

Association between intradialytic blood pressure and left ventricular mass index in end-stage renal disease

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ABSTRACT

Aim To evaluate whether single and two-month average intradialytic blood pressure (BP) measurements, including systolic blood pressure (SBP) and diastolic blood pressure (DBP), are associated with left ventricular mass index (LVMI), and whether intradialytic BP thresholds discriminate left ventricular hypertrophy (LVH) in patients with end-stage renal disease on haemodialysis.

Methods This prospective observational study was conducted at the Dialysis Centre in Doboj between June and September 2017 and included 97 adults on chronic haemodialysis for at least five months. Intradialytic BP was assessed as a single measurement and as a two-month average. LVMI and LVH were evaluated by echocardiography. Multivariable regression examined associations with LVMI after adjustment for age, haemoglobin, and volume overload, while receiver operating characteristic (ROC) curve analysis assessed LVH discrimination.

Results Median dialysis duration was 3.9 years, and 24 patients (25%) had ischemic heart disease. Median LVMI was 117.5 g/m², and LVH was present in 44 patients (45%). After adjustment for relevant covariates, single intradialytic SBP and DBP measurements were not associated with LVMI. In contrast, two-month average SBP ($B = 0.5364$, $p = 0.0075$) and DBP ($B = 0.7308$, $p = 0.0299$) remained independently associated with LVMI. Two-month average SBP showed moderate LVH discrimination (area under the curve (AUC) 0.711; $p = 0.0001$), with an exploratory cut-off of >143 mmHg.

Conclusion Two-month average intradialytic SBP was independently associated with higher LVMI and showed moderate discrimination for LVH but requires external validation.

Keywords: echocardiography, hypertension, hypertrophy, left ventricular, renal dialysis

INTRODUCTION

Cardiovascular disease remains the predominant cause of mortality in patients with end-stage renal disease (ESRD) undergoing haemodialysis, with left ventricular hypertrophy (LVH) being one of the most common cardiac abnormalities in this population (1).

The prevalence of LVH in haemodialysis patients exceeds 70%, driven by chronic pressure and volume overload, arterial stiffness, and anaemia, and is independently associated with increased cardiovascular morbidity and all-cause mortality in ESRD (2).

Blood pressure (BP) in the general haemodialysis popula-

tion typically follows a characteristic pattern, marked by a sharp decline during the dialysis session and a progressive rise throughout the interdialytic period (3,4).

In about 5–15% of haemodialysis patients, BP shows a paradoxical increase during or immediately after the dialysis session, a condition referred to as intradialytic hypertension (5).

It is unknown how elevated intradialytic BP leads to adverse cardiovascular events. Pathophysiological factors contributing to intradialytic hypertension—including extracellular volume overload, heightened sympathetic nervous system activity, endothelial dysfunction, sodium gain from the dialysate, and the removal of antihypertensive medications during haemodialysis—may play a role in elevating cardiovascular risk in affected patients (6).

Pre-dialysis BP has limited value in predicting LVH. BP measurements taken in a standardized manner or as cumulative values during dialysis sessions show a stronger association with LVH. However, there is still no agreement on the most accurate

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BP threshold, timing, or measurement method—whether standardized, home-based, or ambulatory—for assessing the BP burden that best predicts the development or progression of LVH (7,8).

This study aimed to assess whether single and two-month average intradialytic systolic blood pressure and diastolic blood pressure (SBP, DBP) are associated with left ventricular mass index (LVMI), and whether intradialytic BP thresholds discriminate LVH in patients receiving chronic haemodialysis.

MATERIAL AND METHODS

Patients and study design

A prospective observational study was conducted among patients enrolled in the chronic haemodialysis program at the Dialysis Centre in Doboj between June and September 2017.

Patients were eligible for inclusion if they had been receiving haemodialysis for at least five months. This period included a minimum of three months for clinical stabilization after initiation of chronic dialysis, followed by an additional two months required for average BP monitoring.

Exclusion criteria were as follows: presence of atrial fibrillation (due to unreliable BP measurement), use of a central venous catheter for dialysis, liver failure, uncontrolled diabetes mellitus, pregnancy, concurrent medications affecting the autonomic nervous system, secondary hypertension, and incomplete medical data.

A total of 123 patients were screened, of whom 97 were included in the final analysis. The remaining 26 patients were excluded due to dialysis duration shorter than five months, atrial fibrillation, catheter use, or missing data. A minimum sample size of 84 participants was estimated to achieve 80% power at $\alpha = 0.05$ for detecting a moderate association between BP and LVMI.

Methods

BP values were measured using the standard auscultatory indirect method according to the Korotkov method, with a calibrated standard aneroid sphygmomanometer. BP measurements were performed by the same trained dialysis personnel according to a standardized protocol to minimize inter-observer variability. Whenever feasible, blood pressure was measured on the arm without vascular access, and the same arm was used consistently for repeated measurements in each patient. The measurements were performed during dialysis sessions scheduled on Mondays, Wednesdays, and Fridays, over the final two months of the study period, to determine the average BP. The single BP refers to the mid-week intradialytic measurement (second hour of dialysis), while the two-month average BP represents the average of all intradialytic measurements over the entire two-month period. Participants spent five minutes in a seated position before the measurement, with the arm supported at heart level. BP values are expressed in mmHg.

Volume overload was assessed clinically using daily pre-dialysis body weight relative to the prescribed dry weight, supported by routine clinical examination findings such as peripheral oedema and lung auscultation. The prescribed dry weight was periodically reassessed during the observation period based on routine clinical evaluation. Volume overload was expressed as a percentage of dry weight using the following formula: $((\text{pre-dialysis weight} - \text{dry weight}) / \text{dry weight}) \times 100$. The two-month average volume overload (%) was calculated as the mean

of all available pre-dialysis assessments during the two-month period and was entered into regression analyses as a continuous confounder because baseline fluid excess may contribute to intradialytic BP patterns and left ventricular remodelling.

Transthoracic echocardiography was performed at a standardized post-dialysis time point, approximately one hour after completion of the haemodialysis session. We standardized echo post-dialysis to reduce variability; however, residual acute effects of ultrafiltration cannot be fully excluded. Examinations were conducted twice monthly during the study period, and values were averaged for analysis. Echocardiographic measurements used for analysis were averaged from examinations performed during the same two-month observation period used for calculation of the average intradialytic BP. A General Electric LOGIQ C5 Premium ultrasound machine (General Electric, Boston, MA) equipped with an M3S 1.5-4 MHz phased array transducer was used. The assessments included B-mode imaging, colour Doppler, and pulsed Doppler techniques. Participants were positioned in the left lateral decubitus position to optimize image quality. Standard echocardiographic views were obtained, including parasternal long-axis, parasternal short-axis, apical four-chamber, and five-chamber views. The patients were clinically and biochemically stable while on their haemodialysis program and had normal sinus rhythm on electrocardiography. Left ventricular mass was calculated using the Devereux-modified American Society of Echocardiography (ASE) formula from linear measurements (IVSd, LVIDd, and LVPWd). LVMI was obtained by indexing LVM to body surface area. LVH was defined as LVMI $> 95 \text{ g/m}^2$ (women) and $> 115 \text{ g/m}^2$ (men) (9).

All measurements were performed by a board-certified cardiologist and analysed using EchoPac PC software (version 7.0.1), in accordance with the guidelines of the European and American Societies of Echocardiography. For each examination, parameters were measured over consecutive cardiac cycles and averaged, and for each participant, the final values represented the mean of all echocardiographic examinations performed during the study period. For each participant, basic demographic data were collected, a detailed medical history was obtained, medical records were reviewed, and a comprehensive clinical examination was performed.

Statistical analysis

The results were processed using standard statistical methods in SPSS Statistics version 21.0 (IBM Corp., Armonk, NY). Continuous variables were summarized as mean $\pm$ standard deviation (SD) when normally distributed, and as median with interquartile range (IQR) when non-normally distributed. Normality was assessed using the Shapiro–Wilk test and visual inspection of histograms. Both descriptive and analytical statistical methods were applied. Regression analyses were used to assess associations between BP measures and LVMI, with LVMI examined as a continuous outcome. Multivariable models were constructed separately for each BP parameter and adjusted for age, haemoglobin, and two-month average volume overload. Receiver operating characteristic (ROC) curve analysis was used to evaluate the ability of BP measures to discriminate between patients with and without LVH. Cut-off values were derived internally and require external validation. The exploratory cut-off was selected using the Youden index (sensitivity + specificity – 1). Sensitivity, specificity, and the area under the receiver operating characteristic curve (AUC) were calculated.

Table 1. Baseline characteristics and intradialytic blood pressure of included patients

Variable	Value (N=97)
Baseline characteristics	
Ischemic heart disease, N (%)	24 (25%)
Left ventricular hypertrophy, N (%)	44 (45%)
Duration of dialysis treatment (years), median (min–max)	3.9 (0.5–6.2)
Haemoglobin (g/L), median (IQR)	115 (108–120)
Two-month average haemoglobin (g/L), median (IQR)	114.5 (108–120)
Two-month average volume overload (%), mean $\pm$ SD	11 $\pm$ 6
Overall LVMI (g/m ²), median (IQR)	117.5 (98.7–142.9)
LVMI in males (g/m ²), median (IQR)	122.9 (100.1–148.2)
LVMI in females (g/m ²), median (IQR)	112.2 (96.6–130.9)
Intradialytic blood pressures	
Single systolic (mmHg), median (IQR)	136 (124–156)
Single diastolic (mmHg), median (IQR)	69 (62–80)
Two-month average systolic (mmHg), median (IQR)	140 (127–153)
Two-month average diastolic (mmHg), median (IQR)	73 (64–81)

SD, standard deviation; IQR, interquartile range; LVMI, left ventricle mass index.

Table 2. Multiple regression models predicting LVMI based on systolic and diastolic blood pressure parameters, adjusted for age, anaemia, and volume overload

Model	Independent variable	B coefficient	Std. error	Partial <i>r</i>	<i>t</i>	<i>p</i>
Model 1: Single intradialytic SBP	Single intradialytic SBP	0.2805	0.1763	0.2261	1.592	0.1182
	Age	0.3898	0.3173	0.1764	1.229	0.2253
	Single haemoglobin	–0.3032	0.3207	–0.1366	–0.946	0.3491
	Volume overload	1.0755	0.6017	0.2523	1.788	0.0803
Model 2: Two-month average intradialytic SBP	Two-month average intradialytic SBP	0.5364	0.1954	0.2951	2.745	0.0075
	Age	–0.2052	0.2888	–0.0797	–0.710	0.4796
	Two-month average haemoglobin	–0.4760	0.3093	–0.1706	–1.539	0.1277
	Volume overload	1.0231	0.5290	0.2126	1.934	0.0567
Model 3: Single intradialytic DBP	Single intradialytic DBP	0.4719	0.3430	0.1968	1.374	0.1754
	Age	0.6457	0.3426	0.2651	1.884	0.0657
	Single haemoglobin	–0.3355	0.3202	–0.1511	–1.048	0.3000
	Volume overload	1.1141	0.6042	0.2597	1.844	0.0715
Model 4: Two-month average intradialytic DBP	Two-month average intradialytic DBP	0.7308	0.3306	0.2414	2.211	0.0299*
	Age	0.1778	0.3244	0.0616	0.548	0.5851
	Two-month average haemoglobin	–0.6077	0.3038	–0.2196	–2.000	0.0489*
	Volume overload	1.2174	0.5418	0.2451	2.247	0.0274*

SBP, systolic blood pressure; DBP, diastolic blood pressure; LVMI, left ventricular mass index.

RESULTS

The cohort included 97 adults receiving chronic haemodialysis, with a median dialysis duration of 3.9 years (range 0.5–6.2 years). Ischemic heart disease was recorded in 24 patients (25%). Haemoglobin levels were 115 g/L (IQR 108–120) during the month of echocardiography and 114.5 g/L (IQR 108–120) when averaged across two months. The mean two-month volume overload was 11% $\pm$ 6%. The median LVMI was 117.5 g/m² (IQR 98.7–142.9), measuring 122.9 g/m² (IQR 100.1–148.2) in men and 112.2 g/m² (IQR 96.6–130.9) in women. LVH was identified in 44 of 97 patients (45%) (Table 1).

Mid-week intradialytic BP measurements showed a single-session SBP of 136 mmHg (124–156) and DBP of 69 mmHg (62–80). When BP was summarized as a two-month intradialytic average, SBP was 140 mmHg (127–153), and DBP was 73 mmHg (64–81) (Table 1).

In multivariable linear regression adjusted for age, haemoglobin, and volume overload, single intradialytic SBP was not associated with LVMI (B = 0.2805; *p* = 0.1182), while

the two-month average intradialytic SBP remained associated with LVMI (B = 0.5364; *p* = 0.0075); volume overload showed a borderline association in this SBP model (*p* = 0.0567). Single intradialytic DBP was not associated with LVMI (B = 0.4719; *p* = 0.1754), with borderline associations observed for age (*p* = 0.0657) and volume overload (*p* = 0.0715). In the model using two-month average intradialytic DBP, DBP was associated with LVMI (B = 0.7308; *p* = 0.0299), and haemoglobin (B = –0.6077; *p* = 0.0489) and volume overload (B = 1.2174; *p* = 0.0274) were also independently associated with LVMI (Table 2).

The two-month average intradialytic SBP showed moderate discrimination for LVH, with an AUC of 0.711 (95% CI 0.609–0.799; *p* = 0.0001). An exploratory cut-off of >143 mmHg, derived within this cohort, corresponded to 63.6% sensitivity and 73.1% specificity, with a positive likelihood ratio of 2.36 and a negative likelihood ratio of 0.50 (Figure 1). Two-month average intradialytic DBP showed limited discrimination for LVH (AUC 0.587; *p* = 0.140).

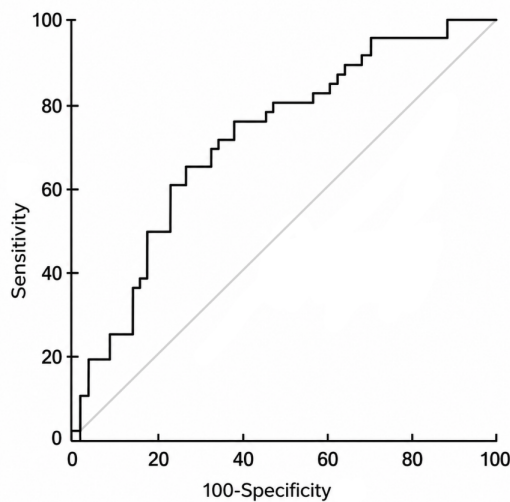


Figure 1. ROC curve for two-month average intradialytic systolic blood pressure predicting left ventricular hypertrophy

DISCUSSION

The principal finding of this study is that the two-month average intradialytic SBP was more strongly associated with LVMI than a single intradialytic SBP measurement in patients receiving chronic haemodialysis. In addition, the averaged intradialytic SBP showed a moderate ability to identify patients with LVH, suggesting that repeated intradialytic BP assessment may provide more clinically relevant information than an isolated measurement. These findings support the importance of longitudinal BP monitoring during haemodialysis when evaluating cardiovascular risk in this population.

This approach is consistent with the National Kidney Foundation Kidney Disease Outcomes Quality Initiative (KDOQI) guidelines (10), which emphasize systematic BP assessment and individualized BP management in haemodialysis rather than relying on single measurements. In this context, our ROC-derived threshold (two-month average SBP >143 mmHg) may be viewed as an exploratory risk stratification signal to identify patients who may benefit from closer cardiovascular evaluation and optimization of volume and antihypertensive therapy. Regarding DBP, the results should be interpreted more cautiously: two-month average DBP was associated with LVMI, but it showed limited ability to discriminate LVH, suggesting DBP is supportive information rather than a primary screening metric.

Prior studies have linked peridialytic BP behaviour and intradialytic hypertension with adverse outcomes, supporting the clinical importance of BP patterns during dialysis (11–13). BP behaviour during dialysis is influenced by various patient-related factors, including comorbidities, autonomic dysfunction, vascular stiffness, impaired vasoreactivity, use of antihypertensive drugs, procedural dialysis factors, and dialysate sodium and calcium concentrations (14).

On the other end of the spectrum, patients with high pre-dialysis SBP may suffer from chronic fluid overload and vascular stiffness. In these patients, further intradialytic pressure increases may reflect poor fluid removal. Recent studies using bioimpedance analysis have shown that patients with intradialytic pressure increases have fluid overload and higher extracellular-to-total body water ratios (15,16).

A growing body of evidence suggests that intradialytic SBP

increases may result from elevated peripheral vascular resistance (PVR). Analysing hemodynamic and biochemical parameters, Chou et al. found inappropriate PVR increases in 30 patients prone to intradialytic hypertension, likely due to altered nitric oxide/endothelin-1 balance and intradialytic hypoxemia, independent of sympathetic stimulation or renin activation (17).

Based on this and earlier work by Raj et al., the pathophysiology of intradialytic BP elevation may be linked to underlying endothelial dysfunction—a precursor of vascular injury and a contributor to increased cardiovascular event risk (18). Endothelial dysfunction may thus offer a mechanistic explanation for the link between intradialytic BP rise and increased mortality.

Among possible mechanisms, extracellular volume overload is often cited as a primary contributor. Bioimpedance spectroscopy has shown that patients with intradialytic hypertension have greater volume overload, and aggressive reduction of dry weight can alter intradialytic BP trajectories (19,20).

Interestingly, Shamir et al. found that patients with intradialytic hypertension had lower pre-dialysis SBP and weight gain compared to others, possibly misleading clinicians into underestimating fluid removal needs—contributing to volume overload (6). Recurrent intradialytic hypertension patients were shown to have higher extracellular volume post-dialysis, as measured by bioimpedance. In a study of more than 500 patients, extracellular water overload (L) post-dialysis was significantly higher in those with intradialytic pressure increases versus those with stable or decreasing pressures (21).

Additionally, inappropriate renin-angiotensin system or sympathetic nervous system activation, or endothelial responses to small intravascular volume reductions during ultrafiltration, may also contribute to BP increases, seen as higher total intradialytic peripheral resistance (22). Antihypertensive medications may also play a role. Some water-soluble agents are removed during dialysis. Renin-angiotensin-aldosterone system activation might worsen BP, while agents like carvedilol may improve endothelial function and reduce risk. Conversely, calcium channel blockers may cause peripheral vasodilation, increasing volume overload and intradialytic hypertension.

Clinical features associated with intradialytic pressure increases include older age, lower body weight, lower serum creatinine and albumin, and more frequent use of antihypertensive medications (11). Lower pre-dialysis urea and albumin levels may reduce osmolarity changes, preventing BP drops. Intradialytic hypertension patients were older, with lower haemoglobin, malnutrition, lower intact parathyroid hormone (iPTH) levels, and higher high-sensitivity C-reactive protein levels, suggesting development of the malnutrition-inflammation-atherosclerosis syndrome. Low iPTH has previously been linked to inflammation and oxidative stress (23). The exact mechanism by which intradialytic BP increases leads to adverse cardiovascular events is unclear. Proposed mechanisms include volume overload, sympathetic overactivity, endothelial dysfunction, sodium loading, and dialyzability of antihypertensives (24).

These may contribute to left ventricular mass increase—a known cardiovascular risk factor (25).

This study has several limitations. Echocardiographic assessments were performed post-dialysis, potentially influenced by acute volume shifts. Because the ROC-derived thresholds were generated and evaluated within the same cohort, these cut-offs may be optimistic and require external validation in independent populations. Volume status was assessed using prescribed dry weight and routine clinical evaluation rather than objec-

tive methods such as inferior vena cava diameter/collapsibility or bioimpedance. Echocardiographic assessment was limited to LVMI/LVH and did not include other potentially relevant parameters, including ejection fraction, chamber dimensions, diastolic function, left atrial size, or valvular disease. Finally, the multivariable models were restricted to a limited number of clinically available confounders, so factors such as dialysis duration, comorbidities, and antihypertensive therapy were not included, and residual confounding cannot be excluded.

CONCLUSION

A higher two-month average intradialytic SBP was independently associated with higher LVMI in patients on chronic haemodialysis. The exploratory cut-off of >143 mmHg showed moderate discrimination for LVH, but this cut-off requires validation in larger independent cohorts before clinical use.

FUNDING

No specific funding was received for this study.

CONFLICTS OF INTEREST

None to declare.

ETHICS STATEMENT

The study was conducted in accordance with the Declaration of Helsinki and principles of good scientific practice. Written informed consent was obtained from all participants for the use of their medical data in this study. All participants had also previously provided written consent for haemodialysis treatment and related diagnostic procedures. The research protocol was approved by the Ethics Committee of the Faculty of Medicine, University of Banja Luka (Approval No. 18/4.22/17).

DATA AVAILABILITY STATEMENT

The datasets generated and analysed during the current study are available from the corresponding author on reasonable request.

AUTHOR CONTRIBUTIONS (CRediT)

Conceptualization: A.M., S.T.; **Data curation:** A.M., A.C., D.B.; **Formal analysis:** A.M., A.C., S.T., D.B.; **Investigation:** A.M., S.T.; **Methodology:** A.M., S.T.; **Project administration:** A.M., S.T.; **Resources:** A.M.; **Software:** A.C., D.B.; **Supervision:** A.C., S.T.; **Validation:** A.M., S.T.; **Visualization:** A.C., D.B.; **Writing – original draft:** A.M., D.B.; **Writing – review & editing:** A.C., D.B. All authors have read and agreed to the published version of the manuscript.

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