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CLINICAL STUDY

Influence of Intraperitoneal Volume on QT Dispersion in Patients with Continuous Ambulatory Peritoneal Dialysis: Acute Cardiac Impact of Peritoneal Dialysis

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Abstract

Aim: The leading cause of mortality in dialysis patients is cardiovascular complications, including ventricular arrhythmias and sudden cardiac death. QT dispersion (QTd), a simple noninvasive arrhythmogenic marker, is used to assess homogeneity of cardiac repolarization. It was also significantly prolonged in continuous ambulatory peritoneal dialysis (CAPD) patients. The acute cardiac effect of increased abdominal pressure due to infused dialysate during CAPD is not clear yet. In this study we aimed to evaluate corrected QTd (cQTd) and cardiac injury markers such as plasma pro-brain natriuretic peptide (proBNP) and troponin I (TnI) in CAPD patients before and after an infusion of peritoneal dialysate fluid. *Methods*: Thirty subjects (16 women, 14 men; mean age, 40.21 $\pm$ 12.34 years) enrolled in our study. QTd, cQTd, maximum QT (QTmax), maximum corrected QT (cQTmax), minimum QT (QTmin), and minimum corrected QT (cQTmin) intervals were measured from standard 12-lead electrocardiography. *Results*: We found that cQTmax, cQTmin, and cQTd were not changed from baseline measurement after infusion of dialysate in CAPD patients (460 $\pm$ 49 vs. 460 $\pm$ 38, $p = 0.9$; 410 $\pm$ 36 vs. 410 $\pm$ 41, $p = 0.8$; 470 $\pm$ 30 vs. 460 $\pm$ 25, $p = 0.7$, respectively). There were no statistically significant differences between before and after peritoneal dialysate according to the levels of proBNP and TnI (155.64 $\pm$ 76.41 vs. 208.30 $\pm$ 118.46, $p = 0.2$; 0.008 $\pm$ 0.007 vs. 0.01 $\pm$ 0.011; $p = 0.4$, respectively). *Conclusion*: In conclusion, we did not find any significant effect of peritoneal dialysate fluid infusion volume on QTd and cardiac injury markers in patients with chronic renal failure receiving CAPD therapy, which is thought to be a safer modality of dialysis.

Keywords: QT dispersion, infused dialysate volume, continuous ambulatory peritoneal dialysis

INTRODUCTION

Cardiovascular mortality and morbidity rates are high in chronic renal failure (CRF) patients. It has been shown that increased QT dispersion (QTd) is also associated with arrhythmias. Previous studies in nonuremic individuals have demonstrated the association between cardiac mortality, in particular sudden death with increased QTd, and defined QTd as the difference between the maximal and minimal QT intervals on a standard electrocardiogram.[1,2] Also increased QTd was significantly higher in hemodialysis (HD) and continuous ambulatory peritoneal dialysis (CAPD) patients when compared with healthy volunteers.[3] This is possibly explained by different mechanisms of arrhythmia in CRF when electrolytic disorders are the main provoking cause. The cardiac effect of increased abdominal pressure due to infused dialysate during CAPD is not clear yet. In this study we aimed to evaluate QTd and corrected QTd (cQTd) in CAPD patients before and after an infusion of peritoneal dialysate fluid.

METHODS

Study Population

The patients who visited the outpatient clinic of the dialysis unit in our hospital enrolled in this study. The patients were on a CAPD program consisting of

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four dwells daily with 2000 cm^3 peritoneal fluid. Standard 12-lead electrocardiography (ECG) was recorded while patients were in supine position with an empty abdomen, after 20–30 min of quiet resting in a semirecumbent position. After infusion of the 2000 mL dialysate, repeated ECG was recorded by the same registered nurse. The plasma pro-brain natriuretic peptide (proBNP) and troponin I (TnI) levels were measured just after the recordings. We also measured proBNP and TnI levels 4 h after an infusion of peritoneal dialysate fluid. Patients with a history of coronary artery disease, diabetes mellitus, left ventricular (LV) wall motion abnormality, LV ejection fraction <50%, primary cardiomyopathy, valvular heart disease, atrial fibrillation and other ECG abnormalities, pulmonary disease, thyroid dysfunction, amyloidosis, and electrolyte imbalance and if the ending of the *T* wave of a patient's ECG could not be determined reliably were excluded from this study. All patients were in sinus rhythm, and none of them were taking medications that could affect QT intervals.

Electrocardiography

All standard 12-lead ECGs were obtained using a recorder (Nihon Kohden, FQW 110-2-140, Nihon Kohden WEP-4208, Tokyo, Japan) set at 25 mm/s paper speed and 10 mm/mV amplitude. QT intervals were measured manually from the point where the Q wave started and the T wave returned to the isoelectric line. To decrease the error probability, QT intervals were measured manually with caliper and magnifying glass. Subjects with U waves on their ECGs were excluded from this study. ECG assessments were done by two medically qualified observers without knowledge of the patient's clinical status.

Whenever possible, 3 consecutive cycles in each of the 12 leads were measured and the mean QT interval calculated. The precedent RR interval to the measured QT was used to calculate the corrected heart rate (cQT) interval using Bazett's formula in milliseconds. Maximum QT (QTmax) was determined as the lead with the longest QT interval. Minimum QT (QTmin) was determined as the lead with the shortest QT interval. The QTd, as previously defined, was calculated as the difference between the QTmax and QTmin intervals obtained in any of the 12 electrocardiographic leads.

Laboratory Measurements

All laboratory measurements for proBNP and TnI were repeated after infusion of 2000 mL dialysate. proBNP and TnI levels were also measured 4 h after infusion. The samples were immediately placed on ice in prechilled ethylenediamine tetraacetic acid tubes. Plasma is obtained by separation from whole blood by centrifugation at 3000 rpm (704 × *g*) for 15 min at 4°C. proBNP and TnI were measured with a commercially available fluorescence immunoassay (Triage Meter Plus, Biosite Diagnostics, San Diego, CA, USA). The normal values were <100 pg/mL for proBNP and <0.4 ng/mL for TnI. The detection limit was 5 pg/mL for proBNP and 0.05 ng/mL for TnI.

Statistical Analyses

SPSS 16.0 statistical program was used for statistical analysis. All values are presented as mean ± standard deviation. Values between different groups were compared using the independent samples *t*-test. Chi-square test was used to assess the differences between categorical variables. The relationship between parameters was determined using the Pearson coefficient of correlation. *p*-Values lesser than 0.05 were considered significant.

RESULTS

Thirty subjects (16 women, 14 men; mean age, 40.21 ± 12.34 years) enrolled in our study. The characteristics of the patients are shown in Table 1. The etiology of the kidney disease was unidentified in 19 patients (63.3%), chronic glomerulonephritis in 6 patients (20%), obstructive uropathy in 3 patients (10%), solitary kidney in 1 patient (3.3%), and vesicoureteral reflux in 1 patient (3.3%).

There were no statistically significant differences among the groups when measured before and after infusion of dialysate in terms of maximum corrected QT

Table 1. Clinical and laboratory characteristics of the subjects.

Age, years	40.21 ± 12.34
Gender (men/women)	14/16
Weight, kg	63.85 ± 16.18
Disease duration, months	30.21 ± 24.31
Systolic BP, mmHg	133.10 ± 20.15
Diastolic BP, mmHg	83.52 ± 12.26
CRP, mg/dL	1.17 ± 1.17
Blood urea nitrogen, mg/dL	59.67 ± 18.73
Creatinine, mg/dL	8.5 ± 3.3
K, mmol/L	4.26 ± 0.73
Ca, mg/dL	8.85 ± 0.84
Mg, mmol/L	0.88 ± 0.17

Notes: BP, blood pressure; CRP, C-reactive protein; K, potassium; Ca, calcium; Mg, magnesium. Values are presented as mean ± standard deviation.

Table 2. Corrected QT dispersion in CAPD patients before and after an infusion of dialysate.

	Before infusion of dialysate	After infusion of dialysate	*p*-Value
cQTmax, ms	460 ± 49	460 ± 38	0.9
cQTmin, ms	410 ± 36	410 ± 41	0.8
cQTd, ms	470 ± 30	460 ± 25	0.7

Notes: cQTmax, maximum corrected QT; cQTmin, minimum corrected QT; cQTd, corrected QT dispersion. Values are presented as mean ± standard deviation.

Table 3. Serum proBNP and TnI levels in CAPD patients before and after an infusion of dialysate.

	Before infusion of dialysate	After infusion of dialysate	4 h after infusion of dialysate	*p*-Value
ProBNP, pg/mL	155.64 ± 76.41	208.30 ± 118.46	204 ± 96.42	0.2
TnI, ng/mL	0.008 ± 0.007	0.01 ± 0.011	0.009 ± 0.008	0.4

Notes: ProBNP, pro-brain natriuretic peptide; TnI, troponin I. Values are presented as mean ± standard deviation.

(cQTmax), minimum corrected QT (cQTmin), corrected QTd (cQTd) (Table 2), and plasma levels of proBNP and TnI (Table 3).

DISCUSSION

In this study we found that infused dialysate volume did not affect QTd in CAPD patients. In addition, the biomarkers of cardiac injury such as plasma proBNP and TnI did not change with dialysate infusion.

Despite the inherent differences between HD and peritoneal dialysis (PD), overall cardiovascular outcomes appear to be similar between the two modalities.[4,5] Selby et al.[6,7] showed that subclinical myocardial ischemia and stunning occur in response to the hemodynamic stress of HD with ultrafiltration. The development of new LV regional wall motion abnormalities during HD, as determined by echocardiography, is indeed due to myocardial hypoperfusion.[8] In contrast to HD, the acute effects of PD do not result in subclinical myocardial ischemia. According to these studies, it emphasized that PD may be a safer and a more eligible modality than HD.[9] However, CAPD may exert significant effects on systemic hemodynamics. These hemodynamic changes appear to be mediated by fluctuations in peripheral resistance, which falls when fluid is drained out of the abdomen and rises upon fluid instillation. This is most likely due to the compression of mesenteric resistance vessels and is consistent with the results observed with serial automated PD exchanges.[10] Moreover in CAPD patients, the higher blood pressure was due to higher heart rate, stroke volume, and cardiac output during 3.86% than 1.36% glucose dwells.[11]

The cardiovascular complications are the leading causes of mortality in dialysis patients including ventricular arrhythmias and sudden cardiac death. The interlead variation in duration of the QT interval on the surface electrocardiogram known as the QTd, a marker of the overall variability of ventricular repolarization, might serve as a surrogate for ventricular arrhythmia. Prolonged cQTd is commonly encountered in dialysis patients.[12] They may predict the risk of malignant arrhythmias. CAPD patients receive dialysis daily and they also have higher rates of QTd. In the literature, conflicting results have been reported on the potential link between CAPD and HD. Some authors suggested that QTd was significantly prolonged in HD patients compared with CAPD patients.[13] It might be due to the effect of different dialysis modalities on electrolytes, especially serum calcium levels. However, Kantarci et al.[3] showed similar QTd between CAPD and HD patients. In this study, cQTmax, cQTmin, and cQTd were not changed from baseline measurement after infusion of dialysate.

The proBNP is a hormone synthesized and released by LV myocytes, and its generation rate is amplified by heart failure or LV hypertrophy. Upon stress-induced secretion of BNP, proBNP in cardiomyocytes is cleaved into the active hormone BNP and inactive N-terminal proBNP.[14] Elevated baseline plasma NT-proBNP levels are associated with a higher risk of cardiovascular events in patients with end-stage renal disease.[15] Thereafter, NT-proBNP levels act as indicators for the changes of LV filling pressure in chronic HD.[16] TnI, a group of cardiac-specific contractile proteins, is a predictor of myocyte death. The increases in BNP and TnI are reflections of hypervolemia in CAPD patients.[17] In our study, there were no statistically significant differences between before and after peritoneal dialysate according to the levels of proBNP and TnI.

In one study, proBNP levels were correlated with several inflammation markers in patients with CAPD and some authors also suggested that CAPD patients have chronic inflammation.[18,19] In our study, while the serum levels of TnI were normal, proBNP levels were higher than cutoff values. On the other hand, after peritoneal dialysate fluid infusion, an increase in proBNP levels has been detected without a statistically significant difference. From a theoretical point, according to our findings we hypothesize that minimal elevation of proBNP levels may be related to chronic inflammation.

There are some limitations to this study. The patient size should be larger in order to comment any sufficient conclusions. Echocardiographic examination and intraperitoneal pressure measurements should be performed before and after the infusion of dialysate. However, in this study we aimed to investigate the effect of dialysate infusion volume on cardiac arrhythmia and QTd independently from intraperitoneal pressure and cardiac filling.

In conclusion, we did not find any significant effect of peritoneal dialysate fluid infusion volume on QTd and cardiac arrhythmias in patients with CRF receiving CAPD therapy, which is thought to be a safer modality of dialysis.

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