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ORIGINAL ARTICLE

Utilising pulse wave velocity in assessing arterial stiffness in patients with mild-to-moderate chronic kidney disease

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ABSTRACT

Introduction: Arterial damage in patients with chronic kidney disease (CKD) is characterized by increased arterial stiffness, which is associated with increased cardiovascular risk, particularly seen in elderly patients with advanced CKD. However, arterial stiffness among the young early CKD is not clear. The aim of this study is to investigate the presence of arterial stiffness using pulse wave velocity (PWV) in the younger-age adults with stages 2 to 4 CKD. **Methods:** Eighty-seven patients with stages 2-4 CKD and eighty-seven control subjects with normal renal function participated in the study. Demographic details, comorbidities, risk factors, medications as well as blood samples were collected. Arterial stiffness was determined using carotid-femoral PWV. **Results:** The mean age for CKD patients was 47+5.4 years. CKD patients had a higher mean PWV (7.8+1.7 m/s) compared to control subjects (5.6+1.0 m/s) ($p<0.001$, 95% CI -2.59-1.77). A significant difference in mean PWV was also found between patients with stage 2 CKD (7.6+1.5 m/s) and control subjects (5.6+1.0 m/s) ($p<0.001$, 95% CI -2.40, -1.49). A stepwise increase in PWV corresponding to CKD stages was observed ($p<0.001$). Furthermore, significant differences were seen in mean PWV in CKD patients with diabetes (8.2+1.8 m/s) compared to non-diabetic CKD patients (7.3 + 1.3 m/s) ($p=0.022$, 95% CI -1.50, -0.12). Multiple linear regression analysis revealed pulse pressure as an independent predictor of abnormal PWV ($r2=0.249$, $p<0.001$). **Conclusion:** In summary, arterial stiffness occurs early in younger CKD stage 2 patients. Increased arterial stiffness occurs in parallel with a decline in glomerular filtration rate in patients with mild-to-moderate CKD.

1. INTRODUCTION

Chronic kidney disease (CKD) contributes significantly to global health issues, particularly to the increased risk of cardiovascular disease. In this regard, arterial stiffness is recognized as an independent predictor for cardiovascular events in patients with CKD [1-4]. Cardiovascular mortality risk increases 30-fold in end-

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stage renal disease (ESRD) compared to age-adjusted normal population without renal impairment [5]. Increased arterial stiffness has been observed in young patients with end-stage renal disease (ESRD) [6], though literature on arterial stiffness in early CKD is lacking, and current reports reveal inconsistent findings. Although several studies report a link between arterial stiffness and early CKD [7-11], numerous other studies mainly in older subjects have found no significant correlation between arterial stiffness and mild-to-moderate CKD [12-14]. The reason for these discrepancies remains unclear. This study, however, investigates arterial stiffness using pulse wave velocity among younger-aged patients with mild-to-moderate CKD, and at the same time determines the risk factors associated with arterial stiffness.

2. MATERIALS AND METHODS

Patients with CKD stages 2 to 4 were recruited from the cardiology clinic, Universiti Teknologi MARA, Sungai Buloh, Malaysia. The inclusion criteria consisted of those aged 18 to 54 years with confirmed CKD, stages 2 to 4, in accordance with the criteria from the Clinical Practice Guidelines for CKD from the National Kidney Foundation–Kidney Disease Outcomes Quality Initiative [estimated glomerular filtration rate (eGFR) >15 to <90 ml/min/1.73m2 derived from CKD Epidemiology Collaboration (CKD-EPI) formula] 15. The exclusion criteria consisted of patients with acute coronary syndrome within three months of study, chronic inflammatory disease, atrial fibrillation, complete heart block, aortic or femoral artery grafts, history of renal transplant, and pregnancy.

The control group was recruited from the family medicine clinic of the same institution, and consisted of only those with normal estimated glomerular filtration rate (eGFR) > 90 ml/min/1.73m2 derived from CKD Epidemiology Collaboration (CKD-EPI) formula, without a history of diabetes mellitus (defined as fasting venous plasma glucose values no greater than 7.0 mmol/L and random glucose values no greater than 11.0 mmol/L, and not on antidiabetic medication), hypertension (defined as systolic blood pressure of < 140 mmHg or and/or diastolic blood pressure of <90 mmHg, and not on any antihypertensive medication), no history of cardiovascular disease events, such as acute coronary syndrome or history of revascularization or positive diagnostic tests (e.g. exercise stress test, dobutamine stress echocardiogram and/or computed tomography angiography), were non-smokers. Comorbidities, concurrent medications, socio-demographic and anthropometric information was collected upon recruitment from all participants. Blood and urine samples were collected from each subject prior to PWV measurement to determine serum creatinine, glycated hemoglobin (HbA1C), total cholesterol, triglyceride, LDL-cholesterol, HDL-cholesterol and urine protein-creatinine ratio (UPCR).

A carotid-femoral PWV (cfPWV) measurement was performed on each patient to assess arterial stiffness. Blood pressure measurement: After at least five minutes of rest, blood pressure was taken using the Omron HBP sphygmomanometer (Model 1120-E, China) while seated and reported as the mean of three measures. Prior to the investigation, and in accordance with the calibration schedule, the Omron HBP T-105 sphygmomanometer was calibrated. blood pressure's systolic (SBP) and diastolic (DBP) differences measurements was used to compute pulse pressure (PP). Mean blood pressure (MAP) was calculated using the formula MAP = DBP+PP/3. For PWV measurement, a skilled medical laboratory technician at the non-invasive cardiac laboratory used the SphygmoCor XCEL PWV + PWA (Model EM4C, Australia) to automatically measure the carotid-femoral PWV, which was used to evaluate arterial stiffness. A thigh blood pressure cuff was applied over the patient's right femoral artery area. The thigh cuff will inflate automatically once a reliable signal from carotid tonometer is detected. Length of the common carotid artery to sternal notch, sternal notch to femoral pulse and femoral pulse to femoral cuff were measured in millimeters. These parameters were recorded into the computerized database. Using an arterial tonometer, transcutaneous recordings of pulse waveforms were performed over the common carotid artery. The PWV was automatically calculated by the PWV device as L/t (in m sec-1), where L is the distance between recording sites recorded over the body's surface and t, the time interval between the pressure wave feet.

Statistical analyses were performed using either t-test, One-Way ANOVA, or Chi-Square testing for categorical variables contained in the Statistical Package for the Social Sciences (SPSS Version 22.0, SPSS

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Inc., Chicago, IL, USA). Correlations between two continuous variables were analysed using Pearson rho test. For variables that had a significant degree of correlation, simple linear regression analysis was done. A multivariate linear regression analysis, which included the variables previously chosen in the linear univariate regression was carried out to assess the variables that were independently linked with PWV. Except where otherwise noted, $p<0.05$ was used as the significance level. Sociodemographic data of the patients are presented as mean + standard deviations, and percentages were used to represent categorical data.

3. RESULTS

Table 1 lists the baseline characteristics of the study population. The mean age of patients with CKD was 47 + 5.4 years. Half (52%) of the CKD patients were obese, class 1 (BMI: 27.5 – 32.4 kg/m2) according to Malaysian Obesity Guidelines 2023 that has adopted Asian population-specific classification (16). Investigation results and medications usage are also shown in Table 1.

Table 1. Sociodemographic, biochemical profile and medication usage of controls and CKD patients

Variables	Controls (n=87)	CKD 2 (n=39)	CKD 3 (n=39)	CKD 4 (n=9)	P-value
Age (years)	45 ± 8	47 ± 6	48 ± 6	48 ± 3	0.422
Male sex (%)	69	97	72	67	0.005
BMI (kg/m^2)	28 ± 3	30 ± 5	29 ± 5	30 ± 6	0.195
SBP (mm/Hg)	120 ± 12	133 ± 17	141 ± 22	152 ± 27	< 0.001
DBP (mm/Hg)	73 ± 8	82 ± 12	82 ± 14	89 ± 11	< 0.001
MAP (mm/Hg)	89 ± 9	99 ± 14	102 ± 16	110 ± 15	< 0.001
PP (mm/Hg)	47 ± 8	51 ± 8	59 ± 13	63 ± 20	< 0.001
HR (bpm)	72 ± 15	69 ± 14	75 ± 12	79 ± 15	0.191
Hypertension (%)	-	95	95	100	0.785
DM (%)	-	41	69	67	0.034
IHD (%)	-	72	62	78	0.498
Smoking (%)	-	62	46	44	0.343
Creatinine (µmol/l)	78 ± 8	106 ± 8	137 ± 24	259 ± 37	< 0.001
eGFR (ml/min per 1.73 m^2)	95 ± 8	72 ± 6	50 ± 7	22 ± 5	< 0.001
HbA1C (%)	6.1 ± 3.1	7.1 ± 2.3 (n=28)	10.0 ± 2.9 (n=24)	8.0 ± 2.7 (n=7)	0.001
Total-C (mmol/l)	4.2 ± 1.2	4.4 ± 1.0	5.0 ± 1.7	5.8 ± 1.4	0.028
TG (mmol/l)	1.6 ± 0.5	1.7 ± 0.8	2.4 ± 1.2	2.6 ± 1.1	0.010
LDL-C (mmol/l)	2.3 ± 1.2	2.6 ± 1.1	2.8 ± 1.4	3.4 ± 0.9	0.344
HDL-C (mmol/l)	1.0 ± 0.2	1.0 ± 0.2	1.1 ± 0.4	1.0 ± 0.2	0.252
UPCR (g/mmol)	0.02 ± 0.05	0.04 ± 0.06	0.15 ± 0.21	0.10 ± 0.16	0.014

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Antihypertensive therapy (%)	-	95	95	100	0.785
Number of antihypertensive	-	2.6 ± 1.2	2.4 ± 1.2	2.7 ± 1.5	0.729
RAS blocker (%)	-	84	87	55	0.071
ACE inhibitor (%)	-	56	58	55	0.999
ARB (%)	-	28	31	0	0.159
CCB (%)	-	39	51	56	0.436
Beta Blocker (%)	-	80	56	56	0.073
Diuretics (%)	-	36	33	67	0.167
Statin (%)	-	97	92	89	0.477

Data are expressed as mean ± SD for the continuous variables and as percentage for categorical variables. Data are analysed using One-Way ANOVA for continuous variables and Chi-Square for categorical variables.
ACE = angiotensin-converting enzyme; ARB = angiotensin-receptor blocker; BMI = body mass index; C = cholesterol; CCB = calcium-channel blocker; CKD = chronic kidney disease; DBP = diastolic blood pressure; DM = diabetes mellitus; eGFR = estimated glomerular filtration rate; HbA1C = glycated haemoglobin; HDL = high density lipoprotein; HR = heart rate; IHD = ischaemic heart disease; LDL = low density lipoprotein; PP = pulse pressure; RAS= renin-angiotensin system; SBP = systolic blood pressure; TG = triglyceride; UPCR = urine protein-creatinine ratio

Source: Ahmad Bakhtiar Md Radzi (2026)

Renin-angiotensin system (RAS) blockers including ACE inhibitors or angiotensin receptor blockers (ARBs), calcium-channel blockers, beta-adrenergic blockers, and diuretics were among the anti-hypertensive drugs taken by CKD patients. A comparison of 87 individuals with stages 2-4 CKD and 87 control participants with normal renal function was made. The CKD group's mean PWV was significantly greater than that of the control group, indicating increased arterial stiffness in the former (Table 2).

Table 2. PWV between control and CKD stage 2-4 patients

Variables	**Control (n=87)**	**CKD 2-4 (n=87)**	**P-value**
PWV (m/s)	5.6 ± 1.0	7.8 ± 1.7	< 0.001

Data are expressed as mean + SD for the continuous variables and as percentage for categorical variables. Data are analysed using independent T-test for continuous variables and Chi-Square for categorical variables.

Source: Ahmad Bakhtiar Md Radzi (2026)

The box-plot analysis in Figure 1 ($p<0.001$) shows a gradual rise in PWV from control subjects (5.6 + 1.0 m/s), CKD stage 2 (7.6 + 1.5 m/s), stage 3 (7.8 + 1.8 m/s), and stage 4 (9.0 + 0.8 m/s) (Table 3). There was a significant difference in the mean PWV between stage 2 CKD patients and controls ($p<0.001$, 95% CI -2.40,-1.49). In stages 2 - 4 CKD, the mean PWV was significantly different between diabetic (8.2 + 1.8 m/s) and non-diabetic (7.3 + 1.3 m/s) populations ($p<0.05$, 95% CI -1.50,-0.12). Age, MAP, PP, and eGFR were significantly and positively associated with PWV among patients with stages 2-4 CKD (Table 4). Only PP was found to be independently linked with PWV ($r2=0.078$; $p<0.05$) in the multivariate analysis using multiple linear regression ($r2=0.248$; $p<0.001$) (Figure 2).

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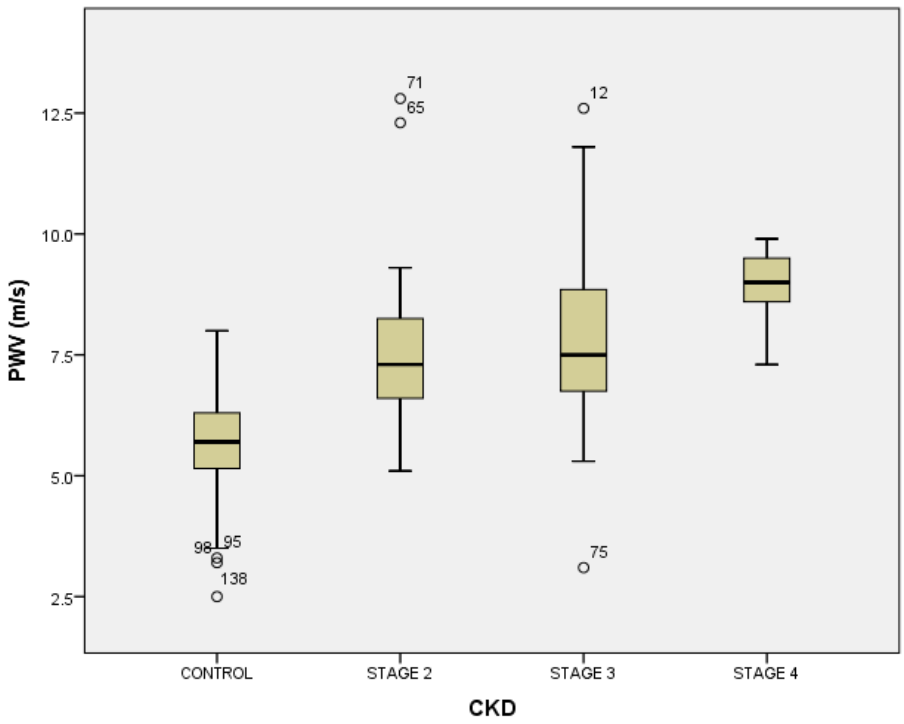

Fig.1 PWV between control and CKD stage 2-4 patients

Source: Ahmad Bakhtiar Md Radzi (2026)

Table 3. Mean of PWV between control and CKD stage 2-4 patients

Variables	Control (n=87)	CKD Stage 2 (n=39)	CKD Stage 3 (n=39)	CKD Stage 4 (n=9)	P-value
PWV (m/s)	5.6 ± 1.0	7.6 ± 1.5	7.8 ± 1.8	9.0 ± 0.8	<0.001

Source: Ahmad Bakhtiar Md Radzi (2026)

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Table 4. Factors associated with PWV (m/s) among CKD stage 2-4 patients (n=87) using simple and multiple linear regression

Variable	SLR[α]				MLR[b]	
	β	r^2	95% CI	p-value	β	p-value
Age (year)	0.066	0.048	0.00, 0.13	0.042	0.042	0.186
MAP (mmHg)	0.033	0.087	0.01, 0.06	0.006	0.008	0.556
Pulse pressure (mmHg)	0.060	0.219	0.31, 0.08	<0.001	0.045	0.009
BMI	0.021	0.004	-0.05, 0.09	0.564	-	-
eGFR (ml/min per 1.73m^2)	-0.027	0.079	-0.04, -0.01	0.008	-0.011	0.276
LDL (mmol/l)	0.135	0.010	-0.18, 0.45	0.400	-	-
TG (mmol/l)	0.014	0.000	-0.33, 0.36	0.936	-	-
Hba1C (%)	0.016	0.001	-0.13, 0.16	0.836	-	-
UPCR (g/mmol)	0.777	0.006	-1.58, 3.14	0.514	-	-

α = Simple linear regression (Outcome as PWV m/s)
b = Multiple linear regression (Outcome as PWV m/s), r^2=0.248, p<0.001
β = crude regression coefficient
Source: Ahmad Bakhtiar Md Radzi (2026)

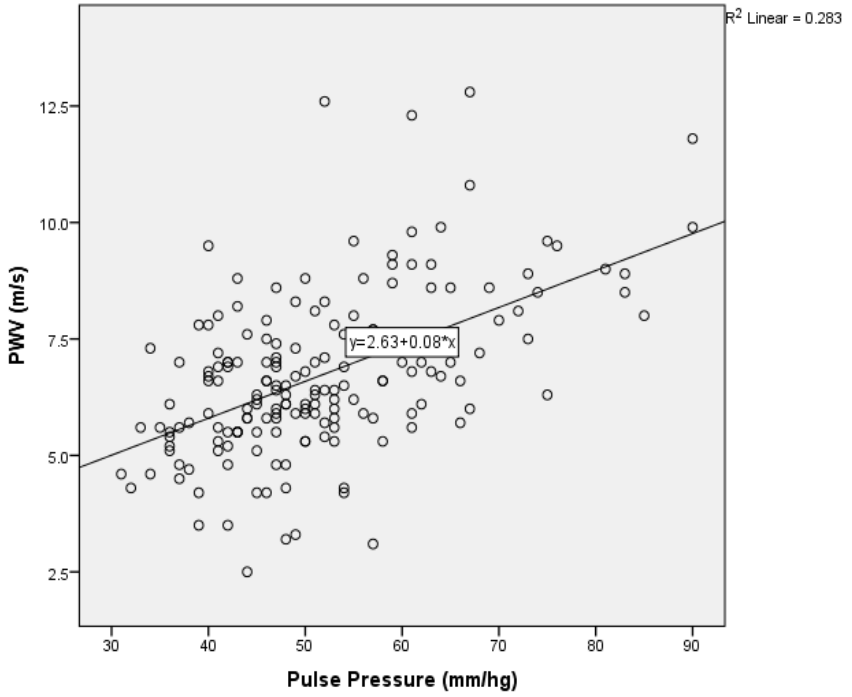

Fig 2. Correlation between PWV and PP in CKD stage 2-4 patients

Source: Ahmad Bakhtiar Md Radzi (2026)

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4. DISCUSSION

In this study, increased arterial stiffness, as determined by measurement of PWV, was observed in younger age patients from as early as stage 2 CKD. Our findings are in line with earlier reports, mostly from studies on older CKD patients, that had reported a link between elevated central arterial stiffness and decreased creatinine clearance. A study on a group of 150 CKD patients (65 + 11 years), e.g. showed higher adjusted PWV than that in healthy individuals, regardless of disease stage [7]. Another study in patients with a mean age of 58.4 + 14.9 years, found that stiffness of the aorta, as measured by cfPWV, was considerably higher in mild-to-moderate CKD patients (GFR<60 ml/min/1.73m2 calculated using MDRD) than that in hypertensive and normotensive participants by 7% and 19% respectively [8]. Furthermore, aortic stiffness (cfPWV) was correlated independently with the pace of renal function decrease in a sample of 120 individuals aged 69 + 12 years with CKD stages 3 and 4 [10]. In another study of 1290 participants (49 + 11 years) with a mixed group of normal and impaired kidney function, individuals in the lowest tertile of kidney function (creatinine clearance 68.5 + 15.6 mL/min/1.73 m2) demonstrated an inverse relationship between creatinine clearance and aortic PWV [11]. The results showed that elevated arterial stiffness manifests early in CKD patients, which is consistent with our study's findings in a cohort of comparatively younger CKD participants.

Our findings are consistent with those in the literature that indicate a link between arterial stiffness and mild-to-moderate CKD. However, some researchers have reported inconsistent findings regarding the relationship between arterial stiffness and renal function. For instance, the Framingham Heart Study, which included 2680 people, found no link between PWV and a decline in eGFR in people with mild-to-moderate chronic kidney disease (defined as having an eGFR of less than 60 ml/min per 1.73 m2) [12]. Most of the participants in this study were community-dwelling people with stage 3 CKD and a mean age of 70. Similar results were found in the Hoorn Study, which found no significant difference in PWV between patients with an eGFR of less than 56 ml/min/1.73 m2 and those with greater 56 ml/min/1.73 m2 [13]. The participants in this study were senior individuals with diabetes, and cfPWV was not used to measure arterial stiffness. Carotid-femoral transit time, which measures aortic (thoracic-abdominal) compliance, was used to quantify aortic stiffness instead. Another study with 913 patients aged 62.5 ± 10 years found that after controlling for various confounders, arterial stiffness, measured using cfPWV and brachial-ankle PWV (baPWV), was not associated with lower eGFR in populations with relatively preserved kidney function (estimated GFR 30 mL/min/1.73 m2) [14].

Overall, discrepancies in research populations, notably disparities in the groups of CKD patients, could partially account for the contradictory results between these studies including our investigation. Our study's findings, which are in line with those of research involving older CKD patients, indicate a stepwise increase in PWV from control normal to CKD stage 2 through stage 4 patients. BaPWV increased progressively with CKD progression from stages 1 through to stage 5 according to a Japanese study that looked at the association between eGFR and the severity of arterial stiffness in the population [17]. Similarly, a Taiwanese study revealed a stepwise increase in PWV from CKD stage 1 through to stage 5, but did not find a significant difference in PWV between patients in CKD stages 3, 4 and 5 [9]. Another study also demonstrated similar stepwise results but did not observe any further increases in PWV from CKD stages 4, 5 to CKD 5D (dialysis) [7]. According to Mourad et al. (2001) [11] only the lowest tertile group of patients with normal and elevated blood pressure (eGFR 68.5 + 15.6 ml/min per 1.73 m2), but not the intermediate tertile group (eGFR 89.4 + 17.4 ml/min per 1.73 m2) demonstrated a strong inverse relationship between PWV and creatinine clearance [11]. There was a tendency of little to no progression of PWV at stages 3, 4 and above. A definite conclusion cannot be made regarding the increment of PWV as renal function declines, which therefore warrants a larger prospective study to address this theory. When compared to patients with normal renal function, people with CKD stages 3 and above have a two-fold greater mortality rate from cardiovascular disease [18].

In the present study, stiffening of the arteries was noted to occur as early as stage 2 CKD. Therefore, CKD stage 2 may be considered as a non-traditional risk marker for cardiovascular event and should be

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considered in high-risk individuals for primary prevention. One of the risk factors for the onset of arterial stiffness in the early stages of CKD may be the micro uremic milieu although significant uremia only becomes apparent in the later stages of CKD. Some of the suggested causes for increased arterial stiffness in CKD patients include chronic inflammation, elevated oxidative stress, abnormalities of calcium-phosphate metabolism, activation of the renin angiotensin system, and fluid volume retention [19-20]. High carotid-femoral PWV in young adults also correlated with other vascular indicators such as augmentation index (AIx), carotid intima media thickness (CIMT), and C-reactive protein (CRP), which may have contributed to the above-mentioned probable mechanisms [21].

4.1 Study Limitations

The current study has certain limitations. It mainly focused on mild-to-moderate CKD population with diabetes mellitus and hypertension in which it compared these patients with those with normal renal function who are non-diabetic and normotensive. This does partly contribute to the confounding variables and makes it a little difficult to directly link the increasing PWV with kidney disease. Ideally, the inclusion of a group with patients who were hypertensive and diabetic but without kidney disease would have been useful to clearly establish the effect of renal disease per se on PWV. Nevertheless, multivariate analysis did show significant link between renal disease and PWV, after considering these variables.

The other limitation was that the number of patients with stage 4 CKD was relatively small compared to that with stage 2 and 3 CKD patients. Besides that, the study participants were recruited from a single centre, which may introduce a sampling bias. Patients on antihypertensive medications may have also affected the mean arterial pressure measurements and thus influenced the overall results. Finally, with GFR values of 60 ml/min/1.73 m2 or higher, the precision of GFR based on creatinine may not be accurate. Future research that calculates the GFR using a more precise and trustworthy marker, such as plasma cystatin C levels, is, therefore necessary.

5. CONCLUSION

This study revealed that arterial stiffness becomes evident in the younger-age CKD patients with hypertension and diabetes from as early as stage 2 CKD. Increased arterial stiffness occurs in parallel with a decline in glomerular filtration rate in patients with mild-to-moderate CKD. Whether the changes in PWV in patients with CKD are exaggerated in the presence of hypertension and diabetes remains to be clearly established.

6. ACKNOWLEDGEMENTS/FUNDING

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7. CONFLICT OF INTEREST

Authors declare none.

8. AUTHORS' CONTRIBUTION

Writing – original draft: Ahmad Bakhtiar Md Radzi.
Writing – review & editing: Ahmad
Bakhtiar Md Radzi, Sazzli Shahlan Kasim.

9. ETHICS STATEMENT

The study was approved by the institutional research ethics committee (600-RMI-5/1/6) and written informed consents were received from all participants.

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