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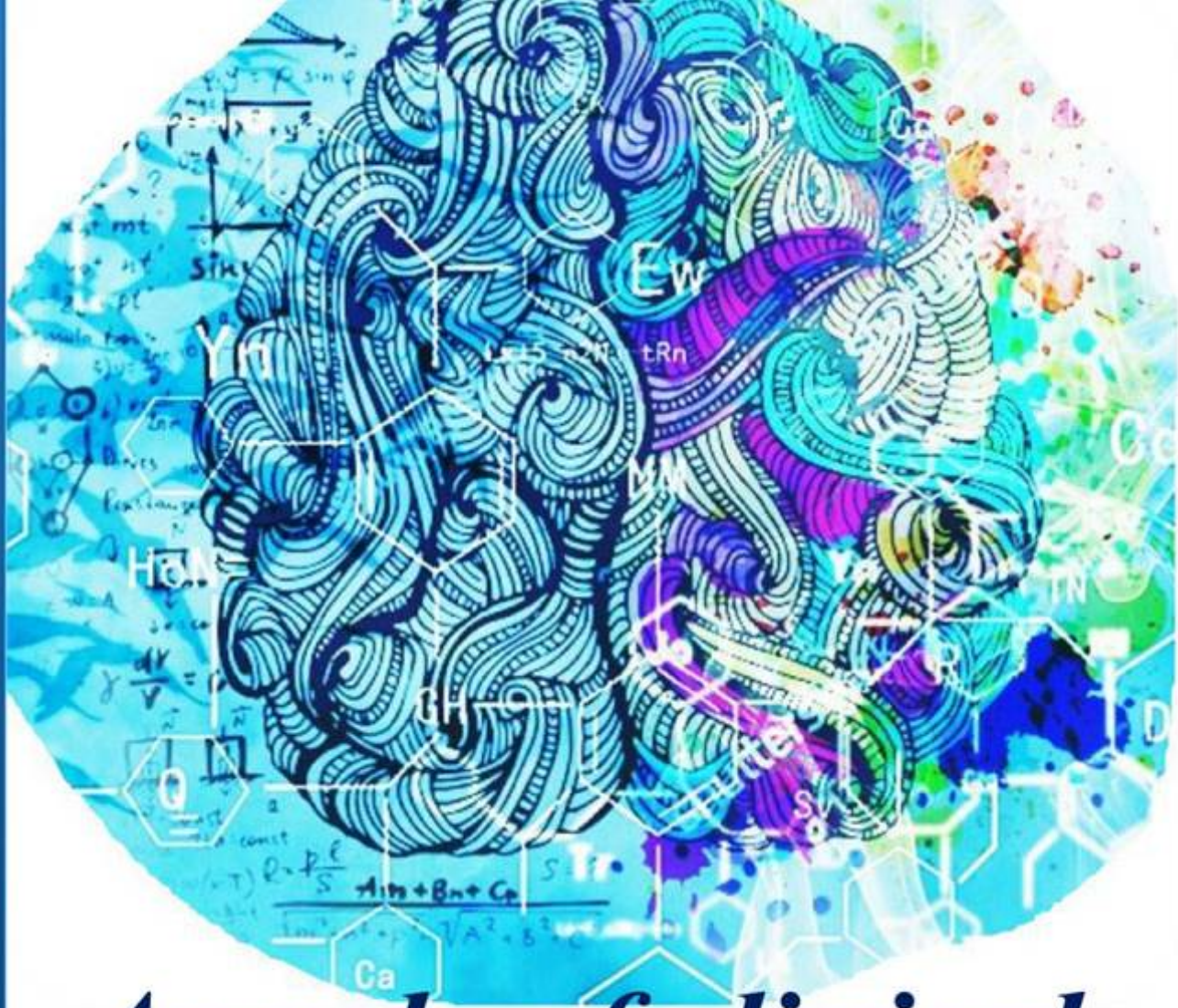

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MOLECULAR GENETIC ANALYSIS OF THE RENIN–ANGIOTENSIN SYSTEM IN CHRONIC KIDNEY DISEASE

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ABSTRACT

This study examined polymorphisms of the AGTR1 and AGTR2 genes in 153 patients with chronic kidney disease (CKD). Molecular genetic analysis revealed an increased frequency of single nucleotide polymorphisms in these genes, which influence the activity of the renin–angiotensin system. It was found that CKD progression is associated with the presence of the C allele of the AGTR1 gene (encoding the angiotensin II type 1 receptor) and the G allele of the AGTR2 gene (encoding the angiotensin II type 2 receptor).

Keywords: chronic kidney disease, renin–angiotensin system, molecular genetic factors.

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АНАЛИЗ МОЛЕКУЛЯРНО-ГЕНЕТИЧЕСКИХ АСПЕКТОВ РЕНИН-АНГИОТЕНЗИНОВОЙ СИСТЕМЫ ПРИ ХРОНИЧЕСКОЙ ПОЧЕЧНОЙ НЕДОСТАТОЧНОСТИ

РЕЗЮМЕ

В исследовании изучался полиморфизм генов AGTR1 и AGTR2 у 153 пациентов с хронической болезнью почек (ХБП). Молекулярно-генетический анализ выявил повышенную частоту однонуклеотидных полиморфизмов в этих генах, которые влияют на активность ренин-ангиотензиновой системы. Было установлено, что прогрессирование ХБП связано с наличием аллеля С гена AGTR1 (кодирующего рецептор ангиотензина I типа) и аллеля G гена AGTR2 (кодирующего рецептор ангиотензина II типа).

Ключевые слова: хроническая болезнь почек, ренин-ангиотензивная сстема, молекулярно-генетические факторы

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СУРУНКАЛИ БУЙРАК ЕТИШМОВЧИЛИГИДА РЕНИН-АНГИОТЕНЗИН ТИЗИМИНИНГ МОЛЕКУЛЯР-ГЕНЕТИК ХУСУСИЯТЛАРИНИ ТАҲЛИЛ ҚИЛИШ

РЕЗЮМЕ

Мақолада сурункали буйрак етишмовчилиги (СБЕ) бўлган беморларда АГТР1 ва АГТР2 генларининг полиморфизми натижалари келтирилган. 153 та СБЕ беморлари орасида ўтказилган молекуляр-генетик таҳлил, ренин-ангиотензин тизими фаолияти билан боғлиқ генларнинг бир нуқтали полиморфизмларининг частотасининг ошишига мойиллик текширилди. СБЕ нинг ривожланиши AGTR 1 генининг С аллели ва AGTR 2 генининг Г аллели билан боғлиқ ҳолда кузатилмоқда.

Калит сўзлар: сурункали буйрак касаллиги, ренин- ангиотензин тизими, молекуляр генетик омиллар.

Introduction. Chronic kidney disease (CKD) is a major global health burden, affecting an estimated 9.1% of the population and contributing significantly to cardiovascular morbidity and premature mortality [3].

The disease is characterized by progressive nephron loss, structural damage to the renal parenchyma, and functional decline in glomerular filtration rate (GFR), ultimately leading to end-stage renal disease (ESRD) [10].

Approximately 13% of the global population is affected by chronic kidney disease (CKD) [2][3], a prevalence that remains consistent across both developed nations with high living standards and developing countries. The incidence of CKD is rising at an annual rate of about 10% [5]. CKD is characterized as a progressive condition where the renal excretory system gradually loses its functional capacity over an extended period, typically at least three months. This disease often develops in the context of other chronic health conditions that impose a persistent burden on kidney function [13][14].

Among the multiple pathophysiological pathways involved in CKD progression, the renin–angiotensin system (RAS) plays a central role in both hemodynamic and non-hemodynamic mechanisms of kidney injury [6]. Activation of RAS promotes vasoconstriction, sodium retention, oxidative stress, and pro-inflammatory signaling, which together accelerate glomerulosclerosis and interstitial fibrosis [1]. Angiotensin II, the main effector peptide of RAS, exerts its biological effects primarily via angiotensin II type 1 (AT1) receptors, encoded by the AGTR1 gene, and type 2 (AT2) receptors, encoded by the AGTR2 gene [2]. (Figure 1)

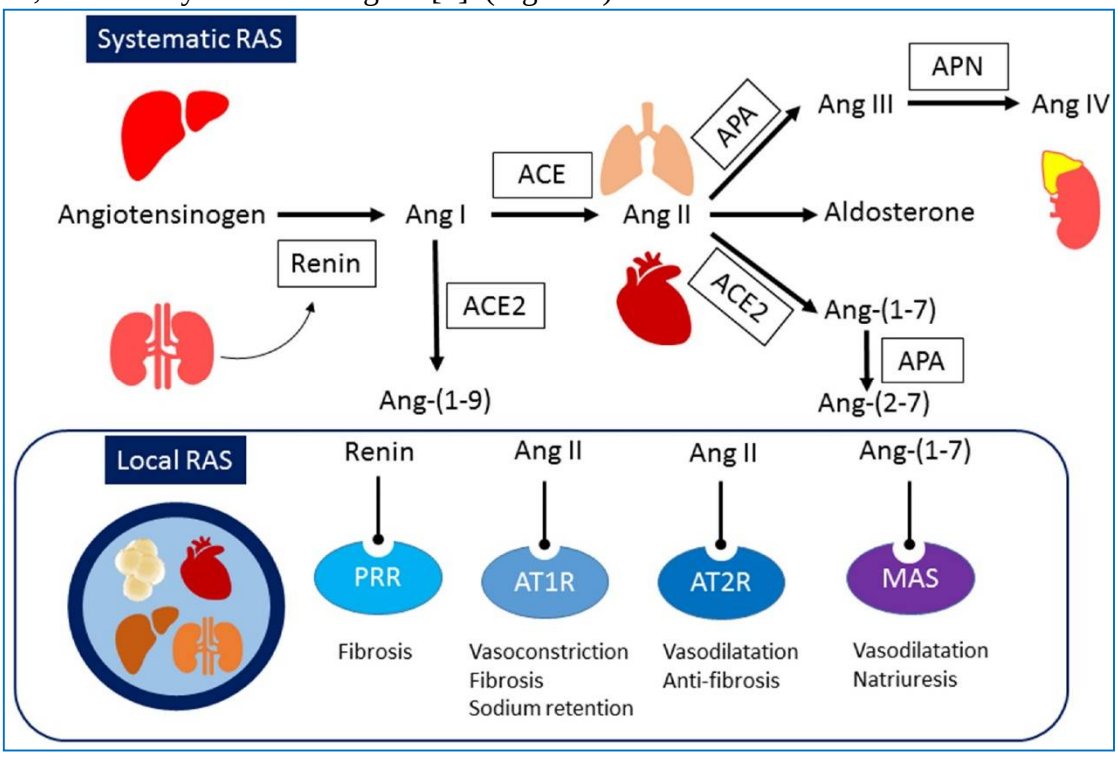


Fig. 1. Adopted from: Tain, You-Lin, and Chien-Ning Hsu. [15] 2024. Schema outlining the major organs and components of the renin–angiotensin system.

Overactivation of AT1 receptors is associated with increased intraglomerular pressure, mesangial proliferation [8], and fibrotic remodeling, while AT2 receptors may mediate vasodilation and anti-fibrotic effects, suggesting a complex balance between receptor subtypes [7].

Genetic variability within AGTR1 and AGTR2, particularly single nucleotide polymorphisms (SNPs) [9], has been shown to influence receptor expression, ligand binding affinity, and downstream signaling cascades [10].

Several studies have demonstrated associations between AGTR1 polymorphisms, such as A1166C, and susceptibility to hypertension, cardiovascular disease, and CKD progression [15].

Similarly, AGTR2 gene variants have been linked to altered renal hemodynamics, increased oxidative stress, and modulation of nitric oxide production, all of which contribute to renal injury [11].

Understanding the distribution of AGTR1 and AGTR2 genotypes in CKD patients, and their relationship with disease stages, could help identify individuals at higher risk for rapid progression [2].

Such insights may enable the development of genotype-guided strategies for RAS blockade and other targeted therapies, potentially improving renal and cardiovascular outcomes [13].

Extensive research in global scientific literature has established a direct link between the renin-angiotensin system (RAS) and renal dysgenesis [7][9]. The renin-angiotensin-aldosterone system (RAAS) plays a critical role in regulating vascular tone and maintaining water-electrolyte homeostasis [6]. The RAAS cascade is initiated by renin, which cleaves angiotensinogen (AGT) to produce the inactive decapeptide angiotensin I (Ang I) [17]. Subsequently, angiotensin I is converted into the biologically active angiotensin II (Ang II) through the action of angiotensin-converting enzyme (ACE) [10]. The physiological effects of Ang II are primarily mediated through its interaction with two distinct receptors: AGTR1 (type I angiotensin receptor) [12] and AGTR2 (type II angiotensin receptor) [4]. Predominantly, Ang II binds to AGTR1, triggering a cascade of intracellular signaling pathways that promote tissue remodeling, vasoconstriction, and aldosterone secretion by the adrenal glands [8][14].

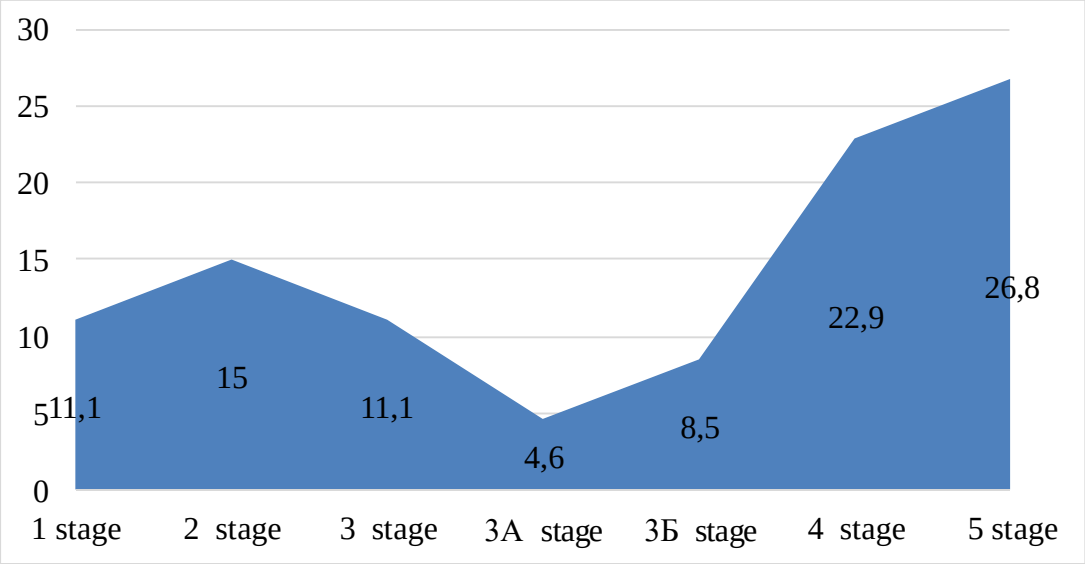
In recent ears, significant scientific attention has been directed toward exploring molecular genetic factors that contribute to the onset and adverse progression of nephrological disorders [6][7][9]. However, existing studies have et to provide conclusive evidence regarding the precise pathogenetic role of specific single-nucleotide polymorphisms (SNPs) in genes of the renin-angiotensin system in the context of CKD, highlighting the need for further investigation [15].

The objective of this study was to investigate the characteristics of polymorphisms in the AGTR1 and AGTR2 genes among patients diagnosed with CKD [16].

Materials and methods of research. A molecular genetic analysis was performed on 153 patients diagnosed with chronic kidney disease (CKD), revealing the distribution of disease stages as follows: stage 1 was observed in 11.1% of cases (17 out of 153 patients), stage 2 in 15.0% (23 out of 153), stage 3 in 11.1% (17 out of 153), stage 3A in 4.6% (7 out of 153), and stage 3B in 8.5% (13 out of 153). This study highlights the varying prevalence of CKD stages within the sampled population. Further investigation into the genetic factors influencing these stages could provide insights into disease progression and potential therapeutic targets.The data obtained is presented in diagram 1. The largest percentage of patients had stages 4 and 5 of CKD (22.9% and 26.8%, respectively). The age range of the patients ranged from 21 to 95 ears, with an average age of 57.9±2.3 ears. Among all the patients who participated in the study, the male sex prevailed over the female sex, their ratio was 1:1.4 (89 men and 64 women).

All participants in the study underwent genetic testing to identify single nucleotide polymorphisms in the targeted genes, employing an allele-specific polymerase chain reaction amplification technique with dedicated test systems. The polymorphisms under investigation were specifically A1166C in the AGTR1 gene and G1675A in the AGTR2 gene.

Fig. 2. Distribution of patients with CKD by disease stages



Statistical analysis of the collected data was carried out using the R programming language [R Core Team. 2015. R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria]. To evaluate differences in the frequencies of genotypes and alleles, the Chi-square test and Fisher's exact test were applied. Findings were deemed statistically significant when the p-value was below 0.05. This methodological framework enhances the reliability of detecting genetic associations in CKD patients. Additionally, such statistical tools are widely recognized for their precision in handling categorical data in biomedical research

Results of the study. Medical and genetic analysis identified that the A1166C polymorphism of the AGTR1 gene occurs in two allelic forms: A (“A”) and C (“C”). The presence of the minor “C” allele was associated with the progression of chronic kidney disease (CKD) and demonstrated lower expression levels compared to the “A” allele (Fig. 2). These results highlight the potential significance of the “C” allele as a genetic marker predisposing to CKD progression.

The medical and genetic investigations revealed that the A1166C polymorphism of the AGTR1 gene manifests as either the A (“A”) or C (“C”) allele. The progression of chronic kidney disease (CKD) is linked to the presence of the minor “C” allele. This allele exhibits reduced expression intensity compared to the “A” allele (Fig. 2). These findings underscore the potential role of the “C” allele as a genetic risk factor for CKD progression. Further studies are warranted to explore the mechanistic pathways underlying this association and its implications for personalized treatment strategies.

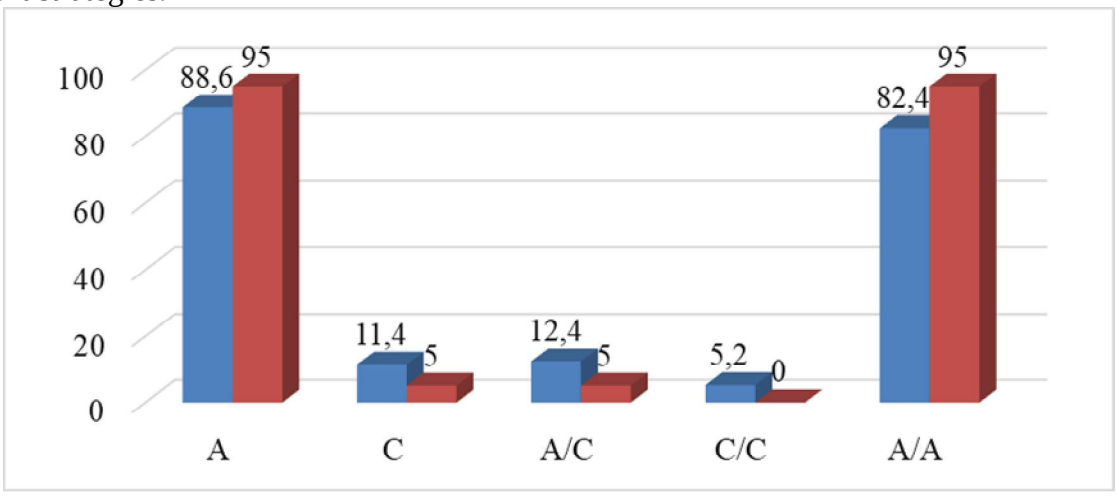


Fig. 3. Frequency of occurrence of alleles and genotypes of the A1166C polymorphism of the AGTR1 gene in patients with CKD

In the group of patients with CKD, it was revealed that the actual frequency of the homozygous genotype C/C is statistically significantly increased compared to the theoretical one (Hobs = 0.19% and Hexp = 0.18%, respectively, $\chi 2 = 0.22$; $p > 0.05$). The observed number of heterozygotes A/A, on the contrary, is insignificantly lower than the expected one (Hobs = 0.45% and Hexp = 0.46%, respectively, $\chi 2 = 0.045$; $p > 0.05$). The indicator of the relative deviation of the expected heterozygosity from the observed one turned out to be negative (deficiency of heterozygotes), i.e., D = -0.02. However, such a moderate deficiency of A/C heterozygotes is compensated by an excess of A/A homozygotes. The observed frequency distribution of the unfavorable genotype C/C also corresponded to the Hardy-Weinberg equilibrium (HWE) (Hobs=0.41% and Hexp=0.41%), as evidenced by the value $\chi 2=0.01$ and $p>0.05$.

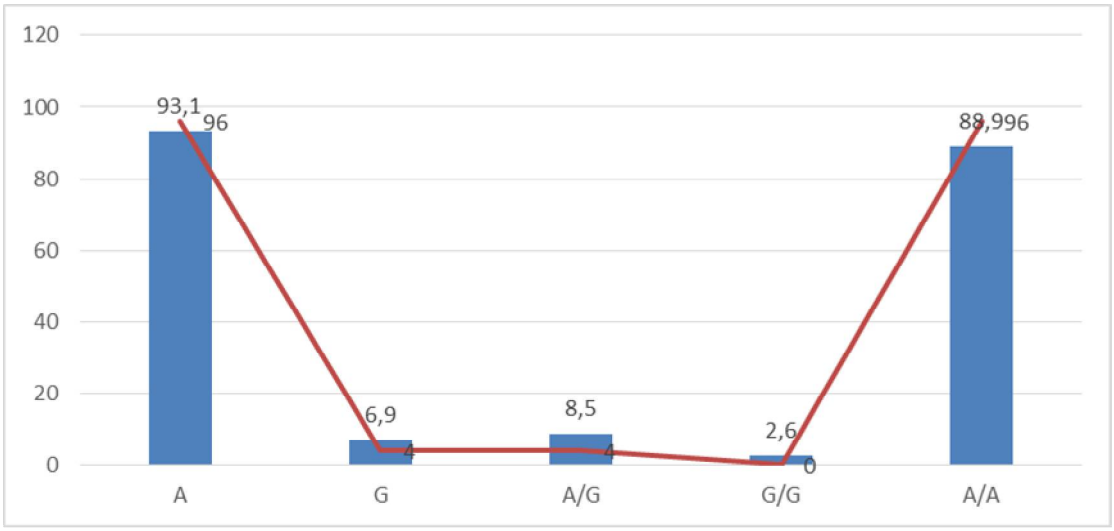


Fig. 4. Analysis of Allele and Genotype Frequencies for A1166C Polymorphism of the AGTR1 Gene in CKD Patients

When assessing the G1675A polymorphism of the AGTR2 gene, two allelic variants, “A” and “G”, were identified. The minor allele “A” was found to be associated with the development of chronic kidney disease (CKD) and is characterized by lower expression levels compared to the “G” allele. In the studied population, the wild-type genotype G/G occurred at a relatively low frequency, which closely matched the expected value (0.11 vs. 0.12; $\chi^2 = 0.03$; $P > 0.05$).

Analysis of the A1166C polymorphism in the AGTR1 gene revealed that the frequency of the heterozygous genotype was slightly higher than the theoretical expectation (0.46 vs. 0.45; $\chi^2 = 0.03$; $P > 0.05$). The relative deviation of observed heterozygosity from the expected value was positive (D = +0.02), indicating a moderate excess of heterozygotes. In contrast, the frequency of the unfavorable G/G genotype was marginally lower than the expected level (0.43 vs. 0.42; $\chi^2 = 0.01$; $P > 0.05$).

Importantly, patients with end-stage CKD were significantly more likely to carry mutant variants of the angiotensin receptor genes, particularly A1166C (AGTR1) and G1675A (AGTR2). These genetic variants are linked to a more severe course of arterial hypertension and may contribute to faster disease progression. The results indicate a possible genetic predisposition to advanced CKD and its complications. Further large-scale studies are required to confirm these associations and to assess their potential role in developing targeted therapeutic strategies.

Conclusion. This study demonstrates that polymorphisms in the AGTR1 and AGTR2 genes are present at notable frequencies among patients with chronic kidney disease, with the C allele of AGTR1 and the G allele of AGTR2 showing potential associations with disease progression. Although observed deviations in genotype distribution [17], including the moderate deficiency of heterozygotes, were not statistically significant, the patterns suggest a possible genetic influence on renal function decline. The predominance of advanced CKD stages (4 and 5) in the study population underscores the clinical importance of early genetic screening to identify individuals at increased risk [8]. Integrating molecular genetic profiling into CKD management may facilitate personalized

therapeutic approaches, particularly in guiding the intensity of renin–angiotensin system blockade. Future large-scale, multi-center studies are warranted to validate these findings and explore their translational potential in precision nephrology.

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ANNALS OF CLINICAL DISCIPLINE

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