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Original Research Article

Hemodynamic Changes in Hemodialysis Patient

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Abstract

Objective: To study the evaluation of hemodynamic during haemodialysis and to determine the frequency of haemodialysis induced hypotension & myocardial ischemia.

Methods: This study was conducted in the Department of Medicine CIMS Chhindwara during a period from September 2020 to October 2022. To assess various complication seen during HD. Present study includes a sum total 300 patients selection on random sampling method. Out of 300 patients there were 129 ARF/AKI and their haemodynamics were studied and results derived.

Results: Eleven of the 21 patients experienced a hypotensive period (H group) during dialysis, while the remaining 10 patients had no such event (NH group). Age, dry weight, time on dialysis, interdialytic weight gain, ultrafiltration rate/kg dry weight, and medication were comparable between the two groups. Before dialysis systolic arterial pressure appeared to be lower in the H group, although this was not significant (127±26 vs.151±46 mmHg. Baseline values in both groups for heart rate, SI, TPRI and CVP also did not show any significant differences. Most hypotensive patients developed severe symptoms, which necessitated intervention.

Conclusion: We conclude that dialysis related hypotension in our patient group did not result from an inability to maintain blood volume or from decreased cardiac filling. Hypotension appeared to result from the inability to adequately increase arteriolar tone and a reduction in left ventricular function.

Keywords: Dialysis related hypotension, Haemodynamics, Haemodialysis (HD), peritoneal dialysis (PD), Renal replacement therapy (RRT)

Introduction

The number of individuals kept alive by dialysis therapy in worldwide continues to increase each year. Large majority are treated with haemodialysis and only 7% to 8% of patients are on peritoneal dialysis (PD).

Whereas the current evidence suggest that both HD & PD provide equal renal replacement therapy (RRT) for most patient modality selection should be based on patient preference.[1-4]

The requirement of dialysis therapy in India has been increasing continuously. Patient of renal failure are managed by dialysis therapy or transplantation. But in India majority of the patients cannot afford transplantation therapy. So, they are managed by dialysis therapy. Dialysis causes many complications in patient. Some of them are fatal. Very few studies have been done on complication during hemodialysis in India. Therefore this study is designed to study of hemodynamics during Hemodialysis (HD).[2-5]

Materials and Methods

This study was conducted in the Department of Medicine CIMS Chhindwara during a period from September 2020 to October 2022.Total 300 numbers of cases were taken for study during this period.

Inclusion Criteria

Diagnosed cases of acute renal failure (ARF) and chronic renal failure (CRF) of all age group and both sexes. Diagnosis is based on history, examination and investigation.

Exclusion Criteria

- HIV Positive patients.
- HBV (HBs Ag) Positive patients.

In present study variations of hemodynamic during hemodialysis cardiac performance will be monitored by measuring blood pressure, heart rate and electrocardiography, based on intradialytic hemodynamic parameters and its comparison with predialytic hemodynamic status. The diagnosis of renal failure patients is done by serum creatinine

level and GFR by using cockroft-gault, Modification of Diet in Renal Disease (MDRD), and modified MDRD.

Observation Chart

The present study included a sum total of 300 hemodialysis sessions. Study was conducted in the dialysis unit. Numbers of patients undergoing for hemodialysis for renal failure patient (ARF/CRF)

Table 1: Distribution of Cases According to their Age & Diagnosis

Age	Arf	Crf	Total
< 20	01(2.70%)	01(2.22%	02(2.44%)
20 – 29	05(13.51%	02(4.44%)	07(8.53%)
30 – 39	16(43.24%)	05(11.11%)	21(25.61%)
40 – 49	11(29.73%)	11(24.44%)	22(26.82%)
50 – 59	02(5.40%)	11(24.44%)	13(15.85%)
>60	02(5.40%)	15(33.33%)	17(20.73%)
Total	37	45	82

Majority of ARF patients were observed in age range of 30 -49 years about 72.97% and CRF patients were observed in age group 40 – 70 years about 82.21%.

Table 2: Numbers of Hemodialysis in Which Complication Seen

Complication	ARF	CRF	Total
Presents	34(26%)	67(39%)	101(33.67%)
Absents	95(73%)	104(61%)	199(66.66%)
Total HD session	129	171	300

Complication seen in total cases 33.67% cases during HD

Table 3: Complication Seen During Hemodialysis in Total of 300 Cases of Hemodialysis

Complication	Number	Percentage
Hypotension	74	24.66%
Nausea and vomiting	38	12.66%
Arrhythmia	15	5%
Muscle cramp	43	14.33%
Hypertension	14	4.66%
Seizure	00	00
Dialysis reaction	00	00
Vascular access complication	00	00
Death	00	00

Table 4: Hypotension Associated Complication (N=300)

Complication	Number of Haemodialysis	Percentage
Nausea and vomiting	22	7.33%
Muscle cramp	13	4.66%
Nausea, vomiting and muscle cramp	25	8.33%

Table 5: Pre And Post Hemodialysis Laboratory Parameters (N=300)

Laboratory parameter	Pre HD	Post HD	Difference	Significance
Urea	183.10 + 59.78	137.80 + 54.30	44.30	t=35.45; p<0.0001
Creatinine	9.45 + 3.93	6.63 + 3.41	2.82	t=31.281; p<0.0001
Na	138.77 + 3.85	140.33 + 1.75	1.56	t=5.58; p<0.0001
K	4.28 + 0.60	4.35 + 0.31	0.07	t=1.63; p>0.05
Ca	8.96 + 0.55	9.29 + 0.47	0.33	t=13.18; p<0.0001
Hb%	9.46 + 3.93	9.21 + 3.93	0.25	t=6.87; p<0.0001

Results

Baseline characteristics: Eleven of the 21 patients experienced a hypotensive period (H group) during dialysis, while the remaining 10 patients had no such event (NH group). Age, dry weight, time on dialysis, interdialytic weight gain, ultrafiltration rate/kg dry weight, and medication were comparable between the two groups. Before dialysis systolic arterial pressure appeared to be lower in the H group, although this was not significant (127±26 vs.151±46 mmHg; Table 2). Baseline values in both groups for heart rate, SI, TPRI and CVP also did not show any significant differences.

- Present study included a total of 300 HD sessions there where 37 ARF/AKI and 45 CRF/CKD patients, male contribution 65% and female 35% who undergone HD.
- Most common age group presenting with renal failure due to various etiology who undergone HD was b/w 20 – 49year in those HD done (N=59 ,71.94%).
- Each HD cycle was conducted for an average three hours of duration. Most common complication seen during HD in present study was hypotension seen in 24% of cases most episodes precipitated during first hour of HD (N=200).
- Most of the cases in hypotension developed remain symptomatic during HD and there were no ECG changes. Hypertension during HD was seen in 5% cases. Most cases seen in end of HD. Cardiac arrhythmia were seen in 5% cases, 80% seen ventricular ectopic and 20% seen atrial ectopic.

Statistical Analysis

The collected data was summarized by using frequency, percentage, mean & S.D. To compare the qualitative outcome measures Chi-square test or Fisher's exact test was used. To compare the quantitative outcome measures independent t test was used. If data was not following normal distribution, Mann Whitney U test was used. SPSS version 22 software was used to analyse the collected data. p value of <0.05 was statistically significant.

Discussion

To assist hemodynamic parameters during HD and pre and post hemodialysis renal parameters. Present study included a total of 300 patients selected on random sample method. Hypotensive episodes are a major complication of hemodialysis. Hypotension during dialysis could be directly related to a reduction in blood volume or to a decrease in cardiovascular activation as a response to decreased cardiac filling. A decreased cardiovascular activation could be due to patient-related or to dialysis-related factors. In order to study the isolated effect of a reduction in filling pressure, lower body negative pressure (LBNP) causes activation of the cardiovascular reactivity with a decrease in cardiac filling, but without the influence of the dialysis procedure that could affect cardiovascular reactivity. In order to study the conditions in which hypotension occurred after the dialysis, we divided the patients into two groups: Hypotensive (H) and non-Hypotensive (NH) during dialysis. [5-8]

Baseline levels did not show any significant differences. During dialysis systolic arterial pressure declined gradually in the H group from 30 minutes before the onset of hypotension. There was a similar decrease of RBV and increase of heart rate in both groups with a large inter-individual variation. At hypotension, H patients showed a significantly smaller increase in TPRI, as compared to NH patients. The reduction in SI tended to be greater at hypotension, while CVP decreased to a similar extent in both groups. Moreover, during LBNP, a similar reduction in CVP resulted in a much smaller decrease in SI. Systolic arterial pressure was only slightly lowered due to a much greater increase in TPRI.

Hypovolemia is generally thought to play an important role in the pathogenesis of intradialytic hypotension [2]. However, in our study hypovolemia did not seem to play a pivotal role in the pathogenesis of hypotension, as the change in blood pressure was not related to the decline in RBV. Moreover, at the onset of hypotension there was a huge variation in decline of RBV. These results are in agreement with our previous results, in which RBV and blood pressure varied significantly during 300 dialysis sessions, even when corrected for ultrafiltration volume [18]. We also found that the change in RBV now of hypotension varied markedly, even within the same patient. It is known that two essentially different patterns of dialysis related hypotension can be distinguished. One of these has a gradual decrease in blood pressure, whereas the other is characterized by a sudden onset of bradycardia. The bradycardia associated hypertension is presumed to result from a Bezold-Jarish reflex, i.e. paradoxical symphaticoinhibition during severe underfilling . All hypotensive episodes observed in our study were preceded by a gradual increase in heart rate. These findings suggest that the hypotensive episodes did not result from severe underfilling or an inability to increase heart rate, but rather from an incapability to maintain SI and/or to increase vascular tone. [9-11]

In hypotensive subjects SI was decreased within one hour of the start of dialysis. The reduction in SI also tended to be greater now of hypotension than the Pathophysiology of haemodialysis-Related Hypotension (IDH) corresponding moment in the non-hypotensive patients. This reduction in SI occurred at a similar reduction in filling pressure, as estimated by CVP. Similar reduction in CVP resulted in a much smaller decrease in SI. Thus the inability to maintain cardiac output appears to be related to the dialysis procedure. Dialysis may impair either systolic or diastolic left ventricular function. We previously observed a decreased myocardial contractile reserve in hypotension prone patients. Further, previous studies have shown that the dialysis procedure appears to interfere with the systolic left ventricular function [10, 11]. However, there is evidence to suggest that increases in cardiac inotropy are not very important during hypovolemic conditions. Alternatively diastolic function can be

reduced by the reduction in filling pressure, as a decreased pressure difference between left atrium and ventricle results into a reduced early left ventricular filling. Moreover, diastolic dysfunction during dialysis has been suggested to result from shifts in ionized calcium. Decreased availability of calcium to the myocardium could impair both myocardial contraction and relaxation. Diastolic dysfunction is a complex process that may also be influenced by ventricular interaction.

The observed episodes of hypotension may also have resulted from the inability to adequately increase vascular tone. The increase in vascular resistance at hypotension in the H group was smaller than the corresponding time in the NH group. During LBNP, the hypotensive subjects were able to increase arteriolar tone and thereby maintain blood pressure despite a similar fall in SI. Therefore, the dialysis procedure appears to interfere with arteriolar tone. [12]

An inadequate increase in arteriolar tone during dialysis is either due to decreased sympathetic activation or to decreased vascular responsiveness. Many previous studies showed that dialysis normally stimulates sympathetic nerve activity during gradual hypotension . On the other hand, it has also been suggested that sympathetic function detoriates during dialysis as plasma norepinephrine levels do not rise appropriately. Analysis with heart rate variability with spectral analysis also failed to show an increase in sympathetic tone during hemodialysis in hypotensive patients [13-15].

The dialysis sessions in our study lasted for four hours. It is possible that prolonging the dialysis session, as described in the Tassin study, would have resulted in an improved blood pressure profiles during hemodialysis. Decreased vascular response during dialysis could result from a positive thermal balance, due to an inability to dissipate the excess of heat [16,17]. Further, a change in the Nitric Oxide–Endothelin–1 balance as a result of mechanical and chemical stimuli may also be involved in the pathogenesis of dialysis induced hypotension [18,19]. Other dialysis related factors that could cause an impaired vascular response include changes in plasma sodium, potassium, acid base composition and use of anti-hypertensive drugs. The decrease in CVP was comparable in both groups, a decrease in venous tone was unlikely responsible for the occurrence of the hypotensive episodes in our study. This agrees with previous study that showed a decreased veno constriction in stable sessions as well. [20-22]

Conclusion

We conclude that dialysis related hypotension in our patient group did not result from an inability to maintain blood volume or from decreased cardiac filling. Hypotension appeared to result from the inability to adequately increase arteriolar tone and a reduction in left ventricular function.

Declarations

Funding: None,

Availability of data and material: Department of Medicine CIMS Chhindwara during a period from September 2020 to October 2022.

Code availability: Not applicable.

Consent to participate: Consent taken.

Ethical Consideration: There are no ethical conflicts related to this study.

Consent for publication: Consent taken

References

1. Henrich WL: Hemodynamic instability during hemodialysis. Kidney Int 1986; 30:605-612.
2. Daugirdas JT: Dialysis hypotension: a hemodynamic analysis. Kidney Int 1991; 39:233-246.
3. Daugirdas JT: Pathophysiology of Dialysis hypotension: an update Am J Kidney Dis 2001;38: S11-S17.
4. Mitra S, Chamney P, Greenwood R, Farrington K: Linear decay in relative blood volume during ultrafiltration predicts hemodynamic instability. Am J Kidney Dis 2002; 40:556-565.
5. Pelosi G, Emdin M, Carpeggiani C, Morales MA, Piacenti M, Dattolo P, Cerrai T, Macerata AL, Abbate A, Maggiore Q: Impaired sympathetic response before intra dialytic hypotension: a study based on spectral analysis of heart rate and pressure variability. CliSci 1999; 96:23-31.
6. London GM: Left ventricular alterations and end-stage renal disease. Nephrol Dial Transplant 2002;17: S29-36.
7. Tisler A, Akocsi K, Harshegyi I, Varga G, Ferenczi S, Grosz M, Kulcsar I, Locsey L, Samik J, Solt I, Szegedi J, Toth E, Wagner G, Kiss I. Comparison of dialysis and clinical characteristics of patients with frequent and occasional hemodialysis-associated hypotension. Kidney Blood Press Res 2002; 25:97-102.
8. Heunissen KML, Kooman JP, van der Sande FM, van Kuijk WHM: Hypotension and ultrafiltrationphysiology in dialysis. Blood Purif 2000; 18:251-254.
9. Irkan E, Devarajan P, Kaskel F: Role of nitric oxide, endothelin –1, and inflammatory cytokines in blood pressure regulation in hemodialysis patients. Am J Kidney Dis 2002:40:76-81.
10. Raj DSC, Vincent B, Simpson K, Sato E, Jones KL, Welbourne TC, Levi M, Shah V, Blandon P, Zager P, Robbins RA: Hemodynamic changes during hemodialysis: Role of nitric oxide and endothelin. Kidney Int 2002; 61:697-704.

11. Henrich WL, Woodard TD, Blachely JD, Gomez-sanchez C, Pettinger W, Cronin RE: Role of osmolarity in blood pressure stability after dialysis and ultrafiltration. Kidney Int 1980; 18:480-488.
12. Krepel HP, Nette RW, Akcahuseyin E, Weimar W, Zietse R: Variability of relative blood volume during hemodialysis. Nephrol Dial Transplant 2000; 15:673-679.
13. Zocalli C, Tripepi G, Mallamaci F, Pannucio V: The heart rate response pattern to dialysis hypotension in hemodialysis patients. Nephrol Dial Transplant 1997; 12:519-523.
14. Poldermans D, Man in't Veld AJ, AJM, Rambaldi R, Bax JJ, Roelandt J, Weimar W, Zietse R: Cardiac evaluation in hypotension prone and hypotension resistant hemodialysis patients. Kidney Int 1999; 56:1905-1911.
15. Rozich JD, Smith B, Thomas JD, Zile MR, Kaiser J, Mann DL: Dialysis induced alterations in left ventricular filling: mechanisms and clinical significance Am J Kid Dis 1991;17:277-285.
16. Barnas MGW, Boer WH, Koomans HA: Hemodynamic patterns and spectral analysis of heart rate variability during dialysis hypotension. JASN 1999; 10:577-2584.
17. Ligtenberg G, Blankestijn PJ, Boomsma F, Koomans HA: No change in autonomic function tests during uncomplicated haemodialysis. Nephrol Dial Transplant 1996; 11:651-656.
18. Baldamus CA, Ernst W, Frei U, Koch KM: Sympathetic and hemodynamic response to Volume removal during different forms of renal replacement therapy. Nephron 1982; 31:324-332. 126 Pathophysiology of Hemodialysis-Related Hypotension
19. Enzmann G, Bianco F, Paolini F, Panzetta G: Autonomic nervous function and blood volume monitoring during hemodialysis. Int J Artif Organs 1995; 18:504-508.
20. Katzarski KS, Charra B, Luik AJ, NisellJ, DivinoFilho JC, Leypoldt JK, Leunissen KML, Laurent G, Bergstrom J: Fluid state and blood pressure control in patients treated with long and short haemodialysis. Nephrol Dial Transplant 1999; 14:369-375.
21. Kooman JP, Gladziwa U, Bocker G, van Bortell LMAB, van Hooff JP, Leunissen KML: Role of the venous system in hemodynamics during ultrafiltration and bicarbonate dialysis. Kidney Int 1992; 42:718-726.
22. Bradley JR Evans DB, Gore SM, Cowley AJ: Is dialysis hypotension caused by an abnormality of venous tone? BMJ 1988; 296:1634-1637.