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Comparison of Cardiovascular Risk Factors in Home Hemodialysis and Kidney Transplant Patients

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Research Article

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Abstract

Background: Arterial stiffness has an independent predictive value for cardiovascular events in patients at high risk for cardiovascular disease, such as those with essential hypertension and type 2 diabetes mellitus. However, data regarding arterial stiffness and related inflammatory markers in patients undergoing home hemodialysis and kidney transplant recipients remain limited. Pulse wave velocity (PWV) and augmentation index are noninvasive markers used to assess vascular damage. This study aimed to compare arterial stiffness and associated cardiovascular risk factors between patients receiving home hemodialysis and kidney transplant recipients.

Methods: A total of 25 patients undergoing home hemodialysis and 38 kidney transplant recipients were included in this retrospective study. Data were obtained from patient records between June 2020 and June 2021, including arterial stiffness measurements performed using the Mobil-O-Graph® system, as well as biochemical, hematological, and inflammatory parameters. Demographic and clinical characteristics were extracted from hospital records.

Results: Median PWV values were 7.1480 m/s in the home hemodialysis group and 7.186 m/s in the kidney transplant group, with no statistically significant difference between groups ($p = 0.918$). Peripheral and central blood pressure values were higher in transplant recipients, whereas PWV values were comparable. Markers of systemic inflammation, including the systemic immune-inflammation index, were significantly higher in the kidney transplant group.

Conclusion: Arterial stiffness as assessed by PWV was similar between patients receiving home hemodialysis and kidney transplant recipients. Despite differences in inflammatory and hemodynamic parameters, home hemodialysis demonstrated comparable arterial stiffness profiles, supporting its role as an effective renal replacement therapy option during the pretransplant period.

1.Introduction

Home hemodialysis is a form of hemodialysis therapy performed in the patient's own home by the patient or a caregiver. It is typically administered three times per week for 6–8 hours, most often overnight while the patient is asleep. The primary goals of home hemodialysis are to improve the effectiveness of dialysis therapy, enhance quality of life, and prolong patient survival [1–3]. Although dialysis and kidney transplantation extend life expectancy, cardiovascular diseases remain the leading cause of mortality in patients with chronic kidney disease (CKD). Approximately half of patients undergoing hemodialysis die from cardiovascular causes, highlighting CKD as a strong risk factor for cardiovascular disease [3–5]. Vascular calcification and arterial stiffness are key indicators of the atherosclerotic process in CKD. Increased arterial stiffness is recognized as an independent risk marker for coronary artery disease, stroke, and cardiovascular mortality in the general population, and it also has predictive value for cardiovascular events in patients with uncomplicated essential hypertension and diabetes mellitus [1–5].

Arterial stiffness is a potentially reversible cardiovascular risk factor that can be assessed using noninvasive methods. Patient survival improves after kidney transplantation, and arterial stiffness has been shown to decrease compared with pretransplant levels. According to the Framingham cardiovascular risk score, the risk of ischemic heart disease is markedly reduced in kidney transplant recipients. However, despite these favorable effects, cardiovascular diseases remain among the leading causes of posttransplant mortality. This persistent risk has been attributed, at least in part, to an increased systemic inflammatory response following transplantation. Systemic inflammation markers such as C-reactive protein, homocysteine, and the neutrophil-to-lymphocyte ratio have been shown to play a role in the development of arterial stiffness [2–4]. Arterial stiffness is associated not only with structural changes in the vascular wall but also with mechanisms involving endothelial function and nitric oxide release, and it is therefore considered an established risk marker that reflects the overall burden of cardiovascular disease. Importantly, arterial stiffness is a potentially reversible risk factor and can be used in patient follow-up to assess treatment effectiveness [4–6]. Currently, arterial stiffness can be reliably evaluated using noninvasive parameters such as pulse wave velocity (PWV), augmentation index (AIx), and pulse pressure, enabling the early detection of atherosclerosis [2–6].

The aim of this study was to compare arterial stiffness and other cardiovascular risk factors between patients receiving home hemodialysis in our unit and kidney transplant recipients followed in our outpatient clinic, using noninvasive methods including pulse wave velocity and augmentation index.

2. Methods

2.1.Patient Selection

This single-center cross-sectional study was conducted in accordance with the principles of the Declaration of Helsink. All patients receiving home hemodialysis (HHD) in the Dialysis Unit of Adnan Menderes University Faculty of Medicine Hospital and all kidney transplant recipients (from deceased or living donors) followed in our nephrology outpatient clinic were included in the study. Patients were evaluated retrospectively. All individuals who had been receiving HHD for at least 6 months or who had undergone kidney transplantation more than 6 months prior to enrollment were eligible for inclusion. Patient data recorded between June 2020 and June 2021 were reviewed. A total of 25 patients undergoing HHD and 38 kidney transplant recipients were included in the study.

2.2.Exclusion Criteria

Patients were excluded if they met any of the following criteria:

- Presence of cardiovascular disease or a history of cardiovascular surgery
- Age <18 years
- Age >80 years
- Presence of active infection

- History of malignancy
- Pregnancy
- Acute coronary syndrome within the previous 3 months
- Surgical intervention within the previous 3 months
- Hospitalization within the previous 2 months
- Duration of follow-up or transplantation <6 months

Before inclusion in the study, routinely performed laboratory test results were reviewed. Detailed discharge summaries were examined retrospectively. Data on routinely measured pulse rate, arterial blood pressure, height, and body weight were obtained from patient records. Additional demographic and clinical information—including age, sex, primary renal disease, presence of hypertension or diabetes mellitus, medication use, smoking and alcohol consumption, duration of HHD, and time since kidney transplantation—were collected from archived medical records. Body mass index (BMI) was calculated using the formula weight (kg) / height² (m²).

2.3.Laboratory Tests

Retrospective biochemical data for all patients were obtained from the hospital electronic database. The following parameters were recorded from patient files: complete blood count; serum urea, creatinine, uric acid, potassium (K), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), total cholesterol (TC), alkaline phosphatase (ALP), calcium, phosphorus, parathyroid hormone (PTH), albumin, and total protein levels; as well as 24-hour urinary proteinuria and microalbuminuria measurements. The calcium–phosphorus product was calculated by multiplying serum calcium and phosphorus levels.

Markers of systemic inflammation were derived from retrospective complete blood count data. The neutrophil-to-lymphocyte ratio (NLR) was calculated as the first inflammatory parameter, and the platelet-to-lymphocyte ratio (PLR) was calculated as the second inflammatory marker. In addition, the systemic immune-inflammation index (SII), another important indicator of systemic inflammation, was calculated using the formula (neutrophil × platelet) / lymphocyte.

Estimated glomerular filtration rate (eGFR) in patients with chronic kidney disease was calculated using the abbreviated Modification of Diet in Renal Disease (MDRD) formula:

eGFR (MDRD)=186 × (serum creatinine)^−1.154 × (age)^−0.203 × (0.742 if female)

2.4.Ankle–Brachial Index Measurement

Previously performed ankle–brachial index (ABI) measurements were obtained retrospectively from patient discharge summaries. Measurements were conducted as follows. Systolic blood pressures of both posterior tibial and dorsalis pedis arteries were measured. A sphygmomanometer cuff was placed proximal to the ankle. The stethoscope was positioned below the medial malleolus for the posterior

tibial artery and on the dorsal surface of the foot for the dorsalis pedis artery. The cuff was inflated until the arterial pulse sound disappeared and then slowly deflated; the pressure at which the pulse sound reappeared was recorded as the systolic pressure. This procedure was repeated for both lower extremities, and values were recorded.

Brachial systolic blood pressure was measured from the arm without an arteriovenous fistula. The cuff was placed proximally on the arm, covering approximately two-thirds of the upper arm, and the stethoscope was positioned over the brachial artery in the antecubital region. The cuff was inflated until the pulse sound disappeared and then gradually deflated; the pressure at which the pulse sound reappeared was recorded as the systolic pressure (Figure 1).

The ABI was calculated by dividing the higher of the posterior tibial or dorsalis pedis systolic pressures by the brachial systolic pressure. ABI values were calculated for both lower extremities, and the lower value was recorded as the patient's ABI. Based on previous studies and current guidelines, an ABI value <0.9 was interpreted as indicative of peripheral arterial disease.

2.5.Arterial Stiffness Measurement

In this study, arterial stiffness data were obtained retrospectively from patient discharge records using a cuff-based oscillometric device, the Mobil-O-Graph PWA/ABPM system (I.E.M. GmbH, Stolberg, Germany), which performs noninvasive measurements via the brachial artery. Prior to arterial stiffness assessment, patients were allowed to rest in a seated position in a quiet room for 15 minutes. Appropriate cuff sizes were selected based on arm circumference, and the cuff was placed just above the elbow on the arm without an arteriovenous fistula or vascular access.

Pulse wave velocity (PWV) analysis was performed from the brachial artery in the seated position, with three consecutive measurements obtained at 5-minute intervals. A wireless Bluetooth connection was established between the device and the dedicated software developed for data acquisition and analysis. Participant age, height, and weight were entered into the software program. Initially, brachial blood pressure was measured to determine the pressure at which the brachial artery was fully occluded. After a 30-second interval, the brachial artery was again completely compressed, and when pulse waves reached the point of absent flow, they were detected, amplified, and recorded by specialized sensors within the device to obtain the peripheral pulse waveform.

The acquired data were automatically classified by the software as either very high quality or low quality, and only very high-quality measurements were included in the analysis. Using the measured brachial pulse waveform, the central aortic pulse waveform was calculated via the dedicated software, thereby completing the pulse wave analysis. The following parameters were obtained: systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), heart rate, pulse pressure (PP), central SBP, central DBP, central PP, cardiac output, peripheral vascular resistance, cardiac index, body surface area, augmentation pressure (AP), augmentation index adjusted to a heart rate of 75 beats per minute (AIx@75), reflection magnitude, and pulse wave velocity. All data were transferred to the software

program, reviewed by the investigators, recorded in patient discharge summaries, and subsequently entered into the Statistical Package for the Social Sciences (SPSS) for analysis.

2.6.Statistical Analysis

Statistical analyses were performed using the Statistical Package for the Social Sciences for Windows, version 21.0 (SPSS Inc., Chicago, IL, USA). The normality of distribution for continuous variables was assessed using the Kolmogorov–Smirnov test. Descriptive statistics were expressed as number and percentage for categorical variables; mean±standard deviation for normally distributed continuous variables; and median (25th–75th percentile) for non-normally distributed continuous variables.

Comparisons of categorical variables were performed using the chi-square test. For comparisons between two independent groups, Student's t test was used for normally distributed continuous variables, and the Mann–Whitney U test was used for non-normally distributed variables. Correlations between variables were assessed using the Pearson correlation coefficient for normally distributed variables and the Spearman correlation coefficient for non-normally distributed variables. A p value <0.05 was considered statistically significant.

Post-hoc power analysis was performed using G*Power software (version 3.1.9.7; Heinrich Heine University, Düsseldorf, Germany). Based on an expected hemoglobin difference of 1.2 g/dL between the kidney transplant and home hemodialysis groups, with a standard deviation of 1.8, an effect size (Cohen's d) of 0.67 was calculated. With 38 patients in the transplant group and 25 patients in the home hemodialysis group, the estimated statistical power was approximately 74% at a two-sided alpha level of 0.05.

2.7.Ethical Approval

This retrospective, single-center study was conducted in accordance with the ethical principles of the Declaration of Helsinki and was approved by the Clinical Research Ethics Committee of Adnan Menderes University Faculty of Medicine (approval date: September 30, 2021; decision no. E-53043469-050.04.04-81949). All eligible patients followed at the study center during the study period were included in the analysis.

3. Results

The mean age was 50.1±12.8 years in the home hemodialysis (HHD) group and 45.1±11.4 years in the kidney transplant group. Seventeen patients (68%) in the HHD group and 26 patients (68.4%) in the transplant group were male. The median duration from initiation of HHD to arterial stiffness measurement was 39.2 (14.0–58.5) months, whereas the median duration from transplantation to arterial stiffness measurement was 41.1 (12.8–46.3) months in transplant recipients (Table 1).

When ankle–brachial index (ABI) values were compared between groups, the mean ABI was 1.2±0.2 in the HHD group and 1.1±0.3 in the transplant group. Comparison of hematological parameters revealed

that mean hemoglobin (p<0.001) (Figure 2A), hematocrit (p<0.001), leukocyte count (p=0.003), platelet count (p=0.003), neutrophil count (p=0.007), and systemic immune-inflammation index (SII) values were significantly higher in the transplant group compared with the HHD group (Figure 2B).

When biochemical findings were compared, mean urea and creatinine levels were significantly higher in the HHD group, whereas mean sodium, calcium, uric acid, and albumin levels were significantly lower compared with the transplant group (p<0.05 for all) (Table 2).

Comparison of Mobil-O-Graph measurements showed that mean peripheral and central systolic and diastolic blood pressure values were significantly higher in the transplant group than in the HHD group, while reflection magnitude was significantly lower in the transplant group (p<0.05 for all). Mean pulse wave velocity (PWV) values were similar between the two groups (p=0.918) (Figure 2C, Table 3).

Correlation analyses demonstrated that PWV was strongly positively correlated with age; moderately positively correlated with body mass index (BMI), uric acid, glucose, and mean systolic blood pressure; and weakly positively correlated with parathyroid hormone, mean heart rate, neutrophil-to-lymphocyte ratio (NLR), and mean diastolic blood pressure (p<0.05 for all). SII showed a strong positive correlation with NLR; moderate positive correlations with uric acid, total protein, platelet-to-lymphocyte ratio (PLR), and mean systolic blood pressure; and a weak positive correlation with C-reactive protein (CRP). In contrast, SII was strongly negatively correlated with mean heart rate (p<0.05 for all) (Table 4).

4. Discussion

With increasing life expectancy in chronic kidney disease (CKD), cardiovascular diseases continue to be the primary determinants of morbidity and mortality. Arterial stiffness has emerged as a comprehensive marker of cardiovascular risk in this patient population and carries prognostic significance both across dialysis modalities and in the post–kidney transplantation period. Although the cardiovascular effects of home hemodialysis (HHD) and kidney transplantation have been addressed in the literature from various perspectives, studies directly comparing these two patient groups in terms of arterial stiffness and associated inflammatory markers remain limited.

In a study by Kim et al. including 50 kidney transplant recipients, the mean preoperative ankle–brachial index (ABI) measured in the right extremity was 1.17±0.11. Postoperative ABI values measured at 1 week, 6 months, and 1 year were 1.14±0.10, 1.15±0.11, and 1.17±0.15, respectively, with no significant differences observed between preoperative and postoperative measurements [7]. In our study, the mean ABI—an indicator of peripheral arterial disease—was 1.1±0.3, and no significant difference was observed between the groups. These findings suggest that there may be no marked difference in the risk of peripheral arterial disease between patients receiving HHD and kidney transplant recipients.

Hemoglobin and hematocrit levels are generally higher in kidney transplant recipients compared with patients with end-stage kidney disease or those undergoing hemodialysis. Several etiopathogenetic mechanisms have been proposed to explain this increase in hemoglobin levels. These include elevated

erythropoietin (EPO) levels following transplantation, increased sensitivity of EPO receptors associated with activation of the renin–angiotensin system in the transplanted kidney, and the resulting enhancement of erythropoiesis. In our study, hemoglobin and hematocrit levels were significantly higher in the kidney transplant group than in the HHD group. We believe that two major factors contribute to this finding: increased responsiveness of EPO receptors to elevated EPO levels after transplantation and the resolution of uremia-induced bone marrow suppression associated with CKD, leading to increased erythropoiesis [2–4,8]. In contrast, urea and creatinine levels were significantly higher in the HHD group, consistent with the persistence of CKD in these patients compared with kidney transplant recipients.

It has been shown that individuals undergoing hemodialysis have relatively stable predialytic plasma sodium concentrations [9,10]. Nevertheless, despite this apparent stability, many patients exhibit a lower sodium set point [10]. A cross-sectional study also reported a high prevalence of hyponatremia in hemodialysis patients both before and after dialysis sessions [10]. Although the mechanisms underlying hyponatremia in hemodialysis patients have not been fully elucidated, it is generally thought to result from excessive interdialytic free water intake. Oliguric or anuric patients with end-stage kidney disease who require hemodialysis depend on dialysis for the removal of sodium and water to maintain homeostasis. Consequently, inadequate fluid removal during hemodialysis predisposes these patients to hyponatremia. Due to differences in definitions and study populations, reported prevalence rates of hyponatremia among hospitalized patients range from 2.5% to 30.0%. In another study analyzing 655,493 non–dialysis-dependent CKD patients in the United States, hyponatremia was identified in 13.5% of patients. In our study, when hyponatremia was defined as a serum sodium level <135 mEq/L, the prevalence was 8%. Hyponatremia is also relatively common in hemodialysis patients, with a reported prevalence of 29.3% among 1,549 oligoanuric hemodialysis patients [10–12]. Consistent with these findings, serum sodium levels were relatively lower in the HHD group compared with the kidney transplant group in our study. We believe that this difference may be related to suboptimal volume management during dialysis in patients receiving hemodialysis.

In patients with chronic kidney disease (CKD), hypoalbuminemia is generally considered a reflection of protein–energy malnutrition (PEM) [13,14]. PEM is defined as a state of metabolic and nutritional alterations characterized by depletion of protein and energy stores, leading to loss of muscle and fat mass and ultimately cachexia [15–17]. Risk factors for PEM—and consequently hypoalbuminemia—in patients with CKD include reduced dietary intake of protein and calories, anorexia secondary to uremic toxins, delayed gastric emptying, systemic inflammation, and comorbid conditions [13]. Patients with CKD who are not receiving kidney replacement therapy are often prescribed dietary restrictions, including recommendations to reduce protein intake. These factors, combined with increased albumin catabolism and losses, further contribute to hypoalbuminemia. Comorbid conditions such as infection and cardiovascular disease may induce a hypercatabolic state, while renal and gastrointestinal protein losses (including albumin) further disrupt albumin homeostasis [17]. In addition, endocrine disturbances associated with advanced uremia—such as insulin resistance, increased glucagon sensitivity, and hyperparathyroidism—may also adversely affect albumin homeostasis [13,14]. Protein–energy malnutrition increases the risk of infection, depression, frailty, and cardiovascular disease, thereby

exacerbating malnutrition and inflammation and reinforcing a vicious cycle that ultimately leads to progressively severe PEM and hypoalbuminemia [13,14,16].

Another important contributor to hypoalbuminemia in patients with end-stage kidney disease (ESKD) is the use of phosphate-binding chelators. Progression to ESKD and initiation of maintenance hemodialysis further predispose an already vulnerable population to hypoalbuminemia. Although patients receiving hemodialysis are advised to consume approximately 1.2 g/kg/day of protein, maintaining such intake is often challenging [14]. In studies conducted among older adults undergoing hemodialysis, neither nutritional intervention alone nor nutritional intervention combined with a 6-month exercise program resulted in significant increases in serum albumin levels [18,19]. An unintended consequence of increased dietary protein intake in hemodialysis patients is an elevated risk of hyperphosphatemia, which is an independent risk factor for mortality in patients with kidney disease and those undergoing hemodialysis [20–23]. Although plant-based diets may reduce phosphorus intake, serum phosphorus levels >5.5 mg/dL are observed in approximately 40% of hemodialysis patients, and nearly 80% of the phosphorus burden in these patients is eliminated through the use of phosphate binders [22–24]. Moreover, it has been shown that the use of sucroferric oxyhydroxide, a phosphate binder with a relatively low pill burden and a tolerable gastrointestinal side-effect profile, allows effective phosphorus control without a concomitant decline in serum albumin levels [23,24].

Inadequate dialysis may also lead to decreased appetite and subsequent malnutrition. In addition, insufficient dialysis can result in dilutional hypoalbuminemia. Similarly, pre-dialysis albumin levels may be falsely low due to fluid retention. For this reason, post-dialysis assessment of serum albumin levels is generally considered preferable to pre-dialysis measurements [24,25]. Albumin loss during dialysis may also occur as a result of exposure to foreign materials, including the dialyzer membrane, which can trigger an inflammatory response. One recognized mechanism of reduced albumin levels is the leakage of albumin across the dialysis membrane into the dialysate, leading to hypoalbuminemia [26]. In our study, serum albumin levels were significantly lower in the HHD group compared with the kidney transplant group. We attribute this finding to ongoing PEM, persistent inflammation, and continued albumin losses associated with hemodialysis in patients receiving HHD.

One of the most important causes of mortality in end-stage kidney disease (ESKD) is the suppression of cardiac contractility due to elevated serum potassium levels, which may lead to bradycardia and cardiac arrest. In patients receiving home hemodialysis (HHD), prolonged and slower dialysis sessions allow for more stable potassium levels, with heart rate remaining within the normal range throughout the day, thereby contributing significantly to reduced cardiac mortality. In a randomized controlled study conducted in patients with ESKD and coronary plaque, patients receiving HHD were compared with those undergoing in-center hemodialysis. Coronary computed tomography follow-up demonstrated no progression of coronary plaques in patients receiving HHD, whereas plaque progression was observed in patients undergoing conventional in-center hemodialysis three times per week for 4 hours. Improved control of dyslipidemia with HHD was identified as a major contributing factor to these findings [6,7]. In

our study, total cholesterol levels were significantly lower in the HHD group compared with kidney transplant recipients.

With advances in hemodialysis, transplantation, and renal replacement therapies (RRT), survival of patients with chronic kidney disease (CKD) has improved. Despite these important developments, cardiovascular disease remains the leading cause of mortality in patients with CKD. One of the most important indicators of cardiovascular mortality is increased arterial stiffness, which is reflected by elevated aortic pulse wave velocity (PWV) [1,2].

Bahous et al. conducted a study in which arterial stiffness was measured noninvasively in 101 living kidney donors and their corresponding 101 recipients, with comparisons made to healthy volunteers. Cardiovascular risk and arterial stiffness, assessed by PWV, were significantly higher in kidney transplant recipients compared with healthy individuals, with a mean PWV of 11.4±2.4 m/s [27]. In another study by Kolonko et al., arterial stiffness measurements performed 8–10 years after transplantation demonstrated significantly higher PWV values (13.1±5.0 m/s) in transplant recipients compared with healthy controls, and pretransplant diabetes was identified as a cardiovascular risk factor in the posttransplant period [28]. In a meta-analysis published in 2017 by Aboubacar et al., which included 12 studies and 450 kidney transplant recipients, arterial stiffness decreased significantly following transplantation. PWV values declined from 12.1±3.1 m/s before transplantation to 10.7±2.1 m/s at 6 or 12 months posttransplantation [29]. In our study, the absence of longitudinal arterial stiffness measurements within the same patient group and the relatively small sample size may explain the discrepancy between our findings and those of this meta-analysis.

Patients receiving HHD have been shown to experience marked regression of cardiovascular disease compared with those undergoing in-center hemodialysis. Long and slow HHD performed at home for 6–8 hours, three times per week, has been associated with normalization of blood pressure, elimination of the need for antihypertensive medications, and reduction in cardiac afterload, leading to regression of left ventricular hypertrophy. In one study, improvement in ejection fraction was observed at 6 months in patients with ESKD and reduced baseline ejection fraction who were receiving HHD [30]. In our study, no significant difference in arterial stiffness, as assessed by PWV, was observed between the HHD and kidney transplant groups. We speculate that the use of immunosuppressive agents after transplantation —which may contribute to atherosclerosis through multiple mechanisms—and the greater cardioprotective advantages of HHD compared with conventional hemodialysis may underlie these findings [30].

Multiple inflammatory processes contribute to the development of arterial stiffness in patients with CKD. Elevated levels of proinflammatory cytokines, such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), are commonly observed in this population. Short bacterial DNA fragments, endotoxins, and small muramyl dipeptides may be present in dialysate and can induce IL-6 production after passing through high-flux membranes. In addition, catheters used for hemodialysis or peritoneal dialysis, as well as synthetic grafts, may serve as potential sources of inflammation. Increased

inflammation in CKD, along with elevated anti–TNF-α and low-density lipoprotein (LDL) levels, contributes to the development of atherosclerosis [31,32]. In a study conducted by Samantha et al. in 2020 involving 378 kidney transplant recipients, systemic immune-inflammation index (SII) values were compared between recipients of living-donor and deceased-donor transplants, with mean SII values of 617 and 545, respectively; the difference approached statistical significance (p=0.07) [33]. To our knowledge, the present study is the first to compare SII between patients receiving HHD and kidney transplant recipients. In our cohort, mean SII was 507.255±326.296 in the HHD group and 848.325±630.289 in the transplant group, with a statistically significant difference (p=0.007). These findings suggest that kidney transplantation is associated with a significant increase in systemic inflammation, which may contribute to the development of atherosclerosis.

One of the main limitations of our study is the relatively small sample size in both patient groups; larger studies are needed to obtain more robust and generalizable results. Another limitation is that arterial stiffness was assessed only using noninvasive parameters such as PWV and augmentation index (AIx), without concurrent evaluation using histological findings or indirect markers of vascular calcification, including fetuin-A, osteoprotegerin, osteopontin, or adiponectin levels.

Conclusion

The findings of this study indicate that arterial stiffness, an established surrogate marker of cardiovascular risk, is comparable between patients receiving home hemodialysis and kidney transplant recipients. Despite the well-documented cardiovascular benefits associated with kidney transplantation, arterial stiffness parameters did not differ significantly between the two groups in our cohort. Our results suggest that home hemodialysis may provide effective cardiovascular protection during the period preceding kidney transplantation. Given that cardiovascular disease remains the leading cause of mortality in patients with chronic kidney disease and that increased arterial stiffness is closely associated with adverse cardiovascular outcomes, the similarity in arterial stiffness observed between the groups supports the role of home hemodialysis as a favorable renal replacement therapy option. Taken together, these findings support the preferential use of home hemodialysis in suitable patients with chronic kidney disease until kidney transplantation is achieved, particularly with regard to cardiovascular risk mitigation.

Declarations

Ethical Approval Statement

Ethics Approval:
This study was approved by the Clinical Research Ethics Committee of Adnan Menderes University Faculty of Medicine (approval number: E-53043469-050.04.04-81949, dated September 30, 2021).

Consent to Participate:
Due to the retrospective nature of the study, the requirement for informed consent was waived by the Ethics Committee.

Consent for Publication:
All authors consent to publication of this manuscript.

Authorship & Conflict of Interest Declaration

Authorship Confirmation:
All authors (Aykut Yaman, Hakan Akdam, Yavuz Yeniçerioğlu) contributed to study design, data collection, analysis, and manuscript preparation. All authors approved the final version.

Conflict of Interest:
The authors declare no competing interests.

Data availability and material statement

The datasets generated and/or analyzed during the current study are not publicly available due to ethical and privacy considerations but are available from the corresponding author upon reasonable request. No additional materials were used in this study.

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Tables

Table 1. Demographic characteristics of the groups.

Parameter	Home Hemodialysis (n=25)	Transplantation (n=38)	p
Male (n, %)	17 (68.0%)	26 (68.4%)	0.972
Age (years)	50.1±12.8	45.1±11.4	0.112
Body mass index (kg/m²)	25.2±5.1	26.1±4.9	0.489
Time since transplantation or home hemodialysis (months)	39.2 (14.0–58.5)	41.1 (12.8–46.3)	0.872
Smokers (n, %)	12 (48.0%)	18 (47.4%)	0.961
Use of renin–angiotensin system blockers (n, %)	0 (0.0%)	4 (10.5%)	0.040

Mean±standard deviation; median (25–75 percentile). BMI: Body mass index.

Table 2. Comparison of ankle–brachial index and laboratory parameters between groups.

Parameter	Home Hemodialysis (n=25)	Transplantation (n=38)	p
Ankle–Brachial Index	1.2±0.2	1.1±0.3	0.279
Hematological data			
Hemoglobin (g/dL)	11.8±1.2	13.7±2.4	<0.001
Hematocrit (%)	36.6±3.8	43.0±7.2	<0.001
MCV (fL)	89.5±6.9	88.9±7.7	0.761
WBC (10^3/μL)	6.9±1.8	8.6±2.6	0.003
Platelets (10^3/μL)	209.7±70.5	269.1±79.6	0.003
Neutrophils (10^3/μL)	4.2±1.5	5.6±2.3	0.007
Lymphocytes (10^3/μL)	1.8±0.6	2.2±1.3	0.097
RDW (%)	14.7±1.6	13.8±1.8	0.032
MPV (fL)	10.8±0.7	10.3±0.9	0.012
Neutrophil/Lymphocyte ratio	2.5±1.2	3.2±2.0	0.088
Platelet/Lymphocyte ratio	137.8±97.9	145.8±64.4	0.695
SII	507.3±326.3	848.3±630.3	0.007
Biochemical data			
Urea (mg/dL)	90.0±32.9	45.4±21.2	<0.001
Creatinine (mg/dL)	7.4±2.5	1.5±0.7	<0.001
Sodium (mEq/L)	137.8±2.6	140.5±2.1	<0.001
Potassium (mmol/L)	4.6±0.4	4.5±0.5	0.598
Calcium (mg/dL)	9.1±0.7	9.7±0.7	0.001
Phosphorus (mg/dL)	3.9±1.2	3.5±0.9	0.109
Calcium–phosphorus product	35.3±10.7	33.7±7.5	0.479
Uric acid (mg/dL)	5.6±1.0	6.3±1.5	0.042
Glucose (mg/dL)	119.4±36.4	118.5±50.9	0.938
Bicarbonate (mmol/L)	21.5±4.3	24.4±3.4	0.005
Albumin (g/dL)	3.7±0.3	4.3±0.4	<0.001
Total protein (g/dL)	6.8±0.5	7.4±0.5	<0.001
C-reactive protein (mg/dL)	5.6±6.3	9.2±20.0	0.313

Alkaline phosphatase (U/L)	143.0±94.5	92.8±44.1	0.006
Total cholesterol (mg/dL)	179.0±27.5	215.5±44.1	<0.001
HDL cholesterol (mg/dL)	48.2±14.8	56.6±19.7	0.073
LDL cholesterol (mg/dL)	99.0±25.7	120.3±36.4	0.018
Triglycerides (mg/dL)	193.4±110.5	189.9±104.7	0.900

Mean±standard deviation; median (25–75 percentile). ABI: ankle–brachial index; MCV: mean corpuscular volume; WBC: white blood cell count; RDW: red cell distribution width; MPV: mean platelet volume; SII: systemic immune-inflammation index; HDL: high-density lipoprotein; LDL: low-density lipoprotein; TG: triglycerides.

Table 3. Comparison of Mobil-O-Graph measurements between groups.

Parameter	Home Hemodialysis (n=25)	Transplantation (n=38)	p
Peripheral systolic blood pressure (mmHg)	117.9±22.4	133.9±18.8	0.003
Peripheral diastolic blood pressure (mmHg)	77.2±16.6	87.2±14.9	0.015
Peripheral mean arterial pressure (mmHg)	94.2±19.6	108.3±15.7	0.003
Heart rate (beats/min)	83.3±14.0	81.1±12.8	0.517
Peripheral pulse pressure (mmHg)	41.1±10.9	46.6±11.0	0.060
Central systolic blood pressure (mmHg)	109.0±20.9	122.4±18.3	0.009
Central diastolic blood pressure (mmHg)	79.4±18.2	89.3±15.2	0.023
Central pulse pressure (mmHg)	33.0±13.1	32.9±9.5	0.996
Cardiac output (L/min)	4.2±0.7	4.4±0.5	0.151
Peripheral resistance (s·mmHg/mL)	1.3±0.3	1.4±0.2	0.419
Cardiac index (L/min/m²)	2.4±0.6	2.5±0.4	0.639
Body surface area (m²)	1.8±0.3	1.8±0.2	0.805
Augmentation pressure (mmHg)	7.0±3.4	7.4±5.3	0.719
Augmentation index (%)	25.9±11.8	24.2±16.1	0.632
Reflection magnitude (%)	61.8±7.4	54.9±10.7	0.003
Pulse wave velocity (m/s)	7.1±1.4	7.2±1.5	0.918

PWV: pulse wave velocity; SBP: systolic blood pressure; DBP: diastolic blood pressure; MAP: mean arterial pressure.

Table 4. Correlation analysis of pulse wave velocity and systemic immune-inflammation index with other parameters.

Parameter	Correlation coefficient	p
Age (years)	0.854	<0.001
Body mass index (kg/m²)	0.452	<0.001
Uric acid (mg/dL)	0.500	<0.001
Glucose (mg/dL)	0.606	<0.001
Parathyroid hormone (pg/mL)	0.286	0.023
MPV	0.273	0.030
NLR	0.317	0.011
Central systolic blood pressure	0.418	0.001
Central diastolic blood pressure	0.275	0.029
Uric acid (mg/dL)	0.392	0.001
Total protein (g/dL)	0.323	0.010
C-reactive protein (mg/L)	0.264	0.037
MPV	−0.272	0.031
NLR	0.742	<0.001
PLR	0.483	<0.001
Central systolic blood pressure	0.352	0.005

PWV: pulse wave velocity; SII: systemic immune-inflammation index; MPV: mean platelet volume; NLR: neutrophil-to-lymphocyte ratio; PLR: platelet-to-lymphocyte ratio.

Figures

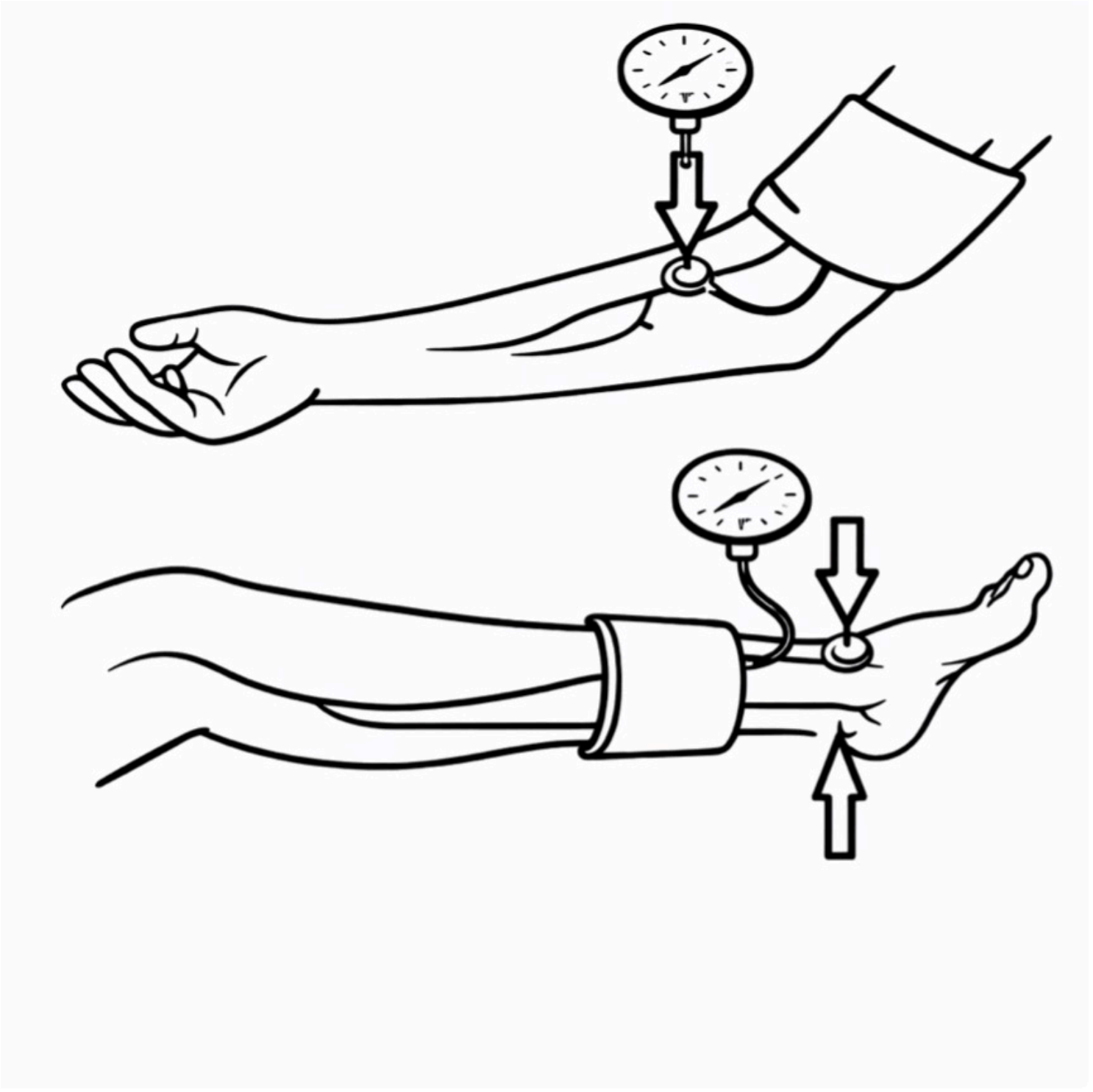


Figure 1

Measurement of the ankle–brachial index.

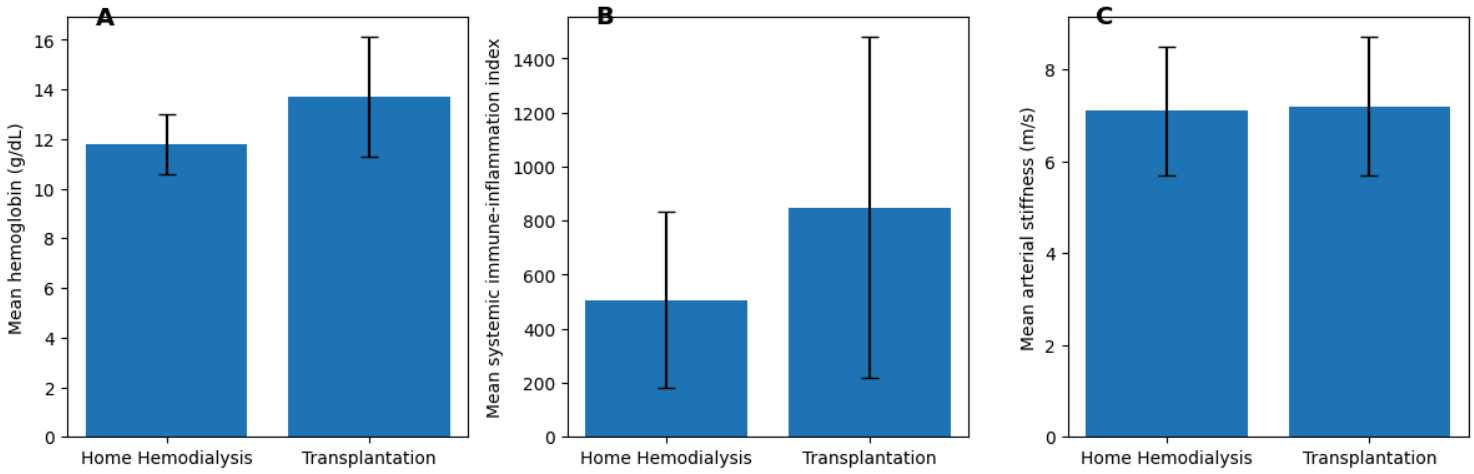


Figure 2

Comparison of mean hemoglobin levels (A), systemic immune-inflammation index (B), and arterial stiffness (C) between home hemodialysis and kidney transplant patients.