ORIGINAL ARTICLE

The Effect of Hemodialysis on Electrocardiographic Parameters

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Background: Cardiovascular complications are the leading causes of premature deaths in hemodialysis patients. Due to rapid changes in volume and electrolyte concentration following dialysis, the some electrocardiographic (ECG) changes or arrhythmias might be seen.

Objective: To investigate the acute effects of hemodialysis on the ECG parameters in patients with chronic end-stage renal disease (ESRD).

Method: We included the consecutive ESRD patients who underwent a hemodialysis. Before and after hemodialysis, some 12 lead ECG parameters were analyzed by two different cardiologists by using electronic digital caliper device.

Results: A total of 62 patients (mean 52 ± 15 years; 65% male) with ESRD undergoing hemodialysis were recruited to the study. P-wave amplitude, QRS amplitude, QRS duration, QTc dispersion, the sum of amplitudes in V1S + V5R derivations, total QRS amplitude, and duration were significantly greater in posthemodialysis patients compared to the prehemodialysis ones. However, T-wave amplitude and QTc duration were significantly lower in posthemodialysis patients.

Conclusion: The ECG changes including prolangated QRS and increased QTc interval after hemodialysis should be kept in mind and assessed carefully in ESRD patients. Prolongation of these parameters may prove to be a further noninvasive marker of susceptibility to ventricular arrhythmias.

Ann Noninvasive Electrocardiol 2015;20(3):253–257

ECG; electrocardiography; hemodialysis

Cardiovascular events are the most common causes of morbidity and mortality in dialysis patients. Whether the rapid changes in volume and electrolyte concentration after hemodialysis lead to malignant arrhythmias has still been a matter of debate.[1–3] In addition to cardiac complications, anemia, hypertension, volume overload, electrolyte imbalance, hyperlipidemia, and arteriovenous fistula can also contribute to decreased lifespan in these patient groups.[4,5] An increase in wave voltage, such as QRS, T and P waves, has been attributed to an increase of the transfer impedance of the body by liquid extraction after hemodialysis. In addition to increasing wavelength, wave durations have also been shown to be effected by hemodialysis.[6,7] In this study, we aimed to investigate the acute effects of hemodialysis on the electrocardiogram (ECG) in patients with chronic end-stage renal disease (ESRD).

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Disclosures: There are no associations that may pose a conflict of interest concerning the submitted article. There is no relationship with industry.

DOI:10.1111/anec.12209

METHOD

We included the consecutive ESRD patients who underwent a hemodialysis. Fasting biochemical tests including creatinine, blood urea nitrogen (BUN), sodium, potassium, calcium, phosphorus, albumin, total cholesterol, triglyceride, high-density lipoprotein cholesterol (HDL), low-density lipoprotein cholesterol (LDL), aspartate transaminase (AST), alkaline phosphatase (ALP), high sensitivity C-reactive protein (hs-CRP), ferritin, iron, total iron binding capacity, parathormone, and complete blood counts (CBC) were analyzed. After hemodialysis, BUN, creatinine, sodium, potassium, and calcium levels were controlled.

A total 12 lead ECGs (Cardioline Delta 60 plus, Remco Italia S. p.A, Milan, Italy) were recorded from all patients just before and immediately after hemodialysis (Fresenius Medical Care 4008 S, Hamburg, Germany). In lead 2, P-wave amplitude, T-wave amplitude, QRS amplitude, QRS duration, PR interval, sum of V1S + V5R, QT interval, and QTc interval were calculated by two different cardiologists with digital caliper device (Control Company, Friendswood, TX, USA; resolution: mm/0.0005''). In all leads, sum of T-wave amplitudes, sum of the QRS periods, QT dispersion, and QTc (corrected QT) dispersion were also measured. QTc values were calculated by Bazett's formula ($QTc = QT / \sqrt{RR}$).

Transthoracic echocardiographic (Vivid 7 Dimension, GE Medical Systems, Horten, Norway) evaluation was performed with the patient in left lateral decubitus position. LV dimensions and wall thicknesses were obtained using M-mode cursor from the parasternal long axis view. LV end-diastolic (LVEDD) and end-systolic diameters (LVESD), and left atrial dimension were all measured using a single reader by the standards of the American Society of Echocardiography.[8] LV ejection fraction was measured in accordance with the Simpson's method.[9]

Exclusion criteria were the patients with short hemodialysis duration (less than 3 months), severe hyperkalemia (higher than 6 mEq/L), pericardial effusion, and acute coronary syndrome.

All statistical analyses were performed using Statistical Package for Social Sciences (SPSS) for Windows 20 (IBM SPSS Inc., Chicago, IL, USA). Normal distributions of variables were evaluated with Kolmogorov-Smirnov test. Numerical variables with a normal distribution were presented as the mean ± standard deviation, numerical variables with a skewed distribution were presented as the median (minimum and maximum), and categorical variables were presented as percentages. Electrocardiographic and echocardiographic data before and after hemodialysis were compared by using paired samples *t*-test or Wilcoxon signed ranks test according to normality test results. Relationships between the continuous variables were evaluated by Pearson's correlation analysis when data were normally distributed or by Spearman's correlation analysis when they were not normally distributed. For all tests, a 2-tailed $P < 0.05$ was considered significant.

Table 1. Etiologies of Renal Disease

Etiologies of Renal Failure	(n: 62)
Diabetes mellitus (n, %)	16 (25.8)
Hypertension (n, %)	16 (25.8)
Chronic glomerulonephritis (n, %)	16 (25.8)
Idiopathic (n, %)	14 (22.6)

RESULTS

A total of 62 patients with ESRD undergoing hemodialysis were recruited to our study. The ages of the patients ranged from 18 to 79 years (mean 52 ± 15 years) and 60% of the patients were male. Etiologies of renal diseases, baseline demographic, and laboratory parameters are given in Tables 1 and 2.

P-wave amplitude, QRS amplitude, QRS duration, QT dispersion (QTd), QTc dispersion, the sum of amplitudes of V1S + V5R derivations, total QRS amplitude, and total QRS duration were significantly greater in the posthemodialysis patients compared to the prehemodialysis patients. However, T-wave amplitude and QTc duration were significantly lower in the posthemodialysis patients. Effects of hemodialysis on electrocardiographic and echocardiographic parameters are shown in Table 3. Control renal functions and electrolyte levels are given in Table 4.

Pre- and postdialysis body weights were found to be 70.79 ± 17.22 kg and 68.70 ± 17.15 kg ($P < 0.001$), respectively. Correlation analysis revealed a negative correlation between QT dispersion and body weight before (r: –0.395, P: 0.003) and after (r: –0.389, P: 0.003) hemodialysis.

Table 2. Basal Demographic Clinical Characteristics and Laboratory Results

	Patients (n = 62)
Age (years)	52 ± 15
Gender (male, %)	37/62(60)
Average time on dialysis after first dialysis (month) (median [IQR])	36 (64)
Number of hemodialysis for a week (mean ± SD)	3 ± 0.2
Ultrafiltration level (mL; mean ± SD)	2196 ± 760
Duration of hemodialysis (hours) (median [IQR])	4(1)
Serum BUN (mg/dL; mean ± SD)	144 ± 34
Serum creatinine (mg/dL; mean ± SD)	8.60 ± 2.20
Serum sodium (mEq/L; mean ± SD)	137 ± 3.5
Serum potassium (mEq/L; mean ±SD)	5.08 ± 0.78
Serum calcium(mg/dL; mean ± SD)	2.19 ± 0.21
Serum phosphorus (mg/dL; mean ± SD)	5.8 ± 1.8
Serum albumin (g/dL; mean ± SD)	3.9 ± 0.4
Serum parathormone (pg/mL; mean ± SD)	361 ± 566
Serum total cholesterol (mg/dL; mean ± SD)	166 ± 41
Serum LDL cholesterol (mg/dL; mean ± SD)	93 ± 32
Serum triglyceride (mg/dL; mean ± SD)	177 ±98
Serum AST (mg/dL; mean ± SD)	18 ± 18
Serum ALT (mg/dL; mean ± SD)	19 ± 19
Serum ALP (mg/dL; mean ± SD)	94 ± 46
Serum hs-CRP (pg/mL; mean ± SD)	5 ± 8.1
White blood cell ($\times 10^3$/mm^3; mean ± SD)	7.8 ± 2.3
Mean platelet volume (fL; mean ±SD)	8.5 ± 0.8
Hemoglobin (g/dL; mean ± SD)	11.7 ±1.6
Serum ferritin (ng/mL; mean ± SD)	559 ±294
Serum iron (μg/dL; mean ± SD)	70 ± 30
Total iron binding capacity (%; mean ± SD)	175 ± 45

Abbreviations as in text.

QTc dispersion was also found to be negatively correlated with body weight before (r: –0.295, P: 0.025) and after (r: –0.311, P: 0.02) hemodialysis.

DISCUSSION

We showed that some electrocardiographic and echocardiographic parameters have changed after hemodialysis in this study. Indeed, the cardiovascular events are the most common cause of death in ESRD patients,[10] and cardiac complications, such as heart failure, pericarditis, coronary arterial disease, and arrhythmias are frequent among hemodialysis patients. Increased QT dispersion is associated with increased morbidity and mortality due to cardiovascular reasons.[11] QT dispersion, obtained through calculation of the difference between the longest and the shortest QT interval in ECG, represents the heterogeneity of myocardial repolarization. A greater QT dispersion causes a decreased level of homogeneity in the ventricular repolarization process, hence a higher ventricular instability might lead to serious ventricular arrhythmias and sudden cardiac death.

Prolongation in the corrected QT dispersion is encountered in the majority of dialysis patients, and increased QT dispersion has been known to be associated with increased risk of sudden cardiac death.[12] Changes in fluid volume and electrolyte level cause an increase in QT dispersion through electrical conduction delay. Perhaps in the future, with the improvement of precision ECG recording devices, hemodialysis may result in a more accurate liquid ultrafiltration and a decreased susceptibility to cardiac arrhythmias.

Increase in the amplitude of QRS wave in patients undergoing hemodialysis has long been reported and myocardial ischemia, volume changes, and cardiac conduction abnormalities are considered to be among the causative factors. The effect of hemodialysis-induced fluid loss may cause an increase in the amplitude of the QRS due to increased body impedance.[13] In general, volume loss through hemodialysis reduces transmissibility, since the ultrafiltrated liquid resistance is lower

Table 3. Comparison of Electrocardiographic and Echocardiographic Parameters before and after Hemodialysis

	Before Hemodialysis	After hemodialysis	P Value
Electrocardiographic findings			
RR interval (ms) (median [IQR])	770 (264)	738 (213)	0.56
PR interval (ms; mean ± SD)	172.07 ± 27.97	171.25 ± 28.39	0.58
P amplitude (mm) (median [IQR])	1.5 (0.5)	1.5 (0.9)	0.001
QRS amplitude (mm) (median [IQR])	7.0 (5.5)	9 (7)	<0.001
QRS duration (ms; mean ± SD)	87.88 ± 21.52	97.38 ± 20.56	<0.001
T amplitude (mm) (median [IQR])	2.1 (2)	2.0 (2.2)	0.001
ST depression (mm; mean ± SD)	0.10 ± 0.22	0.11 ± 0.24	0.30
QT interval (ms; mean ± SD)	378.21 ± 44.34	366.24 ± 38.63	<0.001
Corrected QT (QTc; ms; mean ± SD)	444.87 ± 43.98	430.36 ± 36.84	0.01
QT dispersion (QTd; mean ± SD)	61.96 ± 25.06	74.35 ± 24.95	<0.001
Corrrected dispersion (QTcd; ms; mean ± SD)	71.57 ± 28.40	86.83 ± 27.58	<0.001
Sum of QRS duration (ms; mean ± SD)	1093.2 ± 173.2	1190.1 ± 159.07	<0.001
Sum of QRS amplitude (mm; mean ± SD)	127.77 ± 38.74	148.77 ± 44.81	<0.001
Sum of T-wave amplitude (mm; mean ± SD)	32.70 ± 12.68	27.88 ± 11.02	<0.001
Sum of V1S + V5R (mm; mean ± SD)	20.05 ± 9.27	23.05 ± 11.31	<0.001
Echocardiographic findings			
LVEDD (cm; mean ± SD)	4.99 ± 0.43	4.87 ± 0.42	<0.001
LVESD (cm; mean ± SD)	3.49 ± 0.56	3.43 ± 0.55	<0.001
LVEDV (mL; mean ± SD)	119 ± 24	112 ± 22	<0.001
LVESV (mL; mean ± SD)	52 ± 21	50 ± 20	<0.001
LVEF (%; mean ± SD)	57.00 ± 9.85	56.13 ± 10.45	<0.001
LA size (cm) (median [IQR])	3.9 (0.4)	3.8 (0.35)	<0.001
LA volume (mL) (median [IQR])	66 (16)	66 (16)	<0.001

SD = standard deviation; IQR = interquartile range; LVEDD = left ventricular end diastolic diameter; LVESD = left ventricular end sistolic diameter; LA = left atrium; LVEF = left ventricular ejection fraction; LVEVD = left ventricular end diastolic volume; LVESV = left ventricular end sistolic volume.

Table 4. Comparison of Renal Functions and Electrolyte Levels Taken before and after Hemodialysis

	Before Hemodialysis	After Hemodialysis	P Value
Serum BUN (mg/dL; mean ± SD)	144.71 ± 34.37	53.90 ±19.19	<0.001
Serum creatinine (mg/dL; mean ± SD)	8.60 ± 2.20	3.62 ± 1.29	<0.001
Serum sodium (mEq/L; mean ± SD)	137.08 ± 3.47	135.77 ± 3.49	<0.001
Serum potassium (mEq/L; mean ± SD)	5.08 ± 0.78	3.58 ± 0.56	<0.001
Serum calcium (mg/dL; mean ± SD)	2.19 ± 0.21	2.17 ± 0.18	0.11

Abbreviations as in text.

compared to the other body fluids. Therefore, the transfer impedance increases due to withdrawal of fluid during hemodialysis and QRS amplitude increases (Ohm's Law).[14] This mechanism is not only valid for QRS amplitude, but also for all ECG waves. Accordingly, the P-wave amplitude can be expected to increase after hemodialysis.[15] In our study, we found a statistically significant prolongation in QRS duration, increase in QRS and P-wave amplitudes in lead 2, as well as increase in the sum of QRS durations and the sum of QRS amplitudes in the immediately posthemodialysis period. The electrical stimuli arising from the heart disseminate throughout the body surface with diminishes proportional to the bodily fluid distribution. Such electrical potentials diminish even further in dialysis patients with peripheral edema or in those patients with peripheral edema due to other reasons. These changes in the QRS amplitude are not of cardiac, but extracardiac origin, which was conferred from the studies in which intracardiac recordings performed on edematous patients who were at their maximum body weight, and following loss of edema yielded no change in the QRS amplitude, while successive surface ECG recordings showed changes in the QRS amplitudes in patients with peripheral edema due to other reasons. Other data supporting this condition were provided by studies reporting differences in the amplitude of pacemaker stimuli of surface ECGs, which were measured directly from the pacemakers in patients with peripheral edema. These changes represent the results of measurements and are not ultimate, namely, they do not occur as a result of cardiac electrophysiologic mechanisms.[16]

Decreased level of potassium leads to slower myocardial conduction and acts as a possible causative reason for prolongation of QRS duration, thereby meriting further investigations. With this in mind, hypokalemia may contribute to prolongation of QRS duration through synergetic or additive effect in patients with peripheral edema due to other reasons, although this often was not noted. Moreover, dialysis causes decrease in blood potassium level and such a change in the bodily electrical milieu might be the true reason behind the prolongation of QRS duration following hemodialysis in dialysis patients.[17] Decreased T-wave amplitudes after hemodialysis reported in our study may stem from decreased blood potassium levels. As is well known, T-wave amplitudes decrease with decreasing blood potassium levels.

After fluid withdrawal, increment in the sum of amplitudes of V1S + V5R should be remembered in the hemodialysis patients, these results suggest that hemodialysis is an important factor when the left ventricular hypertrophy criteria are evaluated.[18]

We showed that some ECG parameters in ESRD patients undergoing hemodialysis might change during hemodialysis. Prolongation of these parameters may prove to be a further noninvasive marker of susceptibility to ventricular arrhythmias. It is proposed that these increases in QRS duration are only representing an extracardiac phenomenon mediated by alterations in the composite impedance of the passive body volume conductor, resulting in measurement of augmented QRS complexes after fluid removal. The QRS amplitude increases after hemodialysis, but the cause of this increase is still unclear. These ECG changes should be kept in mind and ECG should be carefully evaluated in the posthemodialysis period.

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