

Original Article

Frequency of Intradialytic Hypotension in Hemodialysis Patients and Its Relationship with Serum Osmolarity and Electrolytes

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Abstract

Objective: Intradialytic hypotension (IDH) is the most common complication of hemodialysis (HD). Many factors (cardiac factors, lack of vasoconstriction, etc.) that cause IDH have been reported so far. However, studies investigating the relationship between IDH and serum osmolarity and serum electrolytes (especially magnesium) are not sufficient. In this study, we aimed to determine the incidence of IDH in hemodialysis patients and to investigate the relationship between serum osmolarity and electrolytes and IDH.

Methods: A total of 94 patients received routine dialysis treatment at our center. Among these, 66 patients who met the inclusion criteria were included in the study. Serum osmolarity, electrolytes (sodium, potassium, calcium, magnesium), glucose and urea levels were measured from three samples drawn at the start, middle (2 hours in), and end of each session. Blood pressure was recorded every 15 minutes during HD with an ambulatory blood pressure device.

Results: IDH was detected in 29 patients (44%). In patients with IDH, blood pressure and blood glucose levels at the beginning, middle, and end of sessions were higher than those without IDH. Magnesium levels were also higher in patients with IDH in samples obtained at the end of the sessions. Independent risk factors for IDH are stated as initial glucose level, magnesium level at the end of the session, and antihypertensive drug use.

Discussion: It is important to control hypertension in patients with IDH and to prevent eating during HD sessions. Prospective studies on serum magnesium levels in the prevention of IDH are needed..

Keywords: Hemodialysis, Hypotension + Renal Dialysis, Osmolar Concentration, Electrolytes

INTRODUCTION

Intradialytic hypotension (IDH) is the most common complication of acute dialysis and occurs in 20%–30% of all hemodialysis sessions - resulting in many adversities (such as decreased life quality, inadequate dialysis, vascular tract problems, myocardial dysfunction, and death) (1). Some observational studies have reported that IDH increases both cardiovascular mortality and all-cause mortality (2, 3).

Causes of IDH consist of low intravascular volume (high ultrafiltration rate, low target dry weight, low dialysate sodium etc.), cardiac factors (decreased cardiac output, heart failure, low dialysate calcium level, etc.), vasoconstriction deficiency (warm dialysate, splenic vasodilatation, autonomic neuropathy, antihypertensive treatment) and changes in osmolarity (4).

In a recent study, it was reported that high plasma osmolarity before dialysis was associated with IDH development (5). In the literature, there is a limited number of studies investigating intradialytic hypotension in terms of its relationships with serum osmolarity and serum electrolytes.

In this study, we aimed to determine the frequency of IDH in patients receiving regular hemodialysis treatment, and to investigate the relationships between IDH development and calculated and measured serum osmolarity and the serum electrolytes.

METHODS

This study was carried out with 94 patients with end-stage renal failure (ESRD) who underwent routine dialysis in the dialysis unit of our hospital. 28 of these

patients were excluded from the study according to the exclusion criteria. 34 (51.5%) of the patients included in the study were male and 32 (48.5%) of them were female.

Exclusion Criteria;

The patients who satisfied at least one of the following criteria were excluded from the study:

- Patients under 18 years old (4 patients)
- Patients who had recently begun dialysis treatment (shorter than 3 months) (4 patients)
- Patients with any type of infection (2 patients)
- Patients with newly diagnosed or metastatic cancer (5 patients)
- Patients using cocaine, intravenous drugs or chemotherapy (1 patient)
- Patients who were very frail, failed to provide informed consent or had communication problems (12 patients)

The study was approved by N.E.U Meram Medical Faculty Ethics Committee with the decision no 2015/390. All patients were informed in detail about the study and signed the informed consent form.

Data concerning age, gender and antihypertensive drug use of each patient in the study group was recorded. All patients were evaluated during weekday dialysis sessions and patients underwent hemodialysis in the same conditions: with a dialysate temperature of 36.5 °C, dialysate sodium of 138 meq/l, dialysate potassium of 2meq/l, dialysate calcium of 1.5 mg/dl and dialysate magnesium of 0.50 mmol/l. Arterial blood pressures were measured and recorded by an ambulatory blood pressure device at 15-minute intervals during the 4-hour dialysis period. Serum osmolarity, glucose, sodium, potassium, calcium, magnesium, and urea levels were analyzed at the beginning of the dialysis, at the second hour of the dialysis session and at the end of the dialysis. The osmolarity level of the patients was measured with Löser Mikro-Osmometer (type 15) (Löser Messtechnik, Germany); sodium, potassium, calcium and magnesium levels were measured with Olympus AU 5800 autoanalyzer-ion selective electrode - ISE Unit (Beckmann Coulter Inc., USA). Glucose and urea levels were measured by Olympus AU 5800 autoanalyzer device (Beckmann Coulter Inc, USA) with the spectrophotometric method. In addition, albumin levels were measured at the beginning of hemodialysis to account for the corrected Calcium level.

During each dialysis session, dialysis nurses recorded whether patients had nausea, muscle cramps, dizziness, sweating, vomiting, anxiety and feeling of lightheadedness.

Intradialytic hypotension was defined according to the KDOQI guideline (≥ 20 mmHg decrease in systolic blood pressure (SBP) or a 10 mmHg decrease in mean blood pressure accompanied by muscle cramps, dizziness, nausea, sweating) (6).

STATISTICAL ANALYSIS

The SPSS 19.0 package program was used for statistical data analyses. Descriptive measurements were calculated for all nominal and ratio scale variables. Nominal scale variables are presented with frequency and percentage ratio, and continuous scale variables with mean $\pm$ SD. Continuous numerical variables were checked by the Kolmogorov-Smirnov Test to determine normality of distribution. Intra-group distributions of the majority of variables were not normal. Therefore, the Mann-Whitney U test was preferred for the comparison of groups. Monte Carlo simulation chi-square test was used to determine the relationship between categorical variables. Type-I error value was set as 5% and $p < 0.05$ was accepted to be statistically significant. Multivariate binary logistic regression analysis was performed to analyze factors which contributed to intradialytic hypotension.

RESULTS

A total of 66 patients with end-stage renal failure (ESRF) were included in the study. Of these, 32 (48%) were female and 34 (52%) were male. The time of day at which dialysis was performed was the morning in 26 (39%), the afternoon in 33 (50%) and the evening in 7 (11%) patients. IDH was detected in 29 (44%) of the 66 patients, while IDH was not detected in 37 (56%). 18 (27%) patients had a diagnosis of diabetes mellitus (DM), while 48 (73%) patients did not have DM.

Patients with and without IDH were compared according to their laboratory characteristics. In patients with IDH, blood glucose values at the beginning of the session, at the second hour of the session and at the end of the session were found to be higher than those without IDH. Magnesium levels were also higher in patients with IDH at the end of the sessions. On the other hand, comparisons revealed that the osmolarity, urea, calcium, sodium and potassium levels of both patient groups were similar at the beginning, at the second hour and at the end of the session (**Table 1**).

When the patients in both groups were compared in terms of clinical and demographic characteristics; it was detected that patients with IDH had higher systolic and diastolic blood pressures and used more antihypertensive drugs. Patients in both groups were similar in terms of ultrafiltration volume and percentages during dialysis, as well as age, gender, the presence of DM, dialysis times, and pre-dialysis weights. Although it did not reach statistical significance, it was seen that patients with IDH mostly underwent hemodialysis in the afternoon and evening sessions (**Table 2**).

When the patients with DM were excluded from the analyses, IDH was detected in 18 patients and not detected in 30. In the comparison of the laboratory findings, clinical assessments and demographic characteristics of these patients in regard to the presence/absence of IDH; it is found that –similar to the overall findings– glucose levels, end-of-session magnesium value, systolic and diastolic blood pressures and antihypertensive drug use in IDH patients were higher than those without IDH

Table 1. Laboratory values and changes of cases according to hypotension conditions)

Parameter		Intradialytic hypotension positive (N=29)	Intradialytic hypotension negative (N=37)	Total (N=66)	P
Measured osmolarity (mOsm/kg)	Beginning	289±12.7	290±6.8	289.6±9.7	0.525
	2nd hour	290.4±4.8	287.8±3.6	288.9±4.3	0.070
	End-of-season	289.2±6.7	288.2±3.8	288.6±5.3	0.917
	Change (0-2)	-1.4±13.3	2.2±6.4	0.6±10.1	0.056
	Change (0-4)	-0.2±15.2	1.8±7.0	0.9±11.3	0.969
Calculated osmolarity (mOsm/kg)	Beginning	302.3±7.2	300.9±7.8	301.5±7.5	0.405
	2nd hour	291.7±5.9	292.0±6.7	291.9±6.3	0.913
	End-of-season	287.3±5.4	284.8±7.5	285.9±6.8	0.165
	Change (0-2)	10.5±7.3	8.9±7.3	9.3±7.3	0.281
	Change (0-4)	14.9±6.8	16.1±9.4	15.6±8.3	0.628
Glucose (mg/dl)	Beginning	188.0±124.5	123.3±56.9	151.8±97.6	0.036
	2nd hour	151.1±50.8	120.4±38.6	133.9±46.6	0.009
	End-of-season	136.1±49.5	105.3±44.7	118.9±49.0	0.002
	Change (0-2)	36.9±102.9	2.9±64.6	17.8±84.6	0.477
	Change (0-4)	51.9±107.4	18.0±70.5	32.9±89.5	0.531
Urea (mg/dl)	Beginning	114.1±28.2	116.7±37.5	115.6±33.5	0.954
	2nd hour	53.8±15.7	57.2±22.9	55.7±2	0.601
	End-of-season	31.2±10.9	34.0±14.9	32.8±13.3	0.522
	Change (0-2)	60.3±16.6	59.5±18.8	59.9±17.7	0.727
	Change (0-4)	82.9±21.1	82.7±25.3	82.8±23.4	0.756
Corrected Ca ⁺² (mg/dl)	Beginning	8.8±0.5	8.7±0.9	8.8±0.8	0.646
	2nd hour	9.6±0.4	9.6±0.6	9.7±0.6	0.093
	End-of-season	10.3±0.5	10.1±0.6	10.2±0.6	0.078
	Change (0-2)	-1.0±0.4	-0.9±0.6	-1.0±0.5	0.227
	Change (0-4)	-1.5±0.7	-1.4±0.9	-1.4±0.8	0.332
Mg ⁺² (mg/dl)	Beginning	2.38±0.40	2.27±0.36	2.32±0.38	0.146
	2nd hour	2.03±0.19	1.96±0.20	1.99±0.20	0.104
	End-of-season	1.96±0.14	1.88±0.15	1.92±0.15	0.024
	Change (0-2)	0.35±0.36	0.31±0.26	0.33±0.31	0.865
	Change (0-4)	0.42±0.37	0.39±0.28	0.4±0.32	0.739
Na ⁺¹ (mEq/L)	Beginning	136.38±2.8	137.3±2.7	136.9±2.8	0.100
	2nd hour	137.2±2.4	137.9±2.9	137.58±2.7	0.266
	End-of-season	137.3±2.5	136.6±3.4	136.9±3.0	0.617
	Change (0-2)	-0.79±2.77	-0.59±2.83	-0.68±2.79	0.979
	Change (0-4)	-0.90±2.88	0.68±4.13	-0.02±3.69	0.189
K ⁺¹ (mEq/L)	Beginning	4.88±0.58	4.83±0.87	4.85±0.75	0.349
	2nd hour	3.63±0.48	3.62±0.39	3.62±0.43	1.000
	End-of-season	3.47±0.31	3.5±0.33	3.49±0.32	0.938
	Change (0-2)	1.25±0.37	1.21±0.58	1.23±0.49	0.269
	Change (0-4)	1.41±0.5	1.33±0.76	1.36±0.65	0.376

(data not shown in the table).

The effect of various variables that differed between the groups for IDH status in all patient groups was analyzed with binary logistic regression models. Antihypertensive drug use, end-of-session magnesium value, initial systolic blood pressure, and initial, second hour and end-of-session glucose levels were taken as independent variables and both “enter” and “conditional forward” logistic regression models were created. In the enter model ($R^2 = 0.381$), the effects of antihypertensive drug use, initial glucose level and end-of-session magnesium were significant ($p = 0.004$, $OR = 0.112$; $p = 0.012$, $OR = 0.984$; $p = 0.014$, $OR = 0.002$ respectively). In the conditional forward (CF) model ($R^2 = 0.344$), the independent variables found to be significant in the

“enter” model were also found to be significant (**Table 3**).

DISCUSSION

In our study, the incidence of IDH in one session of dialysis was found to be 44% in our group of dialysis patients. According to our analysis, in dialysis patients, high blood glucose levels (diabetic or non-diabetic), end-session magnesium levels and use of antihypertensive drugs were determined to be independent risk factors for IDH.

In a recent review, parameters such as, advanced age, female sex, presence of DM, long-term dialysis treatment, increased BMI, low predialysis systolic blood pressure and high ultrafiltration, were listed as risk factors for

Table 3. Clinical and demographic characteristics of cases according to hypotension

Parameter		Intradialytic hypotension positive (N=29)	Intradialytic hypotension negative (N=37)	Total (N=66)	P
Age		51.8±17.0	49.7±17.8	50.6±17.4	0.610
Gender	Male	16 (55.2)	18 (48.6)	34 (51.5)	0.599
	Female	13 (44.8)	19 (51.4)	32 (48.5)	
DM	Positive	11 (37.9)	7 (18.9)	18 (27.3)	0.085
	Negative	18 (62.1)	30 (81.1)	48 (72.7)	
Dialysis time	(month)	41.5±33.4	42.2±45.5	41.9±40.3	0.362
Systolic blood pressure (mmHg)	Beginning	150±33.8	133.1±26.9	140.5±31.0	0.041
	2nd hour	121.4±27.8	141.1±29.2	132.4±30.0	0.011
	End-of-season	119.3±31.2	140.9±29.4	131.4±31.9	0.002
Diastolic blood pressures (mmHg)	Beginning	91.3±19.8	87±20.1	88.9±19.9	0.387
	2nd hour	77.8±16.7	88.1±19	83.6±18.6	0.043
	End-of-season	76.3±18.4	89.7±21	83.8±20.9	0.007
Pulse Pressure (mmhg)	Beginning	46.5±15.9	52.4±14.3	49.8±15.2	0.050
	2nd hour	81.4±15.5	76.1±13.6	78.5±14,6	0.196
	End-of-season	84±15,4	82±12,9	82,8±14	0.543
Pre-dialysis weights	(kg)	66,9±13.4	67,4±15	67,2±14,2	0.882
Post-dialysis weights	(kg)	64,5±13,1	64,9±14,6	64,8±13,9	0.877
Intradialytic UF	(ml/kg/hour)	9±3,6	9,2±4,4	9,1±4	0.964
UF percentage	(%)	3,6±1,4	3,7±1,8	3,6±1,6	0.964
antihypertensive drug use	Yes	17 (58,6)	11 (29,7)	28 (42,4)	0.018
	No	12 (41,4)	26 (70,3)	38 (57,6)	
Hb	g/dL	11,5±1,6	11±1,3	11,2±1,4	0.229
Session time	Morning	8 (27,6)	18 (48,6)	26 (39,4)	0.097
	Noon	17 (58,6)	16 (43,2)	33 (50,0)	
	Evening	4 (13,8)	3 (8,1)	7 (10,6)	

IDH (7). However, in our study; results concerning age, gender, duration of dialysis, the frequency of DM, intradialytic ultrafiltration volume and percentages, measured and calculated serum osmolarities were found to be similar in both patient groups.

There are a limited number of studies investigating the relationship between magnesium levels and IDH. Magnesium plays an important role in maintaining the electrical stability and vascular smooth muscle tone of the myocardium. The first study that investigated the relationship between magnesium level and blood pressure in dialysis patients was performed with 8 HD patients by Kyriazis et al.. In this study, 4 different dialysates were

used in terms of dialysate calcium and magnesium levels. It was found that IDH was more frequent in the group which received 0.25 mmol/L dialysate magnesium and 1.25 mmol/L dialysate calcium. In addition, increasing the dialysate magnesium level to 0.75 mmol/L (in patients receiving 1.25 mmol/L dialysate calcium) was found to prevent the development of IDH (8).

Another study was conducted by Pakfetrat et al. with the participation of 98 hemodialysis patients. In this study, all serum electrolyte measurements including serum magnesium were performed three times (before, at the second hour and at end of the dialysis session). They reported that serum magnesium levels in patients

Table 3. Binary logistic regression models for IDH status

N=66, Hypotensive Situation	Model 1 (Enter), Cox & Snell R ² =0,381		Model 2 (Conditional forward), Cox & Snell R ² =0,109		Model 4 (Conditional forward), Cox & Snell R ² =0,344	
	p	ExpB (CI%95)	p	ExpB (CI%95)	p	ExpB (CI%95)
antihypertensive drug use	0.004	0.112 (0.025- 0.499)			0.004	0.121 (0.029-0.501)
Initial Glucose	0.012	0.984 (0.972- 0.996)	0.014	0.992(0.986-0.998)	0.002	0.983 (0.972-0.994)
2nd hour Glucose	0.422	1.013 (0.982-1044)				
End-of-season Glucose	0.152	0.981 (0.965-1.010)				
End-of-season Magnesium	0.014	0.002 (0.0-0.272)			0.008	0.002 (0.0-0.197)
Initial Systolic blood pressure	0.277	0.987 (0.965-1.010)				

who developed IDH significantly decreased during the session compared to those without IDH (9).

Elsharkawy et al. evaluated 20 dialysis patients that initially underwent acetylated dialysis followed by bicarbonate dialysis. The relationship between the frequency of IDH in both dialysis modalities and serum magnesium changes were investigated. In the acetylated dialysis, the decrease in the serum magnesium level from 3 mg/dl to 1.97 mg/dl at the end of the session was associated with IDH (negative correlation). In the bicarbonate dialysis, the increase in serum magnesium level from 2.73 mg/dl to 4.73 mg/dl was found to be associated with IDH (positive correlation) (10).

In our study, the initial serum magnesium levels of all patients were within normal limits. Furthermore, it was found that the amount of decrease in magnesium levels during dialysis were insignificant in all patients (with and without IDH). The lack of significance in magnesium changes may be associated with the small sample size of the current study. According to the literature on this topic and the results of our study, we believe that a study involving more patients and applying different concentrations of dialysate magnesium according to patient magnesium levels (for instance: dialysate with 0.5 mmol/L magnesium in patients with magnesium levels in the upper limit of normal range, and dialysate with 0.75 mmol/L magnesium in patients with serum magnesium levels in the normal range) could better demonstrate the effects of magnesium levels on the development of IDH. In conclusion; we suggest that serum magnesium levels (in addition to calcium, sodium and potassium) of dialysis patients should be measured regularly to prevent IDH development.

In the current study, we detected that IDH occurred in patients who were found to have high blood glucose levels at the start of the procedure and during the session, regardless of DM. We also detected that high glucose at the beginning of the session was an independent risk factor for IDH; however, this may be explained by the fact that half of our patients were in the noon session and therefore they had eaten.

Postprandial hypotension (PPH) was first described as a clinical problem in 1977 (defined as 20 mmHg or more reduction of SBP within 2 hours after eating) (11). PPH develops as a result of dysfunctions in normal homeostatic mechanisms necessary to maintain blood pressure in response to a reduction in systemic vascular resistance due to splanchnic and peripheral vasodilatation with no compensation an increase in cardiac output (12). This may be explained by the fact that sudden IDH cases cause a decrease in cardiac output due to splanchnic relaxation (due to eating) without a significant increase in hematocrit (13). The first study on PPH in hemodialysis patients was conducted by Richard et al. In this study, a standard meal was given in 62 of the 125 dialysis sessions of 9 non-diabetic hemodialysis patients, and prospective blood pressures of the patients were checked. While symptomatic hypotension developed 13 times in 5 patients who ate, only one of the

patients who did not eat suffered 2 hypotension events. They also reported that hypotension occurred 45 minutes after eating (14).

The general clinical approach to maintaining hemodynamic stability during dialysis as well as concerns about IDH have resulted in the suggestion that antihypertensive medications should not be given before dialysis sessions. However, restricting the use of antihypertensive drugs may cause various significant problems. Firstly, it can lead to the development of intradialytic hypertension (HT) in these susceptible patients. Secondly, this approach will cause inability to treat possible hypertensive episodes between dialysis sessions in hemodialysis patients (15). Thirdly, when rate-control drugs (such as beta-blockers) are not given, life-threatening arrhythmias may develop in some patients (16).

In the selection of antihypertensive agents, basic risk factors and comorbidities of the patient are often considered. ACE inhibitors / ARBs or beta blockers are generally the first agents used to treat HT in dialysis patients. In addition to the positive blood pressure-lowering effects, ACE inhibitors / ARBs contribute to cardiac remodeling and positively influence arteriosclerosis in dialysis patients (17, 18). Additionally, beta-blockers have been shown to reduce the risk of cardiac death in dialysis patients (19). There is evidence that beta blockers are preferable for vasodilation. For instance, Carvedilol is known to improve endothelial function. Thus, it may have additional benefit in reducing the frequency of intradialytic hypertension (20). The dosage chart of medications should be specific for each patient according to the cardiovascular profile. Generally, dialyzable long-acting drugs (such as lisinopril, atenolol) are preferred in patients with IDH and are given on post-dialysis days up to three times a week (to improve patient compliance) or before bedtime (for improvement of overnight results) (21).

Despite all this, in a recent review by Tara I.C., it was explained in detail that, in theory, antihypertensive drugs can both reduce or increase IDH incidence. Moreover, currently there is no prospective-study data supporting the use or avoidance of antihypertensive treatment for the purpose of preventing IDH development (1). However, in another review article written by Patrick B.R and Finnian R.M.C., it was found that the use of antihypertensive drugs impaired the sympathetic nervous system, renin-angiotensin system and vasopressin response; hence reducing cardiac output and preventing the DeJager-Kroger phenomenon (reduction of blood volume in the venous system and enhancement of venous return through arteriolar vasoconstriction which is obtained by activation of the sympathetic nervous system and vasoactive hormones when hypovolemia is present). This reflex is especially seen in the splanchnic area and thus may be associated with IDH development (22).

In our study, we found that antihypertensive use (i.e., the presence of HT) was an independent risk factor for IDH.

A similar finding was also reported recently by Rocha et al. (where HT was reported to be an independent risk factor for IDH) (23).

In a study conducted by Finnian et al. 61% of the 3142 participants were diabetic; it was found that low levels of pre-dialysis sodium, high levels of serum urea nitrogen and serum glucose were associated with further decrease in SBP during dialysis. According to the study, high osmolality at pre-dialysis was related to primary elevated urea and glucose levels, and these were in turn associated with the decrease in intradialytic SBP and IDH (5). In the current study, calculated osmolality was found to be decreased in correlation with the blood glucose and urea decrease during dialysis, but there was no significant change in the measured osmolality. In contrast to previous studies, neither measured or calculated serum osmolality were found to be related to IDH. The most important limitation of our study was the low number of patients included.

CONCLUSION

The findings of our study have shown that serum magnesium levels may be important in the development of IDH, which we believe warrants prospective studies on this subject. Secondly, the development of PPH is a problem for our patients and a very easy practice to avoid this problem, not eating before HD sessions, is being ignored by patients. Our third conclusion concerns the HT in such patients—a problem which has not been fully elucidated so far. The most important cause of HT in patients undergoing HD is hypervolemia and achieving adequate dry weight in patients with IDH remains as an important problem. In patients with both IDH and HT, frequent and long HD sessions may provide better chances to obtain appropriate dry weight and may prevent HT episodes (thus also preventing antihypertensive use) which could contribute to the prevention of IDH.

DECLERATIONS

Ethics Committee Approval: All procedures performed in studies involving human participants were in accordance with the ethical standards of institutional and with the 1975 Helsinki declaration and its later amendments or comparable ethical standards. The study was approved by the local ethics committee (IRB approval number: 2015/390).

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REFERENCES

- Chang TI. Impact of drugs on intradialytic hypotension: Antihypertensives and vasoconstrictors. *Semin Dial.* 2017;30(6):532-536. doi:10.1111/sdi.12633
- Shoji T, Tsubakihara Y, Fujii M, Imai E. Hemodialysis-associated hypotension as an independent risk factor for two-year mortality in hemodialysis patients. *Kidney Int.* 2004;66(3):1212-1220. doi:10.1111/j.1523-1755.2004.00812.x
- Flythe JE, Xue H, Lynch KE, Curhan GC, Brunelli SM. Association of mortality risk with various definitions of intradialytic hypotension. *J Am Soc Nephrol.* 2015;26(3):724-734. doi:10.1681/ASN.2014020222
- Van Buren PN, Inrig JK. Special situations: Intradialytic hypertension/chronic hypertension and intradialytic hypotension. *Semin Dial.* 2017;30(6):545-552. doi:10.1111/sdi.12631
- Mc Causland FR, Waikar SS. Association of Predialysis Calculated Plasma Osmolality With Intradialytic Blood Pressure Decline. *Am J Kidney Dis.* 2015;66(3):499-506. doi:10.1053/j.ajkd.2015.03.028
- K/DOQI Workgroup. K/DOQI clinical practice guidelines for cardiovascular disease in dialysis patients. *Am J Kidney Dis.* 2005;45(4 Suppl 3):S1-S153.
- van der Sande FM, Dekker MJ, Leunissen KML, Kooman JP. Novel Insights into the Pathogenesis and Prevention of Intradialytic Hypotension. *Blood Purif.* 2018;45(1-3):230-235. doi:10.1159/000485160
- Kyriazis J, Kalogeropoulou K, Bilirakis L, et al. Dialysate magnesium level and blood pressure. *Kidney Int.* 2004;66(3):1221-1231. doi:10.1111/j.1523-1755.2004.00875.x
- Pakfetrat M, Roozbeh Shahroodi J, Malekmakan L, Zare N, Hashemi Nasab M, Hossein Nikoo M. Is there an association between intradialytic hypotension and serum magnesium changes?. *Hemodial Int.* 2010;14(4):492-497. doi:10.1111/j.1542-4758.2010.00477.x
- Elsharkawy MM, Youssef AM, Zayoon MY. Intradialytic changes of serum magnesium and their relation to hypotensive episodes in hemodialysis patients on different dialysates. *Hemodial Int.* 2006;10 Suppl 2:S16-S23. doi:10.1111/j.1542-4758.2006.00120.x
- Jansen RW, Lipsitz LA. Postprandial hypotension: epidemiology, pathophysiology, and clinical management. *Ann Intern Med.* 1995;122(4):286-295. doi:10.7326/0003-4819-122-4-199502150-00009
- Ong AC, Myint PK, Potter JF. Pharmacological treatment of postprandial reductions in blood pressure: a systematic review. *J Am Geriatr Soc.* 2014;62(4):649-661. doi:10.1111/jgs.12728
- Daugirdas JT. Pathophysiology of dialysis hypotension: an update. *Am J Kidney Dis.* 2001;38(4 Suppl 4):S11-S17. doi:10.1053/ajkd.2001.28090
- Sherman RA, Torres F, Cody RP. Postprandial blood pressure changes during hemodialysis. *Am J Kidney Dis.* 1988;12(1):37-39. doi:10.1016/s0272-6386(88)80069-6
- Van Buren PN, Kim C, Toto R, Inrig JK. Intradialytic hypertension and the association with interdialytic ambulatory blood pressure. *Clin J Am Soc Nephrol.* 2011;6(7):1684-1691. doi:10.2215/CJN.11041210
- Takeda A, Toda T, Fujii T, Sasaki S, Matsui N. Can predialysis hypertension prevent intradialytic hypotension in hemodialysis patients?. *Nephron Clin Pract.* 2006;103(4):c137-c143. doi:10.1159/000092910
- London GM, Pannier B, Guerin AP, Marchais SJ, Safar ME, Cuche JL. Cardiac hypertrophy, aortic compliance, peripheral resistance, and wave reflection in end-stage renal disease. Comparative effects of ACE inhibition and calcium channel blockade. *Circulation.* 1994;90(6):2786-2796. doi:10.1161/01.cir.90.6.2786
- Ichihara A, Hayashi M, Kaneshiro Y, et al. Low doses of losartan and trandolapril improve arterial stiffness in hemodialysis patients. *Am J Kidney Dis.* 2005;45(5):866-874. doi:10.1053/j.ajkd.2005.02.022
- Abbott KC, Trespalacios FC, Agodoa LY, Taylor AJ, Bakris GL. beta-Blocker use in long-term dialysis patients: association with hospitalized heart failure and mortality. *Arch Intern Med.* 2004;164(22):2465-2471. doi:10.1001/archinte.164.22.2465
- Inrig JK, Van Buren P, Kim C, Vongpatanasin W, Povsic TJ, Toto R. Probing the mechanisms of intradialytic hypertension: a pilot study targeting endothelial cell dysfunction. *Clin J Am Soc Nephrol.* 2012;7(8):1300-1309. doi:10.2215/CJN.10010911
- Agarwal R, Sinha AD, Pappas MK, Abraham TN, Tegegne GG. Hypertension in hemodialysis patients treated with atenolol or lisinopril: a randomized controlled trial. *Nephrol Dial Transplant.* 2014;29(3):672-681. doi:10.1093/ndt/gft515
- Reeves PB, Mc Causland FR. Mechanisms, Clinical Implications, and Treatment of Intradialytic Hypotension. *Clin J Am Soc Nephrol.* 2018;13(8):1297-1303. doi:10.2215/CJN.12141017
- Rocha A, Sousa C, Teles P, Coelho A, Xavier E. Frequency of intradialytic hypotensive episodes: old problem, new insights. *J Am Soc Hypertens.* 2015;9(10):763-768. doi:10.1016/j.jash.2015.07.007