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Journal of Nephrology and Hypertension

Open Access | Research Article

Antihypertensive use and risk of intradialytic hypotension in hospitalized end-stage renal disease patients

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Received: June 19, 2018

Accepted: Aug 02, 2018

Published Online: Aug 09, 2018

Journal: Journal of Nephrology and Hypertension

Publisher: MedDocs Publishers LLC

Online edition: http://meddocsonline.org/

Keywords: Antihypertensive agents; End-stage renal disease; Hemodialysis; Intradialytic hypotension; medication use

Abstract

Background: Intradialytic hypotension (IDH) is the most common complication of Hemodialysis (HD). Administration of antihypertensive medications (AHTs) in the inpatient setting often occurs before dialysis; however, the influence on the rate of IDH is unclear. This study evaluated the association of AHT, nitrates, and other factors with development of IDH in the inpatient setting.

Methods: In this single-center, retrospective study, adult hospitalized patients with end-stage renal disease requiring HD during a 2-year evaluation period were divided into IDH and non-IDH cohorts based on the occurrence of IDH during HD. AHT and nitrate use within twelve hours prior to each HD session was compared. The association between the development of IDH and serum albumin, pre-HD blood pressure, serum sodium, and ultrafiltration rate during HD was also evaluated.

Results: In 104 patients included (50 IDH, 54 non-IDH), a significantly greater proportion of IDH patients received AHTs (82% vs. 63%, p= 0.048). Additionally, arteriovenous graft use and mean pre-dialysis systolic blood pressure (SBP) were significantly higher in the IDH cohort (26% vs. 5% and 141 vs. 134 mmHg, respectively). Sub analysis of IDH dialysis sessions revealed a significant correlation between IDH and total AHT/nitrate doses received (negative) and admission hemoglobin (positive).

Conclusion: In addition to timing of administration of AHTs, pre-dialysis SBP and hemoglobin are important to consider when evaluating risk of developing IDH.

Introduction

Of the approximately 703,000 patients with end-stage renal disease (ESRD) in the U.S., approximately 437,000 patients receive hemodialysis (HD) [1]. Intradialytic hypotension (IDH) is the most common complication of HD, occurring in an estimated 5% to 30% of HD sessions, although some studies cite rates of 50% or greater [2-5]. IDH is defined as a drop of at least 20 mmHg in systolic blood pressure (SBP) or a decrease in mean arterial pressure of 10 mmHg or more with associated hypotensive symptoms. IDH is associated with increased morbidity and mortality [6,7]. In a hypotensive patient receiving dialysis, decreased perfusion of muscles and organs can result in dizziness,

Cite this article: Nishimoto A, Duhart BT, Canada RB, Shoop D, Hudson JQ. Antihypertensive use and risk of intradialytic hypotension in hospitalized end-stagerenal disease patients. J Nephrol Hypertens. 2018; 2: 1007.

fatigue, nausea, cramps, and blurred vision [3]. In severe cases, transient ischemic attacks, syncope, cerebrovascular accidents and seizures can occur [8]. Additionally, hypotensive episodes during HD may require interventions including administration of albumin, fluid replacement, decreasing the ultrafiltration rate, or discontinuing HD [9,10]. These interventions can result in inadequate dialysis or the need for specialized types of renal replacement therapy such as continuous renal replacement therapy (CRRT) [10].

High ultrafiltration rates and impaired cardiovascular response of the patient during dialysis are among the key factors responsible for precipitating IDH [10,11]. While measures such as blood volume monitoring and regulation or preemptive use of midodrine have been attempted to prevent this complication of HD, IDH remains a significant problem [12,13]. As the causes of IDH are multifactorial, investigation into potential contributing factors remains an area of interest. Antihypertensive medications (AHTs) and nitrates are commonly prescribed in the ESRD population. The occurrence of IDH and use of AHT therapy has been evaluated to some extent, but is relatively limited [14-18]. One study showed that IDH was more likely in patients not prescribed AHTs; however, the administration times of these agents prior to HD was based on patient reporting [17]. Nitrate use has also been associated with more frequent episodes of IDH; however, it is unclear whether the timing of administration of these agents relative to HD has any effect on IDH rates [19]. Since administration of AHTs and nitrates in the inpatient setting often occurs prior to dialysis, we were interested in determining if this practice increases the risk of IDH in our inpatient population and identifying other factors that may contribute to IDH.

Methods

The study was a single-center, retrospective evaluation of ESRD patients who received HD during hospitalization at Methodist University Hospital (MUH) from August 2011 through August 2013 and was approved by the Institutional Review Board at the University of Tennessee Health Science Center. Patients were included if they were at least 18 years of age and had received at least two dialysis sessions during hospitalization. Patients were excluded if they received vasopressors at any point during hospitalization, required any other type of dialysis (e.g. sustained low efficiency dialysis, peritoneal dialysis, CRRT), received any type of organ transplant, had a diagnosis of liver disease, or received consecutive dialysis sessions within 24 hours.

The study population was divided into two groups: a control group of patients who did not experience IDH (non-IDH group) and a group of patients who did experience IDH (IDH group). Intradialytic hypotension was defined as a drop in SBP of ≥ 30 mm Hg during intermittent HD, consistent with the definition used by the dialysis staff at our facility. Any AHTs or nitrates given within twelve hours prior to HD were documented with respect to the drug class, the number of doses, and time of administration. Patient demographic information including age, gender, ethnicity, and comorbidities was collected for analysis. Clinical parameters recorded included length of stay, admission sodium, albumin, and hemoglobin. Dialysis information included duration of dialysis, pre-dialysis blood pressure, sodium and albumin on the day of dialysis, ultrafiltration rate, total volume of fluid removed, average blood flow, time of occurrence of IDH during HD, dialysis access type, and interventions for IDH. Data from individual dialysis sessions was excluded if the patient's pre-dialysis systolic blood pressure (SBP) was greater than 180 mm Hg or if there was no documented pre-dialysis blood pressure. A maximum of five dialysis sessions were evaluated per patient.

The primary objective of the study was to evaluate AHT and nitrate use in patients who experienced IDH compared to those patients who did not develop IDH. Secondary objectives were to determine the relationship between pre-dialysis blood pressure, serum sodium levels, serum albumin, and the incidence of IDH. Disease- and dialysis-specific factors associated with development of IDH as well as interventions required to manage IDH were also evaluated.

Statistical analysis

Data were entered into a Microsoft Access (Seattle, WA) database, and analyses were performed with SPSS statistical software (IBM, Armonk, New York). The Shapiro-Wilk goodness-of-fit test was used to test for normality of data in both groups. Univariate analyses using either Fisher's exact test or Chi-square analysis on nominal data, student's t-test for normally distributed data, and Mann-Whitney U for non-normal data distribution were used to compare patient and dialysis characteristics in IDH vs. non-IDH groups. Categorical variables were calculated as percentages while continuous variables were expressed as either mean ± standard deviation or median (interquartile range) for nonparametric data sets.

A point-biserial correlation or phi coefficient was used for all continuous and dichotomous variables, respectively, to identify possible predictors of IDH. A bivariate logistic regression model using both a forward and reverse conditional stepwise approach was then used to devise a prediction model for IDH. A regression model was built to initially include all correlative factors that were statistically significant at the 0.05 or 0.01 level by either point-biserial correlation or phi coefficient. Variables that did not demonstrate significant correlation were then added to the model individually, and the logistic regression was rerun to determine their significance. Only variables significant in both the forward and reverse conditional logistic regression were considered as predictors of IDH. To further define predictors of IDH, the same correlation test was run in a subgroup analysis comparing the non-IDH cohort to a modified IDH cohort compromised of dialysis-specific data from only the dialysis sessions where IDH occurred.

For all statistical tests, a *p*-value less than 0.05 was considered significant. For the univariate analysis and prediction of IDH, AHTs and nitrate use was combined into a single variable, as the number of nitrate doses administered in this study population was too sparse to be statistically meaningful.

Results

A total of 372 patients were screened based on the inclusion and exclusion criteria. In total, 303 dialysis sessions in 104 patients were analyzed. There were 50 patients in the IDH group (accounting for 164 dialysis sessions) and 54 patients in the non-IDH group (accounting for 139 dialysis sessions). When comparing the baseline characteristics of the IDH cohort and the non-IDH cohort, there were no significant differences identified with regard to age, gender, ethnicity, comorbidities, admission labs, or length of stay (Table 1).

A significantly greater number of patients in the IDH group received AHTs within the 12 hours prior to HD compared to patients in the non-IDH group (82% vs. 63%, p = 0.048). Figure 1 shows the percentage of patients in each group who received

AHTs by drug class. While overall usage of AHTs was greater in the IDH group, there were no significant differences in the proportion of patients receiving AHTs when evaluated by individual drug classes.

There were no significant differences in the dialysis-specific characteristics of the two groups (Table 2). Patients were dialyzed for approximately 3.5 hours and had a similar pre-dialysis sodium and albumin. However, patients in the IDH cohort had a significantly elevated pre-dialysis SBP compared to the non-IDH group (141 mmHg and 134 mmHg, respectively, $p<0.05$). Also, a significantly greater percent of patients in the IDH group had an arteriovenous (AV) graft as their vascular access site compared to the non-IDH patients (26% vs. 5%, $p<0.01$).

In the IDH group, hypotension occurred in 45% (73 of 164) of the total dialysis sessions. On average, IDH occurred 1.9±1.0 hours after the start of dialysis and the median blood pressure at event was 99/54 mmHg. The median ultrafiltration amount was 1260 mL at the time IDH occurred. Interventions to address IDH were required in 40% (29/73) of dialysis sessions. The vast majority of the interventions (97%) included decreasing the ultrafiltration rate. Fluid boluses with either normal saline or albumin were administered in seven instances (five normal saline boluses, two albumin boluses). In two cases, dialysis was stopped prematurely because of significant IDH.

When identifying patient factors associated with IDH, a significant correlation was observed between development of IDH and pre-dialysis SBP, AV graft access, and administration of at least one AHT twelve hours prior to the start of HD (Table 3). Pre-dialysis SBP was not significant in the bivariate logistic regression model; however, receiving at least one AHT and graft access type remained significant in the model (Table 3).

In the subgroup analysis using a modified IDH cohort that included only the 73 dialysis sessions in which IDH occurred, pre-dialysis SBP and graft access were both significantly correlated with IDH (Table 3), as in our original analysis. The use of beta-blockers, hydralazine and total AHT and nitrate doses received by a patient were also significantly negatively associated with IDH. The bivariate logistic regression (Table 3) showed significance with pre-dialysis SBP and admission hemoglobin as positive predictors of IDH and total doses of AHTs and nitrates received as a significant negative predictor.

Discussion

In this study, we found that approximately 48% of patients with ESRD on HD experienced IDH, which occurred in 24% (73 of 303) of all HD treatment sessions evaluated. This is within the range of incidence of IDH reported in the literature, which includes incidences as high as 59% [4,5,8,20]. It has been reported that certain subgroups, such as those with impaired autonomic responses, the elderly, and diabetic patients may be at risk for higher rates of IDH, upwards of 50% [4]. Our study did not detect differences in our IDH and non-IDH cohorts with regard to age or diabetes, likely due to the inadequate powering of the sample size. The variability in the incidence of IDH reported in the literature can likely be attributed to inconsistency in defining IDH across studies. For example, in contrast to KDOQI guideline definitions of IDH, Coli *et al*. used a SBP of ≤90 mmHg or a decrease in SBP of either 10% or 25 mmHg, depending on symptoms and initial SBP [7,20]. Our definition of IDH was dependent on the relative drop in SBP and, therefore, patients with elevated SBP may have been more prone to experience IDH per our definition even if they were asymptomatic. This can explain why higher pre-dialysis SBP was a key predictor of IDH in our subgroup analysis.

In addition, our study found that use of AHTs may play a role in the incidence of IDH, particularly with regard to the timing of administration before dialysis. While current clinical guidelines recommend discontinuation of AHTs prior to dialysis, the exact timeframe for withholding these medications is not well-defined [7]. Therefore we chose to evaluate the potential influence of AHTs administered over a prolonged time period prior to dialysis. While there was no difference in the type of AHT given prior to HD, the cumulative number of doses of blood pressure-lowering medications was significantly associated with decreased incidence of IDH. This finding in the subgroup analysis is seemingly congruent to our observation in the overall study cohort, in which the regression model indicated that receiving any AHT medication within 12 hours of dialysis was a negative predictor of IDH. One possible explanation for this may be the tendency of those with persistent complicated hypertension to receive multiple blood pressure-lowering medications. These types of patients may be resistant to significant decreases in blood pressure during dialysis and, therefore, may not be at as great a risk of IDH compared to other populations that use AHTs more infrequently. It has been reported that individuals who do not meet blood pressure goals may have a decreased incidence of IDH [17].

Our analysis revealed possible patient-specific factors that heretofore have never been identified as possible predictors of IDH. Admission hemoglobin levels were the strongest predictor of IDH in our logistic regression model, with high hemoglobin levels associated with a greater risk of developing IDH. While we did not evaluate hemoglobin beyond admission, it is possible that this result relates to fluid and blood volume status. Blood volume monitoring and blood volume controlled HD has been shown to reduce rates of IDH [21-23]. A higher admission hemoglobin may be reflective of a poor intravascular blood volume which may lead to hypotensive episodes during dialysis.

The mechanism by which AV graft access might be related to incidence of IDH cannot be definitively explained. Chang *et al*. showed that frequency of IDH was significantly associated with AV fistula thrombosis, but not graft thrombosis [24]. However, the small number of included patients with graft access (n=15) precluded further evaluation of this observation.

A study evaluating blood pressure control and symptomatic IDH in the UK did report some findings similar to our own [17]. In this study, symptomatic hypotension occurred in 7% of all dialysis treatments. Patients not prescribed AHTs were more likely to suffer symptomatic IDH [17]. Though a clinical definition of IDH (symptomatic hypotensive episodes requiring intravenous fluid resuscitation) was used in this study rather than an objective benchmark, patients who achieved post-dialysis blood pressure targets had a significantly higher incidence of IDH. Of note, the details of AHT administration and whether the medications were held prior to starting dialysis was not reported in the study. Regardless, our results propose that there exists an association with AHTs and IDH, with the number of doses prior to dialysis being an important determinant.

There were inherent limitations due to the retrospective nature of our study. Inter-staff variability in assessing and treating the patient for IDH may have influenced the decision to intervene and the intervention choice. Additionally, the retrospec-

tive nature of this study prevented evaluation of symptomatic patients. While our objective definition of IDH was adopted from the clinical setting of the dialysis unit, the inability to assess IDH-associated symptoms and the fact that our definition of IDH differs to some extent from other established definitions also limits generalizability. External validity is also compromised due to the fact that we are evaluating hospitalized patients as opposed to ambulatory dialysis patients; however, the intent was to evaluate factors that potentially influence development of IDH in the inpatient setting. Furthermore, we were unable to determine the duration of time between the first inpatient dialysis session and dialysis prior to admission, if any. Thus, it is possible that we unintentionally included patients who received HD before hospitalization and then again after admission within 24 hours between sessions (one of our exclusion criteria). Since our subgroup analysis focused on individual dialysis sessions where IDH occurred, there may be a patient-specific prediction bias weighted towards patients who experienced multiple sessions with IDH.

Regarding data collection, we chose to record AHT medications given within twelve hours prior to the start of HD. As such, there may be patients who received AHTs within a wider window prior to HD. We also limited our data collection to the first five dialysis sessions per patient; therefore, our results do not include information on IDH that may have occurred in patients with prolonged hospital stays. Hydration status of the patient and/or the ultrafiltration volume in relation to the patients dry weight was also not evaluated and should be taken into consideration in future investigations. Lastly, the incidence of IDH per patient is not described in our study, as only the first occurrence of IDH in a patient was recorded and subsequent re-hospitalizations for the same patient were omitted.

Conclusion

To our knowledge, this is the first study to evaluate the role of AHT administration and the risk of developing IDH in the inpatient setting. This study suggests that in addition to the timing of administration of AHTs, other factors including pre-dialysis SBP and hemoglobin are important to consider when evaluating risk of developing IDH and adds to the currently limited body of knowledge regarding development of IDH in the inpatient setting. Though further research is warranted, the results of this study may help to elucidate factors that are associated with risk for IDH and contribute to more clearly defining a window of time prior to dialysis in which AHTs should be avoided.

Acknowledgement

The authors would like to thank Dr. Joyce E. Broyles for her help with research oversight and Khalid bin Saleh and Faisal Sehli for their help with data collection. This work was conducted at Methodist University Hospital in Memphis, TN.

Disclosure of interest

The authors declare that they have no conflicts of interest. The results presented in this paper have not been published previously in whole or part, except in abstract format.

Tables

Table 1: Baseline characteristics (*N* = 104).

Variables	IDH (*N* = 50)	Non-IDH (*N* = 54)	*P*-value
Mean age (± SD), years	61 ± 16	58 ± 16	0.33
Female sex, n (%)	30 (60)	29 (53.7)	0.52
Median length of stay (IQR), days	7 (5 – 10)	6 (4 – 8)	0.06
Admission lab values			
Median sodium (IQR), mmol/L	138 (136 – 141)	138 (136 – 140)	0.76
Mean albumin(± SD), g/dL	3.1 ± 0.6	3.0 ± 0.6	0.44
Mean hemoglobin(± SD), g/dL	10.6 ± 2.0	9.9 ± 1.9	0.06
Race, n (%)			
African-American	41 (82)	50 (93)	0.10
Caucasian	8 (16)	3 (5)	0.08
Hispanic	1 (2)	0 (0)	0.48
Multiracial	0 (0)	1 (2)	1.00
Comorbidities, n (%)			
Hypertension	47 (94)	50 (93)	1.00
Diabetes	32 (64)	30 (56)	0.38
Heart Failure	13 (26)	17 (31)	0.54
Coronary Artery Disease	13 (26)	12 (22)	0.65
Stroke or TIA	11 (22)	12 (22)	0.98
Peripheral Artery Disease	8 (16)	7 (13)	0.66
Infection	6 (12)	8 (15)	0.67
Other	3 (6)	4 (7)	1.00

†IDH: Intradialytic Hypotension; TIA: Transient Ischemic Attack; SD: Standard Deviation; IQR: Interquartile Range

Table 2: Dialysis parameters and vascular access type.

Dialysis Parameters‡	IDH (*N*=50)	Non-IDH (*N*=54)	*P*-value
Number of dialysis sessions	3 ± 1	3 ± 1	1.00
Median duration of dialysis (IQR), hours	3.42 (3 – 3.75)	3.49 (3.25 – 3.75)	0.38
Pre-dialysis SBP, mmHg	141 ± 21	134 ± 22	<0.05
Pre-dialysis DBP, mmHg	69 ± 15	70 ± 15	0.25
Pre-dialysis sodium, mmol/L	138 ± 4	138 ± 4	0.51
Pre-dialysis albumin, g/dL	2.8 ± 0.6	2.9 ± 0.6	0.59
Blood flow rate, mL/min	367 ± 48	381 ± 42	0.76
Ultrafiltration rate, mL/min	739 ± 282	723 ± 332	0.22
Ultrafiltration volume, mL	2297 ± 1139	2356 ± 1207	0.56
Vascular access type	**IDH (*N*=50)**	**Non-IDH (*N*=54)**	***P*-value**
Catheter, n (%)	19 (38)	22 (41)	0.78
AV Fistula, n (%)	19 (38)	29 (54)	0.11
AV Graft, n (%)	12 (26)	3 (5)	<0.01

† IDH: Intradialytic Hypotension; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; AV: Arteriovenous; IQR: Interquartile Range

‡Reported as mean ± standard deviation unless otherwise noted.

Table 3: Analysis of factors overall and by subgroup.

Correlation of Patient Factors with IDH		Logistic regression model for predictors of IDH	
Variable	**Point-Biserial/Phi Coefficient Correlation**	***P*-value**	**Odds Ratio (95% CI)**
Total dialysis sessions in IDH patients (*N* = 164)			
Pre-dialysis systolic blood pressure	0.212*	-------	-------
Received any antihypertensive/nitrate	0.194*	0.030	0.312 (0.117 – 0.832)
AV Graft access	0.262**	0.009	0.153 (0.038 – 0.620)
Dialysis sessions involving IDH (*N* = 73)‡			
Pre-dialysis systolic blood pressure	0.380**	0.000	1.064 (1.035 - 1.100)
Number Antihypertensive/nitrate doses received Admission hemoglobin	-0.273** -------	0.003 0.020	0.721 (0.578 - 0.891) 1.343 (1.034 - 1.707)
Beta blocker usage	-0.208*	-------	-------
Hydralazine usage	-0.203*	-------	-------
AV Graft access	0.262**		

*Correlation is significant at the 0.05 level (2-tailed)

**Correlation is significant at the 0.01 level (2-tailed)

‡Included only the 73 dialysis sessions in which IDH occurred

IDH = Intradialytic Hypotension; AV = Arteriovenous

Figures

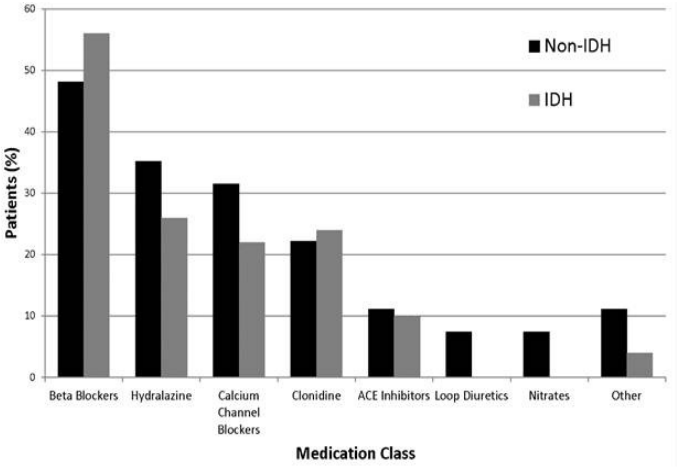

Figure 1: Proportion of Antihypertensive and Nitrate Use, By Medication Class.

Percentage of patients in IDH and non-IDH cohorts who received at least one dose from a respective medication class. Significance was not noted across any medication class between groups.

References

1. United States Renal Data System. 2017 USRDS annual data report: Epidemiology of kidney disease in the United States. National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases. Bethesda, MD. 2017.
2. Fortin PM, Bassett K, Musini VM. Human albumin for intradialytic hypotension in haemodialysis patients. Cochrane Database Syst Rev. 2010: CD006758.
3. Perazella MA. Pharmacologic options available to treat symptomatic intradialytic hypotension. Am J Kidney Dis. 2001; 38: S26-36.
4. Perazella MA. Review Articles: Approach to Patients with Intradialytic Hypotension: A Focus on Therapeutic Options. Seminars in Dialysis. 1999; 12.
5. Sands JJ, Usvyat LA, Sullivan T, Segal JH, Zabetakis P, Kotanko P, et al. Intradialytic hypotension: frequency, sources of variation and correlation with clinical outcome. Hemodial Int. 2014; 18: 415-422.
6. Grange S, Hanoy M, Le Roy F, Guerrot D, Godin M. Monitoring of hemodialysis quality-of-care indicators: why is it important? BMC Nephrol. 2013; 14: 109.
7. K/DOQI Workgroup. K/DOQI clinical practice guidelines for cardiovascular disease in dialysis patients. Am J Kidney Dis. 2005; 45: S1-153.
8. Santos SF, Peixoto AJ, Perazella MA. How should we manage adverse intradialytic blood pressure changes? Adv Chronic Kidney Dis. 2012; 19: 158-165.
9. Knoll GA, Grabowski JA, Dervin GF, O'Rourke K. A randomized, controlled trial of albumin versus saline for the treatment of intradialytic hypotension. J Am Soc Nephrol. 2004; 15: 487-492.
10. Agarwal R. How can we prevent intradialytic hypotension? Curr Opin Nephrol Hypertens. 2012; 21: 593-599.
11. Palmer BF, Henrich WL. Recent advances in the prevention and management of intradialytic hypotension. J Am Soc Nephrol. 2008; 19: 8-11.
12. Lim PS, Yang CC, Li HP, Lim YT, Yeh CH. Midodrine for the treatment of intradialytic hypotension. Nephron. 1997; 77: 279-283.
13. Santoro A, Mancini E, Paolini F, Cavicchioli G, Bosetto A, Zucchelli P. Blood volume regulation during hemodialysis. Am J Kidney Dis. 1998; 32: 739-748.
14. Peters CD, Kjaergaard KD, Jensen JD, Christensen KL, Strandhave C, Tietze IN, et al. Short and Long-Term Effects of the Angiotensin II Receptor Blocker Irbesartan on Intradialytic Central Hemodynamics: A Randomized Double-Blind Placebo-Controlled One-Year Intervention Trial (the SAFIR Study). PLoS One. 2015; 10: e0126882.
15. Sherman RA, Casale P, Cody R, Horton MW. Effect of predialysis verapamil on intradialytic blood pressure in chronic hemodialysis patients. ASAIO Trans. 1990; 36: 67-69.
16. Davenport A, Cox C, Thuraisingham R. Blood pressure control and symptomatic intradialytic hypotension in diabetic haemodialysis patients: a cross-sectional survey. Nephron Clin Pract. 2008; 109: c65-71.
17. Davenport A, Cox C, Thuraisingham R. Achieving blood pressure targets during dialysis improves control but increases intradialytic hypotension. Kidney Int. 2008; 73: 759-764.
18. Leidig M, Bambauer R, Kirchertz EJ, Szabã T, Handrock R, Leinung D, et al. Efficacy, safety and tolerability of valsartan 80 mg compared to irbesartan 150 mg in hypertensive patients on long-term hemodialysis (VALID study). Clin Nephrol. 2008; 69: 425-432.
19. Tisler A, Akocsi K, Harshegyi I, Varga G, Ferenczi S, Grosz M, et al. Comparison of dialysis and clinical characteristics of patients with frequent and occasional hemodialysis-associated hypotension. Kidney Blood Press Res. 2002; 25: 97-102.
20. Coli L, La Manna G, Comai G, Ursino M, Ricci D, Piccari M, et al. Automatic adaptive system dialysis for hemodialysis-associated hypotension and intolerance: a noncontrolled multicenter trial. Am J Kidney Dis. 2011; 58: 93-100.
21. Santoro A, Mancini E. Blood volume monitoring systems and biofeedback. Contrib Nephrol. 2002: 233-244.
22. Santoro A, Mancini E, Basile C, Amoroso L, Di Giulio S, Usberti M, et al. Blood volume controlled hemodialysis in hypotension-prone patients: a randomized, multicenter controlled trial. Kidney Int. 2002; 62: 1034-1045.
23. Micklos L. Does blood volume monitor use decrease episodes of intradialytic hypotension in chronic hemodialysis treatments? Nephrol Nurs J. 2013; 40: 447-450.
24. Chang TI, Paik J, Greene T, Desai M, Bech F, Cheung AK, et al. Intradialytic hypotension and vascular access thrombosis. J Am Soc Nephrol. 2011; 22: 1526-1533.