

Original Research Article

Study of Serum Electrolyte Changes in End Stage Renal Disease Patients Before and After Hemodialysis Sessions: A Hospital Based Study

Dr. U. Sreenivasulu¹, Dr. S.N. Bhagamma², Dr. R. Anuradha³

^{1,2}Assistant Professor, ³Associate Professor, Department of Biochemistry, Government Medical College, Anantapuramu-515001, Andhra Pradesh, India

***Corresponding author**

Dr. U. Sreenivasulu

Email: drusreenivasulu@gmail.com

Abstract: Chronic renal failure (CRF) is a worldwide health problem, a leading cause of mortality in the developed countries. Patients with CRF are at higher risk for cardiovascular disease (CVD). Hyperkalemia is a commonly encountered problem in patients with end-stage renal disease (ESRD). The aim of the present study is to assess the effect of Hemodialysis (HD) on serum electrolytes, which compared with healthy controls and also correlation of serum electrolyte levels before and after hemodialysis was assessed. 40 hemodialysis patients with ESRD were tested to assess their serum electrolytes, Blood urea and Serum Creatinine before and after haemodialysis (HD). Serum electrolytes was estimated by electrolyte kit method, Serum Creatinine was estimated by jaffs method and Blood urea was estimated by Diacetylmonoxime (DAM) method by using semi-auto Analyzer in Biochemistry Department. There was a significant effect of Hemodialysis on serum electrolytes particularly serum potassium levels. The pre-Hemodialysis K⁺ was 5.25 ± 0.72 mmol/L and post-hemodialysis K⁺ was 3.8 ± 0.36 mmol/L (P < 0.0001 ESS). The pre-hemodialysis Na⁺ was 134.5 ± 2.6 and post-hemodialysis Na⁺ was 136.6 ± 5.8 (P = 0.0399 SS). The pre-hemodialysis Cl⁻ was 102.2 ± 2.29 and post-hemodialysis Cl⁻ was 101.3 ± 1.92 (P 0.0605 NS). We conclude that serum potassium was significantly decreased in post-hemodialysis states compared with pre-hemodialysis levels. Although serum sodium, serum chloride levels not significantly affected after hemodialysis. Post-hemodialysis serum potassium decrement (< 3.5 mmol/L) may have an arrhythmogenic potential and Hemodialysis is a prefer technique to correct biochemical abnormalities in ESRD patients.

Keywords: End-stage renal disease, Hemodialysis, Serum electrolytes.

INTRODUCTION

Diseases of kidneys are among the most important causes of death and disability in many countries throughout the world[1]. Chronic renal failure (CRF) refers to chronic deterioration of renal function but significant decrease in urine volume may not always be present. The major causes of CRF include chronic glomerulonephritis, progressive nephrotic syndrome, diabetes mellitus, chronic hypertension, long standing polycystic kidney and chronic pyelonephritis[2].

Disturbances in water, electrolyte and acid – base balance contribute to the clinical picture in CRF[3]. In recent years, diabetes mellitus and hypertension have become recognized as the leading cause of End stage renal disease (ESRD), together accounting for more than 70 percent of all chronic renal failure[1].

Potassium is the major intracellular cation and maintains intracellular osmotic pressure. About 90% of excess potassium is excreted through kidneys and the rest through gastrointestinal tract (GIT). Kidney can

lower renal excretion to 5-10 mmol per day or increase excretion to 450 mmol per day depending on the potassium intake[4].

Hyperkalemia is common complication in end-stage renal disease (ESRD) patients[5]. Plasma potassium level about 5.5 mmol/l is known as hyperkalemia. Since the normal level of k⁺ is kept at a very narrow margin, even minor increase is life – threatening. In hyperkalemia, there is increased membrane excitability, which leads to ventricular arrhythmia and ventricular fibrillation[4].

Dialysis is a process of separating the soluble crystalloids from the colloid is a mixture by means of a dialyser. Dialysis is based on the principle of diffusion equilibrium. In general dialyzing fluid (Dialysate) contains Na⁺, K⁺ and HCO₃⁻ in a higher concentration than normal plasma (urea, urates, creatinine, phosphate, sulphate are absent). If the plasma K⁺ of patient is above normal, K⁺ diffuses out of the blood across the cellophane tubing and in to the dialyzing fluid. Similarly, waste products and excess of the substances

also diffuse in to the dialyzing fluid and thus are removed from the body[6].

Hemodialysis is one of the effective means of treatment of hyperkalemia, uremia, also to correct sodium and serum creatinine levels in renal failure[7]. Severe hyperkalemia might occur in 10-19% of haemodialysis (HD) patients[8]. It has been shown by several studies that a sudden shift and decrease in serum K⁺ causes arrhythmia especially in patients undergoing dialysis[9]. The prevalence of hyperkalemia in any given month of HD patients was reported to be about 8.7-10% depending on individual centers.

Hypokalemia is usually defined as serum K less than 3.5meq/L. It usually occurs as a consequence of K depletion due to either increased excretion or inadequate intake. However, shift of K in the extracellular to intra cellular compartments also can cause hypokalemia. In case of ESRD patients on hemodialysis, hypokalemia is a relatively rare event comparing to hyperkalemia[10].

Sodium is the chief cation of the extracellular fluid. It is required for the maintenance of osmotic pressure and fluid balance. Kidney is the major route of sodium excretion from the body, as much as 800 g Na/day is filtered by the glomeruli, 99% of this is reabsorbed by the renal tubules by an active process[11]. Advanced renal failure typically results in sodium retention[12].

The Present study was done to access the changes in serum potassium, sodium and chloride in pre and post dialysis patients.

MATERIAL AND METHODS

A total of 80 subjects were taken for this study. For them (28 males and 12 females) who were apparently healthy were used as controls while the remaining 40 (28 males and 12 females) were End stage renal disease(ESRD) patients. The age group of included individuals was from 30-60 years. In order to understand the influence of dialysis on serum electrolytes, the patients who were in End stage renal disease were divided in to 2 groups: those who have ESRD but unanalyzed and those on dialysis. Thus, the study was divided in to 3 groups:

Group 1 - healthy controls

Group 2 - ESRD patients who have never under gone hemodialysis

Group 3 - ESRD patients on hemodialysis.

ESRD patients undergoing haemodialysis treatment three times per week for a minimum duration of at least 6 months.

The mean duration for sessions of the dialysis was 4 hours. The patients were dialysed using polysulfone-based dialysis membrane(Haemodialysis Apparatus Dialog+) in dialysis unit at Government General Hospital, Anantapuram, AP.

Exclusion criteria

Subjects suffering from liver diseases, ischemic heart disease and patients taking anti - hypertensives were excluded from this study.

Sample collection

5ml of venous blood sample taken in a plain tubes from each patient just before the haemodialysis session and 10 minutes after the session for measurement of serum K⁺, Na⁺, Cl⁻ as well as serum level of creatinine and urea. After collection, samples were centrifuged and serum was analyzed for estimation of Na⁺, K⁺, Cl⁻ and serum creatinine by using Semi-Auto analyzer following various methods. Serum sodium, potassium, and chloride were estimated by Electrolyte kit method[13]. The Blood urea was estimated by Diacetylmonoxime (DAM)method[14]. The serum creatinine was estimated by Jaffe's method[15].

Statistical Analysis

Data were analyzed using Graph pad software. All the data were expressed in mean and standard deviation. Test of probability less than 0.05(P<0.05) was regarded significant.

RESULTS

The 40 patients comprised 28 males and 12 females with a mean age of 47.52 ±11.09. Out of 40 patients 17(42.5%) patients were diabetic. The mean values of blood urea and serum creatinine in the pre-dialysis pts was higher than healthy controls as shown in Table No.1 and lower than post dialysis patients as shown in Table No2.

The mean levels of serum sodium in pre -HD patients was lower than healthy controls shown in table 1 and in post- HD patients serum Na⁺ levels was slightly higher than pre-HD patients as shown in the Table 2. However, the mean level of serum K⁺ value was significantly high in pre- HD pts compared to healthy controls as shown in Table No.1 and the mean value of K⁺ is significantly lower in post-HD patients compared to pre -HDs patients as shown in Table No2.

The mean Serum chloride level in pre-HD patients was higher in pre-HD patients compared to controls shown in Table No.1 and post-HD group (Group-3) was lower compared to pre-HD patients (Group-2) as shown in Table No.2.

Table-1: Revealed the Mean±SD of serum electrolytes (Na⁺, K⁺, Cl⁻), blood urea and serum creatinine levels in controls (Group-1) and pre-HD ESRD patients(Group-2)

Parameters	Healthy controls(Group-1)	Pre-HD ESRD pts(Group-2)	t-value	p-value
Na+(mmol/L)	138.5±1.58	134.5±2.6	8.3151	<0.0001 ESS
K+(mmol/L)	4.57±0.54	5.25±0.72	4.7786	<0.0001 ESS
Cl-(mmol/L)	100.5±3.4	102.2±2.29	2.6228	0.0105 SS
Blood urea(mg/dl)	29.5±6.2	127.4±6.7	67.828	<0.0001ESS
Serum creatinine(mg/dl)	1.07±0.21	7.05±1.19	31.298	<0.0001 ESS

Na⁺-sodium, K⁺-potassium, Cl⁻chloride, HD- hemodialysis, ESRD-End-stage renal disease,NS-not significant,ESS-Extremely statistically significant,SS-Statically significant.

Table-2: Revealed the Mean±SDserum electrolytes (Na⁺,K⁺,Cl⁻), blood urea and serum creatinine levels in pre-HD patients(Group-2) and post-HDpatients(Group-3)

Parameters	Pre-HDpatients (Group-2)	Post-HDpatients (Group-3)	t-value	p-value
Na+(mmol/L)	134.5±2.6	136.6±5.8	2.0896	=0.0399 SS
K+(mmol/L)	5.25±0.72	3.8±0.36	11.392	<0.0001 ESS
Cl-(mmol/L)	102.2±2.29	101.3±1.92	1.9047	=0.0605 NS
Bloodurea(mg/dl)	127.4±6.7	100.75±4.06	21.5148	<0.0001 ESS
Serum creatinine(mg/dl)	7.05±1.19	5.29±1.18	6.6421	<0.0001ESS

Na⁺ - Sodium, K⁺ - potassium, Cl⁻ - Chloride,HD-Hemodialysis, NS-Not significant, ESS- extremely statically significant, SS- Statistically significant

DISCUSSION

Chronic renal failure (CRF) is a worldwide health problem and is the leading cause of morbidity and mortality in the developed world. Patients with CRF are at high risk for cardiovascular disease (CVD)[16].

In this study we assess the change of electrolytes mainly K⁺ during hemodialysis in patients with ESRD. These patients usually presents with hyperkalemia. Hyperkalemia reduces the resting membrane potential, slows the conduction velocity and increases the rate of repolarization [17]. Hypokalemia on the other hand increases the resting membrane potential, and refractory period, which are potentially arrhythmogenic[18].

All these changes are the signs of membrane instability and cardiac arrest or ventricular fibrillation may follow and thus this situation usually requires careful and prompt management [19].Hemodialysis is an effective treatment in most renal failure patients and forms an alternative to renal transplantation.

Adequate dialysis treatment has prolonged the survival of patients with quality of life. Cardiovascular disease was found to be the most frequent cause of mortality in majority of patients on maintenance hemodialysis [20].

In the present study, mean serum potassium level in post - HD patients was significantly low compared to mean serum level of pre-HD patients as shown in Table No.2. During hemodialysis there is a quick shift of serum K⁺ which leads to hypokalemia[21].

The obtained result was consistent with that reported by Abdul-Majeed H *et al* [22], Nauman Tarief *et al*[23], Barry Kirschbaum[24], Seethalakshmi C *et al*[25] observed that mean serum potassium levels in post-HD (3.72±0.75) were low compared to pre-HD Patients (5.73±1.24).

Removal of serum potassium during the hemodialysis procedure is dependent on the duration, type of dialyzer, blood flow and most importantly dialysate potassium concentration [18]. It has been shown that most of these sudden deaths do not occur during the dialysis session[26] but in the following hours[27].

In the present study mean serum sodium levels in postdialysis patient was slightly higher when compared to mean serum sodium levels of pre dialysis patients. Seethalakshmi C *et al*[25] reported that the mean Na⁺ concentration in post -hemodialysis (135.23±4.38) patients was high compare to pre-hemodialysis patients (133.5±4.75). A study by NaumanTarief *et al* [23] observed that mean serum Na⁺

post - hemodialysis patients (134.24±2.59) were higher when compared to pre-hemodialysis patients (132.67±2.30).

In chronic Hemodialysis patients, sodium (Na⁺) balance largely depends on inter dialytic dietary salt intake and intra dialytic Na⁺ removal [28]. Dialysis patients appear to have a unique set point for serum sodium [29]. In general post-HD serum sodium exceeds pre-HD values by 2 meq/L to 4 meq/L implying that HD removes a hyponatremic ultra filtrate of plasma water and the patients exchangeable Na⁺ pool is incompletely depleted of excess Na⁺ [30].

In the present study mean serum chloride level in post -HD pts was low compared to pre HD pts. A study by Kirschbaum B [24] shows that mean serum chloride level in post HD patients (Mean-100) was low compare to pre HD patients (Mean-103).

CONCLUSION

Serum Sodium and Serum chloride does not have significant post dialysis change, but serum potassium, serum creatinine and blood urea having significant post-dialysis change. Post-HD serum potassium decrement (<3.5 mmol/L) have an arrhythmogenic potential. Hemodialysis is a perfect technique to correct biochemical abnormalities in ESRD patients.

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REFERENCES

1. Hall JE; Diuretics, Kidney Diseases. Guyton and Hall, Text book of medical physiology, 12th edition, Elsevier Publications, chapter 31, 397-410.
2. Aroor AR; Kidney and lungs. Medical Biochemistry ,1st edition, Jaypee publications, chapter 28, 719-729.
3. Pal GK; Kidney function tests, patho physiology of renal failure. Text book of medical physiology, 2nd edition, Ahuja book publishers, chapter 66, 556-560.
4. Vasudevan DM, Sreekumari S, Kannan Vaidyanathan; Electrolyte and water balance. Textbook of Biochemistry for medical students, 7th edition, Jaypee publications, chapter 30, 407-419.
5. Aslam S, Friedman EA, I fudu O; Electrocardiography is unreliable in detecting potentially lethal hyperkalemia in haemodialysis patients. Nephrol Dial Transplant, 2002;17:1639-42.
6. Jain AK; Renal Clearance. Text book of physiology, volume-1, 5th edition, Avichal publishing Company, chapter 57, 555-563.
7. Dushyant MS, Ajay CT; Role of Hemodialysis in Renal failure to correct biochemical parameters. Indian J of Applied Research, 2013; 3(6):414-416.
8. Szerlip HM, Weiss J, Singer I; Profound hyperkalemia without electrocardiographic manifestations. Am J kidney Dis., 1986; 7(6):461-5.
9. Redaelli B, Locatelli F, Limido D et al; Effect of a new model of hemodialysis potassium removal on the control of ventricular arrhythmias. Kidney int., 1996; 50(2):609-17.
10. Tzamaloukas AH, Avasthi PS; Temporal profile of serum potassium concentration in nondiabetic and diabetic out patients on chronic dialysis. Am J Nephrol., 1987;7:101-109.
11. Satyanarayana U, Chakrapani U; Mineral metabolism. Biochemistry, 4th edition, Elsevier Publications, 2013, chapter 18, 403-424.
12. Brennan BL, Yasumura S, Letteri JM, Cohn SH; Total body electrolyte composition and distribution of body water in uremia. Kidney int., 1980;(17):364-371.
13. Schoenfeld RG, Lewellen C; A colorimetric method for determination of serum chloride. Clin chem., 1964;10:533-539.
14. Wybenga DR, Di Giorgio J, Pileggi VJ; Manual and automated methods for urea nitrogen measurement in whole serum. Clin chem., 1971;17:891-895.
15. Toro G, Ackermann PG; Practical clinical chemistry, Boston: Little Brown and Co., 1975;154.
16. Agarwal K, Srivastava RK; Chronic Kidney disease in India: Challenges and Solutions. Nephron clinpract., 2009;111:C197-C203.
17. Fisch C; Relation of electrolyte disturbances to cardiac arrhythmias. Circulation, 1973;47(2):408-19.
18. Webster A, Brady W, Morris F; Recognizing signs of danger: ECG changes resulting from an abnormal serum potassium concentration. Emerg Med J., 2002;19(1):74-7.
19. Tarif N, Yamini H, Bakhsh AJ et al; Electrocardiography and serum potassium before and after hemodialysis sessions. Saudi J Kidney Dis Transpl., 2008;19(1):47-53.
20. Varan HI, Dursum B, Dursum E et al; Acute effects of hemodialysis on oxidative stress parameters in chronic uremic patients: comparison of two dialysis membranes. International journal of nephrology and renovascular disease, 2010;3:39-45.
21. Covic A, Diaconita M, Gusbeth-Tatomir P et al; Hemodialysis increases QT(C) dispersion in ESRD patients without manifest cardiac disease. Nephrol Dial Transplant, 2002;17(12):2170-7.
22. Abdul-Majeed H, Al-Saffer; Pre and post Hemodialysis: The effect of Electrolyte imbalance

- on ECG of patients with end-stage renal disease. Medical journal of Babylon, 2011; 8(2):221-229.
23. Tarif N, Yamini H, Bakhsh AJ, et al; Electrocardiography and serum potassium before and after hemodialysis sessions. Saudi j kidney dis transpl., 2008;19(1):47-53.
 24. Kirschbaum B; The effect of hemodialysis on electrolytes and acid base parameters. clinicachimicaacta., 2003;336:109-113.
 25. Seethalakshmi C, koteeswaran D, Chiranjeevi V; Correlation of serum and salivary biochemical parameters in end stage disease patients undergoing hemodialysis in pre and post dialysis state. J clindign res., 2014; 8(12):12-14.
 26. Karnil JA, Young BS, Lew NL et al; Cardiac arrest and sudden death in dialysis unite. Kidney int., 2001; 60(1)350-7.
 27. Bleyer AJ, Hartman J Brannon PC et al; Characteristics of sudden death in hemodialysis patents. Kidney int., 2006; 69(12):2268-73.
 28. Santos SFF, Peixoto AJ; Sodium balance in maintenance hemodialysis. Semin Dial., 2010;23: 549-555.
 29. Petitcherc T, Jacobs C. Dialysis sodium concentration: what is optimal and can it be individualized? Nephrol Dial transplant, 1995;10:596-599.
 30. Flanigan MJ; Sodium flux and dialysate sodiumin hemodialysis. Semin Dial., 1998;11:298-304.