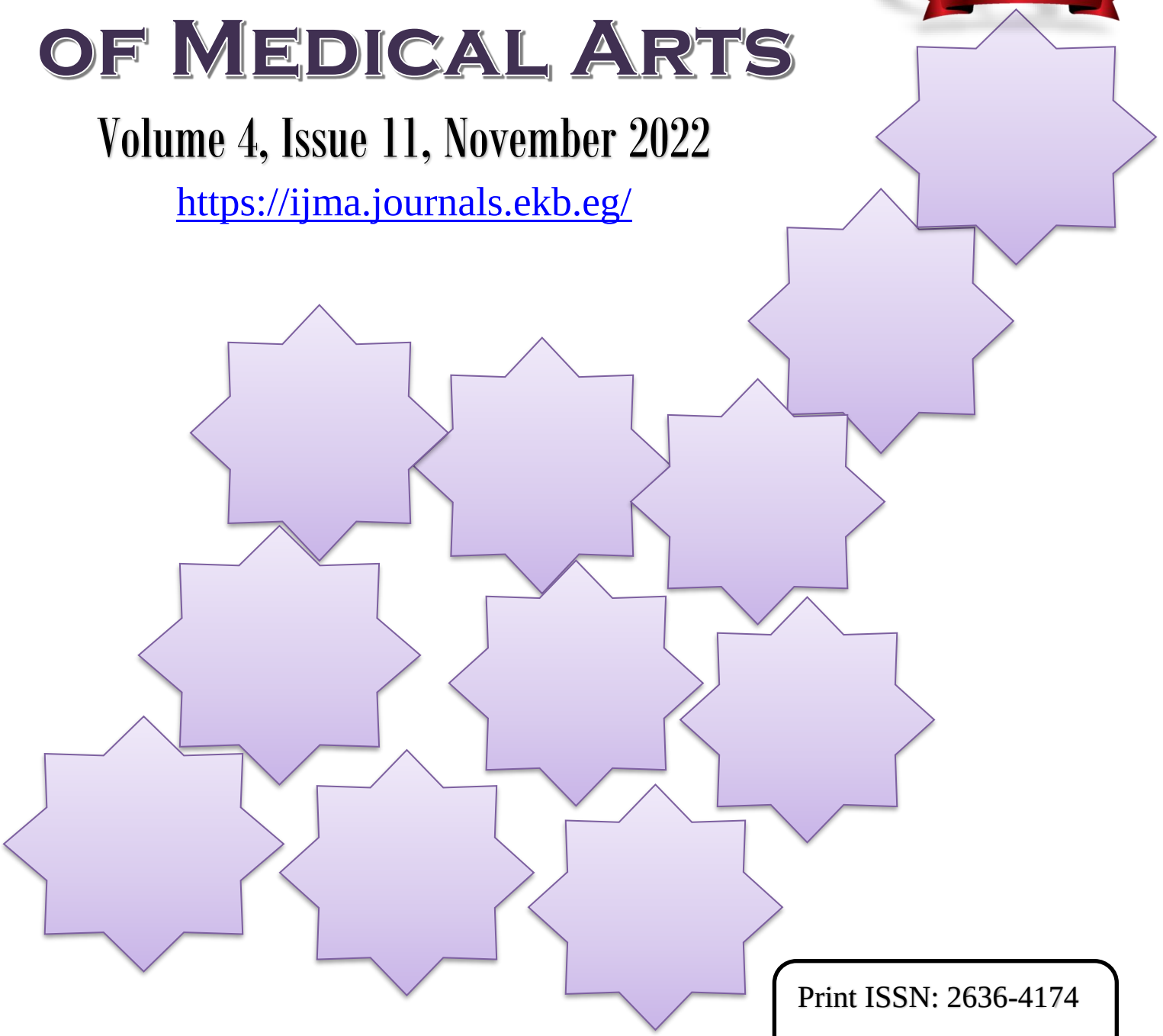
International Journal of Medical Arts

Volume 4, Issue 11, November 2022

https://ijma.journals.ekb.eg/



Print ISSN: 2636-4174

Online ISSN: 2682-3780

Available online at Journal Website
https://ijma.journals.ekb.eg/
Main Subject [Internal Medicine]

Original Article

Echocardiographic Evaluation of Cardiac Dysfunction in Hemodialysis Patients with or without Intradialytic Hypertension

Mohamed Ahmed Siam *[1], Emad Mohamed Allam [2], Ahmed Salama Al-Adl [1], Tamer Mohamed Abd Elkader [1], Marwa Ahmed Byomy [3]

[1] Department of Internal Medicine, Damietta Faculty of Medicine, Al-Azhar University, Damietta, Egypt
[2] Department of Internal Medicine, Faculty of Medicine, Al-Azhar University, Cairo, Egypt
[3] Department of Clinical Pathology, Faculty of Medicine, Al-Azhar University, Cairo, Egypt

Article information

Received: 27-11-2022

Accepted: 25-02-2023

DOI: 10.21608/IJMA.2023.177195.1562

*Corresponding author

Email: mohamedsiam24.msr@gmail.com

Citation: Siam MA, Allam EM, Al-Adl AS, Abd Elkader TM, Byomy MA. Echocardiographic Evaluation of Cardiac Dysfunction in Hemodialysis Patients with or without Intradialytic Hypertension. IJMA 2022 November; 4 [11]: 2807-2812. doi: 10.21608/IJMA. 2023.177195.1562.

ABSTRACT

Background: Intradialytic hypertension [IDH] is an increase in systolic blood pressure [SBP] of more than 10 mm Hg over pre-dialysis SBP, which is a serious consequence of hemodialysis [HD]. Evaluation of IDH-related cardiac dysfunction in HD patients, such as left ventricular hypertrophy [LVH] or pulmonary hypertension [PHTN], may be performed using an echocardiogram, a non-invasive technique.

Objective: This study aimed to evaluate the characteristics of IDH in patients undergoing maintenance HD and the echocardiographic findings in patients with IDH.

Patients and methods: A case-control study was conducted for seven months on 60 patients with end-stage renal disease in Al-Azhar University Hospital, Egypt. The study included adult patients on regular dialysis for more than three months and excluded patients with unstable health conditions. Data were collected through a full history, clinical examination, laboratory tests, and echocardiographic assessments and analyzed using statistical methods such as T-test, ANOVA, and the Chi-square test.

Results: The study showed a statistically significant reduction in left ventricular end-systolic diameter [LVESD], left ventricular end-diastolic diameter [LVEDD], left atrial volume index [LAVI], and pulmonary artery systolic pressure [PASP] in patients with IDH [$p<0.05$]. The reduction in LVESD was from 43.67 ± 3.26 to 42.17 ± 3.03 mm, in LVEDD from 59.6 ± 2.04 to 58.07 ± 3.93 mm, in LAV from 34.67± 2.3 to 31.9 ± 2.04 mm^3, and in PASP from 42.33 ± 6.7 to 40.93 ± 6.84 mmHg.

Conclusion: Poorly managed BP in HD patients is linked to volume overload, increasing the risk of diastolic dysfunction and subsequent heart failure. HD patients with IDH had substantial reductions in LVEDD, LVESD, and PASP after HD sessions.

Keywords: Echocardiographic changes; LVH; Hemodialysis; IDH

INTRODUCTION

To lessen the signs and symptoms of uremia, hemodialysis [HD], an extracorporeal treatment, is recommended. When kidney function is no longer adequate to preserve an individual's well-being or life, HD partly replaces some essential renal functions [1]. The elimination of fluids and salt during HD is known to reduce blood pressure [BP] in most hypertensive patients; however, in a small proportion of individuals, BP paradoxically increases towards the end of HD [2,3]. Clinically, this condition is known as intradialytic hypertension [IDH], and it manifests as an increase in systolic blood pressure [SBP] of more than 10 mm Hg over the pre-dialysis SBP [4]. Patients with IDH can be asymptomatic; however, in some patients, convulsion, anxiety, palpitations, dyspnea, thoracic discomfort, profuse perspiration, and headaches may present [5]. Moreover, patients on hemodialysis may experience muscle cramps, vomiting, nausea, irritable legs, fatigue, syncopal attack, and hypotension, which may lead to discontinuation of the hemodialysis session [6,7].

Patients undergoing HD are more likely to have poorly managed BP because of volume overload. Previous studies on the cause of IDH have pointed to volume overload as a contributing cause [6,7]. High-risk patients for the development of cardiovascular diseases include those who have had IDH more than once. Even if patients do not look clinically volume overloaded, their dry body weight should be reevaluated [8].

Patients on HD have a 9-fold increased risk of cardiovascular mortality compared to the general population. It has been shown that right ventricular [RV] systolic dysfunction is an independent risk factor for mortality, and structural and functional heart disorders are frequent in the HD population [9]. IDH is linked to substantial alterations in echocardiography [ECHO] characteristics and could be considered one of the leading causes of morbidity and mortality in individuals with cardiovascular disorders.

AIM OF THE WORK

This study aimed to assess the characteristics of IDH in patients undergoing maintenance HD, in addition to their echocardiographic findings.

PATIENTS AND METHODS

This case-control study was conducted over a period of seven months, from December 2021 to June 2022, on 60 patients with end-stage renal disease [ESRD] who were admitted to the Nephrology Unit, Al-Azhar University Hospital, New Damietta, Egypt. Patients were classified into two groups: Group I: 30 patients with IDH [cases], and Group II: 30 patients with non-IDH [controls].

Ethical Consideration: The local ethical committee of Al-Azhar university approved the study. All included patients provided their written informed consent.

Inclusion criteria: We included adult patients [>18 years] on regular HD therapy for more than three months and treated with acetate bicarbonate-buffered dialysate, arteriovenous fistula, and arteriovenous graft as HD access.

Exclusion criteria: Patients were excluded if they had unstable hemodynamics, un-controlled hypertension, decompensated liver disease, decompensated heart failure, patients with an expected survival time of less than three months, or patients with a history of trauma, surgery, or severe infection.

Data collection: At presentation, all patients were subjected to full history taking [age, sex, duration of dialysis, ultrafiltration volume, cause of ESRD, and smoking], and clinical examination, including clinical assessment of dry body weight by neck vein examination, edema, ascites, chest examination, cardiac examination, and weighing scale. Moreover, pulse rate and rhythm were evaluated. BP was assessed before, during, and after HD.

The non-fistula side of the upper arm was measured for BP using a digital BP monitor 30 minutes before the start of HD and every hour throughout the HD session. Pre-dialytic BP [pre-HD BP] was taken 30 minutes before HD started and 120 minutes after beginning HD, whereas post-dialytic BP [post-HD BP] was measured immediately after the end of dialysis. Accordingly, the peri-dialytic change in BP will be calculated as follows: Peridialytic BP change = post-HD BP – pre-HD BP.

The post-HD BPs, post-HD BPs and peri-dialytic SBP changes for each patient were documented throughout a 3-month observation

period. Patients with persistent IDH were those who experienced an average peri-dialytic SBP rise of more than 10 mmHg during the course of the whole three-month monitoring period.

Investigations: Regarding the laboratory investigations, all the following tests were performed for all patients: blood urea pre and post-HD, serum creatinine [SCr], potassium [K], Ph, calcium [Ca], sodium [Na], hematocrit [Hct%], hemoglobin [Hb], and white blood cells [WBCs]. The adequacy of HD was assessed by Kt/v. ECG was done on all patients during the HD session.

Transthoracic echocardiographic assessment: Immediately before and after HD, conventional ECHO was performed using a PHILIPS EPIC7 [Philips healthcare, Sanjon, Canada] ECHO device with a transducer [3.5 MHz]. The same sonographer conducted ECHO in the supine or slight left lateral decubitus position before HD sessions and again at the end of HD. The American Society of Echocardiography Guidelines were used to compute all echocardiographic data.

Statistical methods: Data were analyzed using the Statistical Package for Social Sciences, version 23.0 [SPSS Inc., Chicago, USA]. We used the Shapiro-Wilk test to assess the normality of the data; normally distributed data were evaluated using T-test and ANOVA, while non-normally distributed data were assessed using the Mann-Whitney test and the Kruskal-Wallis test. The quantitative data, including age, LVEDD, LVESD, LAVI, EF, and laboratory parameters, were presented as mean ± standard deviations [SD] and ranges. In addition, qualitative variables were presented as frequencies and percentages. The Chi-square [X2] test or Fisher exact test was used in terms of qualitative data. A p-value of less than 0.05 was considered significant.

RESULTS

Overall, the study population had a mean age of 49±11 years, 55% were males, and 45% were females. Both groups had a comparable mean age (p=0.979) and gender (p=0.06). Approximately 20% were current smokers, 27% were ex-smokers, and 53% were ever-smokers. The mean duration of dialysis was 3.73±2.24 years. The associated comorbidities were vasculitis [1.5%], obstructive uropathy [5%], glomerulonephritis [5%], polycystic kidney [5%], systemic lupus erythematosus [7%], HTN [35%], diabetes [DM; 15%], and both HTN and DM [25%]. There was a statistically insignificant difference between Group I and Group II regarding the leading cause of CKD (p=0.618), as shown in table [1]. The mean dry body weight in both groups before HD was comparable [76.77 ± 11.59 and 77.23 ± 10.64; p=0.626]. After HD, 67% of the Group I patients achieved dry body weight, compared to 87% in Group II.

Regarding the SBP, it increased in 50%, decreased in 20%, and did not change in 30%. In those with increased SBP, the pre-HD SBP was 123.17 ± 11.33 mmHg compared to 140.67 ± 9.35 mmHg post-HD, with a median change of 16.6%. In those with decreased SBP, the pre-HD SBP was 144.58 ± 10.544 mmHg, while post-HD was 109.58 ±8.107 mmHg, with a median reduction of -24.1%, as shown in table [2]. Diastolic blood pressure [DBP] increased in 47% of patients, decreased in 20% of patients, and remained constant in 33% of patients.

Regarding the laboratory investigations, Hb, Hct%, WBCs, uric acid, albumin, and SCr were comparable in both groups, with no statistically significant difference. Pre-HD, the urea level was 164.07±32.144 mg/dL in Group I and 151.93±23.95 mg/dL, with no significant difference [p=0.128]. On the other hand, the urea after HD was significantly higher in the IDH group than the non-IDH Group [85.1 ± 16.57 vs. 75.37 ± 11.57; p=0.015], respectively. In terms of electrolytes, serum K, Ca, and pH were comparable in both groups, with no statistically significant difference. On the other hand, the serum Na was significantly elevated in Group I compared to Group II [145.53± 2.36 vs. 135.17± 2.183 mEq/L; p<0.001], as shown in table [3]. Regarding serum parathyroid hormone and Sp Kt/v, we could not find a significant difference between both groups.

Most patients showed sinus rhythm [90%], while only [10%] showed tachy-arrhythmia. Roughly 28% of patients had LVH, 20% had RVH, 16% had inverted T waves with pathological Q, and 12% showed inverted T waves alone, as shown in the table [4].

ECHO findings regarding the pre- and post-HD LVEDD, LVESD, LAVI, EF, and PASP were reported in table [5 and 6].

Table [1]: Comparison between Group I and group II groups as regards the leading causes of CKD

Main Cause	Group I [IDH]		Group II [Control]		Total	
	No.	%	No.	%	No.	%
Congenital	0	0	1	3.16	1	1.5
Diabetes mellitus	6	20	3	10	9	15
Glomerulonephritis	2	6	1	3.16	3	5
Hypertension	8	27	13	44	21	35
Hypertension and Diabetes mellitus	7	23	8	27	15	25
Obstructive Uropathy	2	7	1	3.16	3	5
Polycystic Kidney	2	7	1	3.16	3	5
Systemic lupus erythematosus	3	10	1	3.16	4	7
Vasculitis	0	0	1	3.16	1	1.5
Statistics	X^2= 6.257, p value=0.618					

Table [2]: SBP in pre- and post-HD patient in both groups

Variables		
In those with increased SBP		**SBP [mmHg]**
Pre-dialysis [110-150]	Mean ± SD	123.17 ± 11.333
After 120 minutes [110-150]		132 ± 8.867
Post-dialysis [110-150]		140.67 ± 9.354
% Change	Median	16.6
In those with decreased SBP		**SBP [mmHg]**
Pre-dialysis [120-160]	Mean ± SD	144.58 ± 10.544
after 120 minutes [120-140]		140.83 ± 11.645
Post dialysis [120-140]		109.58 ±8.107
% Change	Median	-24.1

Table [3]: Comparison between Group I and Goup II in terms of laboratory investigations

Laboratory finding	Group I [IDH] Mean ± SD	Group II [Control] Mean ± SD	P value
Hemoglobin level [g/d]	8.73±0.583	8.7±0.466	0.075
Hematocrit %	24.37±1.189	24.1±0.885	0.501
Total Leukocyte count [K/$\mu\ell$]	7112.75±1724.596	6647.33±1705.239	0.463
Creatinine [mg/dl]	8.33±1.605	7.77±1.165	0.651
Serum uric acid [mg/dL]	6.7±8.074	6.5±0.82	0.537
Serum Sodium [mEq/L]	145.53±2.36	135.17±2.183	0.000
Serum Potassium[mEq/L]	4.27±0.583	4.47±0.571	0.068
Serum Calcium[mg/dL]	8.79±0.556	8.13±0.434	0.515
Serum Phosphorus [mg/dL]	5±0.587	4.83±0.648	1.0
Serum Parathyroid Hormone [pg/ml]	362.7±42.655	342.27±52.283	0.925
Serum Albumin [g/dL]	3.5±0.572	3.74 ±0.571	0.263
Urea [mg/dL]			
Pre-HD	164.07±32.144	151.93±23.945	0.128
post-HD	85.1±16.57	75.37±11.568	0.015
Sp KT/V	1.03±0.32	1.13±0.4	0.253

Table [4]: ECG rhythm and findings in the whole study population

		No. [%]
ECG [Rhythm]	Sinus	54 [90]
	Tachyarrhythmia	6 [10]
ECG [Finding]	Inverted T wave	8 [13]
	Inverted T wave & pathological Q	10 [17]
	Left Ventricular Hypertrophy	11[30]
	Right Ventricular Hypertrophy	18 [18]
	Normal	13[22]

Table [5]: Echocardiography parameters pre and post-dialysis with IDH

Variable		Mean ± SD	P value
LVEDD	**Pre**	59.6±2.044	< 0.05
	Post	58.07±3.930	
LVESD	**Pre**	43.67±3.262	<0.05
	Post	42.17±3.030	
LAVI	**Pre**	34.67±2.368	<0.05
	Post	31.9±2.04	
EF	**Pre**	50.53±8.541	0.149
	Post	48.97±8.381	
PASP	**Pre**	42.33±6.764	<0.05
	Post	40.93±6.843	

Table [6]: Echocardiography parameters pre and post-dialysis in the patients with and without IDH

IDH				Non-IDH			t-test	P
LVEDD	**Pre**	**Mean ± SD**	59.6±2.044	**Pre**	**Mean ± SD**	57.27±2.288	4.023	0.704
	Post	**Mean ± SD**	58.07±3.930	**Post**	**Mean ± SD**	56.57±2.417	1.72	0.673
	% change	**Median**	-2.5	**% change**	**Median**	-0.869	-	-
LVESD	**Pre**	**Mean ± SD**	43.67±3.262	**Pre**	**Mean ± SD**	38.93±4.085	4.321	0.08
	Post	**Mean ± SD**	42.17±3.030	**Post**	**Mean ± SD**	38.47±4.305	4.434	0.224
	% change	**Median**	-2.2	**% change**	**Median**	0	-	-
LAVI	**Pre**	**Mean ± SD**	34.67±2.368	**Pre**	**Mean ± SD**	26.5±1.834	17.94	0.087
	Post	**Mean ± SD**	31.9±2.04	**Post**	**Mean ± SD**	23.1±1.845	20.09	0.2
	% change	**Median**	-8.57	**% change**	**Median**	-13.207	-	-
EF	**Pre**	**Mean ± SD**	50.53±8.541	**Pre**	**Mean ± SD**	59.33±5.689	-4.011	0.028
	Post	**Mean ± SD**	48.97±8.381	**Post**	**Mean ± SD**	59.83±6.783	-4.768	0.066
	% change	**Median**	-2.83	**% change**	**median**	0.833	-	-
PASP	**Pre**	**Mean ± SD**	42.33±6.764	**Pre**	**Mean ± SD**	44.53±2.980	-1.908	0.047
	Post	**Mean ± SD**	40.93±6.843	**Post**	**Mean ± SD**	37.30±1.664	2.833	0.954
	% change	**Median**	-2.43	**% change**	**median**	-17.778	-	-

DISCUSSION

A change in BP may result from an extracellular volume response triggered by an increase in serum Na. Serum Na may directly affect the cardiovascular system, the local renin-angiotensin system, and the hypothalamus, all of which may have a role in regulating BP [10]. It is crucial to consider dialysate sodium as a possible cause of IDH. **Peng *et al.*** reported that IDH could be attributed to a low dialysate sodium concentration [7% lower than serum] [10]. This positive sodium gradient could be particularly significant in the pathogenesis of IDH, in addition to increasing interdialytic weight gain and BP. In 206 hemodialysis patients, there was a direct correlation between the dialysate to serum sodium gradient and the change in SBP during dialysis [11].

Our study showed that there was a statistically significant decrease in LVESD and LVEDD post-dialysis in patients with IDH. This is in line with the findings of **Gerede *et al.*** who examined 84 individuals with IDH who were receiving regular HD [12]. Standard echocardiographic measures were performed, and it was discovered that HD considerably reduced LVEDD and LVESD. Additionally, **Kudoh and Yaso**'s investigation showed that in patients with IDH, the LVEDD reduced from [40.3±4.2 mm] to [36.1±4.6 mm] [13]. According to **Oosugi *et al.*** patients with IDH were more likely to decrease LVEDD post-dialysis and have a worse LVEF than those without IDH [14]. In a study by **Wang *et al.*** the LVEDD dropped from 53.5±1.1 mm to 49.5±1.9 mm in 12 patients with renal insufficiency but without the overt cardiac disease who had received HD [15]. In agreement with our findings, **Ene-Iordache *et al*.** [16] found a significant [p<0.001] drop in LA volume from 36.7 ±21.7 mm^3 to 29.5±10.0 mm^3. It was reported that LA volume could predict the prognosis of HD patients at risk of developing IDH [17]. Our study showed that IDH was associated with a statistically significant reduction in PASP. According to a study by **Pabst *et al.*** conducted on 31 dialysis patients, 25 of whom had pulmonary hypertension, there was a statistically significant drop in PASP from (43±16) mmHg to (37±13) mmHg (p= 0.001) [18].

We acknowledge that our study has some limitations, including the small sample size and the single-center setting, which may hinder the generalizability of our findings. Moreover, we could not assess the predictors of the occurrence

of IDH or the lipid profile of the included patients due to the lack of data. In addition, we enrolled only those who were subjected to hemodialysis, which may introduce a risk of selection bias.

Conclusion: Poorly managed BP in HD patients is linked to volume overload, increasing the risk of diastolic dysfunction and subsequent heart failure. HD patients with IDH had substantial reductions in LVEDD, LVESD, and PASP after HD sessions.

Conflict of Interest and Financial Disclosure: None

REFERENCES

1. Tandukar S, Palevsky PM. Continuous Renal Replacement Therapy: Who, When, Why, and How. Chest. 2019 Mar;155(3):626-638. doi: 10.1016/j.chest.2018.09.004.
2. Inrig JK. Intradialytic hypertension: a less-recognized cardiovascular complication of hemodialysis. Am J Kidney Dis. 2010 Mar;55(3): 580-9. doi: 10.1053/j.ajkd.2009.08.013.
3. Malliara M. The management of hypertension in hemodialysis and CAPD patients. Hippokratia. 2007 Oct;11(4):171-4. PMID: 19582188.
4. Assimon MM, Flythe JE. Definitions of intradialytic hypotension. Semin Dial. 2017 Nov;30(6):464-472. doi: 10.1111/sdi.12626.
5. Kanbay M, Ertuglu LA, Afsar B, Ozdogan E, Siriopol D, Covic A, Basile C, Ortiz A. An update review of intradialytic hypotension: concept, risk factors, clinical implications and management. Clin Kidney J. 2020 Jul 8;13(6): 981-993. doi: 10.1093/ckj/sfaa078.
6. Shamir AR, Karembelkar A, Yabes J, Yao Y, Miskulin D, Gassman J, *et al*. Association of Intradialytic Hypertension with Left Ventricular Mass in Hypertensive Hemodialysis Patients Enrolled in the Blood Pressure in Dialysis (BID) Study. Kidney Blood Press Res. 2018;43(3):882-892. doi: 10.1159/000490336.
7. Loutradis C, Sarafidis PA, Ferro CJ, Zoccali C. Volume overload in hemodialysis: diagnosis, cardiovascular consequences, and management. Nephrol Dial Transplant. 2021 Dec 2;36(12): 2182-2193. doi: 10.1093/ndt/gfaa182.
8. Tsai YC, Chiu YW, Tsai JC, Kuo HT, Hung CC, Hwang SJ, *et al*. Association of fluid overload with cardiovascular morbidity and all-cause mortality in stages 4 and 5 CKD. Clin J Am Soc Nephrol. 2015 Jan 7;10(1):39-46. doi: 10.2215/CJN.03610414.
9. Tamulėnaitė E, Žvirblytė R, Ereminienė R, Žiginskienė E, Ereminienė E. Changes of Left and Right Ventricle Mechanics and Function in Patients with End-Stage Renal Disease Undergoing Haemodialysis. Medicina (Kaunas). 2018;54(5):87. doi: 10.3390/medicina54050087.
10. Van Buren PN, Inrig JK. Mechanisms and Treatment of Intradialytic Hypertension. Blood Purif. 2016;41(1-3):188-93. doi: 10.1159/00044-1313.
11. Movilli E, Camerini C, Gaggia P, Zubani R, Feller P, Poiatti P, *et al*. Role of dialysis sodium gradient on intradialytic hypertension: an observational study. Am J Nephrol. 2013;38(5):413-9. doi: 10.1159/000355974.
12. Gerede DM, Turhan S, Kaya CT, Ozcan OU, Goksuluk H, Vurgun VK, *et al*. Effects of Hemodialysis on Tei Index: Comparison between Flow Doppler and Tissue Doppler Imaging. Echocardiography. 2015 Oct;32(10):1520-6. doi: 10.1111/echo.12895.
13. Kudoh Y, Satoh S, Tsuchida A, Hikita S, Sasa Y, Iimura O. The dual effects of hemodialysis on cardiac function assessed by pulsed Doppler echocardiography. Jpn Circ J. 1988 Jan;52(1):13-20. doi: 10.1253/jcj.52.13.
14. Oosugi K, Fujimoto N, Dohi K, Machida H, Onishi K, Takeuchi M, *et al*. Hemodynamic and pathophysiological characteristics of intradialytic blood pressure elevation in patients with end-stage renal disease. Hypertens Res. 2014 Feb;37(2):158-65. doi: 10.1038/hr.2013.123.
15. Wang M, Yip GW, Wang AY, Zhang Y, Ho PY, Tse MK, Lam PK, Sanderson JE. Peak early diastolic mitral annulus velocity by tissue Doppler imaging adds independent and incremental prognostic value. J Am Coll Cardiol. 2003 Mar 5;41(5):820-6. doi: 10.1016/s0735-1097(02)02921-2.
16. Ene-Iordache B, Perico N, Bikbov B, Carminati S, Remuzzi A, Perna A, *et al*. Chronic kidney disease and cardiovascular risk in six regions of the world (ISN-KDDC): a cross-sectional study. Lancet Glob Health. 2016 May;4(5):e307-19. doi: 10.1016/S2214-109X(16)00071-1.
17. Kim SJ, Han SH, Park JT, Kim JK, Oh HJ, Yoo DE, *et al*. Left atrial volume is an independent predictor of mortality in CAPD patients. Nephrol Dial Transplant. 2011 Nov;26(11):3732-9. doi: 10.1093/ndt/gfr118.
18. Pabst S, Hammerstingl C, Hundt F, Gerhardt T, Grohé C, Nickenig G, Woitas R, Skowasch D. Pulmonary hypertension in patients with chronic kidney disease on dialysis and without dialysis: results of the PEPPER-study. PLoS One. 2012; 7(4):e35310. doi: 10.1371/journal.pone.0035310.

International Journal of Medical Arts

https://ijma.journals.ekb.eg/
Print ISSN: 2636-4174
Online ISSN: 2682-3780