Factors influencing P terminal force in lead V1 of the ECG in hemodialysis patients

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Submitted: 2 September 2015
Accepted: 17 November 2015

Abstract

Introduction: Atrial fibrillation (AF) is a highly prevalent arrhythmia in hemodialysis (HD) patients, and an HD session may be a trigger for AF episodes. An abnormal P-terminal force in lead V1 (PTFV1) may predict new-onset AF in HD patients. The aim of the study was to assess the influence of the HD process on PTFV1 and to evaluate possible factors influencing PTFV1 in a group of selected HD patients.

Material and methods: One hundred and fifty-three selected HD patients entered the study. Blood chemistry, electrocardiography, and impedance cardiography were evaluated before and after HD. Echocardiography was performed on the morning after dialysis. Abnormal PTFV1 was defined as PTFV1 > 40 mm × ms.

Results: Abnormal PTFV1 was found in 35.3% of patients before dialysis and in 48.4% of patients after dialysis. The results of multiple regression analysis revealed that the independent predictors of pre-dialysis abnormal PTFV1 were: left atrial volume index (p = 0.002), left ventricular mass index (p = 0.014), and pre-dialysis thoracic fluid content (p = 0.021) values. The independent predictors of HD-induced abnormal PTFV1 values were larger differences between pre-dialysis and post-dialysis values of serum potassium (p < 0.001) and mean arterial pressure (p = 0.008).

Conclusions: Abnormal PTFV1 is prevalent in HD patients. The HD process adversely affects PTFV1 values. Pre-dialysis abnormal PTFV1 is mainly associated with structural heart abnormalities and hydration status. HD-induced abnormal PTFV1 is associated predominantly with serum potassium changes as well as HD-induced hypotension. Our results suggest possible risk factors for AF; however, their clinical significance needs to be confirmed in follow-up studies.

Key words: hemodialysis, atrial fibrillation, P-terminal force in lead V1, hydration status, potassium, hypotension.

Introduction

Chronic kidney disease is an independent risk factor for cardiovascular events [1–4]. Atrial fibrillation (AF) is a highly prevalent arrhythmia in hemodialysis (HD) patients. AF is estimated to occur in approximately...
18% of HD patients, which is 2 to 3 times higher than reported in the general population, even after adjustment for age, gender, and comorbidities [1, 5]. Like in all other patients, the presence of AF is associated with increased mortality in HD patients [5, 6].

High prevalence of traditional risk factors can explain, at least partly, the high burden of AF in HD patients. However, risk factors for AF in HD patients do not mirror those reported in the general population. Some risk factors are specific to renal failure or related to the HD session itself [2, 5, 7, 8]. Recent studies have documented a distinct relationship between AF and the HD procedure, and that an HD session is considered a trigger for AF episodes. The arrhythmogenic effect of the HD procedure is multifactorial in origin and is attributed, among other reasons, to changes of electrolyte concentrations, changes in acid base status, a rapid decrease in circulating blood volume, and secretion of catecholamines [5–7].

An electrocardiogram (ECG) carries important information about electrophysiological properties of the heart. P wave parameters measured on ECG are commonly used as a noninvasive tool to evaluate left atrial (LA) abnormalities and carry important information about atrial electrophysiology, as well as structure and function. Abnormal P-terminal force in lead V1 (PTFV1) is a strong indicator of an enlarged, poorly functioning LA and is associated with increased risk of AF, stroke, left ventricular hypertrophy (LVH), heart failure as well as risk of death due to all-cause, cardiovascular (CV) disease, and ischemic heart disease mortality [9–12]. Recent studies have demonstrated that PTFV1 is prevalent in HD patients, and the presence of a PTFV1 > 0.04 mm × ms predicts new-onset AF in these patients [13–15]. To the best of our knowledge, there are no data in the literature on the influence of the HD process on PTFV1.

We designed this study to: (i) assess the influence of the HD process on PTFV1 and (ii) evaluate the possible factors influencing PTFV1 in a group of selected HD patients.

**Material and methods**

**Patients**

The study included adult chronic HD patients treated at two dialysis centers in Lublin. The exclusion criteria were: HD treatment less than 3 months, AF or flutter, and severe valvular disease. All patients gave written consent, and the studies were approved by members of the local ethics committees.

**Hemodialysis**

The HD patients were dialyzed three times weekly. Bicarbonate dialysate containing (in millimoles per liter) 32 bicarbonate, 137–140 sodium, 2.5–4.0 potassium (K), 0.50 magnesium and 1.25 or 1.5 calcium was used in all HD patients. During HD, no medication was applied except heparin.

**Electrocardiography**

Surface 12-lead resting ECGs were recorded 10 min before and immediately after (not exceeding 15 min) a single HD session with a computer-based electrocardiograph (CardiA, Imed Ltd., Hungary). Electrodes were not removed during the HD session. PTFV1 was defined as the duration in seconds of the terminal (negative) part of the P wave multiplied by its depth in millimeters. Abnormal PTFV1 was defined as PTFV1 > 40 mm × ms [10]. The determinations of PTFV1 were made by the consensus of two observers, who were blinded to all of the patients’ clinical data.

**Biochemical measurements**

The following parameters were measured by automated analyzers before dialysis: intact parathyroid hormone, albumin, C-reactive protein, cardiac troponin T, N-terminal pro-hormone brain natriuretic peptide, total cholesterol, high-density lipoprotein cholesterol and triglycerides. Low-density lipoprotein cholesterol was calculated using the Friedewald equation. Blood was obtained after at least 8 h fasting. The following parameters were measured by automated analyzers both before and after dialysis: serum sodium, K, calcium, phosphate, magnesium, hemoglobin (Hb). Post-dialysis blood samples were taken immediately after the end of an HD session (after 10–15 s of 50–100 ml/min blood flow).

**Impedance cardiography**

The impedance cardiography measurements were performed in patients 20 min before and immediately after (not exceeding 15 min) a single HD session with a BioZ monitor (CardioDynamics, Int. Corp., San Diego, CA, USA). Sensors were placed as recommended by the manufacturer, and all measurements were performed according to the manufacturer’s guidelines [13] as described in detail elsewhere [16]. The following parameters were determined: stroke index (SI), systemic vascular resistance index (SVRI), cardiac index (CI) and thoracic fluid content (TFC).

**Echocardiography**

Two-dimensional echocardiographic examination was performed using a 2.5-MHz transducer by a cardiologist who was blinded to the clinical data of the study subjects. The left ventricular mass was indexed for body surface area to obtain
the left ventricular mass index (LVMI). Left ventricle hypertrophy was defined by an LVMI over 131 g/m² in male or over 110 g/m² in female subjects [17]. Left ventricular ejection fraction (LVEF) was measured by Simpson’s method. The LA volume was calculated with the biplane area method in apical 4-chamber views, and this was indexed for body surface area to obtain the LA volume index (LAVI) [18]. All echocardiographic measurements were performed on the morning after dialysis [17] according to the recommendations of the American Society of Cardiology.

**Statistical analysis**

Statistical analysis was carried out using Statistica version 10. Results were tested for normality by using the Kolmogorov-Smirnov test. When normally distributed, continuous variables were expressed as mean ± SD, and as median and range when non-normally distributed. The statistical significance of the differences between pre- and post-dialysis results were compared using Student’s t-test for paired data or using the Mann-Whitney U-test when appropriate. Categorical data were expressed as frequencies and percentages and were compared using the χ² test. Multiple regression analysis was performed to estimate the potential influence of various factors on the PTFV1. Probability values of p < 0.05 were accepted as significant.

**Results**

Of the total of 189 HD patients initially identified, 28 (14.8%) patients were excluded due to AE 6 due to HD treatment less than 3 months, and 2 due to valvular disease. The remaining 153 HD patients (81 females and 72 males), aged 44–87 years (mean: 67.97 ±9.18), who remained on HD from 3 to 101 months (mean: 38.51 ±22.34) entered the study. The causes of end-stage renal disease were diabetes (n = 66), glomerulonephritis (n = 32), hypertensive nephropathy (n = 11), obstructive nephropathy (n = 6), polycystic kidney disease (n = 5), chronic pyelonephritis (n = 3), and unknown/uncertain (n = 28). Out of 153 patients who qualified for the study, 122 (79.7%) were taking angiotensin-converting enzyme inhibitors/angiotensin receptor blockers, 131 (85.6%) β-blockers, 98 (64.1%) statins and 104 (70.0%) calcium blockers.

Before the HD, abnormal PTFV1 was found in 54 (35.3%) of all patients. Of the 54 patients with abnormal pre-dialysis PTFV1, post-dialysis abnormal PTFV1 remained in 53 patients, whereas in 1 patient normalization of PTFV1 was observed. The HD process induced a significant increase in the PTFV1 value from 28.32 ±25.89 mm × ms to 35.4 ±33.01 mm × ms (p = 0.004). However, no significant increase in PTFV1 values was observed in HD patients in whom abnormal pre-dialysis PTFV1 values were observed (pre- vs. post-dialysis PTFV1 was 51.2 ±7.72 and 53.9 ±7.05 respectively, p = 0.205). After dialysis, abnormal PTFV1 was observed in 74 (48.4%) patients. Of 74 patients with abnormal post-dialysis PTFV1, 21 had pre-dialysis PTFV1 values < 40 mm × ms, and the HD process induced an increase of PTFV1 values. No differences were observed in PTFV1 between females and males either before or after dialysis. Statistical analysis was performed separately for patients with abnormal pre-dialysis PTFV1 and for patients in whom the HD process induced abnormal PTFV1 values. Baseline characteristics of patients with abnormal pre-dialysis PTFV1 as well as HD-induced abnormal PTFV1 values are shown in Tables I and II, respectively.

Patients with abnormal pre-dialysis PTFV1 were older (p < 0.001), more often had a history of myocardial infarction (MI) (p < 0.001), and had higher prevalence of diabetes (p = 0.011). With regard to echocardiographic parameters, patients with abnormal pre-dialysis PTFV1 values had higher left ventricular mass (LVMI) (p = 0.001), LAVI (p < 0.001) and LAVI (p < 0.001) values, lower LVEF values (p = 0.010), and higher prevalence of LVH (p = 0.003). Additionally, patients with abnormal pre-dialysis PTFV1 values had lower Hb (p = 0.015) and higher TFC values (p = 0.004) in comparison to patients with normal pre-dialysis PTFV1.

Patients in whom the HD process induced abnormal PTFV1 were on dialysis for longer (p = 0.011), had a lower pre-dialysis SI value (p = 0.003), higher parathormone level (p = 0.008), larger differences between pre-dialysis and post-dialysis values (Δ) of systolic blood pressure (Δ systolic BP) (p = 0.008), higher Δ mean arterial pressure (ΔMAP) (p = 0.006), higher Δ potassium (ΔK) (p < 0.001), higher Δ magnesium (p = 0.003), and higher ΔTFC values (p = 0.003).

The results of multiple regression analysis showing independent variables influencing pre-dialysis as well as dialysis-induced abnormal PTFV1 are presented in Tables III and IV, respectively. The independent predictors of pre-dialysis abnormal PTFV1 were LAVI, LVMI, and pre-dialysis TFC value. The independent predictors of HD-induced abnormal PTFV1 values were ΔK and ΔMAP.

**Discussion**

Our study generated four major findings: (i) the prevalence of PTFV1 is high in HD patients, (ii) the HD process adversely affects PTFV1 values, (iii) pre-dialysis abnormal PTFV1 is mainly associated with structural heart abnormalities and hydration status, and (iv) HD-induced abnormal PTFV1 is
| Parameter                  | All patients (N = 153) | Pre-dialysis PTFV1(+) (N = 54) | Pre-dialysis PTFV1(−) (N = 99) | P-value |
|---------------------------|------------------------|-------------------------------|-------------------------------|---------|
| Age [years]               | 67.97 ±9.18            | 72.96 ±8.42                   | 65.53 ±8.12                   | < 0.001 |
| HD vintage [months]       | 38.51 ±20.34           | 39.16 ±21.46                  | 38.23 ±20.06                  | 0.527   |
| MI (%)                    | 26.1                   | 35.2                          | 21.2                          | < 0.001 |
| Diabetes mellitus (%)     | 42.5                   | 48.1                          | 39.4                          | 0.011   |
| Hypertension (%)          | 77.1                   | 77.7                          | 78.8                          | 0.412   |
| β-Blockers (%)            | 85.6                   | 85.2                          | 85.9                          | 0.764   |
| ACE/ARB (%)               | 79.7                   | 79.6                          | 79.8                          | 0.821   |
| Statins (%)               | 64.1                   | 68.5                          | 61.6                          | 0.072   |
| LVM [g]                   | 251.4 ±89.50           | 284.9 ±78.4                   | 232.8 ±85.7                   | 0.001   |
| LVMI [g/m²]               | 143.3 ±43.64           | 176.8 ±38.26                  | 125.0 ±45.13                  | < 0.001 |
| EF (%)                    | 64.7                   | 72.2                          | 60.6                          | 0.003   |
| LAVI [ml/m²]              | 36.29 ±9.82            | 31.96 ±7.45                   | 38.6 ±9.12                    | < 0.001 |
| Hemoglobin [g/dl]         | 11.45 ±1.11            | 10.91 ±0.95                   | 11.75 ±1.14                   | 0.015   |
| Total cholesterol [mg/dl] | 183.2 ±39.06           | 189.8 ±38.99                  | 182.03 ±37.76                 | 0.325   |
| LDL cholesterol [mg/dl]   | 115.6 ±30.12           | 117.6 ±29.16                  | 115.0 ±30.17                  | 0.612   |
| HDL cholesterol [mg/dl]   | 41.43 ±17.03           | 40.62 ±17.44                  | 41.79 ±15.64                  | 0.698   |
| Triglycerides [mg/dl]     | 169.1 ±63.23           | 159.9 ±70.43                  | 171.03 ±63.77                 | 0.542   |
| PTH, range [pg/ml]        | 382 (0.0–1124)         | 401 (0.0–1124)                | 379 (0.0–825)                 | 0.412   |
| Albumin [g/dl]            | 3.71 ±0.41             | 3.65 ±0.46                    | 3.74 ±0.40                    | 0.476   |
| CRP range [mg/dl]         | 7.68 (0.22–21.1)       | 8.13 (0.22–13.76)             | 7.09 (0.79–21.1)              | 0.658   |
| Troponin T, range [μg/l]  | 0.049 (0.00–0.725)     | 0.056 (0.00–0.435)            | 0.045 (0.00–0.725)            | 0.347   |
| NT-proBNP [fmol/ml]       | 189.3 ±86.2            | 196 ±82.24                    | 186.9 ±88.34                  | 0.101   |
| Sodium [mmol/l]           | 137.9 ±2.66            | 137.8 ±2.54                   | 137.9 ±2.59                   | 0.823   |
| Potassium [mmol/l]        | 5.79 ±0.78             | 5.71 ±0.75                    | 5.81 ±0.70                    | 0.673   |
| Magnesium [mmol/l]        | 1.02 ±0.13             | 1.00 ±0.19                    | 1.03 ±0.12                    | 0.348   |
| Calcium [mmol/l]          | 2.48 ±0.25             | 2.46 ±0.24                    | 2.48 ±0.23                    | 0.538   |
| Phosphate [mmol/l]        | 2.24 ±0.75             | 2.29 ±0.68                    | 2.23 ±0.74                    | 0.107   |
| MAP [mm Hg]               | 117.3 ±11.18           | 120.1 ±10.97                  | 116.3 ±11.06                  | 0.423   |
| Systolic BP [mm Hg]       | 139.2 ±8.24            | 137.4 ±8.12                   | 139.9 ±8.46                   | 0.572   |
| Diastolic BP [mm Hg]      | 75.31 ±4.44            | 77.75 ±4.47                   | 74.56 ±4.43                   | 0.243   |
| Si [ml/bpm/m²]            | 40.32 ±8.03            | 38.53 ±6.79                   | 40.90 ±8.45                   | 0.254   |
| SVRI [dyn/s/cm⁻5/m²]      | 2681 ±567.6            | 2954 ±547.2                   | 2597 ±604.4                   | 0.219   |
| CI [l/min/m²]             | 3.11 ±0.51             | 2.86 ±0.46                    | 3.23 ±0.51                    | 0.086   |
| TFC [kOhm⁻¹]              | 36.08 ±7.48            | 39.02 ±6.67                   | 34.51 ±7.49                   | 0.004   |

PTFV1(+) – patients with abnormal PTFV1 values, PTFV1(−) – patients with normal PTFV1 values, MI – myocardial infarction, ACE/ARB – angiotensin-converting enzyme inhibitors/angiotensin receptor blockers, LVM – left ventricular mass, LVMI – left ventricular mass index, LVH – left ventricular hypertrophy, EF – ejection fraction, LAVI – left atrial volume index, PTH – parathormone, CRP – C-reactive protein, NT-proBNP – N-terminal pro-hormone brain natriuretic peptide, MAP – mean arterial pressure, BP – blood pressure, SV – stroke volume, SI – stroke index, SVRI – systemic vascular resistance index, CI – cardiac index, TFC – thoracic fluid content.
Table II. Baseline characteristics of patients with HD-induced abnormal PTFV1

| Parameter                  | HD-induced PTFV1(+) \((N = 21)\) | The rest of HD patients \((N = 132)\) | \(P\)-value |
|---------------------------|------------------------------------|---------------------------------------|--------------|
| Age [years]               | 67.12 ± 7.16                       | 68.09 ± 8.86                          | 0.473        |
| HD vintage [months]       | 49.32 ± 14.65                      | 36.79 ± 18.96                         | 0.011        |
| MI (%)                    | 28.6                               | 27.3                                  | 0.566        |
| Diabetes mellitus (%)     | 38.1                               | 46.2                                  | 0.213        |
| Hypertension (%)          | 76.2                               | 77.3                                  | 0.721        |
| \(\beta\)-Blockers (%)    | 85.7                               | 85.6                                  | 0.895        |
| ACE/ARB (%)               | 76.2                               | 80.3                                  | 0.419        |
| Statins (%)               | 66.7                               | 63.6                                  | 0.588        |
| LVM [g]                   | 261.3 ± 73.7                       | 249.8 ± 85.9                          | 0.378        |
| LVMI [g/m²]               | 151.1 ± 39.5                       | 142.1 ± 44.4                          | 0.413        |
| LVH (%)                   | 71.4                               | 63.6                                  | 0.308        |
| EF (%)                    | 59.14 ± 5.06                       | 58.2 ± 6.13                           | 0.642        |
| LAVI [ml/m²]              | 40.62 ± 7.89                       | 35.65 ± 9.11                          | 0.118        |
| Hemoglobin [g/dl]         | 11.89 ± 0.87                       | 11.38 ± 1.08                          | 0.422        |
| Total cholesterol [mg/dl] | 176.8 ± 30.1                       | 184.9 ± 38.3                          | 0.333        |
| LDL cholesterol [mg/dl]   | 118.7 ± 27.0                       | 114.7 ± 29.5                          | 0.688        |
| HDL cholesterol [mg/dl]   | 33.15 ± 13.9                       | 42.7 ± 17.4                           | 0.121        |
| Triglycerides [mg/dl]     | 164.5 ± 57.9                       | 169.9 ± 62.77                         | 0.615        |
| PTH, range [pg/ml]        | 596 (356–1124)                     | 332 (0.0–702)                         | 0.008        |
| Albumin [g/dl]            | 3.42 ± 0.356                       | 3.76 ± 0.412                          | 0.341        |
| CRP, range [mg/dl]        | 9.76 (0.96–18.5)                   | 6.99 (0.22–21.1)                      | 0.236        |
| Troponin T, range [μg/l]  | 0.058 (0.00–0.689)                 | 0.043 (0.00–0.725)                    | 0.346        |
| NT-proBNP [fmol/ml]       | 207.0 ± 77.4                       | 183.2 ± 72.9                          | 0.189        |
| Sodium predialysis [mmol/l]| 137.4 ± 2.22                      | 138.0 ± 2.62                          | 0.809        |
| Δ sodium [mmol/l]         | 0.215 ± 0.007                      | 0.213 ± 0.009                         | 0.711        |
| Potassium predialysis [mmol/l]| 6.17 ± 0.69                | 5.69 ± 0.73                           | 0.201        |
| Δ potassium [mmol/l]      | 1.99 ± 0.016                       | 1.46 ± 0.023                          | < 0.001      |
| Magnesium predialysis [mmol/l]| 1.03 ± 0.17                    | 1.02 ± 0.18                           | 0.456        |
| Δ magnesium [mmol/l]      | 0.121 ± 0.023                      | 0.081 ± 0.015                         | 0.004        |
| Calcium predialysis [mmol/l]| 2.40 ± 0.24                     | 2.41 ± 0.26                           | 0.729        |
| Δ calcium [mmol/l]        | −0.11 ± 0.08                       | −0.14 ± 0.09                          | 0.462        |
| Phosphate predialysis [mmol/l]| 2.08 ± 0.69                    | 2.27 ± 0.73                           | 0.324        |
| Δ phosphate [mmol/l]      | 1.07 ± 0.11                        | 1.12 ± 0.14                           | 0.653        |
| MAP predialysis [mm Hg]   | 119.1 ± 9.76                       | 116.8 ± 10.97                         | 0.397        |
| Δ MAP [mm Hg]             | 6.82 ± 2.25                       | 4.01 ± 2.39                           | 0.004        |
| Systolic BP predialysis [mm Hg]| 143.5 ± 8.32              | 138.9 ± 8.97                          | 0.229        |
| Δ systolic BP [mm Hg]     | 10.82 ± 5.28                      | 6.68 ± 6.14                           | 0.008        |
Table II. Cont.

| Parameter | HD-induced PTFV1(+) \((N = 21)\) | The rest of HD patients \((N = 132)\) | \(P\)-value |
|-----------|---------------------------------|---------------------------------|-------------|
| Diastolic BP predialysis [mm Hg] | 73.07 ±3.65 | 75.95 ±4.07 | 0.413 |
| Δ diastolic BP [mm Hg] | –1.65 ±0.31 | –1.54 ± 0.34 | 0.521 |
| SI predialysis [ml/bpm/m²] | 34.12 ±7.43 | 41.30 ±7.89 | 0.003 |
| Δ SI [ml/bpm/m²] | 6.18 ±2.37 | 7.05 ±2.76 | 0.165 |
| SVRI predialysis [dyn/s/cm⁻⁵/m²] | 2834.1 ±501.7 | 2658.2 ±555.9 | 0.279 |
| Δ SVRI [dyn/s/cm⁻⁵/m²] | –592.2 ±213.4 | –566.3 ±255.6 | 0.411 |
| CI predialysis [l/min/m²] | 3.01 ±0.37 | 3.14 ±0.42 | 0.358 |
| Δ CI [l/min/m²] | 0.311 ±0.045 | 0.299 ±0.049 | 0.419 |
| Body weight loss [kg] | 3.199 ±0.895 | 2.882 ±1.273 | 0.108 |
| TFC predialysis [kOhm⁻¹] | 38.33 ±6.99 | 35.97 ±7.04 | 0.122 |
| Δ TFC [kOhm⁻¹] | 8.87 ±3.25 | 5.03 ±3.66 | 0.003 |

PTFV1(+): patients with abnormal PTFV1 values, PTFV1(−): patients with normal PTFV1 values, MI – myocardial infarction, ACE/ARB – angiotensin-converting enzyme inhibitors/angiotensin receptor blockers, LVM – left ventricular mass, LVMI – left ventricular mass index, LVH – left ventricular hypertrophy, EF – ejection fraction, LAVI – left atrial volume index, PTH – parathormone, CRP – C-reactive protein, NT-proBNP – N-terminal pro-hormone brain natriuretic peptide, MAP – mean arterial pressure, BP – blood pressure, SV – stroke volume, SI – stroke index, SVRI – systemic vascular resistance index, CI – cardiac index, TFC – thoracic fluid content.

Table III. Factors influencing pre-dialysis abnormal PTFV1 estimated by multivariate stepwise regression analysis

| Dependent variable | Independent variables | B | St. error | \(\beta\) | \(P\)-value |
|--------------------|-----------------------|---|-----------|---------|------------|
| PTFV1              | LAVI                  | 0.497 | 0.029 | 0.343 | 0.002 |
|                    | LVMI                  | 11.23 | 6.61 | 0.295 | 0.014 |
|                    | Pre-dialysis TFC      | 7.664 | 3.989 | 0.297 | 0.021 |

Model \((R = 0.634, R^2 = 0.351)\)

Table IV. Factors influencing HD-induced abnormal PTFV1 estimated by multivariate stepwise regression analysis

| Dependent variable | Independent variables | B | St. error | \(\beta\) | \(P\)-value |
|--------------------|-----------------------|---|-----------|---------|------------|
| PTFV1              | ΔK                    | 3.45 | 1.13 | 0.412 | < 0.001 |
|                    | ΔMAP                  | 5.76 | 2.38 | 0.396 | 0.008 |

Model \((R = 0.560, R^2 = 0.314)\)

predominantly associated with K as well as a drop in blood pressure.

The results of our study are in agreement with the results of previous studies, which indicated that the prevalence of abnormal PTFV1 is high in HD patients [11, 12]. In our study the prevalence of pre-dialysis abnormal PTFV1 was 35.3% and was very similar to the prevalence (34%) found by Nishi et al. [12]. Interestingly, Bilen et al. [11] found higher prevalence (66%) of abnormal PTFV1 in a small group of HD patients without clinically significant CVD.

According to our knowledge, ours is the first study to show that the HD process adversely affects PTFV1 values. It indirectly indicates that an HD session may be a trigger for AF episodes. It is in agreement with the results of previous studies [6, 7, 19], which indicated that the HD session increases the risk for AF. Moreover, recent studies have demonstrated that abnormal PTFV1 is associated with decreased LA functions [11], and may predict new-onset AF in HD patients [12].

The results of our study revealed that pre-dialysis abnormal PTFV1 values were associated with structural heart abnormalities and hydration status. The re-entrant nature of AF requires areas of conduction delay to initiate and sustain arrhythmia, and pathological structural remodeling con-
Recent studies have demonstrated that IDH might actively contribute to the development of AF, and the sequelae of cardiac remodeling. Intradialytic hypotension can cause systemic hypoperfusion, including myocardium [26, 27]. This ischemic effect, though transient, may induce reversible myocardial dysfunction, which may potentially increase PTFV1; however, the ultimate cause remains unknown. Our study suggests that a drop in blood pressure should be especially avoided in patients prone to AF.

The present study has some important limitations. The main limitation is that there was no follow-up of our patients. This causes that both the prevalence of AF during follow-up and its relation with our results remain unknown. Therefore, our results can only indirectly suggest the potential factors associated with increased risk for AF in HD patients. The second limitation is the relatively small number of patients and the impossibility of controlling all possible factors that might influence PTFV1. Further studies are required to confirm our results as well as to determine their possible clinical importance in HD patients.

In conclusion, abnormal PTFV1 is prevalent in HD patients. Pre-dialysis abnormal PTFV1 is mainly associated with structural heart abnormalities and hydration status. HD-induced abnormal PTFV1 values are associated with serum K and blood pressure drop. Our results suggest only possible risk factors for AF in HD patients. However, their clinical significance needs to be confirmed in follow-up studies.

**Conflict of interest**

The authors declare no conflict of interest.

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