https://nephropathol.com DOI: 10.34172/jnp.27651 J Nephropathol. 2026;15(4):e27651

Journal of Nephropathology

The association between integrin subunit alpha V and vascular endothelial growth factor A polymorphisms and peritoneal dialysis adequacy

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ARTICLE INFO

Article type:
Original Article

Article history:
Received: 14 May 2025
Revised: 29 July 2025
Accepted: 20 Apr. 2026
Published online: 10 Jun. 2026

Keywords:
Vascular endothelial growth factor
Dialysis
Integrin subunit alpha V
Peritoneal equilibration test

ABSTRACT

Introduction: Chronic kidney disease often progresses to end-stage renal disease (ESRD), necessitating renal replacement therapies such as peritoneal dialysis (PD). Long-term PD is frequently associated with inflammation, which can affect dialysis efficacy and patient outcomes. Genetic polymorphisms in genes involved in the inflammation and vascular function, including integrin subunit alpha V (ITGAV) and vascular endothelial growth factor A (VEGFA), may influence PD outcomes.

Objectives: This study aims to elucidate the correlation between ITGAV (rs39111238, rs3738919, rs3768777) and VEGFA (rs699947, rs3025039, rs833061) polymorphisms, and their influence on renal-peritoneal Kt/V and PD outcomes.

Patients and Methods: In this cross-sectional study, 46 ESRD patients undergoing chronic PD at Shahid Modarres hospital in Tehran (2020–2021) were enrolled. Demographic and clinical data were collected, and peritoneal membrane transport was assessed using the peritoneal equilibration test (PET). Patients were categorized as high (HT) or low (LT) transporters. Genotyping for ITGAV (rs39111238, rs3738919, rs3768777) and VEGFA (rs699947, rs3025039, rs833061) polymorphisms was conducted using sequencing methods. Associations between genotypes and dialysis adequacy were considered.

Results: Among 46 participants, 21 were high transporters and 25 were low transporters. A significant association was observed between VEGFA rs3025039 and PET classification, while no patients carrying the CC genotype among high transporters. In addition, VEGFA rs833061 TT genotype was positively associated with renal Kt/V (B = 0.93; 95% CI: 0.11–1.75). Moreover, no significant associations were found for ITGAV polymorphisms.

Conclusion: VEGFA polymorphisms, particularly rs3025039 and rs833061, may influence peritoneal membrane transport and dialysis adequacy in PD patients. Further studies with larger cohorts and broader genetic screening are warranted to validate these findings.

Original Article

Implication for health policy/practice/research/medical education:
In this cross-sectional study, 46 end-stage renal disease (ESRD) patients undergoing chronic peritoneal dialysis (PD), vascular endothelial growth factor A polymorphisms, particularly rs3025039 and rs833061, may influence peritoneal membrane transport and dialysis adequacy in PD patients.

Please cite this paper as: Alirezaei A, Rostamiasl A, Tehrani Moayed V, Shakiba Y. The association between integrin subunit alpha V and vascular endothelial growth factor A polymorphisms and peritoneal dialysis adequacy. J Nephropathol. 2026;15(4):e27651. DOI: 10.34172/jnp.27651.

Introduction

Chronic kidney disease is a significant clinical condition that represents diminished renal function extending for at least 3 months (1). This disease may progress to end-stage renal disease (ESRD), in which long-term renal function isn't sustainable for homeostasis without renal replacement therapy (2). While conservative measures including fluid restriction and electrolyte balance are often considered, patients end up receiving either hemodialysis, peritoneal dialysis (PD), or renal transplantation upon eligibility (2-

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5). While PD is a critical treatment option, its long-term use is often accompanied by inflammatory processes that may lead to peritoneal dysfunction and elevated morbidity and mortality rates (6). Inflammation in PD patients is a complex interplay of local and systemic factors, with pro-inflammatory cytokines like interleukin-6 (IL-6) and tumor necrosis factor-alpha playing pivotal roles (6,7). Moreover, peritoneal adipocytes, mesothelial cells, and specifically peritoneal adipose tissue have a profound role in development and progression of peritoneal dysfunction and fibrosis partially by the release of adipokines and cytokines associated with inflammatory cascade (7-11).

Individual variations in peritoneal membrane are important factors for efficient PD, which are demonstrated by peritoneal solute transfer rate (PSTR) in clinical practice. PSTR is conventionally measured by peritoneal equilibration test (PET) (12-15). Although, peritoneal dysfunction secondary to long-term PD may lead to increased PSTR, this subsequently leads to ultrafiltration failure, decreasing efficacy of dialysis, and increasing morbidity (12,16). Among previous studies using diverse modalities to investigate PSTR and factors associated with it, genetic studies have identified that different single nucleotide polymorphisms (SNPs) are related to variations in PSTR among the patients (17).

Genetic polymorphisms in genes involved in inflammation and vascular function, such as Integrin subunit alpha V (ITGAV) and vascular endothelial growth factor-A (VEGFA), are potential determinants of PD outcomes (18,19). The ITGAV gene encodes the integrin αV subunit, a key player in cellular processes including inflammation, fibrosis, and adhesion. Polymorphisms within ITGAV, such as rs39111238, rs3738919, and rs3768777, have been implicated in various inflammatory conditions and cancer (20-22). These genetic variants may influence integrin expression and function, thereby affecting inflammatory responses and peritoneal membrane permeability (19, 23).

Similarly, VEGFA, encoding vascular endothelial growth factor A, is crucial for angiogenesis and vascular permeability. Polymorphisms in VEGFA, including rs699947, rs3025039, and rs833061, have been linked to altered VEGF expression and various pathological conditions, including diabetic retinopathy, cancer, and inflammatory diseases (23-26). These genetic variations may contribute to vascular changes within the peritoneal membrane, affecting solute transport and overall PD outcomes (19,27).

Objectives

This study aims to investigate the association between ITGAV (rs39111238, rs3738919, rs3768777) and VEGFA (rs699947, rs3025039, rs833061) polymorphisms, their distribution, and their impact on peritoneal Kt/V, renal Kt/V, and peritoneal transport characteristics as assessed by the PET. A better understanding of these genetic factors may lead to personalized treatment strategies for PD patients, improving outcomes and survival.

Patients and Methods

Study design and population

This descriptive cross-sectional study included a limited sample of ESRD patients undergoing chronic PD at Shahid Modarres Hospital, Tehran, between 2020 and 2021. The study-enrolled adults over 20 years old receiving PD, excluding those with recent infectious diseases, chronic inflammatory conditions, malignancy, or a history of peritonitis within the preceding two months. Baseline demographic characteristics and laboratory parameters such as hemoglobin levels, platelet counts, serum albumin levels, and urine volume were recorded. The PET was performed to evaluate peritoneal transport characteristics, following standard methodology as described in PD guidelines and handbooks (11). Dialysate and plasma creatinine concentrations were measured after a 4-hour dwell with a 2.5% glucose solution, and the dialysate-to-plasma creatinine ratio (D/P Cr) was calculated to determine peritoneal transport rates (7). Peritoneal equilibration test results were subsequently categorized into high transport (HT) and low transport (LT) groups (9). Peritoneal and renal Kt/V were calculated as a measure of dialysis adequacy using a standard laboratory method based on urea clearance (25,26).

Sample collection and genotyping

Genomic DNA was isolated from 5 cc of EDTA-treated blood samples collected from each patient using a genomic DNA purification kit. The samples were initially stored at -20 °C until polymerase chain reaction (PCR) amplification, after which they were preserved long-term at -80 °C. Genotyping for polymorphisms in the ITGAV (rs39111238, rs3738919, rs3768777) and VEGFA (rs699947, rs3025039, rs833061) genes was conducted using the TaqMan allelic discrimination assay, according to the established protocols (27). Each PCR reaction contained 100 ng of DNA, 12.5 µL of TaqMan genotyping master mix, and 0.6 µL of predesigned primers and probes, bringing the total volume to 25 µL. The PCR cycling conditions were as follows; 50 °C for 2 minutes, 95 °C for 10 minutes, followed by 45 cycles of 95 °C for 15 seconds and 60 °C for 1 minute. The PCR was performed using 96-well plates on an ABI StepOnePlus instrument. Alleles and genotypes were identified using StepOne software version 2.1 (Applied Biosystems, Foster City, CA, USA).

Statistical analysis

Data analysis was conducted using Stata version 17 (STATACORP, USA). Continuous variables were reported as mean± standard deviation (SD), while categorical variables were presented as frequencies or percentages. Fisher's exact test was conducted to compare categorical variables between high and low PET results, and independent T-tests were applied for continuous variables. The association between peritoneal Kt/V and urine volume was examined using Pearson's correlation. Genotype and allele frequencies were compared using the chi-square test. Associations between ITGAV and VEGFA gene polymorphisms and peritoneal and renal dialysis sufficiency (Kt/V) were evaluated using linear models, with results expressed as 95% confidence intervals (CIs). The relationship between gene polymorphisms and transport category (low versus high) was assessed using univariate and multivariate logistic regression models with different adjustment sets. Statistical significance was determined by a *P* value of less than 0.05.

Results

A total of 46 patients were included in the analysis. The average age at enrollment was 42.8 years old. Patients were more likely to be men and the majority had varying degrees of anemia (97.8%). In addition, 21 patients were designated as high transporting while 25 were considered low transporters based on PET. Baseline demographics and genetic polymorphisms are compared between high transport and low transport groups as demonstrated in Table 1.

Dialysate Kt/V was on average 0.55 units higher in high transporter patients. Intriguingly, high transporters on average had lower residual diuresis compared to low transporters and there was a significant correlation between urine volume and renal Kt/V (Pearson's correlation coefficient: 0.93, *P*<0.001). On the other hand, there was no noticeable correlation between dialysate Kt/V and residual urine volume (Pearson's correlation: -0.15, *P*=0.323). Notably, albumin measurement was also not correlated with renal or dialysate Kt/V (Table 2).

Logistic regression was conducted to estimate odds ratios for PET transport status, while linear regression models were applied to evaluate the associations with renal and dialysate Kt/V (Table 3). Among the evaluated genotypes, none of the ITGAV paired alleles were associated with low transporter status. VEGFA rs3025039 genotype was the only polymorphism associated with PET results, in which none of the patients with CC alleles were observed among those with poor PD function. While none of the genotypes were associated with dialysate Kt/V, a positive association was seen with VEGFA rs833061 TT allele (0.93-point increase) and renal Kt/V.

Discussion

The existing evidence in the literature suggests that VEGFA has a significant role in endothelial proliferation and glomerular repair and glomerular and peritubular capillary stabilization. Interestingly, under expression and deletion of the VEGFA gene in mice leads to proteinuria and thrombotic microangiopathy in the glomeruli (24,28). However, overexpression of the VEGFA gene is associated with glomerulomegaly, basement membrane thickening and proteinuria (29). As such, the precise expression of VEGFA gene is crucial for the maintenance of proper renal functionality. Integrins, known as transmembrane receptors, are composed of a total of eighteen distinct subunits of α and eight subunits of β. Nevertheless, to date, only 24 combinations of α and β have been identified (30,31). Integrins play a crucial role in the cellular proliferation and migration (31). Based on previous studies, deletion or inhibition of specific combinations of αV subunits have significant anti-fibrotic effect in mice (32-35). Although, overexpression of αVβ3 has been observed in autosomal dominant polycystic kidney disease (36). On the other hand, subunits of α8 play a vital role

Table 1. Baseline characteristics of patients enrolled in the study,

Demographics and characteristics	High transport (n=21)	Low transport (n=25)	Total (n=46)	*P* value*
Age +/–SD	39.86 (13.21)	45.20 (10.59)	42.76 (12.02)	0.135
Sex (%)				
Female	7 (33.3%)	9 (36.0%)	16 (34.8%)	0.850
Male	14 (66.7%)	16 (64.0%)	30 (65.2%)	
Hemoglobin level (g/dL)	10.61 (0.92)	10.91 (1.16)	10.78 (1.06)	0.354
Platelet count (count/1000 µL)	217.52 (67.00)	217.72 (86.60)	217.63 (77.42)	0.993
Albumin (g/dL)	3.29 (0.50)	3.43 (0.45)	3.37 (0.47)	0.334
Urine Volume (mL)	1,100 (734)	1,514 (473)	1,325 (634)	**0.025**
Dialysate Kt/V	2.21 (0.60)	1.66 (0.41)	1.91 (0.57)	**<0.001**
Renal Kt/V	1.93 (1.43)	2.50 (1.01)	2.24 (1.24)	0.121
BSA (m^2)	1.56 (0.16)	1.48 (0.14)	1.52 (0.15)	0.058

**P* values were obtained using chi-square test or independent T-tests as appropriate.

Table 2. Allele nucleotide distribution in genetic loci evaluated in the patients according to PET status

Genetic polymorphisms	High transport (n=21)	Low transport (n=25)	Total (n=46)	*P* value*
ITGAV rs3738919-Genotype				
AA	9 (42.9%)	11 (44.0%)	20 (43.5%)	0.938
CA	12 (57.1%)	14 (56.0%)	26 (56.5%)	
ITGAV rs3768777-Genotype				
AA	3 (14.3%)	4 (16.0%)	7 (15.2%)	0.580
AG	8 (38.1%)	6 (24.0%)	14 (30.4%)	
GG	10 (47.6%)	15 (60.0%)	25 (54.3%)	
ITGAV rs39111238-Genotype				
CC	0 (0.0%)	1 (4.0%)	1 (2.2%)	0.643
CG	10 (47.6%)	12 (48.0%)	22 (47.8%)	
GG	11 (52.4%)	12 (48.0%)	23 (50.0%)	
VEGFA rs699947-Genotype				
AA	4 (19.0%)	4 (16.0%)	8 (17.4%)	0.874
AC	11 (52.4%)	15 (60.0%)	26 (56.5%)	
CC	6 (28.6%)	6 (24.0%)	12 (26.1%)	
VEGFA rs3025039-Genotype				
CC	4 (19.0%)	0 (0.0%)	4 (8.7%)	**0.037**
CT	17 (81.0%)	23 (92.0%)	40 (87.0%)	
TT	0 (0.0%)	2 (8.0%)	2 (4.3%)	
VEGFA rs833061-Genotype				
CC	4 (19.0%)	4 (16.0%)	8 (17.4%)	0.874
CT	11 (52.4%)	15 (60.0%)	26 (56.5%)	
TT	6 (28.6%)	6 (24.0%)	12 (26.1%)	

**P* values were obtained using chi-square test or independent T-tests as appropriate

Table 3. Univariate and adjusted models evaluating the association between ITGAV/VEGFA polymorphisms and PET status

Polymorphism	Genotype	PET transport status (low vs high; OR; 95% CI)		Renal Kt/V (Beta, 95% CI)		Dialysate Kt/V (Beta, 95% CI)	
		Model 1*	Model 2#	Model 1*	Model 2#	Model 1*	Model 2#
ITGAV rs3738919	AA (Ref.)	Ref	Ref	Ref	Ref	Ref	Ref
	CA	0.95, 95% CI: (0.30–3.08)	1.38, 95% CI: (0.38–4.96)	-0.60, 95% CI: (-1.33–0.13)	-0.45, 95% CI: (-1.19–0.29)	-0.60, 95% CI: (-1.33–0.13)	-0.45, 95% CI: (-1.19–0.29)
ITGAV rs3768777	AA (Ref.)	-	-	-	-	-	-
	AG	0.56, 95% CI: (0.08–3.52)	0.80, 95% CI: (1.00–6.28)	0.67, 95% CI: (-0.49–1.83)	-0.73, 95% CI: (-0.44–1.91)	0.05, 95% CI: (-0.58–0.48)	0.00, 95% CI: (-0.55–0.56)
	GG	1.12, 95% CI: (0.21–6.14)	1.22, 95% CI: (1.17–8.50)	0.21, 95% CI: (-0.86–1.28)	0.04, 95% CI: (-1.05–1.14)	-0.26, 95% CI: (-0.75–0.23)	-0.14, 95% CI: (-0.66–0.38)
ITGAV rs39111238	CG (Ref.)	Ref	Ref	Ref	Ref	Ref	Ref
	CC	-¥	-¥	1.43, 95% CI: (-1.12–3.98)	1.17, 95% CI: (-1.38–3.73)	0.20, 95% CI: (-0.98–1.39)	0.34, 95% CI: (-0.83–1.52)
	GG	0.91, 95% CI: (0.28–2.93)	0.97, 95% CI: (0.28–3.43)	-0.31, 95% CI: (-1.06–0.43)	0.97, 95% CI: (-0.97–0.52)	0.22, 95% CI: (-0.12–0.57)	0.18, 95% CI: (-0.16–0.53)
VEGFA rs699947	AA (Ref.)	Ref	Ref	Ref	Ref	Ref	Ref
	AC	1.36, 95% CI: (0.28–6.68)	0.95, 95% CI: (0.17–5.29)	-0.41, 95% CI: (-1.40–0.57)	-0.61, 95% CI: (-1.57–3.98)	0.05, 95% CI: (-0.42–0.53)	0.10, 95% CI: (-0.37–0.57)
	CC	1.00, 95% CI: (0.17–5.98)	0.70, 95% CI: (0.10–4.85)	0.43, 95% CI: (-0.68–1.55)	0.32, 95% CI: (-0.76–1.41)	0.05, 95% CI: (-0.48–0.59)	0.07, 95% CI: (-0.46–0.60)
VEGFA rs3025039	CT (Ref.)	Ref	Ref	Ref	Ref	Ref	Ref
	CC	-¥	-¥	-0.40, 95% CI: (-1.71–0.91)	-0.65, 95% CI: (-2.04–0.74)	0.20, 95% CI: (-0.41–8.17)	0.52, 95% CI: (-0.13–1.17)
	TT	-¥	-¥	1.17, 95% CI: (-0.64–2.98)	-0.65, 95% CI: (-0.06–0.44)	-0.03, 95% CI: (-0.88–0.82)	-0.22, 95% CI: (-1.04–0.60)
VEGFA rs833061	CT (Ref.)	Ref	Ref	Ref	Ref	Ref	Ref
	CC	0.73, 95% CI: (0.15–3.60)	1.05, 95% CI: (0.19–5.81)	0.41, 95% CI: (-0.57–1.40)	0.61, 95% CI: (-0.36–1.57)	-0.05, 95% CI: (-0.53–0.42)	-0.10, 95% CI: (-0.57–0.37)
	TT	0.73, 0.18–2.89)	0.73, 95% CI: (0.17–3.18)	0.85, 95% CI: (-0.01–1.70)	**0.93, 95% CI: (0.11–1.75)**	0.00, 95% CI: (-0.41–0.41)	-0.03, 95% CI: (-0.43–0.37)

Model 1 *: Each polymorphism as exposure without further adjustment; Model 2#: adjusted with sex, age, and body surface area.
OR: Odds Ratio, CI: Confidence interval, -¥ : Not in the model.
Ref: Genotypes designated as reference categories served as the baseline for all comparisons, and accordingly, no odds ratios or regression coefficients were generated for these groups.

in stabilizing the structure and function of mesangial cells (21). The interactions of integrins with various ligands are determined by the specific combination of integrins present. Multiple combinations of αV subunits (αVβ1, αVβ3, αVβ5, αVβ6, αVβ8) interact with arginine-glycine-aspartic acid (37). Drawing on the aforementioned data and findings from genome-wide association studies that have mentioned the genetic influence on kidney diseases. This study aimed to investigate the association between specific polymorphisms in the ITGAV and VEGFA genes and PD characteristics. The results highlighted the potential association of two polymorphisms in VEGFA (rs3025039) and ITGAV (rs833061) genes with PD success and renal Kt/V in ESRD patients, respectively. The lack of significant associations between most of the studied polymorphisms and PD outcomes may be attributed to several factors. First, the sample size was relatively small, which could limit the statistical power to detect associations. Second, PD outcomes are likely influenced by a complex interplay of multiple genetic and environmental factors, and the polymorphisms studied here represent only a small subset of potential genetic determinants. Additionally, variations in treatment protocols, patient adherence, and comorbidities could also confound the observed associations. Despite these limitations, this study contributes to the growing body of evidence on the genetic underpinnings of PD outcomes. By identifying specific genetic markers associated with peritoneal transport characteristics, we can move closer to personalized treatment strategies that consider an individual's genetic makeup. Such personalized approaches could improve patient outcomes by tailoring dialysis protocols to the genetic profiles of patients, thereby optimizing dialysis adequacy and reducing complications. Future research should focus on expanding the sample size and including a broader range of genetic markers to validate and extend these findings. Longitudinal studies are also needed to assess the long-term impact of genetic variations on PD outcomes and to explore potential interactions between genetic and environmental factors. Furthermore, integrating genomic data with clinical and biochemical parameters could provide a more comprehensive understanding of the mechanisms driving variability in PD outcomes.

Conclusion

In conclusion, this study highlights the potential role of VEGFA polymorphisms in influencing peritoneal transport characteristics and dialysis adequacy in PD patients. While ITGAV polymorphisms were not significantly associated with PD outcomes, the findings underscore the complexity of genetic influences on dialysis efficacy. Continued research in this area is essential to develop personalized treatment strategies that enhance patient care and outcomes in ESRD.

Limitations of the study

This study has several limitations. First, our study was conducted within a single population; thus, the results of this research may not be generalizable to other groups due to genetic diversity. Second, this single-center study had a limited sample size, indicating that additional research with a larger number of patients is required for further investigation. Notably, this study focuses on certain polymorphic variations in ITGAV (rs39111238, rs3738919, rs3768777) and VEGFA (rs699947, rs3025039, rs833061). Moreover, further studies with an adequate design are essential to validate our findings and investigate all the genetic variants.

Authors' contribution

Conceptualization: Amirhesam Alirezaei.
Data curation: All authors;
Formal analysis: Amirhesam Alirezaei.
Investigation: Amirhesam Alirezaei, Ali Rostamiasl.
Methodology: Amirhesam Alirezaei, Ali Rostamiasl.
Project administration: Amirhesam Alirezaei, Yadollah Shakiba.
Software: Amirhesam Alirezaei, Ali Rostamiasl.
Supervision: Yadollah Shakiba.
Validation: Yadollah Shakiba.
Visualization: All authors.
Writing–original draft: Ali Rostamiasl and Vesal Tehrani Moayed.
Writing–review and editing: All authors.

Ethical issues

The study was conducted in accordance with the principles outlined in the Declaration of Helsinki. Approval for this study was granted by the appropriate institutional review board, and informed consent was obtained from all participants. All experiments conducted was approved by Shahid Beheshti University of Medical Sciences institutional ethics committee (Ethical code#IR.SBMU.MSP.REC.1400.447). Informed consent to participate was obtained from patients. The authors have fully complied with ethical issues, such as plagiarism, data fabrication, and double publication.

Data aailability statement

The data generated in this study is available on reasonable request from the corresponding author.

Conflicts of interest

The authors declare that they have no competing interests.

Funding/Support

The authors declare that no funding or support were received for the preparation of this manuscript.

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