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Association of the Glu34Lys Polymorphism of the SERPINA1 Gene with the Severity of Liver Fibrosis in Patients with Non-Alcoholic Fatty Liver Disease

Tursunov Mahmud Muhammadovich
Department of Family Medicine and Internal Medicine, Central Asian Medical University, Fergana, Uzbekistan

Musashaykhova Shahnoza Mamirbekovna
Department of Family Medicine Training, Andijan State Medical Institute, Andijan, Uzbekistan

Salahiddinov Zuhriddin Salahiddinovich
Department of Family Medicine Training, Andijan State Medical Institute, Andijan, Uzbekistan

Musashayxov Umidjon Xusanboyevich
Department of Propedeutics of Internal Medicine, Andijan State Medical Institute, Andijan, Uzbekistan

Abstract

Objective: To evaluate the association of the Glu34Lys polymorphism of the SERPINA1 gene with the development and severity of liver fibrosis in patients with non-alcoholic fatty liver disease (NAFLD).

Materials and Methods: This case-control study was conducted between 2024 and 2026 and initially included 208 participants: 106 patients with NAFLD and 102 individuals in the comparison group. The primary genetic analysis included 49 patients with NAFLD and liver fibrosis and 102 conditionally healthy controls. Liver fibrosis was assessed by transient elastography using FibroScan and classified according to the METAVIR system. The Glu34Lys polymorphism of the SERPINA1 gene was investigated using polymerase chain reaction (PCR)-based molecular genetic methods. Associations were estimated using odds ratios (ORs) with 95% confidence intervals (CIs); $p < 0.05$ was considered statistically significant.

Results: The minor Lys allele was detected in 5.1% of patients with NAFLD and liver fibrosis and in 1.5% of controls. Lys allele carriage showed a trend toward higher odds of liver fibrosis (OR = 3.6; 95% CI, 0.92-14.11; p = 0.20). The heterozygous Glu/Lys genotype was present in 10.2% of patients with fibrosis and 2.9% of controls and likewise showed a trend toward higher odds of fibrosis (OR = 3.8; 95% CI, 0.94-15.04; p

= 0.20). Liver stiffness was significantly greater in Glu/Lys carriers than in Glu/Glu carriers (12.2 ± 0.1 vs 8.6 ± 0.3 kPa, respectively; $p < 0.05$).

Conclusion: The Glu34Lys polymorphism of the SERPINA1 gene, particularly the Glu/Lys genotype, may be associated with greater severity of liver fibrosis in patients with NAFLD. Although allele and genotype frequency differences did not reach statistical significance, the association of Glu/Lys with increased liver stiffness suggests that this variant may represent a potential molecular genetic marker of fibrosis severity. Larger independent cohorts are required to confirm these findings.

Keywords: Non-alcoholic fatty liver disease; NAFLD; SERPINA1; Glu34Lys; Genetic polymorphism; Liver fibrosis; Liver stiffness; Transient elastography; Fibro Scan.

Introduction

Non-alcoholic fatty liver disease (NAFLD) is one of the most prevalent forms of chronic liver disease and may progress from simple steatosis to steatohepatitis, progressive fibrosis, cirrhosis, and hepatocellular carcinoma. Long-term follow-up studies have shown that fibrosis progression in NAFLD is closely associated with clinical outcomes, and advanced fibrosis is an important predictor of both all-cause and liver-related mortality [1,2]. Multicentre transcriptomic studies have also demonstrated disease-specific molecular changes across the spectrum of fibrosis progression [3]. Accordingly, early identification of fibrosis and assessment of individual factors that contribute to its progression are clinically important.

Molecular genetic determinants are increasingly recognised as modifiers of NAFLD susceptibility and severity. The PNPLA3 rs738409 variant is associated with hepatic fat accumulation and inflammatory injury [4], while histology-based studies have identified associations between genetic variants and fibrosis-related phenotypes [5]. Carriage of TM6SF2 rs58542926 has been linked to advanced fibrosis and cirrhosis [6], and the MBOAT7 rs641738 variant has been associated with NAFLD severity and fibrogenesis [7]. In contrast, a functional HSD17B13 variant has been associated with protection against steatohepatitis and severe chronic liver disease [8]. Polymorphisms in PNPLA3, IL28B, PPAR, and other genes have also been investigated in biopsy-proven NAFLD cohorts [9], whereas reduced MBOAT7 function has been linked to profibrotic mechanisms in human and experimental studies [10]. Genetic risk has additionally been associated with liver stiffness measured by magnetic resonance elastography [11]. These findings support further investigation of individual genetic determinants of fibrosis in NAFLD, including variants of SERPINA1.

Study aims. To evaluate the association of the Glu34Lys polymorphism of the SERPINA1 gene with the development and severity of liver fibrosis in patients with NAFLD.

Study objectives:

1. To determine the distribution of alleles and genotypes of the SERPINA1 Glu34Lys polymorphic locus in patients with NAFLD and liver fibrosis.
2. To analyse the association between Glu34Lys polymorphism variants and the likelihood of liver fibrosis.
3. To assess the association between SERPINA1 genotypes and liver stiffness measurements.

Materials and Methods

Study Design and Participants

This case-control study was conducted from 2024 to 2026 among patients admitted to the Departments of Internal Medicine and Gastroenterology of Fergana City Hospital No. 1.

A total of 208 individuals were initially enrolled. The main group consisted of 106 patients diagnosed with NAFLD. Their mean age was 45.7 ± 1.5 years; 51 (48.2%) were men and 55 (51.8%) were women.

Patients with NAFLD were divided into two subgroups according to the presence of liver fibrosis:

1. Subgroup 1: 49 patients with NAFLD and liver fibrosis.
2. Subgroup 2: 57 patients with NAFLD but without detectable fibrosis (F0).

Among the 49 patients with fibrosis, stages were distributed as follows: F1, 14 patients (28.6%); F2, 15 patients (30.6%); and F3, 20 patients (40.8%).

The comparison group comprised 102 conditionally healthy individuals without complaints or medical history suggestive of liver disease and without evidence of NAFLD on examination. The group included 54 men and 48 women, with a mean age of 49.2 ± 4.2 years. For molecular genetic analysis, genomic DNA samples stored in the DNA bank of the Republican Specialized Scientific and Practical Medical Center of Hematology, Ministry of Health of the Republic of Uzbekistan, were used.

For the primary comparative genetic analysis, 49

patients with NAFLD and liver fibrosis were compared with 102 conditionally healthy controls.

Clinical and Anthropometric Assessment

All patients underwent routine clinical examination. Anthropometric assessment included body weight, height, waist circumference, and body mass index.

Clinical features of NAFLD were evaluated on the basis of general clinical status, presenting signs and symptoms, and standard instrumental examination findings.

Instrumental Assessment

- all patients underwent transabdominal ultrasonography of the liver, gallbladder, and extrahepatic bile ducts.
- the degree of hepatic parenchymal fibrosis was assessed by elastography using a FibroScan 530 Compact device with a Probe XL (SN: 92266)-G3.2 transducer.
- liver stiffness was measured in kilopascals (kPa). Fibrosis stages were classified according to the METAVIR system as F0, F1, F2, and F3.
- to assess the clinical relevance of the SERPINA1 polymorphism, genotype variants were analysed in relation to liver stiffness values obtained by FibroScan.

Biochemical Assessment

Venous blood was analysed for lipid profile, liver enzymes, coagulation parameters, and glycaemia to characterise clinical and metabolic features associated with NAFLD and its progression.

Biochemical analyses were performed using a CYANSmart CY009 automated biochemical analyser.

Molecular Genetic Analysis

The molecular genetic analysis focused on the Glu34Lys polymorphic locus of the SERPINA1 gene.

Biological material was collected according to standard procedures, with respect for participants' rights, after provision of study information and acquisition of voluntary informed consent.

Blood samples were collected into standard ethylenediaminetetraacetic acid (EDTA)-containing Vacutainer tubes (Becton Dickinson International, USA).

Polymerase chain reaction (PCR) was performed according to the relevant amplification protocols using the following thermocyclers:

- Applied Biosystems 2720 (USA);
- CG1-96, Corbett Research/QIAGEN (Germany);
- Rotor-Gene Q, QIAGEN (Germany).

For the SERPINA1 Glu34Lys polymorphism, the frequencies of the Glu and Lys alleles and the Glu/Glu and Glu/Lys genotypes were analysed. Associations of genotype and allele variants with liver fibrosis in NAFLD were evaluated by comparison with the control group.

In addition, the relationship between SERPINA1 genotypes and liver stiffness was assessed using FibroScan measurements.

Statistical Analysis

Statistical analyses were performed using Microsoft Office Excel and StatSoft Statistica 10.0. Continuous variables were expressed as mean ± standard error. Between-group differences in quantitative variables were assessed using Student's t-test.

The chi-square (χ^2) test was used to compare the distribution of genetic variants. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated to estimate the association of genotypes and alleles with liver fibrosis.

A two-sided p value < 0.05 was considered statistically significant. Accordingly, genetic associations with $p \geq 0.05$ were interpreted as trends rather than statistically established risk factors.

Deviation from Hardy-Weinberg equilibrium was assessed using GenePop (Genetics of Population).

Results

Comparative analysis of the SERPINA1 Glu34Lys polymorphism in patients with NAFLD and liver fibrosis and in controls demonstrated differences in the distribution of alleles and genotypes.

The major Glu allele predominated in both groups, with a frequency of 94.9% among patients with NAFLD and fibrosis and 98.5% among controls. The minor Lys allele was detected in 5.1% and 1.5% of alleles, respectively. Although the Lys allele was more frequent among patients with fibrosis, the between-group difference was not statistically significant. Lys allele carriage nevertheless showed a trend toward higher odds of liver fibrosis ($\chi^2 = 3.4$; $p = 0.20$; OR = 3.6; 95% CI, 0.92-14.11).

Table 1. Distribution of alleles and genotypes of the SERPINA1 Glu34Lys polymorphism in patients with NAFLD and liver fibrosis and in controls

Allele/genotype	AFLD + fibrosis, n=49	ntrol group, n=102	χ^2	p	OR	95% CI
Glu	93 (94.9%)	201 (98.5%)	3.4	0.20	0.3	0.07-1.09
Lys	5 (5.1%)	3 (1.5%)	3.4	0.20	3.6	0.92-14.11
Glu/Glu	44 (89.8%)	99 (97.1%)	3.5	0.20	0.3	0.07-1.07
Glu/Lys	5 (10.2%)	3 (2.9%)	3.5	0.20	3.8	0.94-15.04

Note: OR, odds ratio; CI, confidence interval. Differences were considered statistically significant at $p < 0.05$.

Table 1. Distribution of alleles and genotypes of the SERPINA1 Glu34Lys polymorphism in patients with NAFLD and liver fibrosis and in controls. Note: OR, odds ratio; CI, confidence interval; NAFLD, non-alcoholic fatty liver disease.

As shown in Table 1, the homozygous Glu/Glu genotype was present in 89.8% of patients with NAFLD and liver fibrosis and in 97.1% of controls. This difference was not statistically significant ($\chi^2 = 3.5$; $p = 0.20$; OR = 0.3; 95% CI, 0.07-1.07).

The heterozygous Glu/Lys genotype was identified in 10.2% of patients with NAFLD and fibrosis and in 2.9% of controls. This genotype showed a trend toward higher odds of liver fibrosis ($\chi^2 = 3.5$; $p = 0.20$; OR = 3.8; 95% CI, 0.94-15.04). Given the p value and the 95% CI crossing the null value, this finding should be interpreted as a trend toward association rather than as an independently established risk factor.

To explore a potential relationship between the SERPINA1 Glu34Lys polymorphism and fibrosis severity, liver stiffness measurements were compared by genotype. Mean liver stiffness was 12.2 ± 0.1 kPa in Glu/Lys carriers and 8.6 ± 0.3 kPa in Glu/Glu carriers, representing a statistically significant difference ($p < 0.05$; Figure 1).

Figure 1. Liver stiffness according to SERPINA1 Glu34Lys genotype

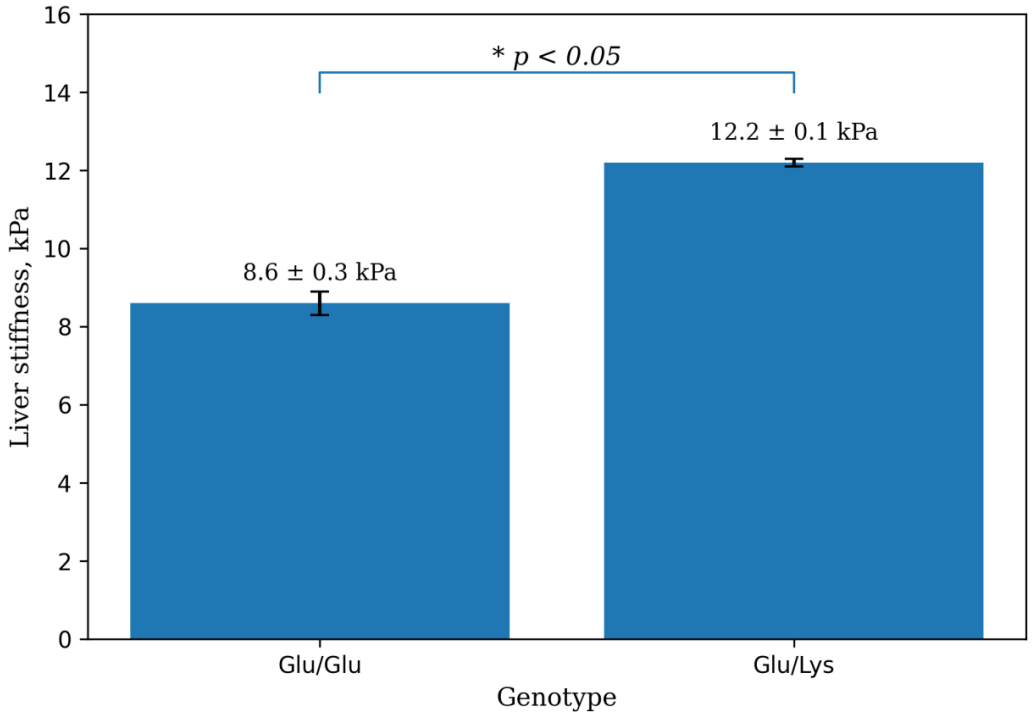


Figure 1. Liver stiffness according to SERPINA1 Glu34Lys genotype. Mean liver stiffness was 8.6 ± 0.3 kPa in Glu/Glu carriers and 12.2 ± 0.1 kPa in Glu/Lys carriers; *p < 0.05. Error values are reported as presented in the manuscript.

As illustrated in Figure 1, despite the absence of statistically significant differences in allele and genotype frequencies between groups, carriers of the minor Lys allele and the Glu/Lys genotype showed a pronounced trend toward greater likelihood of liver fibrosis. The significant difference in elastography measurements suggests a possible association between the Glu/Lys genotype and greater fibrosis severity in patients with NAFLD.

Taken together, the SERPINA1 Glu34Lys polymorphism, particularly the Glu/Lys genotype, may contribute to interindividual variation in the severity of the fibrotic process in NAFLD.

Discussion. The present findings suggest a possible association between the investigated SERPINA1 variant and the severity of liver fibrosis in NAFLD. In this cohort, the minor Lys allele and the heterozygous Glu/Lys genotype were more frequent among patients with NAFLD and fibrosis than among controls; however, the differences in allele and genotype frequencies were not statistically significant. The corresponding OR estimates should therefore be interpreted as signals of a possible association rather than evidence of an independent risk factor.

These observations should be considered in the context of evidence on other clinically relevant SERPINA1 variants. In a multicentre cohort of patients with biopsy-proven NAFLD, carriage of the Pi*Z variant was substantially more frequent among patients with cirrhosis than among those without fibrosis and was associated with increased cirrhosis risk [12]. This supports the broader concept that specific SERPINA1 variants may modify the severity of chronic liver injury.

Prospective phenotyping of adults with the Pi*ZZ genotype demonstrated a high prevalence of liver fibrosis and metabolic abnormalities, with severity influenced by additional metabolic factors [13]. In the UK Biobank, heterozygous Pi*Z carriage was likewise associated with increased cirrhosis risk, and the genetic effect was modified by interaction with other risk factors [14].

A large study of adults with alpha-1 antitrypsin deficiency showed that both homozygous and selected heterozygous SERPINA1 variants were associated with hepatobiliary abnormalities and fibrosis [15]. However, findings are not entirely consistent across populations: in a Lithuanian cohort, Pi*Z was not associated overall with fibrosis or cirrhosis, whereas Pi*S was associated with higher fibrosis risk [16]. This heterogeneity highlights the potential influence of ethnicity, sample size, and coexisting metabolic factors.

Observations in adults with Pi*ZZ-associated lung disease have also shown that advanced liver fibrosis may occur in the presence of conventional risk factors and may warrant targeted screening [17]. The relationship between alpha-1 antitrypsin deficiency and chronic liver disease in adults was first described in early clinical studies [18], while contemporary genetic analyses support pleiotropic effects of SERPINA1 and other loci on cirrhosis susceptibility [19].

Genetic effects in NAFLD may be deleterious or protective. For example, the HSD17B13 rs72613567:TA variant has been associated with a lower frequency of fibrosis and reduced expression of inflammatory genes in the liver [20]. These data emphasise that the effect of a single locus should be interpreted within the broader context of polygenic susceptibility and the patient's metabolic phenotype.

The most robust finding of the present study was the association between the Glu/Lys genotype and greater liver stiffness (12.2 ± 0.1 vs 8.6 ± 0.3 kPa for Glu/Glu; $p < 0.05$). The clinical relevance of liver stiffness assessment is supported by a prospective study in which FibroScan demonstrated good diagnostic performance for fibrosis staging in patients with NAFLD when compared with liver biopsy [21].

Other original studies also support quantitative assessment of liver stiffness. Magnetic resonance elastography has demonstrated high accuracy for fibrosis detection in biopsy-proven NAFLD

[22], while transient elastography has shown significant correlation with histological fibrosis stage [23]. Combining FibroScan with non-invasive indices such as the fibrosis-4 index (FIB-4) or the NAFLD fibrosis score may further improve detection of advanced fibrosis [24].

Overall, the present data should be regarded as preliminary evidence that the investigated SERPINA1 variant may modify fibrosis severity in NAFLD. The higher liver stiffness observed in Glu/Lys carriers supports the biological plausibility of this hypothesis; however, the lack of statistically significant differences in allele and genotype frequencies, the small number of minor-allele carriers, and the wide confidence intervals require confirmation in larger independent cohorts. In addition, the nomenclature of the investigated SERPINA1 variant should be standardised against the genotyping assay and reference sequence to ensure direct comparability with international studies.

Conclusion. The SERPINA1 Glu34Lys polymorphism

may be associated with the development and severity of liver fibrosis in patients with NAFLD.

The minor Lys allele and the heterozygous Glu/Lys genotype were more frequent among patients with NAFLD and fibrosis than among controls. Lys allele carriage showed a 3.6-fold increase in the estimated odds of fibrosis, while the Glu/Lys genotype showed a 3.8-fold increase; however, neither association reached statistical significance. These estimates should therefore not be interpreted as independently proven risk effects.

The principal statistically significant finding was the association between the Glu/Lys genotype and greater liver stiffness. Liver stiffness was 12.2 ± 0.1 kPa in Glu/Lys carriers versus 8.6 ± 0.3 kPa in Glu/Glu carriers ($p < 0.05$).

These findings support consideration of the SERPINA1 Glu34Lys polymorphism, particularly the Glu/Lys genotype, as a potential molecular genetic marker of greater fibrosis severity in NAFLD, while underscoring the need for validation in larger independent populations.

Conclusions

1. The minor Lys allele of the SERPINA1 Glu34Lys polymorphism was more frequent in patients with NAFLD and liver fibrosis than in controls (5.1% vs 1.5%), with a trend toward higher odds of fibrosis (OR = 3.6; 95% CI, 0.92-14.11; p = 0.20).

2. The heterozygous Glu/Lys genotype was present in 10.2% of patients with NAFLD and fibrosis compared with 2.9% of controls and showed a trend toward higher odds of fibrosis (OR = 3.8; 95% CI, 0.94-15.04; p = 0.20).

3. Liver stiffness was significantly greater in Glu/Lys carriers than in Glu/Glu carriers (12.2 ± 0.1 vs 8.6 ± 0.3 kPa; $p < 0.05$).

4. The SERPINA1 Glu34Lys polymorphism may be considered a potential genetic marker of fibrosis severity in NAFLD; however, its prognostic significance requires cautious interpretation because allele and genotype frequency differences were not statistically significant.

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Conflict of interest statement

The authors declare that they have no conflicts of interest.

Author contributions

Tursunov Mahmud Muhammadovich: Conceptualization, study design, clinical interpretation of liver fibrosis findings, supervision of the research, and critical revision of the manuscript. Musashaykhova Shahnoza Mamirbekovna: Patient recruitment, clinical data collection, and investigation. Salahiddinov Zuhriddin Salahiddinovich: Data curation, statistical analysis, and interpretation of the results. Musashayxov Umidjon Xusanboyevich: Methodology, coordination of molecular genetic analyses, literature review, and preparation of the initial manuscript draft. All authors have read and approved the final version of the manuscript and agree to be accountable for their respective contributions.

Data availability statement

The de-identified data supporting the findings of this study are available from the corresponding author upon reasonable request, subject to applicable ethical and institutional approvals.

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