ORIGINAL ARTICLE

A POSSIBLE ASSOCIATION OF ABCB1 (MDR1) GENE rs1045642 VARIANT (SNP) WITH THE DEVELOPMENT OF RENAL GLOMERULAR DISEASES

ABCB1 (MDR1) Geni rs1045642 Varyantının (SNP) Renal Glomeruler Hastalık Gelişimiyle Olası Bir İlişkisi

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ABSTRACT

Objective: Renal glomerular disease is an issue that causes proteinuria, edema, and sometimes hematuria and associated with tiny filters in the kidney called glomerulus. In this retrospective study, we aimed to analyze the possible association of renal glomerular diseases with the ABCB1 (MDR1) gene C3435T polymorphism (rs1045642), with respect to their genetic basis.

Material and Methods: A total of 52 cases with renal glomerular disease were compared with 58 healthy controls for the ABCB1 C3435T polymorphism screened by strip assay and NGS. Allele frequencies and genotype distributions of the relevant gene variant were analyzed.

Results: The patient group included focal segmental glomerulosclerosis, membranous glomerulonephritis, IgA nephropathy, diabetic nephropathy, amyloidosis, and similar conditions. The C allele (wild type) and CC genotype of the MDR1 C3435T single nucleotide polymorphism (SNP) were higher in the control group compared to patients, while the T allele (mutant type) and TT genotype were significantly higher in patient group than controls. The patients were older than the healthy controls.

Conclusion: ABCB1 (MDR1) C3435T polymorphism cause a decrease in P-glycoprotein function. In this context, our data suggests that the C3435T variant (SNP) contributes to the development of renal glomerular diseases.

Keywords: *Renal Glomerular Disease; ABCB1 Gene; Polymorphism; P-glycoprotein*

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ÖZET

Amaç: Renal glomerüler hastalık, proteinüri, ödem ve bazen hematüriye neden olan ve böbrekte glomerulus adı verilen küçük filtrelerle ilişkili bir sorundur. Biz bu retrospektif çalışmada, renal glomerüler hastalıkların ABCB1 (MDR1) geni C3435T polimorfizmi (rs1045642) ile olası ilişkisini, genetik temelleri bağlamında analiz etmeyi amaçladık.

Gereç ve Yöntemler: Renal glomerüler hastalığı olan toplam 52 vaka, strip assay ve NGS ile taranan ABCB1 C3435T polimorfizmi açısından 58 sağlıklı kontrol ile karşılaştırıldı. İlgili gen varyantının alel frekansları ve genotip dağılımları analiz edildi.

Bulgular: Hasta grubunda fokal segmental glomeruloskleroz, membranöz glomerulonefrit, IgA nefropatisi, diyabetik nefropati, amiloidoz ve benzeri rahatsızlıklar yer alıyordu. MDR1 C3435T tek nükleotid polimorfiminin (SNP) C aleli (doğal tip) ve CC genotipi kontrol grubunda hastalara göre daha yüksek bulunurken, T aleli (mutant tip) ve TT genotipi hasta grubunda kontrollere kıyasla anlamlı olarak daha yüksek çıktı. Hastalar sağlıklı kontrollerden daha yaşlıydı.

Sonuç: ABCB1 (MDR1) C3435T polimorfizmi P-glikoprotein fonksiyonunda azalmaya neden olmaktadır. Bu bağlamda, verilerimiz C3435T varyantının (SNP) renal glomerüler hastalıkların gelişimine katkıda bulunduğunu düşündürmektedir.

Anahtar Kelimeler: *Renal Glomeruler Hastalık; ABCB1 Geni; Polimorfizm; P-glikoprotein*

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INTRODUCTION

Renal glomerular disease is a condition that affects the small filters of the kidneys called the glomerulus (part of the nephron), which are responsible for cleaning the blood. Proteins and sometimes erythrocytes pass into the urine due to glomerular lesions in this disease. Glomerulopathies mainly consist of two subgroups known as glomerulonephritis and glomerulosclerosis. It is possible to encounter different clinical pictures from mild findings to severe life-threatening forms. Drug therapies and various diseases affecting the kidneys can lead to nephritic or nephrotic syndrome. Nephrotic syndrome (NS) is a renal disorder accompanied by proteinuria, hypoalbuminemia, edema and hyperlipidemia (1). Primary causes of this glomerular disease include minimal change disease, focal segmental glomerulosclerosis, Immunoglobulin A nephropathy (IgAN), membranous nephropathy, membranoproliferative glomerulonephritis, while secondary causes comprise diabetes, amyloidosis, various infections and autoimmune diseases such as systemic lupus erythematosus (SLE) and Sjögren's disease, etc. (2). Most cases of NS are described as idiopathic or primary, and in adults, primary NS often presents as membranous nephropathy and focal segmental glomerulosclerosis (3).

Minimal change disease (MCD) is a significant cause of NS characterised by edema and proteinuria and represents 15% of adult idiopathic NS patients, but occurs at a much higher frequency at younger ages (4). In MCD, the glomeruli appear normal on light microscopy, whereas the only histological abnormality revealed in a kidney biopsy analysis is the deletion of the podocyte foot process on electron microscopy (5). Focal segmental glomerulosclerosis (FSGS) is the prototype of podocyte loss-related glomerulosclerosis, and the glomerular pathology of the disease is distinguished by focal and segmental localization of sclerotic lesions (6). Immunoglobulin A nephropathy is the most common primary glomerular disease characterized by mesangial immune-complex deposition and involves a high risk of renal failure (7). Membranous nephropathy (MN) is a glomerulopathy with subepithelial immune deposits in the glomerular capillary structure, and its clinical presentation consists of NS or asymptomatic proteinuria and is particularly common in adults over the age of fifty (8).

Diabetes mellitus is a chronic and metabolic disease that causes hyperglycemia as a result of impaired insulin secretion or utilization, and this essentially leads to various complications. Microvascular complications of diabetes include diabetic nephropathy. Diabetic nephropathy is a major disorder resulting in end-stage renal disease (9) and is also a leading cause of secondary NS (10). Systemic lupus erythematosus is a chronic autoimmune disease that can disrupt kidney function (lupus nephritis, LN), even lead to renal failure. LN can be divided into several types based on where immune complexes accumulate in the glomeruli, the presence of mesangial proliferation, the extent of glomerular damage (global or segmental), and whether glomerular involvement is acute (inflammatory) or chronic (sclerotic), etc. (11).

Familial Mediterranean fever (FMF) is a systemic autoinflammatory disease usually identified in the Mediterranean region and is characterized by recurrent fever, polyserositis, and chest, abdominal or joint pain. It is caused by mutations of the *MEFV* gene, which encodes a protein called pyrin. This disease is inherited in an autosomal recessive pattern and AA amyloidosis is a serious complication of FMF that causes renal failure and can eventually lead to death (12).

The multidrug resistance-1, *MDR1* (ABCB1) gene is a member of the ATP-binding cassette family and is known to encode a transmembrane protein, P-glycoprotein (P-gp) (13). P-gp has a protective function that transports a large number of endogenous and exogenous substances to outside of the cell using the energy obtained from ATP hydrolysis (14). Some *ABCB1* gene variants have been associated with response to medications, and predisposition to certain diseases in different populations. There are studies investigating the relationship between inflammatory bowel diseases and *ABCB1* (MDR1) gene polymorphisms. For example, Mijac et al. suggested an association between three different MDR1 gene variants and ulcerative colitis (15). Furthermore, Ahmed et al. claim that a certain *MDR1* polymorphism in the Asian population may increase the risk of Parkinson's disease (16). In this context, we aimed to analyze the association of the C3435T SNP of the *ABCB1* gene with several renal diseases, some of which progress to nephrotic syndrome.

MATERIAL AND METHODS

The study was conducted at Sivas Cumhuriyet University Faculty of Medicine, Department of Medical Genetics. A total of 52 adult patients diagnosed with renal glomerular disease and 58 healthy controls were included in this analysis. The study was carried out in accordance with the Declaration of Helsinki. Informed consent forms were obtained from the participants. Ethics committee approval was received from the Health Sciences Research Ethics Committee of Sivas Cumhuriyet University with the decision dated 24.04.2025 and numbered 2025-04/22. Immunofluorescence, light microscopy, electron microscopy, renal biopsy and some other methods had been applied to our patients previously for diagnostic purposes. The age and gender of the patients and healthy subjects were listed and the clinical and laboratory characteristics of the patients were recorded. The results related to the *ABCB1* C3435T variant detected in peripheral blood by the Reverse Hybridization Strip Assay (Vienna Lab, Strip Assay GmbH, Austria) and Next-Generation Sequencing/NGS (MiSeq, Illumina, San Diego, CA, USA) methods were taken from the participants' records and analyzed. Subsequently, the genotype distribution and allele frequencies of the two groups were compared. Statistical analyses were performed using SPSS (version 23.0). Mean age and gender were determined in both groups. Independent samples T-test was used to compare the means. Chi-square test was applied for categorical variables.

RESULTS

Fifty-two patients with renal diseases such as focal segmental glomerulosclerosis, membranous glomerulonephritis, IgA nephropathy, diabetic nephropathy and renal amyloidosis, and 58 healthy controls were evaluated for the *MDR1* C3435T polymorphism. The mean age of the patients was higher than the healthy control group (P=0.02). The characteristics of the participants were presented in Table 1.

Despite the classical treatment, a certain level of proteinuria was still present in 27 patients (52%) and edema in 24 patients (46.2%). Thirteen of the patients (25%) had hypertension and 5 of them (9.6%) had hematuria. In addition, renal amyloid deposits were present in 5 patients (9.6%) on the basis of FMF. The pathological background included a complex condition with additional disorders, but mainly there were 11 cases of focal segmental glomerulosclerosis (21.2%), 11 membranous glomerulonephritis (21.2%), 8 IgA nephropathy (15.4%), 6 diabetic nephropathy (11.5%), 6 mesangioproliferative glomerulonephritis (11.5%), 5 FMF-induced renal amyloidosis (9.6%), 3 SLE nephritis (5.8%) and 2 cases of minimal change disease (3.8%). Statistically significant differences were found between the patient and control groups in terms of the distribution of genotypes and allele frequencies in *ABCB1* C3435T polymorphism (Table 2). Of the genotypes, CT and TT rates were higher significantly in the patient group compared to controls (P=0.035). Naturally, the T allele was also found to be higher in patients (P=0.022). Indeed, the frequency of the T allele and CT-TT genotype of *MDR1* C3435T in patients suggests the pathogenic role of this variant (SNP) in renal glomerular diseases.

Table 1. Characteristics of the patients and control group.

Feature	Patients	Controls
Age	45.5±12	40.6±9.8
Sex	M 28 (53.8%)	M 28 (48.3%)
	F 24 (46.2%)	F 30 (51.7%)
Hypertension	13 (25%)	-
Diabetes Mellitus	6 (11.5%)	-
FMF	8 (15.4%)	-
Residence	Urban 38 (73%)	Urban 51 (88%)
	Rural 14 (27%)	Rural 7 (12%)

Table 2. The distribution of genotypes, and allele frequencies of ABCB1 C3435T variant in patients and controls.

Genotype	Patients	Controls	P value
CC	16 (30.8%)	32 (55.2%)	0.035
CT	26 (50%)	18 (31%)	
TT	10 (19.2%)	8 (13.8%)	
Total	52	58	
Alleles	**Patients**	**Controls**	**P value**
C	58 (55.8%)	82 (70.7%)	0.022
T	46 (44.2%)	34 (29.3%)	
Total	104	116	
Odds ratio	0.523	0.300 - 0912	

DISCUSSION

The *MDR1* (ABCB1) (7q21.12) is a polymorphic gene that encodes a transmembrane protein called P-glycoprotein (P-gp), which is a member of the superfamily of ATP-binding cassette transporters and located in the plasma membrane (17). P-gp is involved in the eflux of various compounds such as toxins, drugs and certain xenobiotics from inside to outside of the cell (18). Some researchers suggest that *MDR1* may have a significant effect in apoptosis and immune system regulation and is associated with predisposition to some diseases (19).

Some *MDR1* variants (SNPs) have been linked to the development of various diseases. Youssef et al. stated that the *MDR1* C3435T polymorphism may be a risk factor for increased susceptibility and earlier onset of nephrotic syndrome (20). Brinar et al. proposed that certain *MDR1* SNPs are associated with inflammatory bowel diseases, particularly ulcerative colitis (21). The study by Jeleń et al. reveals a possible association of the *ABCB1* gene C3435T polymorphism with susceptibility to the development of depression (22). The *ABCB1* (MDR1) C3435T polymorphism (rs1045642) has been declared to cause a decrease in mRNA expression and P-gp function, suggesting an increase in the risk of cancer with reduced P-gp action (23). In this way, an association of C3435T polymorphism with cancer prognosis and predisposition to various cancers has been reported in a certain number of studies. For example, a meta-analysis conducted by Wang et al. suggests that the *MDR1* C3435T polymorphism increases the risk of kidney and breast cancer (24).

In this current research, a significant part of our patients consisted of cases diagnosed with FSGS. This disease is a histological variant of glomerular lesion involving some different clinicopathological conditions characterized by damage within the podocyte on a pathophysiological basis (25). Etiologically, FSGS can be subdivided into primary (idiopathic), secondary (infections, medications, vascular problems, etc.), or genetic (26). In fact, FSGS reflects a histopathological pattern based on podocyte damage and depletion as a consequence of different etiologies, and its diagnosis is made possible by the integration of clinical history (including medications), laboratory findings (serology, serum albumin, proteinuria, etc.) and histopathology of the kidney (27). Primary FSGS is usually associated with sudden onset of proteinuria and marked nephrotic syndrome, and there are studies in the literature that gives prevalence rates ranging from 54% to 90% for nephrotic syndrome in primary FSGS (28). However, among the FSGS cases we analysed, in addition to nephrotic syndrome, nephrotic-range proteinuria (≥3.5 g/24h and the absence of hypoalbuminemia), and even subnephrotic proteinuria (<3.5 g/24 h) were found.

Membranous glomerulonephritis (MGN), a significant cause of nephrotic syndrome, is usually observed in the fifties and more common in men. While a significant proportion of cases are primary (idiopathic), about one-third arise due to infections, cancers and autoimmune diseases (29). This condition results from the formation of an immune complex structure in the subepithelial part of the glomerular basement membranes (30).

Immunoglobulin A nephropathy (IgAN), a common primary glomerulopathy, is one of the major causes of chronic renal disease and thus end-stage renal disease (31). This condition is characterised by the deposition of IgA-containing immune complexes in the glomeruli against an inflammatory background and the clinical presentation may vary from microscopic hematuria and proteinuria to nephrotic syndrome or rapidly progressive glomerulonephritis (32). Although IgA nephropathy, defined as mesangioproliferative nephritis with IgA deposition, is accepted one of the most common primary glomerular disease, non-IgA mesangioproliferative glomerulonephritis is also a prevalent condition, declared in many developing populations (33). In this current research, IgA nephropathy was also present in a certain number of our cases (8 patients/15.4%). Mesangioproliferative glomerulonephritis (MesPGN) emerges on the basis of mesangial cell proliferation with an increase in the mesangial matrix and/or depositions in the mesangial region and can be seen in various conditions such as IgA nephropathy, chronic obstructive pulmonary disease, IgM nephropathy, SLE, postinfectious GN, etc. (34).

Diabetes mellitus is an endocrine disease characterized by high blood sugar levels due to the pancreas's inability to produce enough insulin or insulin resistance in the body. If the disease is not treated, it can cause various

problems in the eyes, kidneys, cardiovascular and nervous systems. Diabetic nephropathy is one of the microvascular complications of diabetes and is characterized by serious albuminuria and progressive decrease in glomerular filtration rate (GFR); which is usually accompanied by an increase in blood pressure (35). The most important findings detected in kidney biopsies of these patients are glomerular lesions, especially diffuse or nodular mesangial enlargement and basement membrane thickening (36). In this study, there were 6 cases of diabetes mellitus among our patients, and all of them suffered from diabetic nephropathy.

Familial Mediterranean Fever is an autoinflammatory disease characterized by the episodes of polyserositis and fever and can lead to serious complications in the long term, including renal amyloidosis (37). In FMF, the formation of inflammatory markers such as IL-1 and IL-18 due to *MEFV* gene mutation (defective pyrin) causes serositis and recurrent FMF attacks result in the deposition of serum amyloid A protein in tissues (38). FMF patients who do not receive conventional treatment (colchicine) or alternative treatment (IL-1 receptor antagonist, monoclonal antibody, etc.) have a high risk of developing renal failure due to amyloidosis during their lifetime. Renal amyloidosis (AA) is a significant complication of the disease and may lead to end-stage renal disease. The pathological issue of five patients in our study was FMF-induced renal amyloidosis and one of them had undergone kidney transplantation. These patients probably had not received regular treatment and follow-up.

Systemic lupus erythematosus is an autoimmune disease in which the immune system attacks healthy tissues, characterized by excessive B and T cell responses and sensitization to self-antigens (39). Lupus nephritis (LN) is a glomerulonephritis corresponding to one of the serious complications of SLE and the choice of medication for this condition is immunosuppressive agents such as glucocorticoids, cyclophosphamide, etc. (40). Three cases of SLE nephritis were included in this analysis.

In order of frequency, the patients we analyzed in this study were people with focal segmental glomerulosclerosis, membranous glomerulonephritis, IgA nephropathy, diabetic nephropathy, mesangioproliferative glomerulonephritis, FMF-induced renal amyloidosis, SLE nephritis and minimal change disease (Figure 1). Despite the patients were seen as heterogeneous groups, there was a common denominator defined as glomerular pathology in all of our cases. If the *ABCB1* C3435T polymorphism causes a decrease in p-glycoprotein expression and thus impaired detoxification, it may also be effective in glomerulopathies that occur in various diseases. Based on the present findings, we accepted that some of the patients in our study were somewhat resistant to treatment. A significant number of our patients

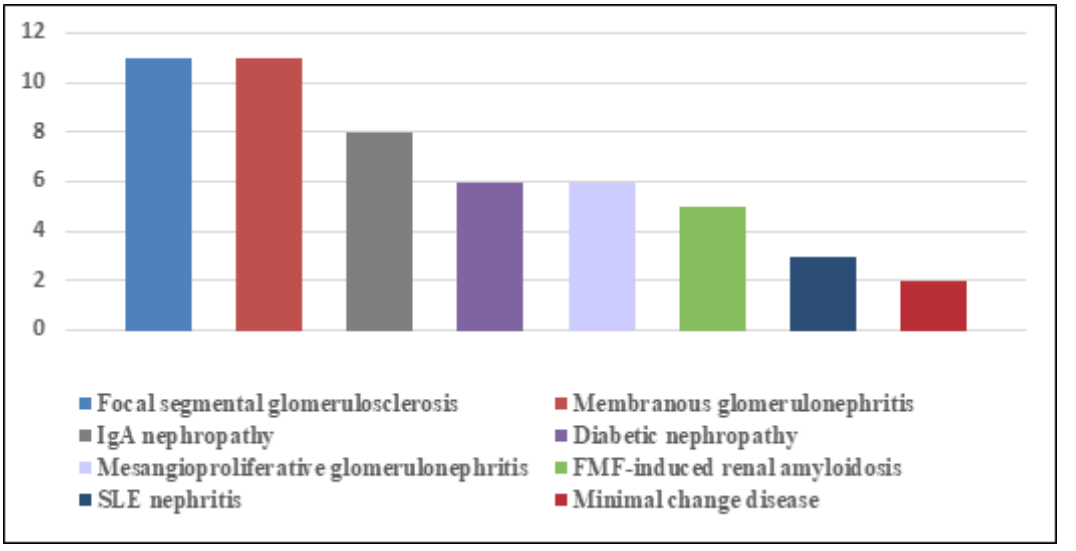


Figure 1. The distribution of the renal glomerular diseases in our patients.

had already developed nephrotic syndrome. Although all cases were treated with certain medications (Methylprednisolone, candesartan, canakinumab, ramipril, amlodipine, furosemide, cyclophosphamide, etc.), there were still patients with high micro total protein (MTP), edema, hematuria, hypertension, hyperlipidemia and some other findings. Some patients were receiving peritoneal dialysis or hemodialysis. Even, a renal transplantation was performed on a patient with renal amyloidosis who had been followed up for 16 years. The T allele frequency of *ABCB1* C3435T polymorphism was higher in our patients than in the control group (P=0.022). In this regard, this T allele and TT genotype may be associated with drug resistance.

CONCLUSION

The *ABCB1* (MDR1) C3435T polymorphism was significantly different in our patients with renal glomerular disease compared to the healthy control group. The T allele (mutant type) and TT genotype were higher in the patient group. This points to a possible pathogenic effect of C3435T polymorphism in renal glomerular diseases. The data we obtained are consistent with the fact that the *MDR1* C3435T polymorphism reduces the protective function of P-glycoprotein. The limitation of our study is that we could not provide clear data on drug resistance due to the relatively small number of cases and the inability to follow up the patients. However, our data clearly suggest that the C3435T variant (SNP) of the *ABCB1* gene is effective in the development of renal glomerular disease.

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