



Article

Arterial Stiffness in Kidney Transplant Recipients: A Cross-Sectional Tunisian Study

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Abstract

Background: Arterial stiffness assessed by measuring pulse wave velocity (PWV) is a well-established predictor of cardiovascular mortality. To our knowledge, no studies on arterial stiffness in kidney transplant recipients (KTRs) from Tunisia have been conducted. The present study aimed to assess arterial stiffness in Tunisian KTRs and to identify the key predictors associated with its increase. **Methods:** We conducted a cross-sectional, single-center study enrolling Tunisian KTRs aged 18 years or older with a minimum post-transplant follow-up of six months. Arterial stiffness was measured as pulse carotid–femoral PWV (CF-PWV) by a Complior device. A CF-PWV ≥ 10 m/s was defined as elevated. **Results:** Fifty-four KTRs were included (mean age: 42.55 ± 10.61 years). Among them, 19 (35.2%) had a CF-PWV ≥ 10 m/s. The univariate analysis showed a significant association between elevated CF-PWV and the following parameters: age, hypertension prior to transplantation, dyslipidemia, donor age, parameters obtained through office blood pressure measurement (systolic blood pressure (SBP), diastolic blood pressure (DBP), and pulse pressure (PP)), central SBP recorded by the Complior device, nocturnal SBP obtained through 24 h ambulatory blood pressure monitoring (ABPM), and fasting blood glucose. A multivariable analysis with CF-PWV ≥ 10 m/s as a dependent variable retained the following independent factors: dyslipidemia ($p = 0.015$; OR = 60.32), donor age ($p = 0.014$; OR = 1.16), SBP obtained through office blood pressure measurement ($p = 0.015$; OR = 1.25), and fasting blood glucose ($p = 0.034$; OR = 22.35). **Conclusions:** Given the major impact of cardiovascular disease on post-transplant outcomes, understanding the determinants of arterial stiffness is crucial for improving patient care. Routine PWV assessment may not be feasible in all centers due to cost or limited equipment availability. Therefore, identifying the clinical and biological markers associated with arterial stiffness offers a low-cost and widely accessible alternative for evaluating cardiovascular risk. These findings may support the development of a simple risk score to help nephrologists detect and manage high-risk KTRs more effectively.



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Keywords: arterial stiffness; pulse wave velocity; kidney transplant recipients; cross-sectional study; North Africa

1. Introduction

Kidney transplantation (KT) remains the gold standard treatment for patients with end-stage renal disease (ESRD), offering improved survival, quality of life, and cost-effectiveness compared to dialysis [1,2]. Globally, the burden of chronic kidney disease (CKD) is rising, with an estimated prevalence of 13.4%, and its progression to ESRD poses significant clinical and economic challenges, particularly in low- and middle-income countries [3]. In Europe and North Africa, access to transplantation and pre-transplant care varies widely, influenced by infrastructure, technological capacity, and healthcare policies [4,5]. Recent innovations in dialysis management, including remote monitoring and wearable technologies, have significantly enhanced pre-transplant care making it more personalized, accessible, and effective. These innovations hold promise for improving patient outcomes and quality of life [6]. Concurrently, the adoption of multidisciplinary care models and refined nurse–patient communication strategies has demonstrated measurable improvements in nephrology outcomes [7]. Together, these technological and relational advancements form the cornerstone of contemporary CKD management, which prioritizes early risk stratification, cardiovascular protection, and individualized treatment pathways [8]. Despite these improvements, cardiovascular disease remains the leading cause of mortality post-transplantation [2]. Among the non-invasive predictors of cardiovascular risk, arterial stiffness measured via pulse wave velocity (PWV) has gained recognition as a well-established predictor of cardiovascular mortality [9]. While several studies have investigated changes in arterial stiffness before and after KT, few have explored the factors associated with increased PWV in stable KTRs, and to our knowledge, none have addressed this issue in North Africa [10]. The primary aim of this study was to assess arterial stiffness in a group of KTRs in a North African setting.

Based on this objective, the following research questions were formulated:

What is the prevalence of elevated arterial stiffness in this population?

Which clinical and biological factors are associated with increased arterial stiffness?

2. Materials and Methods

2.1. Study Population

We conducted a cross-sectional, single-center study at Rabta University Hospital in Tunisia between March and October 2024, enrolling KTRs who met the following inclusion criteria:

- Age ≥ 18 years;
- Time from KT ≥ 6 months.

Exclusion criteria included the following:

- A history of acute or chronic rejection;
- Infectious complications within the past month;
- Surgery within the last month;
- Cardiovascular complications within the last three months;
- Malignant tumors;
- Acute renal failure within the past three months;
- Heart failure or arrhythmias;
- End-stage renal disease.

Additionally, patients who declined to participate in the study were excluded.

The patients eligible for the study were informed about the study's objectives, and they gave their written consent to participate.

2.2. Study Design

This study was reported in accordance with the STROBE (Strengthening the Reporting of Observational Studies in Epidemiology) guidelines for cross-sectional studies [11]. The completed STROBE checklist is provided as Supplementary File S1.

The study protocol was approved by the local Research Ethics Committee of Rabta University Hospital (05476338).

The investigation was conducted in accordance with the principles outlined in the Declaration of Helsinki.

Physical examination, carotid–femoral PWV (CF-PWV) measurement, and biological assessments were performed on the same day, during routine follow-up visits to our department.

Data on KTRs and their donors were collected from medical records.

Arterial blood pressure was assessed using both office blood pressure measurements and 24 h ambulatory blood pressure monitoring (ABPM).

Body mass index (BMI) was calculated by dividing body weight (kg) by the square of height (m²). Post-transplant diabetes mellitus was defined according to the 2024 international consensus guidelines [12].

2.3. Office Blood Pressure Measurement

Peripheral blood pressure was measured in the dominant arm or the non-fistula arm using a validated oscillometric device (Omron M6 Comfort (HEM-7221-E), Kyoto, Japan), in accordance with the European Society of Hypertension (ESH) recommendations [13]. Measurements were taken with the patient in a supine and relaxed position after at least five minutes of rest. Three consecutive readings were recorded. The final blood pressure value was calculated as the average of the measurements.

2.4. Arterial Stiffness Measurement

All CF-PWV measurements were carried out by two experienced physicians using a Complior device (Complior[®] Analyse, Alam Medical, France, (REF: VAB00002)). Complior was made available to us by the Research Laboratory LR00SP01, Charles Nicolle University Hospital, Tunis Tunisia.

The assessments took place in a quiet room after the patient had rested in a supine position for 15 min. Two sensors were positioned: one at a palpable pulse point on the carotid artery and the other at a palpable pulse point on the femoral artery. The distance (d) between the carotid and femoral pulse points was measured. The subtraction method was employed to determine the carotid–femoral distance (d) by subtracting the distance between the carotid artery and the suprasternal notch from the distance between the suprasternal notch and the femoral artery. The Complior device automatically recorded the time (t) between the onset of the pulse wave at the carotid artery and its arrival at the femoral artery. CF-PWV was calculated by the device using the formula $PWV = d/t$ [14].

The central systolic (cSBP) and central diastolic blood pressure (cDBP) values were recorded using the Complior device.

The included patients were categorized into two groups based on an estimated cut-off value of 10 m/s, which corresponds to a high cardiovascular risk according to the European Society of Hypertension (ESH) guidelines [13]. A CF-PWV ≥ 10 m/s was defined as elevated.

2.5. Laboratory Analyses

Laboratory results, including serum creatinine, hemoglobin, calcium, phosphorus, fasting blood glucose, glycated haemoglobin (HbA1c), uric acid, cholesterol, HDL-cholesterol, triglyceride, intact parathyroid hormone (iPTH), complete blood count, calcineurin in-

hibitors trough level, and the spot urine protein–creatinine ratio (UPCR), were measured on the same day as the PWV assessment.

Blood samples presented in this study were drawn in the morning after a 12 h fasting period. Creatinine was analyzed by Jaffe’s colorimetric kinetic method.

The estimated glomerular filtration rate (mL/min/1.73 m² SC) was calculated with the MDRD formula [8].

2.6. Statistical Analysis

Analyses were performed using SPSS version 24. Quantitative variables with a normal distribution were expressed as means $\pm$ standard deviation (SD), while those with a non-normal distribution were presented as medians with interquartile ranges. Normality was assessed using the Kolmogorov–Smirnov test. Qualitative variables were described as frequencies (percentages). The comparison of qualitative variables was performed using the Chi-square test. For quantitative variables, the Student’s *t*-test or the Mann–Whitney U test was used, depending on the data distribution. A binary logistic regression was performed to identify factors independently associated with elevated VOP. Variables were selected for inclusion in the multivariate model based on univariate analysis results ($p < 0.05$). A backward stepwise approach was used, starting with all preselected variables and progressively removing the least significant ones. Model fit was assessed using the Hosmer–Lemeshow test. Adjusted odds ratios (aOR) with 95% confidence intervals were reported. A p -value < 0.05 was considered statistically significant for all analyses.

3. Results

3.1. Characteristics of the Study Population

Among 120 patients who underwent transplantation in our department, 54 KTRs were included. The demographic characteristics and comorbidities of the study population are presented in Table 1.

Table 1. Characteristics of kidney transplant recipients.

Characteristics	Value
Time on dialysis before transplant, mean $\pm$ SD, months	27.5 [17–39.75]
Age, mean $\pm$ SD, years	42.55 $\pm$ 10.61
Gender ratio (male to female)	2.6
Smoking before transplant, <i>n</i> (%)	15 (27.8%)
Smoking after transplant, <i>n</i> (%)	3 (5.6%)
Hypertension, <i>n</i> (%)	35 (64.8%)
Hypertension prior to transplantation, <i>n</i> (%)	34 (38.9%)
New onset hypertension, <i>n</i> (%)	6 (11.1%)
Diabetes, <i>n</i> (%)	21 (38.9%)
Diabetes prior to transplantation, <i>n</i> (%)	5 (9.3%)
Post-transplant diabetes mellitus, <i>n</i> (%)	16 (29.6%)
Dyslipidemia, <i>n</i> (%)	16 (29.6%)

SD: Standard deviation.

The etiologies of kidney disease were as follows: chronic glomerulonephritis in 22 patients (40.7%), diabetes in 5 patients (9.2%), chronic tubulointerstitial nephritis in 15 patients (27.8%), unknown in 10 patients (18.5%), hereditary nephritis in 4 patients (7.4%), and hypertensive nephropathy in 3 patients (5.9%).

Among included patients, 35 (64.8%) had been on hemodialysis, and 17 (31.3%) had been on peritoneal dialysis prior to transplantation, with a median dialysis duration of 27.5 months (17–39.75). Preemptive transplantation was performed in two patients (3.7%).

Among diabetic patients prior to transplantation, three (5.6%) had type 1 diabetes.

Patients with diabetes received insulin therapy in 9 cases, oral antidiabetic medication in 9 cases, and a combination of both insulin and oral antidiabetics in 3 cases.

Five patients achieved blood pressure control without the need for antihypertensive medications following transplantation.

The immunosuppressive medication included 10 mg per day of prednisone ($n = 54$; 100%), Tacrolimus

($n = 43$; 79.6%), Cyclosporin A ($n = 7$; 12.9%), mycophenolate mofetil

($n = 53$; 98.1%), sirolimus ($n = 4$; 7.4%) and azathioprine ($n = 1$; 1.8%).

All included patients had calcineurin inhibitor trough levels within the therapeutic target range.

3.1.1. Donor Characteristics

A total of 51 patients (94.4%) received a kidney from a living related donor, and 3 patients (5.4%) received a deceased donor graft.

The mean age of donors was 47.07 ± 12.3 years.

3.1.2. CF-PWV Measurement

The mean CF-PWV was 8.8 ± 2 m/s. Nineteen patients (35.2%) had a CF-PWV ≥ 10 m/s.

3.2. Factors Associated with Elevated CF-PWV

3.2.1. Univariate Analysis

High CF- PWV was significantly associated with age, hypertension prior to transplantation, dyslipidemia, donor age (Table 2), parameters obtained through office blood pressure measurement (systolic blood pressure (SBP), diastolic blood pressure (DBP), and pulse pressure (PP)), cSBP recorded by the Complior device, nocturnal SBP obtained through 24 h ABPM (Table 3), and fasting blood glucose (Table 4).

Table 2. Demographic and clinical data according to pulse wave velocity.

Variables	CF-PWV < 10	CF-PWV ≥ 10 m/s	<i>p</i> Value
Time on dialysis before transplant, median [Q1–Q3], months	23 [17–36]	35 [15–77]	0.068
Age, mean $\pm$ SD, years	39.54 ± 9.75	48.10 ± 10.07	0.004
Gender			
Male, <i>n</i> (%)	27 (77.1)	12 (63.2)	0.273
Female, <i>n</i> (%)	8 (22.9)	7 (36.8)	
Smoking, <i>n</i> (%)	13 (37.1)	6 (31.6)	0.638
Hypertension, <i>n</i> (%)	20 (57.1)	15 (78.9)	0.109
Hypertension prior to transplantation, <i>n</i> (%)	17 (48.6)	17 (89.5)	0.003
Diabetes, <i>n</i> (%)	10 (29.4)	10 (52.6)	0.094
Diabetes prior to transplantation, <i>n</i> (%)	2 (5.7)	3 (15.8)	0.332
Post-transplant diabetes mellitus, <i>n</i> (%)	9 (25.7)	7 (36.8)	0.392
Dyslipidemia, <i>n</i> (%)	7 (20.0)	9 (47.4)	0.035

Table 2. Cont.

Variables	CF-PWV < 10	CF-PWV ≥ 10 m/s	<i>p</i> Value
BMI, mean ± SD, kg/m ²	26.09 ± 4.63	25.68 ± 3.56	0.742
Donor Age, mean ± SD, years	44.22 ± 12.73	52.31 ± 12.73	0.024

CF-PWV: carotid–femoral pulse wave velocity; SD: Standard deviation; BMI: Body mass index.

Table 3. Univariate comparison of renal transplant recipients according to pulse wave velocity and arterial blood pressure parameters.

Parameter	All	CF-PWV < 10	CF-PWV ≥ 10	<i>p</i> Value
Office blood pressure measurement				
SBP, mean ± SD, mm Hg	128.03 ± 16.70	121.05 ± 11.55	140.89 ± 17.31	0.001
DBP, mean ± SD, mm Hg	83.90 ± 12.59	80.40 ± 11.12	90.36 ± 12.87	0.008
MBP, mean ± SD, mm Hg	97.38 ± 14.03	93.60 ± 10.94	104.36 ± 16.57	0.006
PP, mean ± SD, mm Hg	43.74 ± 11.72	40.05 ± 8.81	50.52 ± 13.53	0.001
cSBP, median [Q1–Q3], mm Hg	131 [124.75–138]	110 [106–122]	127 [116–127]	0.001
cDBP, mean ± SD, mm Hg	79.90 ± 11.53	88.06 ± 11.87	131 [124.75–138]	0.068
24 h ABPM				
Global SBP, mean ± SD, mm Hg	131.39 ± 12.48	128 ± 10.1	137.2 ± 14.2	0.026
Global DBP, mean ± SD, mm Hg	80.78 ± 8.13	79.8 ± 7.6	82.4 ± 8.8	0.350
Global PP, mean ± SD, mm Hg	50.4 ± 8.7	54.7 ± 11.5	47.9 ± 5.5	0.018
Diurnal SBP, mean ± SD, mm Hg	132.68 ± 13.14	129.7 ± 10.5	137.6 ± 15.9	0.750
Diurnal DBP, mean ± SD, mm Hg	82.21 ± 8.36	81.5 ± 7.6	83.4 ± 9.7	0.5
Nocturnal SBP, mean ± SD, mm Hg	124.44 ± 13.85	119.8 ± 12.6	132.2 ± 12.6	0.006
Nocturnal DBP, mean ± SD, mm Hg	75.50 ± 10.06	73.5 ± 10.1	78.8 ± 9.4	0.118
nocturnal dipping status, <i>n</i> (%)	15 (27.8%)	7 (20%)	8 (42%)	0.08

CF-PWV: Carotid–femoral pulse wave velocity; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; MBP: Mean blood pressure; PP: Pulse pressure; c SBP: Central systolic blood pressure; c DBP: Central diastolic blood pressure; ABPM: Ambulatory blood pressure monitoring; SD: Standard deviation.

Table 4. Univariate comparison of renal transplant recipients according to pulse wave velocity and laboratory findings.

Parameter	Value	CF-PWV < 10	CF-PWV ≥ 10 m/s	<i>p</i> Value
Estimated glomerular filtration rate, mean ± SD, mL/min/1.73 m ² SC	65.2 ± 25.9	67.2 ± 24.3	61.6 ± 29	0.460
Calcium, mean ± SD, mmol/L	2.31 ± 1.20	2.32 ± 0.10	2.29 ± 0.14	0.381
Phosphorus, mean ± SD, mmol/L	1.01 ± 0.18	1.02 ± 0.20	0.98 ± 0.15	0.474
Fasting blood glucose, median [Q1–Q3], g/L	1 [0.9–1.2]	0.94 [0.9–1.01]	1.13 [0.93–1.76]	0.027
HbA1c%, median [Q1–Q3]	5.5 [5.0–6.62]	5.5 [5.0–6.0]	6.1 [1–7.9]	0.157
Cholesterol, mean ± SD, g/L	1.73 ± 0.41	1.71 ± 0.40	1.79 ± 0.43	0.522
HDL- cholesterol, median [Q1–Q3], g/L	0.46 [0.32–0.64]	0.44 [0.32–0.65]	0.48 [0.39–0.61]	0.542

Table 4. Cont.

Parameter	Value	CF-PWV < 10	CF-PWV ≥ 10 m/s	<i>p</i> Value
Non-HDL-cholesterol, mean ± SD, g/L	1.21 ± 0.14	1.0 ± 0.39	0.88 ± 0.43	0.390
LDL- cholesterol, mean ± SD, g/L	0.55 ± 0.02	1.28 ± 1.16	1.16 ± 0.44	0.809
Triglyceride, mean ± SD, g/L	1.30 ± 0.47	1.37 ± 0.3	1.38 ± 0.4	0.723
uric acid, mean ± SD, mg/L	401 ± 98	379 ± 102	409 ± 93	0.664
iPTH, median [Q1–Q3], pg/L	115 [94.5–205]	104 [94.5–176.0]	177 [81.25–253]	0.101
Hemoglobin, mean ± SD, g/dL	13.5 ± 2.05	13.5 ± 1.95	13.7 ± 2.2	0.733
Tacrolimus trough level, mean ± SD, ng/mL	5.68 ± 2.1	5.81 ± 1.91	5.46 ± 2.49	0.675
UPCR, mean ± SD, mg/mmol	34 ± 8	33 ± 4	31 ± 6	0.881

CF-PWV: carotid–femoral pulse wave velocity; iPTH: intact parathyroid hormone; UPCR: spot urine protein–creatinine ratio. SD: Standard deviation.

3.2.2. Multivariate Analysis

In the multivariate analysis, dyslipidemia, donor age, SBP assessed by office blood pressure measurement, and fasting blood glucose were independently associated with CF-PWV (Table 5).

Table 5. Multivariate regression analysis of predictors for elevated CF-PWV.

Factor	Odds Ratio (95% Confidence Interval)	<i>p</i> Value
Dyslipidemia	60.32 [2.2–165]	0.015
Donor age	1.16 [1.03–1.32]	0.014
SBP	1.25 [1.068–1.46]	0.006
Fasting blood glucose	22.35 [1.26–39]	0.034

SBP: systolic blood pressure assessed by office blood pressure measurement.

4. Discussion

Successful KT improves arterial stiffness and reduces cardiovascular risk [15]. However, several factors are associated with an increase in arterial stiffness in KTRs [16].

Cardiovascular risk factors in KTRs can be divided into traditional and non-traditional factors. Traditional risks include age, co-morbidities such as hypertension, diabetes, and dyslipidaemia, and non-traditional risks include immunosuppression medications, time since transplantation, and inflammation [17].

Several studies have established a strong correlation between age and CF-PWV [18,19]. In a longitudinal study, Feng et al. [18] demonstrated that advancing age was significantly associated with increased CF-PWV. However, in our study, age was not identified as an independent factor associated with elevated CF-PWV in the multivariate analysis.

This could be attributed to the age of our study population (42.5 years), which is relatively young compared to most cohorts showing age-related increases in PWV [18,19]. In younger populations, PWV may not vary enough with age to detect significant differences [18].

We could not find an association between smoking and elevated PWV. The relation between smoking and changes in PWV remains a topic of debate in the literature. Some

studies showed that long-term smoking contributes to increased PWV however, others suggested potential reversibility upon smoking cessation [20]. Due to the lack of data on the effects of pre-transplant smoking on vascular changes following transplantation, and since we did not assess the amount of time smoking before transplantation, we are unable to draw any conclusions regarding our results.

Hypertension remains a leading predictor of CVD and graft dysfunction in KTRs [21]. It contributes to increased arterial stiffness by promoting vascular hypertrophy and extracellular matrix remodeling, primarily through enhanced collagen deposition [22]. In our study, no significant difference in arterial stiffness was observed between hypertensive and non-hypertensive patients. However, regardless of the patient's current blood pressure status, our findings indicate a significant association between CF-PWV and pre-transplant hypertension, highlighting the potential long-term vascular impact of hypertension prior to kidney transplantation. There are several reports evaluating the effect of SBP on arterial stiffness [18,19]. In a cross-sectional study including 330 kidney transplant patients, Mitchell et al. [19] showed that SBP was associated with high CF-PWV. Feng et al. [18] also found the same results.

Liu et al. [23] reported that increased DBP was independently associated with high CF-PWV.

The impact of dyslipidemia on arterial stiffness is well recognized, as it promotes inflammatory responses that play a key role in the development and progression of atherosclerosis [24].

As all patients in our study were receiving corticosteroids and calcineurin inhibitors it was not possible to specifically assess the independent impact of these immunosuppressive agents on PWV. However, their metabolic effects were indirectly reflected in our findings, as we observed a significant association between elevated PWV and both dyslipidemia and fasting blood glucose.

Indeed, the long-term use of corticosteroids and calcineurin inhibitors is associated with increased metabolic risk [25]. Both tacrolimus and cyclosporine impair insulin sensitivity [25]. Long-term cyclosporine therapy is particularly associated with an increased risk of dyslipidemia, due to its non-competitive inhibition of sterol 27-dehydroxylase (CYP27A1), which results in elevated levels of total cholesterol, LDL-C, and triglycerides. Tacrolimus may also induce dyslipidemia, but typically to a lesser extent [25,26].

Glucocorticoids further contribute to metabolic disturbances by increasing peripheral insulin resistance, enhancing hepatic gluconeogenesis, and impairing pancreatic beta-cell function, resulting in elevated blood glucose levels. They also promote dyslipidemia by raising total cholesterol, LDL-C, and triglycerides, and potentially lowering HDL-C [25].

Similarly to our result, Chen et al. [27] suggested that dyslipidemia is a major contributing factor to arterial stiffness.

Interestingly, we found a significant association between donor age and elevated CF-PWV across the uni- and multivariate analyses. In a longitudinal study, Delahousse et al. [28] demonstrated that arterial stiffness was strongly and independently related to donor age. They showed that arterial stiffness improved in recipients of young-donor kidneys, whereas it worsened in recipients of old-donor kidneys. This finding prompts an interesting question about the underlying mechanisms linking donor age to increased aortic stiffness in the recipient. Vascular histological alterations in grafts from older donors, along with the potential impact of age-related changes in the kidney's metabolic or endocrine function, including the renin-angiotensin system, could help explain this result, as these factors influence the mechanical properties of large arteries [28].

Our study has some limitations. Due to its cross-sectional design, along with the relatively small sample size, some factors may not have been detected or fully explored. The study was conducted in a North African center, where most patients had received

a kidney from a living related donor and had relatively good graft function. Therefore, these findings may not fully apply to other transplant populations, particularly in regions with different healthcare systems, donor types, or immunosuppressive protocols. Further multicenter studies including more diverse patient populations are warranted to confirm and expand the applicability of these results. In addition, although inflammatory markers such as C-reactive protein (CRP) and interleukin-6 could provide further insights into cardiovascular risk, CRP was negative in all patients and was only available through a semi-quantitative method ($\text{CRP} < 6 \text{ mg/L}$), which limited its analytical usefulness. Interleukin-6 measurement is not part of routine clinical practice in our center and was therefore not available for this study. Nonetheless, despite these limitations, our study provides valuable insights for nephrologists involved in the management of KTRs. It is the first study conducted in Tunisia to investigate the factors associated with elevated CF-PWV in this population. Given that arterial stiffness and its determinants can vary across different ethnic groups or races, this study offers important insights into region-specific cardiovascular risk profiles and contributes to the global understanding of post-transplant vascular health. Furthermore, the use of ABPM allowed a more accurate assessment of blood pressure and its impact on arterial stiffness, in contrast to traditional office measurements, which are often subject to the white-coat effect.

Perspective for Clinical Practice

Our findings highlight the relevance of assessing arterial stiffness as part of cardiovascular risk evaluation in KTRs, as it is frequently elevated and associated with several clinical and biological parameters. However, since PWV measurement requires specific equipment and expertise, its routine use may be limited, particularly in low-resource settings. In this context, identifying surrogate clinical and biological markers, such as donor age, systolic blood pressure, dyslipidemia, and fasting blood glucose, offers a practical and accessible approach to estimate cardiovascular risk. These routinely available parameters could form the basis for a simplified clinical tool to help stratify patients according to their arterial stiffness profile. Such an approach would support nephrologists in adapting cardiovascular prevention strategies, even in the absence of direct vascular assessments. It could contribute to narrowing the gap between advanced diagnostics and everyday clinical practice, especially in regions with limited access to PWV technology. Furthermore, a long-term follow-up of our study population would be essential to confirm or refute the predictive value of these parameters in relation to cardiovascular events. This would help validate their integration into clinical decision-making algorithms and guide future interventional strategies aimed at improving post-transplant cardiovascular outcomes.

5. Conclusions

This study highlights the frequent presence of elevated arterial stiffness among KTRs and its significant association with common clinical and biological parameters. Given the cross-sectional design and limited sample size, causal relationships cannot be established, and our results should be interpreted with caution. Larger prospective studies are needed to validate these associations and determine their predictive value for cardiovascular outcomes. From a clinical standpoint, identifying easily measurable surrogate markers offers a practical approach for risk stratification in routine practice. Future work should explore the development of a composite risk score based on these markers, and its validation in larger and diverse transplant populations. A long-term follow-up of our current study population will also be crucial to confirm the clinical utility of these parameters in guiding preventive strategies and improving a cardiovascular prognosis after KT.

Supplementary Materials: The following supporting information can be downloaded at: <https://www.mdpi.com/article/10.3390/transplantology6040032/s1>.

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Informed Consent Statement: Informed consent was obtained from all subjects involved in the study.

Data Availability Statement: The data presented in this study are available on request from the corresponding author due to privacy reasons.

Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

KT	Kidney transplantation
PWV	Pulse wave velocity
KTRs	Kidney transplant recipients
CF-PWV	Carotid–femoral PWV
CVD	Cardiovascular disease
ABPM	Ambulatory blood pressure monitoring
BMI	Body mass index
SBP	Systolic blood pressure
DBP	Diastolic blood pressure
MBP	Mean blood pressure
PP	Pulse pressure
c SBP	Central systolic blood pressure
c DBP	Central diastolic blood pressure

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