Hemodialysis-induced ST-segment Deviation

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Abstract

ECGs of 59 patients undergoing hemodialysis (HD): 52% males, age 59 ± 13 years, renal disease duration 9.7 ± 6.7 years, hemodialysis duration 5.2 ± 4.4 years were recorded. Serum electrolytes (potassium-K, sodium-Na, phosphorus-Ph and calcium-Ca), urea and creatinine levels were evaluated before and after HD. ECG analysis on an average P-QRS-T interval in order to avoid accidental events or noise was performed.

Pre- and post-HD ECG measurements (mean ± *standard deviations) in lead V2 were:*

ST-dev_pre: 0.13±0.18 mV; *ST-dev_post:* 0.15±0.22 mV; *p*=0.03

QRS-ampl_pre: 1.34±0.65 *mV*; *QRS-ampl_post:* 1.55±0.79 *mV*; *p*<<0.001

T-ampl_pre: 0.43±0.31 mV; *T-ampl_post:* 0.36±0.28 mV; *p*=0.0016.

HD leads to a significant increase in the QRS and decrease of the T-wave amplitude and a considerable shift in the ST-segment. The decrease of the T-wave amplitude and the upward shift of the ST-segment could be explained by potassium decrease during HD. QRS amplitude increase could be explained by the decrease of the extracellular fluid and blood volume and hence a decrease of the cardiac preload.

1. Introduction

Hemodialysis (HD) is the most common method used to treat end-stage renal disease. It removes waste products and free water from the blood, restoring a proper balance of electrolytes. This procedure causes substantial changes in the electrical activity of the heart, observed by analysis of electrocardiograms (ECG).

A session of dialysis results in sudden shifts in volume and electrolytes within a short time that alters the physiological milieu and could lead to sudden changes in the myocardial vulnerability to serious arrhythmias. The most frequent cause of arrhythmias appears to be related to changes in fluid status and electrolytes, particularly potassium [1]

Initial evidence of HD-induced myocardial ischemia has previously come from ECG-based studies demonstrating silent ST-segment depression that occurs during dialysis at rates that vary between 15 and 40% [2]. Singh et al. [3] assessed dialysis-induced ischemia using sestamibi single-photon emission computed tomography. In an unselected group of ten dialysis patients without a history of coronary artery disease, seven developed perfusion defects during dialysis.

Hemodialysis is often associated with a risk of cardiac dysfunction. In a paper of Nakamura et al. [4], measurements and analysis of ST-elevation in pre- and post-HD patients are used for prediction of coronary artery disease and cardiac events. The authors report HD- induced ST-elevation of $\geq 1 \text{ mV}$, in 18 out of 61 patients. During follow-up of 21 ± 2 months, all patients from the group with ST-elevation, as well as 21 from the rest of the study group experienced cardiac events. The authors explain this common manifestation of symptomatic and silent myocardial ischemia by reduced coronary artery oxygen delivery and increased myocardial oxygen demand during HD. Taki et al. [5] also report 'oxidative stress' in HD patients.

Another reason for the ST-elevation increase during HD could be associated to the hemodynamic instability, and especially to the reduction in myocardial blood flow [6]. Blood flow reduction during HD was analyzed by serial measurements using positron emission tomography. The aim of this study is to investigate the ECGs of patients undergoing hemodialysis and to assess the QRS and T-wave changes, as well as the ST-segment deviation induced by the procedure.

Objective of the current research is the analysis of the ECG changes induced by HD in order to obtain risk markers of arrhythmia, heart failure or cardiac death.

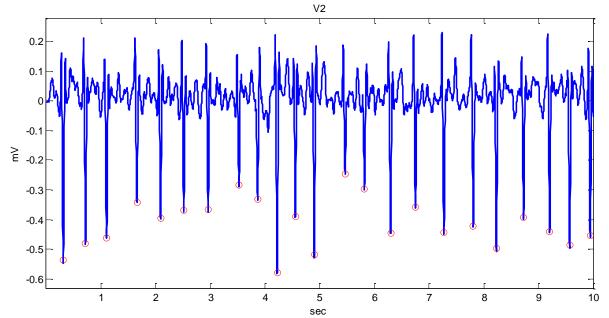


Figure 1. Lead V2 of a patient with atrial fibrillation. The red circles are the fiducial points achieved by cross correlation

2. Hemodialysis Database

We studied 58 patients, age 59 ± 13 years, 52% males, with renal disease duration 9.7 ± 6.7 years and hemodialysis duration 5.2 ± 4.4 years. Digital ECGs (1minute duration, 12-standard leads, 500 Hz sampling rate) were recorded before and after hemodialysis session. Serum electrolytes (potassium-K, sodium-Na, phosphorus-Ph and calcium-Ca), urea and creatinine levels were evaluated before and after hemodialysis. Percentage change of the above-mentioned parameters during hemodialysis was estimated. We also calculated mean hemodialysis clearance.

All patients signed an informed consent for personal data analysis. The study protocol is in accordance with the Declaration of Helsinki.

3. Method

The ECG signals were preprocessed to eliminate or suppress power-line interference [7], drift [8] and electromyographic noise [9,10].

QRS detection was applied [11] and fiducial points for best matching of successive P-QRS-T intervals were achieved by cross correlation. The fiducial points' location was needed because the applied QRS detection algorithm triggers at arbitrary moments during the QRS.

ST-elevation has been measured in V2-lead, 80 ms after the end of QRS (the J point). Lead V2 is shown in Figure 1. It can be seen that measurement of ST-elevation and amplitudes of QRS and T-wave in any selected P-QRS-T interval of the figure is unreliable. In order to

avoid inaccuracies in the measurement, it is performed on an average P-QRS-T interval. The best fitting is achieved by correlation analysis, (Figure 2). Determining the beginning and the end of the QRS (marked with red circles) and the location where the ST-elevation should be measured (green circle) is done automatically [12]. To avoid error caused by residual drift of the zero line, the amplitude of ST-elevation is measured according to the amplitude at the beginning of the QRS complex, and not according to ECG zero line.

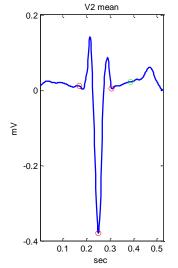


Figure 2. Mean P-QRS-T interval of the ECG in Fig.1. The red circles are the QRS onset and offset. The green circle is the point where the ST-elevation is measured (80ms after the QRS-offset)

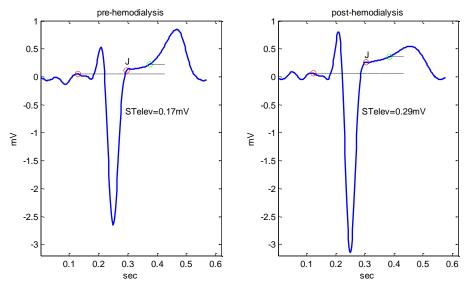


Figure 3. P-QRS-T interval of one and the same individual, pre- and post-hemodialysis. The pre-HD ST-elevation = 0.17 mV, while the post-HD ST-elevation = 0.29 mV. The QRS amplitude increases with approximately 1.8 mV, while the T-wave amplitude decreases with approximately 0.4 mV.

4. Results

Pre- and post-HD measurement of ST-deviation was performed in every patient of the group. The result of the analysis for ST-deviation showed: 51 cases had both preand post- ST-elevation, 3 subjects had both pre- and post-ST-depression, and 5 patients had mixed ST elevation/depression. HD-induced ST-deviations (higher post-deviations compared to pre-deviations) were observed in 35 individuals. ST deviation increased in this group from 0.09 ± 0.10 mV to 0.19 ± 0.27 mV (by 0.096mV ±0.26), p=<<0.001.

The pre- and post-HD measurements of ST-deviation, QRS amplitude and T-wave amplitude are presented in Table 1.

Table 1. Parameters' change during hemodialysis

Parameter	Pre- dialysis	Post- dialysis	Р
ST dev. (mV)	0.13±0.18	0.15 ± 0.22	0.03
QRS ampl. (mV)	1.34 ± 0.6	1.54 ± 0.8	< 0.001
T ampl. (mV)	0.43 ± 0.31	0.36 ± 0.28	0.0016

Example of HD-induced ST-elevation is shown in Figure 3.

During HD systolic blood pressure decreased from 137 ± 23 to 126 ± 21 mmHg, and diastolic blood pressure – from 83 ± 12 to 76 ± 9 mmHg, p <0.001 for both.

5. Discussion

In the present study with 58 HD-patients, we found

that HD is associated with a significant change in QRS and T wave amplitude and with a considerable shift in ST segment. We have already reported [13] for changes of vectorcardiographic (VCG) parameters during hemodialysis: QRS-loop and T-loop areas, QRS- and Tmaximal vectors' amplitudes, spatial angle between QRS and T-wave. Analysis of the current results shows that dialysis leads to a significant increase of the QRS amplitude (p<<0.001). This is in accordance with the increase of the QRS-maximal vector in [13], which has been shown to be independently predicted and negatively correlated to hemodialysis duration in years, i.e. the longer a patient has been on this treatment; the smaller is the change in QRS amplitude during HD. The fact that the decrease of patient's blood volume and intra-cellular liquid is leading to QRS amplitude increase has been discussed in Simov et al. [14]. The authors have mentioned that it is a common practice to dehydrate by medication patients who have underwent coronary artery bypass grafting. They have shown that the QRS amplitude increases proportionally to the prolongation (and hence to the extent of dehydration) from the time of ECG recording after the surgery.

QRS amplitude increase after HD has also been shown in previous studies [15,16], where it was related to the extracellular water and blood volume loss. The mechanism involved is most probably change of electrical resistance of the tissues surrounding the heart caused by loss of interstitial fluid. Since QRS augmentation has been linked to volume shift during the procedure, our finding could mean that percentage change in maximal QRS vector could be regarded as an indirect marker of the efficiency of HD. There are also conflicting studies: Madias and Narayn [17] report that in 26 dialysis sessions of one and the same patient, the weight loss, which is correlated to the water clearance, cannot be directly determined by the QRS amplitude change.

The T-wave amplitude change during HD has not been studied as much. In our group, it turned out that this parameter decreased significantly during the procedure (p=0.0016) and the decrease could be partially explained and inversely correlated to sodium (Na) concentration change, which has been previously demonstrated [13]. The decrease in T-wave amplitude and the upward shift of the ST-segment could be explained by potassium (K) decrease during HD. Baseline ST-segment shift, however, showed mean positive deviation, while ST depression is typical for mild hyperkalaemia.

There was an increase (upward shift) in ST-deviation with 0.02 mV (p=0.03) in the entire group. We are tempted to relate T-wave deviation during HD to myocardial ischemia and it has been previously demonstrated that HD could induce myocardial ischemia [2-4]. Unfortunately, we have not evaluated our patients for inducible myocardial ischemia. On the other hand, electrolyte shift is a very plausible reason for a deviation in the ST segment.

According to Saravanan and Davidson [1], the HD causes sudden shifts in volume and electrolytes within a short time that alters the physiological milieu. This leads to reduced coronary artery oxygen delivery while increasing myocardial oxygen demand during HD [4], the so-called 'oxidative stress' [5]. The HD-induced 'silent ischemia', expressed as a ST-deviation in ECG, leads to sudden changes in the myocardial vulnerability to serious arrhythmias [1]. The ST-deviation can be used for prediction of coronary artery disease [3].

6. Conclusion

HD is associated with a significant increase in QRS amplitude and decrease in T wave amplitude. There is also a significant shift in ST segments during HD. The etio-pathogenesis and future significance of these changes is not thoroughly explained and requires further investigation.

Acknowledgements

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