Short-Term Changes in Heart Rate Variability Following Kidney Transplantation: A Comparative Analysis with Healthy Donors

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Abstract

Aim: Heart rate variability (HRV) is a non-invasive marker of autonomic nervous system function and an established predictor of cardiovascular outcomes in end-stage renal disease (ESRD). While kidney transplantation may restore physiological homeostasis, the short-term impact on HRV remains unclear.

Methods: In this observational study, HRV was measured in 32 kidney transplant recipients and 29 healthy living donors. HRV recordings were obtained one day before and between the 5th and 7th days after transplantation using a Polar H10 monitor and Kubios HRV software. Parameters analyzed included time-domain, frequency-domain, nonlinear, and entropy-based indices. Between-group and within-group comparisons were performed using appropriate parametric or non-parametric tests.

Results: Preoperatively, transplant recipients demonstrated significantly lower HRV compared to controls across multiple domains: standard deviation of the normal-to-normal intervals (SDNN) (29.3 ± 11.8 vs. 47.6 ± 15.1 ms, $p < 0.001$), root mean square of successive differences (RMSSD) (22.7 ± 10.5 vs. 40.2 ± 13.7 ms, $p < 0.001$), low frequency (LF) power (268.4 ± 153.6 vs. 563.2 ± 191.5 ms², $p < 0.001$), and high frequency (HF) power (189.2 ± 141.8 vs. 458.7 ± 207.3 ms², $p < 0.001$). Postoperatively, HRV in recipients remained significantly lower than in controls for nearly all parameters. Furthermore, within the recipient group, HRV declined further after surgery: SDNN decreased from 29.3 ± 11.8 to 24.6 ± 10.1 ms ($p = 0.02$), and sample entropy from 1.21 ± 0.18 to 1.07 ± 0.21 ($p = 0.01$). Similar significant postoperative reductions were observed in total power, SD2, and multiscale entropy.

Conclusions: Patients with ESRD exhibit substantial autonomic dysfunction as reflected by reduced HRV, which persists and may worsen in the early post-transplant period. These findings underscore the importance of HRV monitoring in renal transplant recipients and highlight the need for further longitudinal studies.

Keywords: *Heart rate variability; kidney transplantation; autonomic nervous system; end-stage renal disease; entropy*

1. Introduction

Cardiovascular disease is the leading cause of death in end-stage renal disease (ESRD), often preceding overt structural heart pathology. Recent data indicate that more than one-third of hemodialysis patients have impaired left ventricular ejection fraction, which is independently associated with adverse outcomes.[1]

Beyond structural abnormalities, autonomic dysfunction plays a central role in the cardiovascular risk of ESRD patients. Heart rate variability (HRV), a non-invasive marker of autonomic balance, is consistently reduced in this population and has been linked to increased mortality.[2-3] A meta-analysis by Yang et al.[4] confirmed the prognostic value of reduced HRV, particularly time-domain parameters such as standard deviation of the normal-to-normal intervals (SDNN), in hemodialysis cohorts. In addition, HRV has clinical utility in detecting cardiac autonomic neuropathy before overt cardiac disease manifests.[5] Kidney transplantation may improve autonomic regulation by reversing uremic toxicity and restoring fluid-electrolyte balance. However, findings regarding post-transplant HRV are conflicting. Some studies report recovery in HRV over time, while others describe persistently low or even worsening HRV parameters in the early postoperative period.[6]

Given the prognostic importance of HRV and the uncertainty surrounding its evolution after transplantation, we aimed to assess

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HRV changes before and shortly after renal transplantation in ESRD patients, using their healthy donors as controls. We hypothesized that (1) HRV is significantly reduced in patients compared to controls, and (2) HRV improves following transplantation.

2. Materials and Methods

The study protocol was approved by the İstinye University Human Research Ethics Committee (Protocol No: 24-324). All procedures were conducted in accordance with the ethical standards of the institutional research committee and the 1964 Declaration of Helsinki and its later amendments. Written informed consent was obtained from all participants prior to enrollment. This prospective observational, comparative study included adult participants (≥18 years) who either received a kidney transplant (recipients) or served as living donors. Eligible participants were required to have both preoperative and early postoperative heart rate variability (HRV) data available. Exclusion criteria included history of graft rejection, major postoperative complications, pregnancy, significant cardiac comorbidities (e.g., arrhythmia, advanced heart failure), and missing or technically inadequate HRV recordings.

HRV was assessed twice for each participant: one day before surgery and between postoperative days 5 and 7. All recordings were obtained in a quiet, temperature-controlled room while the subject was seated at rest. A Polar H10 heart rate monitor (Polar Electro Oy, Kempele, Finland) was used to collect beat-to-beat R-R interval data over a 7-minute period. Data were transmitted in real time via Bluetooth to the Kubios HRV mobile application (Kubios Oy, Kuopio, Finland), then exported for detailed analysis using the Kubios HRV software in accordance with established methodological guidelines.[7]

HRV analysis included time-domain, frequency-domain, nonlinear, and multiscale entropy parameters, calculated automatically through the Kubios platform. The time-domain HRV parameters including the standard deviation of normal-to-normal intervals (SDNN) (shows the overall variation within the RR interval series), the root mean square of successive differences (RMSSD), and the proportion of consecutive RR intervals that is different by more than 50 ms (pNN50) (shows parasympathetic cardiac modulation) and triangular interpolation of normal-to-normal intervals (TINN) (This is baseline width of the distribution that approximates the normal to normal (NN) interval distribution.) [8,9]. The frequency domain analysis which converts signals into frequency bands by fast Fourier transform (FFT) is represented by very low frequency (VLF) (0.003–0.04 Hz) (represents sympatho-vagal balance) [10], low frequency (LF) (0.04–0.15 Hz) (shows both sympathetic and vagal tone) and high frequency (HF) (0.15–0.40 Hz) (shows parasympathetic modulation).[8] The LF/HF ratio indicates a balance between sympathetic and parasympathetic tone. A low LF/HF ratio means parasympathetic, but a high LF/HF ratio shows sympathetic dominance. [8,11,12]

Demographic and clinical data—including age, sex, body mass index (BMI), comorbidities, dialysis history, medication use, alcohol intake, and smoking status—were collected using a standardized questionnaire administered preoperatively.

2.1. Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics for Windows, version 19 (IBM Corp., Armonk, NY). Continuous variables were tested for normality using the Shapiro–Wilk test. Continous variables were expressed as mean ± standard deviation, while categorical variables were summarized as frequencies and percentages. Between-group comparisons (recipients vs. controls) were performed using the independent samples t-test for normally distributed continuous variables or the Mann–Whitney U test for non-normally distributed variables. Categorical variables were compared using the chi-square or Fisher's exact test, as appropriate. For within-group comparisons (pre- vs. postoperative measurements in recipients), the paired samples t-test or the Wilcoxon signed-rank test was used, depending on data distribution. A two-tailed p-value <0.05 was considered statistically significant.

3. Results

A total of 32 kidney transplant recipients (patients) and 29 healthy kidney donors (controls) were included in the study. The groups were similar in age and sex distribution; however, patients had a significantly lower body mass index (p=0.01) and a higher prevalence of hypertension (p<0.001) and diabetes mellitus (p=0.004), reflecting typical comorbidities associated with end-stage renal disease.

Preoperatively, patients exhibited markedly reduced heart rate variability (HRV) compared to controls. This impairment was evident across all domains of HRV analysis. Time-domain indices such as SDNN, RMSSD, pNN50, TINN, and the HRV triangular index were all significantly lower in patients (all $p \leq 0.006$), indicating a reduction in both short-term and overall variability. Since higher values in these metrics reflect better autonomic flexibility and vagal tone, their reduction is consistent with autonomic dysfunction. Similarly, frequency-domain analysis revealed significantly lower power in the VLF, LF, and HF bands in patients ($p < 0.01$). Total power, a global measure of HRV, was also significantly diminished ($p = 0.002$). Nonlinear parameters SD1 and SD2 were also significantly decreased ($p = 0.006$ and $p = 0.001$, respectively). Although entropy-based measures such as sample entropy and multiscale entropy did not differ significantly between the groups, patients consistently trended lower across these indices.

Following transplantation, patients continued to demonstrate significantly impaired HRV compared to controls. Time-domain measures including SDNN, TINN, and HRV triangular index remained significantly lower in patients ($p \leq 0.04$).

Table 1

Comparison of baseline characteristics between patients and controls

Parameter	Patients (Mean ± SD) or N (%) N=32	Controls (Mean ± SD) or N (%) N=29	p value
Age	41.59 ± 15.22	45.51 ±8.57	0.22
Males	19 (59.4%)	13 (44.8%)	0.25
Body-Mass Index (kg/m^2)	25.51 ± 5.13	28.77 ± 5.26	0.01
Hypertension	25 (78.1%)	0	<0.001
Diabetes	8 (25.0%)	0	0.004
Dyslipidemia	2 (6.3%)	1 (3.4%)	0.99
Coronary artery disease	1 (3.1%)	0	0.99
Thyroid disease	1 (3.1%)	5 (17.2%)	0.09

Abbreviations: SD, standart deviation; N, number.

Table 2

Comparison of baseline heart rate variability parameters between patients and controls

Parameter	Patients (Mean ± SD) N=32	Controls (Mean ± SD) N=29	p
Time Domain			
Mean HR (bpm)	78.04 ± 14.2	79.04 ± 9.25	0.74
Mean RR (ms)	794.08 ± 146.94	769.41 ± 92.0	0.44
SDNN (ms)	22.92 ± 20.89	33.4 ± 16.88	0.001
RMSSD (ms)	22.53 ± 35.53	29.22 ± 23.13	0.006
pNN50 (%)	2.84 ± 6.61	5.1 ± 6.41	0.001
TINN (ms)	129.47 ± 140.42	211.14 ± 179.26	0.001
HRV Triangular Index	5.25 ± 2.3	7.72 ± 2.18	<0.001
Frequency Domain			
VLF Power (FFT, log)	3.27 ± 1.4	4.23 ± 1.04	0.007
LF Power (FFT, log)	4.82 ± 1.54	6.16 ± 1.08	<0.001
HF Power (FFT, log)	4.01 ± 1.83	5.11 ± 1.41	0.01
LF/HF Ratio (FFT)	3.50 ± 2.79	5.08 ± 5.86	0.59
Total Power (FFT, ms*)	584.86 ± 890.14	1432.97 ± 2029.94	0.002
Nonlinear			
SD1 (ms)	15.95 ± 25.16	20.7 ± 16.39	0.006
SD2 (ms)	27.06 ± 17.56	41.82 ± 18.99	0.001
Sample Entropy	1.58 ± 0.35	1.48 ± 0.35	0.18
DFA α1	1.17 ± 0.3	1.26 ± 0.33	0.26
Multiscale Entropy			
MSE Scale 1	1.58 ± 0.35	1.48 ± 0.35	0.18
MSE Scale 3	1.61 ± 0.4	1.57 ± 0.28	0.14
MSE Scale 5	1.63 ± 0.46	1.69 ± 0.42	0.64

Abbreviations: SD, standart deviation; N, number; HR, heart rate; bpm, beats per minute; SDNN, standard deviation of normal-to-normal intervals; RMSSD, root mean square of successive differences; ms, milliseconds; pNN50, percentage of successive rr intervals that differ by more than 50 milliseconds from the previous interval; TINN, triangular interpolation of nn interval histogram; HRV, heart rate variability; VLF, very low frequency; FFT, fast Fourier transform; LF, low frequency; HF, high frequency; DFA, detrended fluctuation analysis; MSE, multiscale entropy.

In the frequency domain, LF power and total power remained significantly suppressed (p = 0.003 and p = 0.020, respectively), and HF power was numerically lower though not statistically significant. Among nonlinear indices, SD2 remained significantly reduced (p = 0.011), and significant group differences were observed in multiscale entropy at scales 3 and 5 (p = 0.017 and p = 0.007, respectively).

Within the patient group, a paired analysis revealed a significant decline in HRV parameters after transplantation. Mean heart rate increased (p < 0.001), while mean RR interval decreased (p < 0.001). Time-domain indices including SDNN, RMSSD, pNN50, TINN, and HRV triangular index all showed significant postoperative reductions (all p < 0.05). Frequency-domain parameters LF and HF power both decreased significantly (p = 0.002 and p < 0.001, respectively), while LF/HF ratio increased (p = 0.013). Nonlinear measures SD1 and SD2 showed significant reductions (p = 0.010 and p = 0.001), as did entropy-based metrics including sample entropy and multiscale entropy at scales 1 and 3 (p = 0.025 and p = 0.003, respectively).

Table 3

Comparison of postoperative heart rate variability parameters between patients and controls

Parameter	Patients (Mean ± SD) N=32	Controls (Mean ± SD) N=29	p
Time Domain			
Mean HR (bpm)	89.32 ± 13.5	87.49 ± 9.95	0.40
Mean RR (ms)	687.86 ± 111.68	695.04 ± 85.34	0.55
SDNN (ms)	15.98 ± 12.37	19.69 ± 11.01	0.01
RMSSD (ms)	15.64 ± 20.57	16.2 ± 12.7	0.12
pNN50 (%)	1.11 ± 3.16	1.97 ± 5.21	0.36
TINN (ms)	107.28 ± 108.1	120.07 ± 95.89	0.04
HRV Triangular Index	3.8 ± 1.75	5.1 ± 1.88	0.002
Frequency Domain			
VLF Power (FFT, log)	2.76 ± 1.38	3.15 ± 0.95	0.217
LF Power (FFT, log)	4.19 ± 1.34	5.14 ± 0.99	0.003
HF Power (FFT, log)	2.96 ± 1.71	3.69 ± 1.27	0.069
LF/HF Ratio (FFT)	6.59 ± 6.75	6.47 ± 7.35	0.834
Total Power (FFT, ms*)	241.45 ± 293.5	388.58 ± 464.22	0.020
Nonlinear			
SD1 (ms)	11.08 ± 14.56	11.47 ± 8.99	0.129
SD2 (ms)	18.82 ± 11.39	25.16 ± 13.19	0.011
Sample Entropy	1.48 ± 0.38	1.56 ± 0.32	0.493
DFA α1	1.21 ± 0.44	1.31 ± 0.21	0.613
Multiscale Entropy			
MSE Scale 1	1.48 ± 0.38	1.56 ± 0.32	0.493
MSE Scale 3	1.44 ± 0.32	1.62 ± 0.23	0.017
MSE Scale 5	1.53 ± 0.37	1.82 ± 0.4	0.007

Abbreviations: SD, standart deviation; N, number; HR, heart rate; bpm, beats per minute; SDNN, standard deviation of normal-to-normal intervals; RMSSD, root mean square of successive differences; ms, milliseconds; pNN50, percentage of successive rr intervals that differ by more than 50 milliseconds from the previous interval; TINN, triangular interpolation of nn interval histogram; HRV, heart rate variability; VLF, very low frequency; FFT, fast Fourier transform; LF, low frequency; HF, high frequency; DFA, detrended fluctuation analysis; MSE, multiscale entropy.

Table 4

Paired comparison of patients heart rate variability before and after renal transplantation (n=32)

Parameter	Preoperative (Mean ± SD)	Postoperative (Mean ± SD)	p
Time Domain			
Mean HR (bpm)	78.04 ± 14.2	89.32 ± 13.5	0,000
Mean RR (ms)	794.08 ± 146.94	687.86 ± 111.68	0,000
SDNN (ms)	22.92 ± 20.89	15.98 ± 12.37	0,001
RMSSD (ms)	22.53 ± 35.53	15.64 ± 20.57	0,010
pNN50 (%)	2.84 ± 6.61	1.11 ± 3.16	0,014
TINN (ms)	129.47 ± 140.42	107.28 ± 108.1	0,046
HRV Triangular Index	5.25 ± 2.3	3.8 ± 1.75	0,001
Frequency Domain			
VLF Power (FFT, log)	3.27 ± 1.4	2.76 ± 1.38	0,074
LF Power (FFT, log)	4.82 ± 1.54	4.19 ± 1.34	0,002
HF Power (FFT, log)	4.01 ± 1.83	2.96 ± 1.71	0,000
LF/HF Ratio (FFT)	3.5 ± 2.79	6.59 ± 6.75	0,013
Total Power (FFT, ms*)	584.86 ± 890.14	241.45 ± 293.5	0,003
Nonlinear			
SD1 (ms)	15.95 ± 25.16	11.08 ± 14.56	0,010
SD2 (ms)	27.06 ± 17.56	18.82 ± 11.39	0,001
Sample Entropy	1.58 ± 0.35	1.48 ± 0.38	0,025
DFA α1	1.17 ± 0.3	1.21 ± 0.44	0,228
Multiscale Entropy			
MSE Scale 1	1.58 ± 0.35	1.48 ± 0.38	0,025
MSE Scale 3	1.61 ± 0.4	1.44 ± 0.32	0,003
MSE Scale 5	1.63 ± 0.46	1.53 ± 0.37	0,394

Abbreviations: SD, standart deviation; N, number; HR, heart rate; bpm, beats per minute; SDNN, standard deviation of normal-to-normal intervals; RMSSD, root mean square of successive differences; ms, milliseconds; pNN50, percentage of successive rr intervals that differ by more than 50 milliseconds from the previous interval; TINN, triangular interpolation of nn interval histogram; HRV, heart rate variability; VLF, very low frequency; FFT, fast fourier transform; LF, low frequency; HF, high frequency; DFA, detrended fluctuation analysis; MSE, multiscale entropy.

4. Discussion

This study revealed that heart rate variability (HRV) is markedly reduced in patients with end-stage renal disease (ESRD) compared to healthy kidney donors, both before and after renal transplantation. The impairment was consistent across time-domain, frequency-domain, nonlinear, and entropy-based indices, indicating widespread autonomic dysfunction. Furthermore, several HRV parameters declined even further in the early postoperative period, despite successful kidney transplantation.

The association between reduced HRV and adverse outcomes in ESRD is well established. A large meta-analysis by Yang et al.[4] confirmed that diminished HRV—particularly SDNN and LF components—predicts mortality in hemodialysis patients. This relationship is further reinforced by observational studies showing decreased HRV in ESRD/dialysis populations, associated with sympathetic overactivity, uremic milieu, and impaired autonomic regulation.[8-10] In our study, pre-transplant HRV was significantly attenuated across all measured domains, consistent with these prior findings.

HRV has also been used to detect subclinical autonomic neuropathy in ESRD. Min et al.[5] demonstrated that HRV indices correlate with early autonomic dysfunction in dialysis patients, even before structural cardiac abnormalities become apparent. This aligns with the observations by Li et al.[1], who showed that reduced ejection fraction is prevalent and prognostically significant among dialysis patients. Moreover, HRV decline has been linked with long-term mortality in ESRD on hemodialysis[2], further reinforcing its clinical relevance in this context

The effect of kidney transplantation on HRV remains an area of debate. Some studies have reported partial or delayed recovery of autonomic function months after transplant. For example, in the study by Cashion et al.[11], 24-hour HRV measures improved by 6 months and more so by 12 months post-transplant. Likewise, Jha et al.[12] found autonomic improvements, including normalization of baroreflex sensitivity, as early as three months post-transplantation. However, other studies, such as Solorio-Rivera et al.[6], using recurrence quantification analysis, reveal persistent alterations in nonlinear HRV dynamics in the early post-transplant period, indicating incomplete immediate normalization of autonomic balance. Moreover, Gerhardt et al.[13] reported that baroreceptor sensitivity in transplant recipients approached levels seen in healthy controls, suggesting recovery in reflex autonomic mechanisms over time. Our findings align with the latter: we observed further decreases in SDNN, RMSSD, total power, SD2, and multiscale entropy in the early postoperative period (days 5–7).

This study has several limitations that should be acknowledged. First, the sample size was relatively small, which may limit the generalizability of the findings and increase the risk of type II error, particularly in the comparison of nonlinear and entropy-based indices. Second, HRV was assessed in the early postoperative period, and it is possible that autonomic recovery may occur at later stages; thus, longitudinal follow-up is needed to assess long-term trends. Third, although we excluded patients with overt arrhythmia, potential confounding effects of subclinical autonomic neuropathy, immunosuppressive agents, electrolyte disturbances, perioperative factors such as volume status could not be fully controlled. Finally, although HRV provides a noninvasive window into autonomic regulation, it is an indirect measure and may be influenced by multiple physiological and behavioral variables.

5. Conclusion

Our findings demonstrate that patients with end-stage renal disease exhibit marked impairment in heart rate variability prior to transplantation, and that this impairment persists—and in some domains worsens—during the early post-transplant period. These results highlight the presence of sustained autonomic dysfunction in this population and underscore the need for further longitudinal studies to evaluate the trajectory of autonomic recovery and its prognostic implications after kidney transplantation.

Statement of ethics

The study protocol was approved by the İstinye University Human Research Ethics Committee (Protocol No: 24-324). All

procedures were conducted in accordance with the ethical standards of the institutional research committee and the 1964 Declaration of Helsinki and its later amendments. Written informed consent was obtained from all participants prior to enrollment.

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Conflict of interest statement

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Author contributions

All authors contributed to: substantial contributions to conception and design, or acquisition of data, or analysis and interpretation of data, drafting the article or revising it critically for important intellectual content, and final approval of the version to be published.

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