ATATÜRK ÜNİVERSİTESİ YAYINLARI
ATATURK UNIVERSITY PUBLICATIONS

Research Article DOI: 10.62425/pharmata.1780600

Examining the Relationship Between PWV Change and eGFR Change Over Time in Chronic Kidney Disease Patients

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ABSTRACT

Objective: This study aimed to investigate the longitudinal relationship between pulse wave velocity (PWV), a measure of arterial stiffness, and estimated glomerular filtration rate (eGFR), an indicator of renal function, in patients with chronic kidney disease (CKD).

Methods: A total of 55 CKD patients (Stage 3–5), not on renal replacement therapy, were prospectively followed for approximately four years. PWV and eGFR were measured at baseline and at the end of follow-up. Patients were divided into two groups based on eGFR progression: stable and progressive decline. Demographic, clinical, and laboratory data were collected. The association between changes in PWV (ΔPWV) and eGFR (ΔeGFR) was analyzed.

Results: No significant correlation was found between changes in PWV and changes in eGFR (P>.05). While there was a significant decline in eGFR in the progressive group compared to the stable group (P=.001), ΔPWV did not significantly differ between the groups. Variables such as age, blood pressure, and baseline PWV were not significantly different between groups. Parathormone and phosphorus levels were higher, and hemoglobin and albumin levels were lower in patients with eGFR progression.

Conclusion: Over a four-year follow-up, changes in arterial stiffness (PWV) were not significantly associated with changes in renal function (eGFR) among CKD patients. The lack of correlation suggests that separate pathophysiological mechanisms may govern vascular stiffness and kidney function deterioration. Further studies, including molecular-level analyses, are required to clarify these complex interactions.

Keywords: *Chronic kidney disease, arterial stiffness, pulse wave velocity, glomerular filtration rate, renal function decline, prospective study*

INTRODUCTION

Substantial data are available about the association between worsening renal function and nontraditional risk factors in Chronic Kidney Disease (CKD) patients.[1,2] The relationship between arterial stiffness and worsening kidney function studied by lots of scientist. There are confusing datas in the scientific literature regarding the relationship between arterial stiffness and kidney function. Some studies reported that arterial stiffness may increase as kidney function decreases.[3,4] Conversely, there were studies which reported that increased arterial stiffness can lead to deterioration of kidney function.[5-7] This situation might be explained by limited elasticity effect of stiffened arterial vessels on the pressure of ventricular ejection so that more pressure is transmitted to the end organs. Renal endotelial and smooth muscle cells can be damaged by this high pressure and disrupts the function of vessels, leading to a decline of kidney function.[8]

In similar cross-sectional observational studies conducted before our study, the relationship between Pulse Wave Velocity (PWV) and estimated Glomerular filtration rate (eGFR), was generally examined only at a single time point. Therefore, we believe that the true/exact relationship between arterial stiffness and renal function has not been elucidated. We conducted this prospective study in patients with chronic kidney disease who had not renal replacement treatment, to evaluate whether arterial stiffness change is associated with eGFR change and attempted to identify which factors affect the change in eGFR and PWV.

Received 23.09.2025
Accepted 07.11.2025
Publication Date 26.11.2025

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Cite this article: Karaköse S, Erkuş E, Güney İ. Examining the Relationship Between PWV Charge and eGFR Change Over Time in Chronic Kidney Disease Patients. *Pharmata.* 2025;5(3):77-82

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METHODS

This prospective observational study was conducted at the University of Health Sciences, Konya Research, and Training Hospital. The CKD patients who were followed at the nephrology outpatient clinic and fulfilling the inclusion criterias were accepted to the study. The inclusion criteria for the study were; coming to outpatient clinic follow-up for 4 years, having stage 3-5 CKD, being 18 years of age or older, not having an active infection, not having a cardiovascular disease status, and agreeing to participate in the study.

Exclusion criteria for the study included having Stage 1-2 CKD or receiving renal replacement therapy, dying within 4 years, being under 18 years of age, leaving our center within the 4-year period and not coming for follow-up, not agreeing to participate in the study follow-up, having a vascular disease affecting PWV measurement, and missing the necessary tests for inclusion in the study.

Demographic datas and anthropometric values, blood pressures, and smoking status of the patients were recorded at the beginning of the study. Body mass index (BMI) was calculated by dividing weight to the square of the height (Mosteller RD formula).[9] Blood pressure (BP) measurements were performed by a trained physician using a manual mercury sphygmomanometer (OMRON Helthcare Europe, Hoofddorp, The Netherlands) with the patient in a sitting position and the arm at heart level for at least 5 minutes. BP was measured twice and the average of the two measurements was used for calculation. Hypertension was defined as Systolic BP (SBP) of at least 140 mmHg and/or diastolic BP (DBP) of at least 90 mmHg or reported use of antihypertensive medication. European Society of Hypertension guidelines were applied during blood pressure measurements.[9]

Serum urea, creatinine, sodium, potassium, calcium, phosphorus, parathormone (PTH), albumin, hemoglobin, ferritin, total cholesterol, HDL, LDL, triglyceride, uric acid, CRP, creatine and protein in spot urine were recorded from routine laboratory tests. MDRD (Modification of Diet in Renal Disease) formula was used to calcuate the eGFR (CKD stage 3, eGFR=30-59 mL/ min/1.73 m^2; CKD stage 4, eGFR=15-29 mL/min/1.73 m^2; and CKD stage 5, eGFR <15 mL/min/1.73 m^2).[10]

PWV is a technique in which large-diameter arterial elasticity is assessed by analysis of the peripheral arterial waveform. Blood pressure and age are known determinants of measured arterial PWV. Left ventricular systole produces a pulse wave that propagates along the arterial network and reflects segmental arterial elasticity. Dividing the distance traveled by the pulse wave by the time taken to travel the distance gives the PWV [PWV (m/s) = distance (m)/transit time (s)]. As arterial wall stiffness increases, waves within the lumen travel at a higher speed. PWV can be easily measured along two accessible arterial segments.

Mobil-O-Graph NG (Stemberg Germany Stolberg Germany) arteriography device was used to evaluate the PWV. This device allows the measurement of all arterial stiffness parameters from the upper extremity through the brachial artery by the oscillometric method. The same device was used to detect cardiac output, augmentation index (AIx; @75), total peripheral resistance, and central systolic and diastolic blood pressures. Central blood pressures, AIx, cardiac output, total peripheral resistance and PWV measurements were performed as previously described by Afsar et al.[11]. Cardiac index was calculated by dividing cardiac output by body surface area. The measurements were made after resting for at least five minutes in a room away from external stimuli, which was reserved for the study. In the last hour before the procedure, patients were asked not to take stimulants such as tobacco and coffee.[11,12]

The patients were followed approximately four years. The primary endpoints were renal replacement treatment (renal transplantation, hemodialysis or peritoneal dialysis) and death. During follow-up, patients' medical histories were checked, physical examinations were repeated and blood pressure was measured. At the end of follow up period PWV measurements were repeated using the same techniques and the same protocol. Those with a decrease in eGFR during the 4-year follow-up period were called the eGFR progressive group, and those with no change in eGFR were called the stable group. We investigated the relationship between PWV and eGFR changes in this follow up period.

The analysis of the study was performed by using SPSS 22.0 (IBM SPSS Corp., Armonk, NY, USA) software. The Kolmogorov-Smirnov test was used to determine whether the data was normally distributed or not. The results are given as mean with standard deviation for normal distribution and as median (minimum-maximum) for skewed distribution. In pairwise comparisons, the T-test was used for normal distributions and the Mann Whitney-U test was performed for skewed distributions. The independent predictors of end-stage renal disease (ESRD) and mortality were determined by performing Cox-regression (Backward Stepwise) analysis. The independent predictors were those with *P*<.05 in binary comparisons. Type-I error value was taken as 5% in all analyzes and P values less than .05 were accepted as statistically significant.

RESULTS

Among the 55 patients included in the study, the primary etiological causes of CKD were 9 (16.4%) DM, 18 (32.8%) HT, 6 (10.9%) glomerulonephritis, 1 (1.8%) polycystic kidney disease, 2 (3.6%) amyloidosis, 3 (5.5%) chronic tubulointerstitial nephritis, 2 (3.6%) urological problems and 14 (25.5%) urological problems.

Of the patients included in our study, 19 were male and 36 were female. The mean age of the patients included in the study was 57.29±10.68. The follow-up period of the patients was 48.45±3.98 months. Initial eGFR was 43.62±11 ml/min, PWV was 8.32±1.81m/s. General characteristics of the patients are summarized in Table 1.

The patients were divided into two groups. The first group consisted of 36 patients with stable eGFR during follow-up. The second group consisted of 19 patients with progression in eGFR. When the groups were compared, a statistically significant difference was found between the final eGFRs (*P*=.001). In parallel with this result, a statistically significant difference was found in Δ eGFR between the groups (*P*=.001).

PTH levels were found to be higher in the group with eGFR progression and a statistically significant difference was observed between the groups (*P*=.001). There was no statistically significant difference between the groups with stable eGFR and those with eGFR progression in terms of final PWV and ΔPWV. The results of the parameters examined in the patient follow-ups are given in Table 2.

Table 1: Analysis of Demographic Data

Variables (n= 55)	
Age (years)	57.29±10.68
Gender (male) (n/%)	19 (34.5)
Follow -up time	48.45±3.98
eGFR (ml/min) at the beginning	43.62±11
Pulse wave velocity (m/s)	8.32±1.81
BMI (kg/m^2)	31.63±6.11
Smoking (n/%)	2 (3.6)
DM (n/%)	9 (16.4)
HT	12 (21.8)
BUN	72.04±30.31
Creatinine (mg/dl)	1.92±0.66
Uric acid (mg/dl)	7.16±1.83
Potassium (mmol/L)	4.73±0.54
Calcium (mg/dl)	9.23±0.47
Phosphorus (mg/dl)	3.31±0.53
Albumin (g/L)	4.08±0.25
Ferritine (µg/L) (median, min-max)	87.09 (3-280)
PTH (pg/mL)	150,55 (27.7-799)
Total cholesterol (mg/dl)	203.52±39.15
Trigliseride (mg/dl) (median, min-max)	172.06 (48-679)
LDL (mg/dl)	127.48±37.11
HDL (mg/dl)	42.91±10.74
CRP (mg/dl)	10.25 (3.28-78.21)
Hg (g/dL)	13.25±1.93
SBP (mm/Hg)	131.13±16.52
DBP (mm/Hg)	84.72±10.63

BMI: Body mass ındex DM: Diabetes Melltius HT: Hypertension BUN: Blood urea nitrogen PTH: Parathormone LDL: Low-density lipoprotein HDL: High-density lipoprotein CRP: C-reactive protein Hg: Hemoglobine SBP: Systolic blood pressure DBP: Diastolic blood pressure

There was no correlation between initial eGFR and PWV, final eGFR and PWV, and between Δ PWV and Δ eGFR (Table 3).

DISCUSSION

Although there is a lot of data in the literature on the factors affecting PWV change, there is no clear data examining the effect of eGFR change on Pwv change. In a study conducted by Paolo Meani and colleagues in Italy with 333 hypertensive patients, a significant relationship was observed between increased PWV and uncontrolled blood pressure.[13] In this study, it was emphasized that although there were positive changes in PWV with blood pressure regulation in the 3.7-year follow-up, the patient age factor could also lead to changes in PWV. The researchers did not find any significant difference between the controlled hypertension group and the

uncontrolled hypertension group in terms of age, but they reported a significant difference in terms of PWV.[13] In a similar study conducted by Yang Wang et al. who followed 202 people in China for 4 years, it was found that increasing SBP caused PWV progression.[14] There are several similar studies; in two studies following hypertensive patients, there was an increase in PWV values[15,16] and in one study[17], there was a decrease in PWV values. In a study conducted in 2023, a significant relationship was found between the decrease in eGFR and SBP and pulse pressure, but no relationship was found with DBP.[18] Again, in a study conducted by Heleniak et al. in 2021 in 344 kidney transplant patients, it was found that there was no difference between PWV and eGFR (<45 ml/min or >45 ml/min) values.[19] In 2020, Loutradis et al. found no significant difference between 24-hour PWV measurements in 96 diabetic and non-diabetic patients with stage 2-4 CKD.[20]

Table 2: Analysis of Groups According to eGFR Change Status

Variables	eGFR stable group (n= 36)	eGFR progressive group (n= 19)	*P*
Age (years)	62.75±10.22	61.05±11.53	.590
Gender (male) (n/%)	14 (38.9)	5 (26.4)	.360
Follow up time (months)	47.88±4.76	49.52±1.26	.149
First eGFR (ml/min)	45.57±10.61	39.91±11.17	.071
First PWV (m/s)	8.32±1.87	8.31±1.71	.978
Last eGFR (ml/min)	**45.89±12.05**	**23.85±7.56**	**.001**
Last PWV (m/s)	9.09±1.75	8.96±2.18	.806
ΔPWV (m/s) (numerical, mean±std)	-0.77±1.5	-0.65±0.61	.745
Δ eGFR (ml/min) **(numerical,mean±std)**	**-0.31±8.89**	**16.06±7.31**	**.001**
ΔPWV (m/s) (%) (min-max)	-16.39 (-36 - 33)	-7.15 (-16 - 15)	.478
ΔeGFR (ml/min) **(%) (min-max)**	**-2.39 (-47 - 23)**	**39.46 (25-59)**	**.001**
Creatinine (mg/dl)	**1.87±0.64**	**3.26±1.22**	**.001**
Potassium (mmol/L)	4.81±0.52	4.91±0.53	.472
Calcium (mg/dl)	9.31±0.38	8.97±0.75	.036
Phosphorus (mg/dl)	3.61±0.74	4.33±1.11	.006
Albumin (g/L)	3.91±0.33	3.71±0.36	.045
Ferritine (µg/L) (median, min-max)	90.85±66.09	79.97±57.46	.547
PTH (pg/mL)	**146.29±91.46**	**299.66±173.86**	**.001**
CRP (mg/dl)	11.66±7.58	7.59±7.36	.346
Hg (g/dL)	13.31±2.23	11.61±1.51	.005
SBP (mm/Hg)	123.33±16.15	122.05±17.23	.786
DBP (mm/Hg)	86.13±12.25	84.26±11.12	.581

PTH: Parathormone CRP: C-reactive protein Hg: Hemoglobine SBP: Systolic blood pressure DBP: Diastolic blood pressure

Table 3: Correlation analysis of PWV and eGFR

Variables	r	*P*
Between first eGFR and PWV	0.08	.951
Last eGFR and PWV	0.096	.485
Between Δ PWV (%) and ΔeGFR (%)	0.072	.611

PWV: Pulse wave velocity eGFR: Glomerular filtration rate

In our study, no significant difference was found between the group with eGFR progression and the group without progression in terms of age, PWV, SBP and DBP. The lack of difference in terms of SBP, DBP and age in the groups with and without eGFR progression allowed us to comment more easily on the relationship between eGFR and PWV. Similar findings to our study were found in a study conducted by Kim Chang Seong et al. with 913 patients, when corrected for age and various variables that affect eGFR (gender, mean arterial pressure, diabetes, cerebrovascular disease, smoking history, ejection fraction, hemoglobin, uric acid, hs-CRP, LDL cholesterol levels and ACE inhibitors, ARBs or statin use), no significant relationship was observed between PWV and eGFR. Furthermore, when corrected for concomitant diseases and medications, no significant relationship was observed between the decrease in eGFR and PWV. They reported that a high Pulse Pressure remained associated with rapid kidney function decline.[21]

In a study conducted by Lilitkarntakul et al. with 136 individuals, it was observed that carotid – femoral PWV increased in a correlated manner as the CKD stage increased.[22] Briet et al. reported that, as a result of their study with 180 patients and 3.5 years follow up period, increased carotid stiffness was associated with CKD progression.[2]

In our study, we did not find any significant difference between the last PWV and ΔPWV between the group with increasing eGFR and the groups with stable eGFR at the end of the 4-year follow-up. Although there is no consistency between the results of the studies given above and our study, in a study conducted by Chue et al. in England, patients with Stage 2-3-4 CKD were followed for approximately 3 years, it was observed that there was no significant relationship between the decrease in eGFR and PWV, as in our study.[23]

In our study, there was no statistical difference between the groups in terms of blood pressure and age during fo]llow up period, the effects of which have been reported in many studies on PWV, and this allowed for a more accurate evaluation of the effects of changes in eGFR on PWV compared to other studies. In our study, when the group with stable eGFR and the groups with progressively decreasing eGFR were examined at the end of 4 years of follow-up, it was seen that the difference in PWV between the groups was not significant.

In addition to hemodynamic factors, biochemical parameters such as phosphorus and PTH levels may also play a role in CKD progression. Patients with non-dialysis-dependent (NDD) CKD with hyperphosphatemia account for only 0.3% of total CKD patients.[24] Phosphorus elevation classically occurs when GFR falls below 30 mL/min.[25] Data in the literature support our study, indicating that phosphorus levels were high in the group with eGFR progression, and PTH levels were increased to suppress this and maintain calcium balance. As the CKD stage progresses and eGFR progression progresses, maintaining phosphorus, calcium, and PTH balance becomes more difficult.

Study Limitations

Limitations of the study include the decrease in the number of patients followed over the years, the fact that it was a single-center study, and the inability to record patients' medications due to changes over time.

CONCLUSION

When all these studies are examined, the relationships between PWV and kidney disease appear to be complex and their interactions are not clearly known. There are many factors that affect eGFR and PWV and can cause progression of PWV and CKD through different mechanisms.

The fact that the patients had CKD may have caused arterial stiffness to worsen rapidly and PWV to reach its highest value in a short time. Therefore, in our study, despite the change in eGFR, no significant change in PWV may have been detected. Studies have revealed contradictory findings, making it difficult to obtain clear data. In order to obtain clearer data on this subject, it is thought that studies at the molecular level are required.

Ethics Approval and Consent to Participate: This study has been approved by the Health Sciences University Konya City Hospital Ethics Committee. (Date: 01.08.2019, Approval No. 48929119/774/28-25).

Informed Consent: The datasets used and/or analyzed during the current study were available from the corresponding author on reasonable request.

Peer-review: Externally peer-reviewed.

Author Contributions: SK designed the study. All authors conducted the study. EE collected and analyzed the data. SK and EE participated in drafting the manuscript, and both authors contributed to the critical revision of the manuscript for important intellectual content. All authors gave final approval of the version to be published. All authors participated fully in the work, took public responsibility for appropriate portions of the content, and agreed to be accountable for all aspects of the work in ensuring that questions related to the accuracy or completeness of any part of the work are appropriately investigated and resolved.

Conflict Of Interest: The authors have declared that no conflict of interest exists.

Financial Disclosure: The authors declared that this study has received no financial support.

Use of Artificial Intelligence: No artificial intelligence tools were used.

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