IOSR Journal of Biotechnology and Biochemistry (IOSR-JBB)
ISSN: 2455-264X, Volume 4, Issue 4 (Jul. – Aug. 2018), PP 49-53
www.iosrjournals.org

Impact of Dialysis on serum electrolytes in End stage renal disease

Dr. Monisha.M*[1], Dr. Radhika.G[2], Dr. Muraliswaran.P[3],
[*1]*(III year Post Graduate, Department of Biochemistry, SVMCH&RC, Puducherry, India)*
[2]*(Professor and Head, Department of Biochemistry, SVMCH&RC, Puducherry, India)*
[3]*(Professor, Department of Biochemistry, SVMCH&RC, Puducherry, India)*
[4]*(Assistant Professor, Department of Biochemistry, SVMCH&RC, Puducherry, India)*

Abstract:*Chronic renal failure is a leading cause of death in developed countries. Over years there has been increase in the number of patients with chronic kidney disease(CKD). Hemodialysis (HD) is one of themain treatmentfor end-stage renal disease (ESRD). Here we have studied the impact of hemodialysis on electrolyte balance, urea and creatinine levels in patients on maintenance hemodialysis. The current study aims to assess the impact of dialysis on serum electrolytes in ESRD. A cross sectional study which was done in maintenance hemodialysis patients. 47 patients were selected according to their inclusion and exclusion criteria and their blood samples were analysed for various parameters. Statistical Analysis was done by SPSS software.There is increase in mean sodium from 134.17±3.19 to 138.38 ±3.70(p<0.0001) and chloride from 102.29±4.14 to 107.36±3.93 (p<0.0001). There is reduction in potassium levels from 5.83± 1.09 to 4.91±1.07 (p<0.0001) after dialysis.Serum sodium and chloride shows significant increase and potassium shows significant reduction post dialytically.*

Keywords: *End stage renal disease, hemodialysis, electrolytes.*

Date of Submission: 22-08-2018 Date Of Acceptance: 04-09-2018

I. INTRODUCTION

CKD was first defined by GFR less than 60 ml/min/1.73 m^2 for the duration of 3 months or longer with disease severity was staged only by GFR[1]. The prevalence of CKD is 17.2% with stage I, 7% in stage II, 4.3% in stage III, 4.3% in stage IV, & 8% in stage V respectively[2]. The major complications of CKD are cardiovascular disease, anemia, infectious complications, neuropathy and abnormalities related to mineral bone metabolism[3]. The disturbances in acid-base and electrolyte balance in CKD can have a greater impact on a patient's well-being and also leads to increased morbidity and mortality[4]. Dysnatremia is one of the common electrolyte disorder in clinical practice which is observed in many of the medical conditions, including chronic kidney disease[5,6]. HD removes sodium by the method of ultrafiltration and diffusion. Literature document hyperkalemia occurs very commonly in CKD patients due to progression of reduced urinary output, reduced potassium clearance, shift of potassium from the intracellular to the extracellular space in the in renal failure, and the usage of drugs such as ACE and ARB inhibitors[7-12]. Very high serum potassium levels predispose to arrhythmia and sudden death in CKD[13]. The aim of this study was to evaluate the pre and post dialysis electrolyte changes in ESRD patients.

II. MATERIAL AND METHODS

It is a Cross sectional Study with samples collected from 47 patients with ESRD undergoing dialysis which is conducted after obtaining Institutional ethics clearance and informed consent. Their mean duration of dialysis was 3.5- 4 hours. All the patients were treated with bicarbonate dialysate with electrolyte concentration as follows: Na^+-135mmol/L; K^+- 2 mmol/L; bicarbonate – 32mmol/L. Blood flow rate was 300 ml/minute with a dialysate flow of 500 ml/min. Dialysis was done using polysulfone based dialysis membrane.

Study Design: Cross sectional study
Study Location: This was a tertiary care teaching hospital based study done in Department of Biochemistry, at Sri Venkateshwaraa medical college hospital and research centre, Puducherry.

Sample size: 47 patients.

Subjects & selection method: The study population was drawn from dialysis unit who presented to Sri Venkateshwaraa medical college hospital and research centre with End stage renal disease undergoing maintainance hemodialysis.

Inclusion criteria:
Subjects undergoing maintenance hemodialysis of age group 35 – 70 years (both sexes) were included under the study.

Exclusion criteria:
The subjects diagnosed with liver, cardiac diseases and those who are taking drugs like ACE and ARB inhibitors were excluded from the study.

Procedure methodology
5ml of venous blood sample is collected from each patient before and after haemodialysis. Urea, creatinine, sodium, potassium and chloride were analysed. The Blood urea was estimated by Glutamate Dehydrogenase (GLDH) method[14] and serum creatinine was estimated by Jaffe's method[15] in autoanalyser. Serum sodium, potassium, and chloride were estimated by Electrolyte test kit .

Statistical analysis

The data was entered in MS Excel and expressed in Mean and standard deviation. To find the significant difference between pre and post dialysis, paired t test was performed. ANOVA was done to find the significant among the three groups. Statistical Analysis was done by SPSS software. p value<0.05 is considered as statistical significant.

III. RESULT

The 47 patients included based on the inclusion and exclusion criteria, in those 40 (85%) males and 7 (15%) females with mean age group 54.48±9. When the subjects were grouped, we found 10 (21%) of patients have diabetes with CKD, 25 (53%) have hypertension with CKD and 12 (26%) have both the co- morbidities with CKD. There is increase in percentage of patients with hypertensive nephropathy in accordance with the study Hörl MP et al who stated the majority of patients undergoing hemodialysis are hypertensive[16].

Table 1: Characteristic of pre and post hd electrolytes, urea and creatinine.

BIOCHEMICAL PARAMETERS	PRE HD	POST HD	p VALUE
SODIUM	134.17± 3.19	138.38 ±3.70	0.0001
POTTASIUM	5.83± 1.09	4.91± 1.07	0.0001
CHLORIDE	102.29±4.14	107.36±3.93	0.0001
UREA	117.61±24.54	97.76±24.07	0.0001
CREATININE	9.21±2.64	7.48±2.43	0.0001

Table 1 shows the comparison between pre and post HD values of electrolytes, urea and creatinine. The paired t-test analysis was done in pre and post-blood urea, creatinine and electrolytes which was significant with a p-value of < 0.0001.

Table 2: ANOVA RESULTS-PRE AND POST HD ELECTROLYTES, UREA AND CREATININE

		Sum of Squares	df	Mean Square	F	Sig.
pre- Na+	Between Groups	53.530	2	26.765	2.039	.142
	Within Groups	577.577	44	13.127		
	Total	631.106	46			
PRE- K+	Between Groups	11.586	2	5.793	2.638	.083
	Within Groups	96.627	44	2.196		
	Total	108.213	46			
PRE- CL-	Between Groups	29.194	2	14.597	.939	.399
	Within Groups	683.657	44	15.538		
	Total	712.851	46			
PRE UREA	Between Groups	1776.480	2	888.240	1.507	.233
	Within Groups	25936.627	44	589.469		
	Total	27713.106	46			
PRE- CREAT	Between Groups	10.556	2	5.278	.701	.502
	Within Groups	331.317	44	7.530		
	Total	341.872	46			

On subgroup analysis, there is no significant difference among the groups, still, potassium showed maximum F value which indicates that it shows some difference among the subgroups as shown in Table 2.

IV. DISCUSSION

Hemodialysis is the first line of treatment in almost all patients with ESRD which would postpone renal transplantation. In this study we assessed the pre and post dialytic changes in electrolytes in ESRD patients. Adequate dialysis has prolonged the survival of patients with improved quality of life. Cardiovascular disease was found to be the most frequent cause of mortality in majority of patients on maintenance hemodialysis[17].

Hemodialysis proved an effective impact on serum urea and creatinine level which is reduced post dialytically. Analytical results showed that after dialysis, all the patients had reduction in urea from 117.61±24.54 to 97.76±24.07 and serum creatinine from 9.21 ±2.64 to 7.48 ±2.43 with a statistically significant paired t-test which is consistent with the study of seethalakshmi et al study where the pre and post dialysis urea level was 132.34+32.81 and 51.20±16.19 and creatinine levels were 8.68±2.77 and 4.12±1.48 respectively[18]. Urea and creatinine, being small molecules, is removed mainly due to the counter- current flow of the blood and dialysate which maximize the concentration gradient of solutes between the blood and dialysate, which helps to remove more urea and creatinine from the blood after dialysis[19].

Present study results showed hyponatremia in pre dialysis state and gets corrected after dialysis, which is in accordance with NaumanTarifet al study where mean sodium level is higher in post dialytic serum[20]. The malfunctioning of aldosterone and renin-angiotensin system contributes to the decreased pre HD sodium. The raise in post HD sodium is due to the reduced intra dialytic removal.

The ESRD patients usually presents with hyperkalemia which predispose to cardiovascular effects like decrease in the action potential, widening of QRS complex and prolongation of PR interval[21-25]. ECG changes occurs during haemodialysis due to quick shift of serum K^+ which leads to hypokalemia[26] which requires careful monitoring and intervention. But in this study hyperkalemia persists pre dialytically which turns normal after dialysis. The removal of potassium by haemodialysis is largely determined by the potassium concentration gradient between the plasma and the dialysate[27].

V. CONCLUSION

Current study there was significant effects of HD on serum electrolytes, urea and creatinine. Improvement in electrolytes like serum sodium and potassium have prognostic significance in chronic renal failure. Therefore, electrolyte values and dialysate fluid should be considered before dialysis for all patients. It is recommended that the dialysate to be altered in accordance with pre dialytic electrolyte changes for each and every patient to prevent or treat serious effects due to electrolyte imbalance.

REFERENCES

[1]. National Kidney Foundation K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification, and stratification. American journal of kidney diseases. 2002;39(Suppl 1):S1–S266.

[2]. Singh AK, Farag YM, Mittal BV, Subramanian KK, Reddy SR, Acharya VN et al. Epidemiology and risk factors of chronic kidney disease in India - results from the SEEK (Screening and Early Evaluation of Kidney Disease) study. BMC Nephrol. 2013;14:114.

[3]. Levey AS, Stevens LA and Coresh J. Conceptual model of CKD: Applications and implications. Am J Kidney Dis. 2009;53(3Suppl 3):S4-16.

[4]. Raphael KL, Zhang Y, Wei G, Greene T, Cheung AK, Beddhu S. Serum bicarbonate and mortality in adults in NHANES III. Nephrol DialTranspl. 2013;28(5):1207-1213.

[5]. Bettari L, Fiuzat M, Shaw LK et al. Hyponatremia and long-term outcomes in chronic heart failure—an observational study from the Duke Data bank for Cardiovascular Diseases. J Card Fail. 2012;18:74-81.

[6]. Kovesdy CP, Lott EH, Lu JL et al. Hyponatremia, hypernatremia, and mortality in patients with chronic kidney disease with and without congestive heart failure. Circulation. 2012; 125: 677–684.

[7]. Saddadi F, Alidadi A, Hakemi M, Bahar B. Nephrotic Syndrome After Hematopoietic Stem Cell Transplant: Outcomes in Iran. Experimental and clinical transplantation. 2017;15(Suppl 1):90-92.

[8]. Kraft MD, Btaiche IF, Sacks GS, Kudsk KA. Treatment of electrolyte disorders in adult patients in the intensive care unit. Am J Health Syst Pharm. 2005;62(16):1663-1682.

[9]. Williams ME. Endocrine crises: hyperkalemia. Crit CareClin. 1991;7(1):155-174.

[10]. Weir MR. Are drugs that block the renin-angiotensin system effective and safe in patients with renal insufficiency?AmJHypertens. 1999;12(12),(Suppl 3):195S-203S.

[11]. Reardon LC, Macpherson DS. Hyperkalemia in outpatients using angiotensin converting enzyme inhibitors. Arch Intern Med. 1998;158(1):26-32.

[12]. EspinelE, Joven J, GilI et al. Risk of hyperkalemia in patients with moderate chronic kidney disease initiating angiotensin converting enzyme inhibitors or angiotensin receptor blockers: a randomized study. BMC Res Notes. 2013;6:306.

[13]. An JN, Lee JP, Jeon HJ, Kim H, Oh YK, Kim YS, Lim CS: Severe hyperkalemia requiring hospitalization: predictors of mortality. Crit Care. 2012;16(6):R225.

[14]. Eric J. Sampson and Marie A. Baird. Chemical Inhibition Used in a Kinetic Urease/ Glutamate Dehydrogenase Method for Urea in Serum. 1979;25(10):1721-1729.

[15]. Harry Husdan and Abraham Rapoport. Estimation of Creatinine by the Jaffe Reaction. A Comparison of Three Methods. Clinical chemistry. 1968; 14(3):222-238.

[16]. Hörl MP, Hörl WH: Hemodialysis -associated hypertension: patho- physiology and therapy. Am J Kidney Dis. 2002;39(2):227–244.

[17]. Varan HI, Dursum B, Dursum E et al. Acute effects of hemodialysis on oxidative stress parameters in chronic uremic patients: comparision of two dialysis membranes. International journal of nephrology and renovascular disease. 2010;3:39-45.

[18]. C.Seethalakshmi, D.Koteeswaran, V. Chiranjeevi. Correlation of Serum and Salivary Biochemical Parameters in end Stage Renal Disease Patients Undergoing Hemodialysis in Pre and Post –Dialysis State. Journal of Clinical and Diagnostic Research. 2014;8(12):12-14.

[19]. Blake P, Daugirdas J. Physiology of Peritoneal Dialysis. In: Handbook of Dialysis. 2008;4th ed:323-338.

[20]. Tarif N, Yamini H, Bakhsh AJ et al. Electrocardiography and serum potassium before

[21]. and after hemodialysis sessions. Saudi J Kidney Dis Transpl. 2008;19(1):47-53.

[22]. Dananberg J. Electrolyte abnormalities affecting the heart. In: Schwartz GR, editor. Principles and practice of emergency medicine. 4thed. Baltimore: Williams & Wilkins; 1999.p.425–7.

[23]. Quick G, Bastani B. Prolonged asystolichyperkalemic cardiac arrest with no neurologic sequelae. Ann Emerg Med. 1994;24(2):305–11.

[24]. Guyton AC, Hall JE. Textbook of medical physiology. 9th ed. Philadelphia: WB Saunders; 1996. p.375–80.

[25]. Roden DM, Lazzara R, Rosen M, Schwartz PJ, Towbin J, Vincent GM. Multiple mechanisms in the long-QT syndrome. Current knowledge, gaps, and future directions. The SADS Foundation Task Force on LQTS. Circulation. 1996;94(8):1996–2012.

[26]. Dittrich KL, Walls RM. Hyperkalemia: ECG manifestations and clinical considerations. J Emerg Med. 1986;4(6):449–55.

[27]. Covic A, Diaconita M, Gusbeth-Tatomir P, Covic M, Botezan A, Ungureanu G et al. Haemodialysis increases QT(c) interval but not QT(c) dispersion in ESRD patients without manifest cardiac disease. Nephrol Dial Transplant. 2002;17(12):2170-7.

[28]. Shermaan Ra, Hwang ER, Bernholc As, EisingerRp. Variability in potassium removal by haemodialysis. Am JNephrol. 1986;12:125-129.

ACKNOWLEDGEMENT

We would like to thank Dr.N. Bhuvaneshwari, Department of Nephrology, SVMCH&RC for her assistance in research work.

Dr. Monisha.M"Impact Of Dialysis On Serum Electrolytes In End Stage Renal Disease " IOSR Journal of Biotechnology and Biochemistry (IOSR-JBB) 4.4 (2018): 49-53.