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Research Article

# Associations of Hemodialysis Procedures with Stimulation of Supraventricular Arrhythmias

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## Abstract

Evidence suggests higher mortality after two-day gaps in thrice-weekly hemodialysis, with mortality increases of 20-40%. Supraventricular arrhythmias (SVAs), notably atrial fibrillation, are common during dialysis, increasing mortality risk. Hemodialysis induces fluid, electrolyte, and hemodynamic shifts, precipitating arrhythmias. Investigating these associations is crucial for devising preventive strategies and improving outcomes in haemodialysis patients.

**Materials and Methods:** A Holter 24-hour electrocardiogram (ECG) was recorded in 72 hemodialysis patients starting at the beginning of the first session of the week. The associations between an increase in supraventricular premature contractions (SVPCs) during hemodialysis and peridialytic changes in blood electrolytes, mean arterial pressure (MAP), ultrafiltration volume (UFR/DW), ultrasonic cardiographic (UCG) parameters, and other factors were tested using multiple regression analysis. Patients' backgrounds were also compared among tertiles of 24-hour total SVPC counts.

**Results:** SVPCs increased during hemodialysis and six patients developed atrial fibrillation, and 25% maintained low SVPC rates throughout the recording. Patients in the highest tertile for 24-hour SVPC counts had the lowest body mass index (BMI) and the fewest cardiovascular diseases (CVD). They also had the smallest left atrial (LAD) and left ventricular end-systolic dimensions (LVDs). Multiple regression analysis showed that larger changes in mean arterial pressure (MAP) during dialysis were significantly associated with a higher percentage increase in SVPC in all four models. Smaller LAD and larger ultrafiltration volumes (UFR/DW) were also associated with a percentage increase in SVPC.

**Conclusions:** Larger changes in intradialytic mean arterial blood pressure caused by excessive ultrafiltration may increase the risk of developing SVPCs during hemodialysis, particularly in undernourished patients.

**Keywords:** hemodialysis; supraventricular premature contraction; atrial fibrillation; supraventricular arrythmias; ultrafiltrate; mean arterial pressure; Holter electrocardiogram; sudden death

## List of abbreviations

**Af:** atrial fibrillation
**AV:** arteriovenous
**BMI:** body mass index
**BNP:** b-type natriuretic peptide
**CKD:** chronic kidney disease
**ECG:** electrocardiogram
**IVC:** inferior vena cava
**IVST:** interventicular septum thickness
**IQR:** interquartile range
**LAD:** left atrial dimension
**LVDd:** left ventricular end-diastolic dimension
**LVDs:** left ventricular end-systolic dimension
**LVEF:** left ventricular ejection fraction
**MAP:** mean arterial pressure
**PVCs:** premature ventricular contractions
**SD:** standard deviation
**SE:** standard error
**SVA:** supraventricular arrythmia
**SVPC:** supraventricular premature contraction
**UCG:** ultrasonic cardiography

**UFR:** ultrafiltration rate

**VIF:** variance inflation factor

## Introduction

Evidence has accumulated indicating that excess mortality and hospitalization are often observed in patients after the two-day gap in thrice-weekly hemodialysis compared with one-day intervals [1-3]. This dialysis-related mortality is mainly attributed to arrhythmia, including cardiac arrest or sudden cardiac death [4]. Increases in mortality ranging between 20% and 40% have been reported compared with the rest of the week, although findings vary from the first day of the week in the US to the last day of the week in Europe and Japan [2].

Supraventricular arrhythmias (SVAs), especially atrial fibrillation (Af), are increasingly common among patients undergoing hemodialysis [5-8]. The incidence of SVAs has been found to be high during hemodialysis sessions, and peridialytic SVAs are not necessarily symptomatic nor persistent but do increase the risk of mortality [9].

The hemodialysis procedure, while life-sustaining, poses a paradoxical challenge to cardiovascular health. It is associated with abrupt shifts in fluid and electrolyte balance, rapid alterations in hemodynamics, and fluctuations in sympathetic tone, all of which can precipitate arrhythmias [8, 10]. SVAs, particularly atrial fibrillation and atrial flutter, are frequently encountered during or shortly after hemodialysis sessions, further exacerbating the already elevated risk of mortality in this population [9, 11].

The etiology of increased SVAs during hemodialysis may be multifactorial. Electrolyte imbalances, including potassium fluctuations and calcium-phosphate derangements, may play a pivotal role in triggering arrhythmic events. Additionally, volume shifts, hemodynamic instability, and endothelial dysfunction induced by the hemodialysis process contribute to the proarrhythmic milieu [8,10]. Our previous study demonstrated that the stimulation of premature ventricular contractions (PVCs) was mainly associated with blood electrolyte changes such as potassium and calcium ion during hemodialysis [12].

Understanding the precise mechanisms underlying the association between hemodialysis and SVAs is imperative for devising effective preventive strategies and optimizing patient outcomes. Yet, despite the clinical significance of this relationship, there remains a paucity of comprehensive studies elucidating the intricate interplay between hemodialysis procedures and supraventricular arrhythmias.

This study aims to address this critical gap in knowledge by investigating the associations of hemodialysis procedures with the stimulation of supraventricular arrhythmias. By delineating the risk factors, temporal patterns, and clinical implications of these arrhythmias in the context of hemodialysis, we endeavor to provide actionable insights for risk stratification, tailored monitoring strategies, and targeted interventions to mitigate the burden of cardiovascular morbidity and mortality in this vulnerable population.

## Materials and Methods

### Study Population:

Five hundred sixty-seven patients underwent maintenance hemodialysis between April 2017 and December 2018 at the dialysis center of the Itabashi Chuo Medical Center in Tokyo, Japan. After excluding those with continuous atrial fibrillation, permanent pacemakers, or those taking antiarrhythmic drugs other than beta-blockers, seventy-two agreed to participate and underwent Holter electrocardiography. Complete data sets were obtained from 52 patients for further analysis. The patients' ages ranged from 46 to 87 years, with a mean of 69.7 ± 9.4 years. According to the patient report from referral clinics, the etiologies of end-stage renal failure included diabetic kidney disease (N=32), hypertensive nephrosclerosis (N=14), glomerulonephritis (N=4), and unknown causes (N=2). Vascular access types included arteriovenous (AV) fistula (N=36), AV graft (N=6), catheter (N=5), and subcutaneously fixed superficial artery (N=5). 28 patients had cardiovascular disease, and 10 were prescribed beta-blockers. Antihypertensive medications included calcium antagonists (N=26), angiotensin receptor antagonists (N=23), and alpha-blockers (N=6). 39 patients received erythropoiesis-stimulating agents. Informed consent was obtained from all participants, and the study was approved by the ethics committee of Itabashi Chuo Medical Hospital, adhering to the Declaration of Helsinki.

### Hemodialysis Prescription:

Hemodialysis was conducted three times a week for 3 hours (N=2), 3.5 hours (N=1), or 4 hours (N=49) per session, with blood flow rates ranging from 150 to 250 ml/min and dialysate flow rates maintained at 500 ml/min. The mean ultrafiltration rate (UFR) was 2.48 L/session. The dialysate composition was $Na^+$ 140 mEq/L, $K^+$ 2.0 mEq/L, $Ca^{2+}$ 3.0 mEq/L, $Mg^{2+}$ 1.0 mEq/L, $Cl^-$ 110 mEq/L, $CH_3COO^-$ 8 mmol/L, and $HCO_3^-$ 30 mEq/L.

### Protocol:

Holter 24-hour ECG was set up before the first session of the week. SVPCs were automatically counted, with cardiologists reviewing the counts' precision. Blood samples were withdrawn hourly from the arterial side of the vascular access for immediate analysis of $Na^+$, $K^+$, $Cl^-$, $Ca^{2+}$, and $HCO_3^-$ concentrations using a blood gas analyzer and other blood biochemical parameters, including magnesium level, using an automatic analyzer.

## Statistical Analyses:

Baseline demographics, dialysis parameters, and laboratory characteristics are presented as the mean ± standard deviation (SD) or standard error (SE) or the median and interquartile range (IQR) for continuous variables, and percentage (n/N) is used for categorical variables. Time-averaged laboratory or prescription values were analyzed using the mean or median value of all sessions. The Friedman test for nonparametric data was employed to examine the significance of the time-dependent change in SVPC frequency. ANOVA and the Kruskal–Wallis test were used to analyze baseline characteristics and predialysis laboratory data according to tertiles of the observed number of SVPC counts during hemodialysis for continuous variables, and the chi-square test was used for categorical variables. Multiple regression analysis was used to analyze associations of electrolytes and intradialytic changes in fluid or electrolytes with hemodialysis-related PVC. The number of dependent variables in a single model was limited to four or five, and automatic weighted regression was applied to correct for heteroscedasticity. Combinations of independent variables with a high variance inflation factor (VIF > 10) were excluded from the model to avoid multicollinearity among the independent variables. All analyses were computed using MedCalc. ver. 19.5.1 (MedCalc Software Ltd, Belgium), with $p < 0.05$ considered indicative of significance.

## Results

### 1.Frequency of SVPCs during and after hemodialysis sessions:

The frequency of SVPCs steadily increased after the initiation of hemodialysis, peaking in the third and fourth hours **(Figure 1a).** The frequency subsequently decreased after dialysis cessation. When the percent increase in SVPCs was examined every 4 hours, both the first 4 hours (during dialysis) and the second 4 hours showed a significant increase in SVPCs compared to the final 4 hours, as indicated by repeated ANOVA **(P<0.05, Figure 1b).** Six patients exhibited atrial fibrillation, and eighteen patients (25%) had SVPCs below 10 counts/hour throughout the recording period.

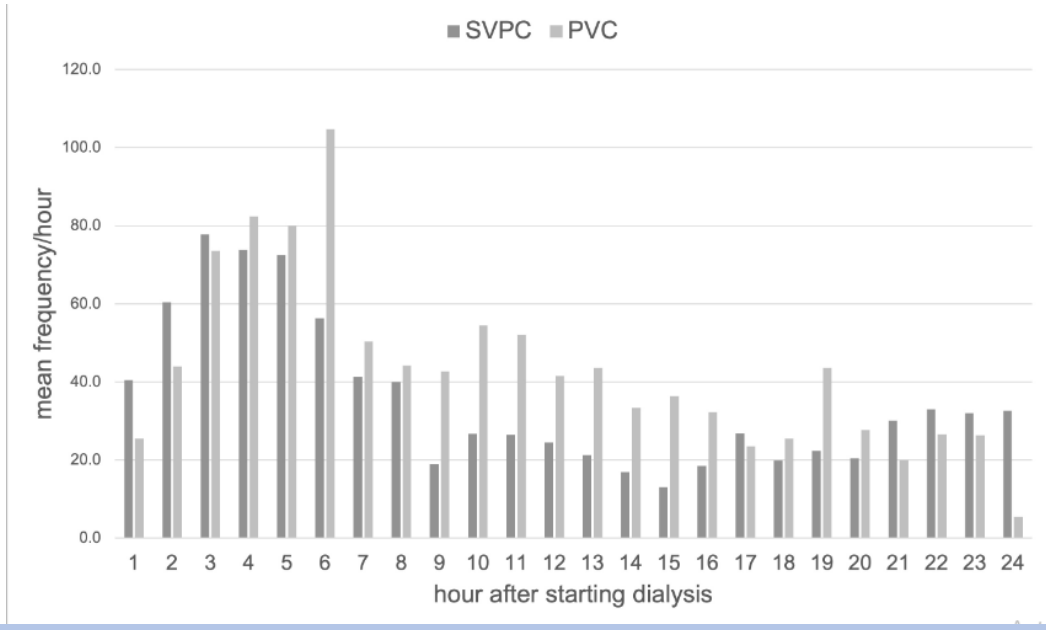


**Figure 1a:** Changes in SVPC and PVC frequency after the beginning of dialysis. Each bar represents mean. SVPC, supraventricular premature contraction; PVC, premature ventricular contraction

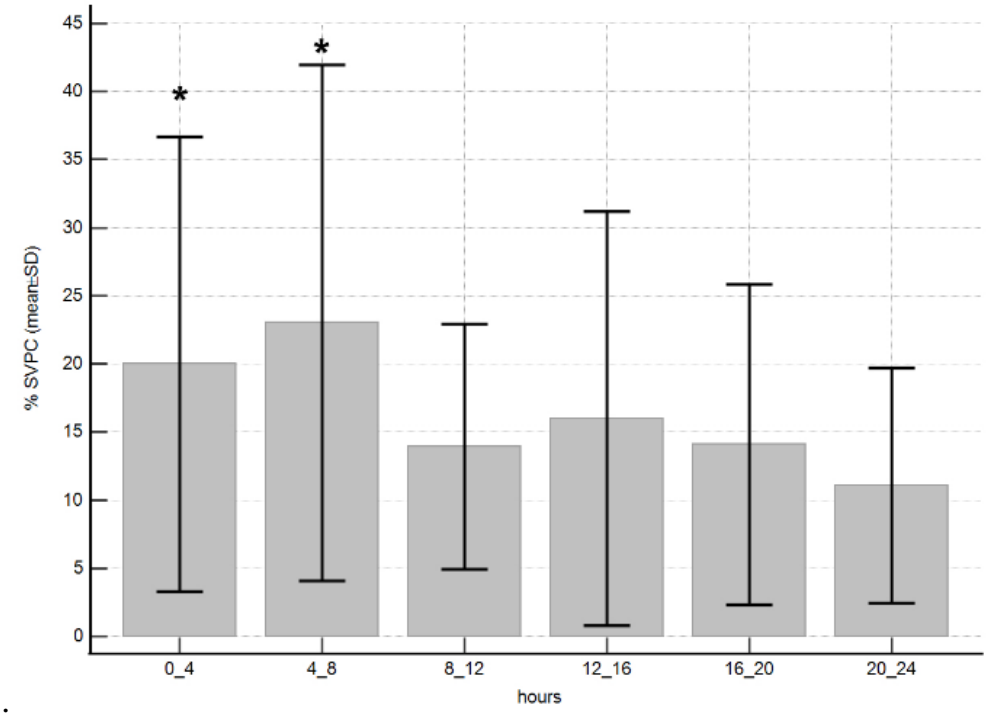


**Figure 1b:** Changes in %SVPC every 4 hour after the beginning of dialysis. Each bar represents a standard error of mean. Repeated ANOVA Measure was employed to compare mean difference among factors and significant difference was found in both 0_4th hour and 4_8th hour compared with 20_24$^{th}$ hour (* P<0.05). SVPC, supraventricular premature contraction.

### 2.Patient characteristics among tertiles of total 24-hour SVPC counts:

Multiple parameters were compared among the tertiles of total 24-hour SVPC counts **(Table 1).** The highest SVPC count tertile was associated with the lowest body mass index (BMI) and the least cardiovascular disease (CVD) history. The variation in dialytic changes in serum $K^+$ was statistically significant among the three groups, but this significance was nullified when patients were divided into two groups (data not shown). There was no significant difference in other parameters among the three groups. In terms of echocardiographic parameters (Table 2), the highest tertile of SVPC counts exhibited the smallest left atrial diameter (LAD) and left ventricular end-systolic dimensions (LVDs).

| . | Lowest SVPC (18±12), n=17 | Middle SVPC (115±42), n=18 | Highest SVPC (2425±5935), n=17 | *P value* |
|---|---|---|---|---|
| Age (years) | 68±9 | 68±9 | 74±10 | 0.97 |
| Sex (F/M) | 7/10a | 2/16 | 7/10 | 0.08 |
| Dialysis vintage (days) | 2333±1857 | 2174±2042 | 1966±2378 | 0.89 |
| Diabetes (%) | 71 | 67 | 59 | 0.76 |
| BMI (kg/m$^2$) | 31.0±37.1 | 22.2±4.6 | 20.8±2.7 | <0.05 |
| CVD (%) | 76 | 44 | 35 | <0.05 |
| β-blocker (%) | 35 | 22 | 18 | 0.47 |
| CRP | 1.21±2.07 | 0.67±1.07 | 1.77±4.38 | 0.13 |
| Albumin (g/dL) | 3.65±0.48 | 3.57±0.31 | 3.51±0.49 | 0.68 |
| BUN (mg/dL) | 49.2±15.6 | 53.1±11.6 | 47.9±21.7 | 0.11 |
| Predialysis $sHCO_3^-$ (mEq/L) | 21.5±2.2 | 21.9±3.1 | 21.5±3.2 | 0.23 |
| Intradialytic change in $sHCO_3^-$ （mEq/L） | 3.8±2.3 | 4.1±2.2 | 3.8±2.0 | 0.97 |
| Predialysis $sK^+$ (mEq/L) | 4.23±0.76 | 4.40±0.91 | 4.13±0.76 | 0.86 |
| Intradialytic change in $sK^+$ （mEq/L） | -1.10±0.47 | -0.96±0.80 | -1.06±0.44 | <0.05 |
| Predialysis $cCa^{2+}$ (mEq/L) | 9.22±0.88 | 8.63±0.64 | 8.78±0.53 | 0.268 |
| Intradialytic change in $cCa^{2+}$ （mEq/L） | 0.060±0.079 | 0.074±0.043 | 0.098±0.052 | 0.28 |
| Predialysis $sMg^{2+}$ (mg/dL) | 2.53±0.31 | 2.66±0.36 | 2.39±0.44 | 0.54 |
| Intradialytic change in $sMg^{2+}$ （mg/dL） | -0.38±0.20 | -0.41±0.26 | -0.26±0.29 | 0.65 |
| Hemoglobin (g/dL) | 10.6±2.3 | 10.4±1.3 | 10.1±1.9 | 0.19 |
| Ferritin | 85.5±40.4 | 108±94 | 180±198 | 0.07 |
| Predialysis MAP (mmHg) | 118±13 | 124±17 | 117±20 | 0.69 |
| Intradialytic change in MAP (mmHg) | -15.6±21.8 | -9.6±20.2 | -16.6±±35.3 | 0.59 |
| UFR (mL/hour) | 0.67±0.23 | 0.64±0.24 | 0.62±0.27 | 0.60 |
| UFR/DW (mL/kg/hour) | 11.3±4.5 | 11.2±4.9 | 11.8±4.8 | 0.99 |
| Postdialysis BNP (pg/mL) | 659±670 | 651±1156 | 366±519 | 0.28 |

**Table 1**. Comparison of patient characteristics among tertiles of total 24-h SVPC count

Each value is presented as the mean ± standard deviation (SD) or as percent (n/N) for categorical variables. The statistical method to compare the difference of continuous variables and categorical data is ANOVA test and chi-square test, respectively.

| Ucg parameters | Lowest SVPC counts (n=12) | Middle SVPC count (n=17) | Highest SVPC counts (n=12) | Levene's test |
|---|---|---|---|---|
| LVEF (%) | 58.2±15.1 | 64.0±11.5 | 66.7±10.3 | P=0.11 |
| IVST | 11.0±1.7 | 10.5±1.4 | 10.6±1.3 | P=0.58 |
| LAD (mm) | 46.1±8.8 | 43.4±6.1 | 41.4±3.1 | P=0.002 |
| LVDs | 35.8±10.7 | 31.8±7.5 | 27.8±3.6 | P=0.032 |
| LVDd | 51.4±7.8 | 49.0±5.8 | 44.7±3.3 | P=0.057 |
| FS (fractional shortening) | 30.1±10.3 | 35.1±8.2 | 35.0±9.3 | P=0.26 |
| IVC (mm) | 17.6±4.9 | 15.7±3.8 | 16.3±2.9 | P=0.82 |

**Table 2.** Comparison of ucg parameters among tertiles of total 24-h SVPC count

Each value is presented as the mean ± standard deviation (SD). The ANOVA test was employed to compare the difference of continuous variables. P value less than 0.05 is considered significant.

### 3.Effect of Ultrafiltration and Blood Pressure Changes on the Stimulation of SVPCs during Hemodialysis:

Multiple regression analysis was conducted to examine the association of dialysis-related parameters with an increase in SVPC frequency during the hemodialysis session. Due to the limitation of sample size, four ± one independent variables were chosen for each model. Initially, no significant associations were found in models that primarily included predialysis levels and/or peridialytic changes in blood electrolyte levels ($Na^+$, $K^+$, $Ca^{2+}$, and $Mg^{2+}$). Subsequently, models incorporating various markers of volume or sympathetic stimulation, ultrafiltration rate (UFR), intradialytic changes in mean arterial pressure (MAP), serum b-type natriuretic peptide (BNP), and left atrial diameter (LAD) were tested (Table 3). Larger dialytic changes in mean arterial pressure (MAP) were consistently associated with a higher percentage increase in SVPC across all models. This association appeared strongest as both backward and stepwise methods showed significance in every model. In contrast, smaller left atrial diameters (LAD) were associated with a higher percentage increase in SVPC only using the backward method. Larger ultrafiltration volumes (UFR/DW) were associated with a percentage increase in SVPC only in Model 4, which included dialytic changes in $Mg^{2+}$ for adjustment. Higher postdialysis BNP levels were associated with a percentage increase in SVPC in every model; however, the coefficient values for BNP were very small, indicating a minimal impact on these changes.

| | Correlation coefficient | | | |
|---|---|---|---|---|
| | Model 1 β±SE | Model 2 β±SE | Model 3 β±SE | Model 4 β±SE |
| Intradialytic changes in MAP (mmHg) | 0.19±0.09** (R=0.32, P<0.05) | 0.19±0.09** (r=0.32, P<0.05) | 0.19±0.09** (r=0.32, P<0.05) | 0.19±0.09** (r=0.32, P<0.05) 0.18±0.09* (r=0.33, P<0.05) |
| UFR/DW (mL/Kg/h) | 0.58±0.36 (ns) | 0.45±0.40 (ns) | 0.61±0.37 (ns) | 0.81±0.35* (r=0.37, P<0.05) |
| Postdialysis BNP (pg/mL) | 0.0047±0.0023* (r=0.32, P<0.05) | 0.0047±0.0023* (r=0.33, P<0.05) | 0.0047±0.0023* (r=0.33, P<0.05) | 0.0044±0.0021* (r=0.34, P<0.05) |
| LAD | -0.75±0.30* (r=-0.38, P=0.018) | -0.75±0.30* (r=-0.38, P=0.018) | -0.75±0.30* (r=-0.38, P=0.018) | -0.60±0.28* (r=-0.35, P<0.05) |
| Intradialytic changes in $K^+$ (mEq/L) | NA | -2.8±3.3 | NA | NA |
| Intradialytic changes in $Ca^{2+}$ (mEq/L) | NA | NA | -15.7±33.0 (ns) | NA |
| Intradialytic changes in $Mg^{2+}$ (mEq/L) | NA | NA | NA | -16.3±6.9 (r=-0.38, P=0.024) |

* Significant by the backward method of multiple regression analysis. ** Significant by the stepwise method of multiple regression analysis. β, correlation coefficient; S.E., standard error, r, coefficient of the partial determination; P, p value for each correlation coefficient. NA, not available.

**Table 3.** Associations of dialysis parameters with % increase in SVPCs during hemodialysis

## Discussion

We investigated 72 hemodialysis patients using a Holter ECG recorder to quantify SVPCs and their correlation with the dialysis procedure. In 75% of the recordings, SVPCs continuously increased during dialysis, peaking around the termination of the session and persisting for another 2 hours before gradually decreasing over the subsequent 24-hour period. This pattern closely mirrors that observed for premature ventricular contractions (PVCs) in our previous study involving the same cohort [12], indicating that the hemodialysis procedure itself exacerbates the risk of SVAs as well as PVCs. Extended monitoring using a loop ECG recorder in a previous study also revealed a secondary peak occurring 36-48 hours after the initial hemodialysis session, just prior to the second session of the week, suggesting that alterations in the volume status of patients may trigger SVPCs [8, 11].

These characteristics of patients with high SVPC frequency during hemodialysis contrast with those observed for PVCs [12], suggesting that the generation of SVPCs may not necessarily be caused by underlying cardiovascular diseases.

In the same cohort, an increase in the number of PVCs, particularly immediately after dialysis, was associated with an intradialytic positive change in $K^+$ levels when patients were dialyzed against 2 mEq/L of $K^+$ dialysate. Additionally, predialysis serum $K^+$ levels were negatively associated with an increase in PVC frequency. Since serum $K^+$ levels usually decreased when the predialysis level exceeded the threshold (2.46 mEq/L) against 2.0 mEq/L of dialysate $K^+$, these results indicate that a very low predialysis $K^+$ level was a high-risk factor for generating PVCs (12). However, the present study did not find any difference or association of $K^+$ or $Ca^{2+}$ with the stimulation of SVPCs during hemodialysis. Only a weak association in serum electrolyte changes was found between negative changes in serum $Mg^{2+}$ during hemodialysis and SVPCs.

Instead, the present study showed that an increase in SVPCs during hemodialysis was positively associated with an ultrafiltration rate in one multiple regression model, with a consistent association with dialytic changes in MAP in every model. Furthermore, there was an association between SVPC stimulation and a smaller LAD. Thus, unlike the stimulation of PVCs, the stimulation of SVPCs by the hemodialysis procedure seems to be highly dependent on changes in MAP due to excess ultrafiltration rate, but not on changes in electrolytes. Additionally, SVPCs occurred more often in patients with less CVD history.

Several observational studies have investigated possible causes of arrhythmogenicity during hemodialysis. One observational study examined the role of SVAs in mortality among dialysis patients by recording two full weeks of Holter recording and following up for up to 10 years [9]. They found that right atrial enlargement and age were associated with SVAs during hemodialysis, and even asymptomatic SVAs during hemodialysis increased the risk of mortality [9]. Recently, another investigation reported associations between intradialytic electrolyte changes and any kind of arrhythmias during dialysis among 66 patients using loop recorder implantation [11]. A cross-sectional study on 152 patients using a 48-hour Holter monitor reported that older age, elevated preload, and lower cardiac output were independently associated with clinically significant arrhythmias of any kind [8]. However, neither of the latter two studies investigated individual arrhythmias separately. Since our results demonstrated that SVPCs were mainly associated with changes in volume and/or sympathetic activity, while PVCs were associated with changes in serum electrolytes due to dialysis, the etiology of SVPCs during hemodialysis must be different from that of PVCs.

This study showed that SVPCs were stimulated during dialysis mainly by larger intradialytic changes in blood pressure in patients with smaller left atrial loads, faster ultrafiltration rates, and lower BMIs. This suggests that a too-low setting of dry weight may exaggerate SVPCs not because of underlying cardiac disease. This is in contrast to the causes of PVCs observed in our previous study [12]. PVCs tended to increase due to basal, intradialytic, and interdialytic electrolyte changes in patients with lower left ventricular ejection fraction (LVEF) and larger preload [12].

Our study has limitations due to its relatively small sample size and single-center design that may cause methodological biases. Since this study was not a controlled study that compared different dialysis prescriptions, it cannot provide evidence proving a cause-effect relationship. Nevertheless, the advantage of this study is that it is the first to successfully demonstrate an association between the generation of SVPCs and dialysis prescriptions/patient characteristics.

## Conclusions

In conclusion, SVPCs increased during hemodialysis in 75% of patients during the 24-hour recording on the first day of the week of hemodialysis. There was a significant association of SVPCs with the magnitude of intradialytic MAP changes. Additionally, there was a weak association with faster ultrafiltration rates and larger dialytic serum $Mg^{2+}$ changes. SVPCs tended to increase in patients with smaller BMIs, smaller LAD,

and fewer cardiovascular comorbidities. From these results, excess ultrafiltration in less nourished patients may render them vulnerable to SVPCs.

## Declarations

### Ethics approval and consent to participate

All procedures performed in this study were in accordance with the ethical standards of the institution or practice at which the studies were conducted (IRB approval number: 20161227). Informed consent was obtained from all individual participants included in the study.

## Consent for publication

Not applicable.

## Availability of data and materials

It is not possible to share the research data publicly, because the original dataset consisted of age, sex and dialysis vintage along with the information of institute that could compromise individual privacy.

## Competing interests

The authors declare that they have no competing interests.

## Funding

Not applicable.

## Authors' contributions

YT and EA were involved in the study design, data analysis and wrote the manuscript. All other authors were also involved in data collection and analysis of Holter ECG. All authors critically revised the report, commented on drafts on the manuscript, approved the manuscript to be published, and agreed to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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