐĐ).

Conflict of interest The authors of this work have nothing to disclose.

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