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Assessment of Clinical Characteristics in Chronic Kidney Disease Patients

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Abstract

➢ ***Background:***

Chronic renal failure is the progressive loss of function of kidney and patient requires a long treatment. The aim of this study is to monitor and management of the biochemical parameters in chronic kidney disease patients before and after treatment.

➢ ***Method:***

The current study is prospective, observational study conducted over a period of six months including 105 diseased subjects from a private hospital located at Karimnagar,Telangana state. Diseased subjects are the patients with Chronic Kidney disease. The biochemical parameters like serum electrolytes, serum creatinine and blood urea, were measured using standard techniques in chronic kidney disease cases and the findings were compared with parameters of same cases before treatment. Results were analyzed using Graph pad prism 8 software.

➢ ***Results:***

Serum sodium in the diseased subjects is 137.69 ± 8. 59 before treatment and after treatment of subjects is 138.13 ± 2.98 with p <0.516, which is statistically not significant. Similarly, Serum chloride in the diseased subjects is 102.71 ± 11.01 before treatment and after treatment of subjects is 103.88 ± 4.16 with p <0.205 which is also statistically not significant. However, Serum potassium in the diseased subjects before and after treatment is 4.42 ± 0.97 and 4.14 ± 0.34 respectively with p <0.0001 which is statistically significant. Serum creatinine in the diseased subjects is 3.18 ± 1.60 before treatment and after treatment of subjects is 1.09 ± 0.31 with p <0.0001 which is statistically significant. Serum urea in the diseased subjects is 67.13 ± 22.12 before treatment and after treatment of subjects is 27.41 ± 5.04 with p<0.0001 which is statistically significant.

➢ ***Conclusion:***

In the present study management of treatment in CKD patients is done by monitoring the biochemical parameters before and after treatment .Thus routine investigation of biochemical parameters of CKD patients especially serum creatinine to prevent complications of chronic kidney disease like cardiovascular diseases, dyslipidaemia, hyperkalaemia, metabolic acidosis, anaemia, and bone and mineral disorders is recommended. Correction of these abnormalities help to reduce the morbidity and mortality related to CKD.

I. INTRODUCTION

Kidneys are the most vital organs of human body. Its malfunction can lead to serious illness or even death. Its two important functions are to flush out harmful and toxic products and to maintain balance of water, fluids, minerals and chemicals(1).The prevalence of CKD is increasing all over the world day by day and the global annual growth number of the ESRD patients is reported at 7%.[2]In India, etiologies hypertension and diabetes accounts for about 40-60% cases of CKD.[3]Few studies in India states that males are more prone to CKD than that of females. The ratio of

male and female prone to CKD is 70:30 respectively for every hundred CKD patients.[4]When discussing the pathophysiology of CKD, renal structural and physiological characteristics, as well as the principles of renal tissue injury and repair should be taken into consideration.(5) Chronic kidney disease pathophysiology is initially described as diminished renal reserve or renal insufficiency, which may progress to renal failure (end-stage renal disease). Initially, as renal tissue loses function, there are few abnormalities because the remaining tissue increases its performance (renal functional adaptation). Reduced renal function interferes with the kidneys' ability to maintain fluid and electrolyte homeostasis. The ability to concentrate urine declines early and is followed by decreases in ability to excrete excess phosphate, acid, and potassium.(6)

II. SIGNIFICANCE OF ELECTROLYTES

The body contains a variety of ions, or electrolytes. All the ions in plasma contribute to the osmotic balance that controls the movement of water between cells and their environment. In terms of body function, six are the most important electrolytes: sodium, potassium, chloride, bicarbonate, calcium, and phosphate. These ions helps in endocrine secretion, nerve excitability, membrane permeability and controlling the movement of fluids between the compartments. They enter the body through the digestive tract and excretion of these ions occurs mainly through the kidneys, with lesser amounts lost in sweat and in feces.

- *Sodium*

Sodium, the major cation of the extracellular fluid. It is responsible for one-half of the osmotic pressure gradient that exists in between the interior of cells and their surrounding environment. ***Hyponatremia*** is a lower-than-normal concentration of sodium, associated with excess water accumulation in the body, which dilutes the sodium. An absolute loss of sodium may be due to a reduced intake of the ion coupled with its continual excretion in the urine. ***Hypernatremia*** is an abnormal increase of blood sodium. It can result from water loss from the blood, which results in the hemoconcentration of all blood constituents.

- *Potassium*

Potassium is the crucial intracellular cation. It helps in establishing the resting membrane potential in neurons and the muscle fibers after membrane depolarization and action potentials. The lower levels of potassium in the blood and CSF are due to sodium-potassium pumps in cell membranes, which maintains the normal potassium concentration gradients between ICF and ECF. ***Hypokalemia*** is an low potassium blood level can arise from decreased intake, frequently related to starvation. In diabetic patients when insulin is administered and glucose is taken up by the cells, potassium passes through cell membrane along with the glucose, thereby decreasing the amount of potassium in the blood and IF, which can cause hyperpolarization of the cell membranes of neurons, decreasing their responses to stimuli. ***Hyperkalemia*** an elevated potassium blood level, can result from increased dietary intake of potassium.

- *Chloride:*

Chloride is the chief extracellular anion. Chloride functions to balance cations in the ECF, maintaining the electrical neutrality of the fluid. ***Hypochloremia*** is lower-than-normal blood chloride levels, occurs because of defective renal tubular absorption. ***Hyperchloremia*** or higher-than-normal blood chloride levels, occurs due to dehydration, excessive intake of dietary salt.(7)

III. MATERIALS AND METHODS

It is a prospective, observational study conducted over a period of six months including 105 diseased subjects from a private hospital located at Karimnagar,Telangana state.

- *Inclusion Criteria:*

All patients with chronic kidney disease (including patients with hemodialysis)

Patients of both sexes were included in this study.

- *Exclusion Criteria:*

Patients below 18 years of age.

Patients who are not willing to sign in informed consent form.

Patients with pregnancy and lactating mothers.

Patients meeting inclusion and exclusion criteria were selected for the study.All the relevant patient data was collected from the sources and documented in a suitably designed data collection form.The data collected was analyzed and comparisons were made by using Graph pad prism 8 software.

IV. RESULTS

➢ *Gender Distribution in Diseased Subjects:*

In a total of 105 disease subjects, 66 (62.85%) were Males and 39 (37.14%) were Females with various renal diseases.

Gender	No. of Subjects	Percentage of Diseased
MALE	66	62.85
FEMALE	39	37.14

Table 1:- Gender Distribution of CKD Subjects

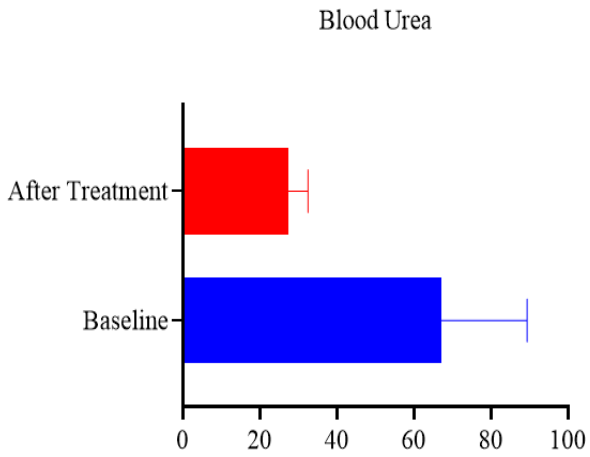

Fig 1:- Comparision of blood urea

Lab Tests	Baseline	After Treatment
Blood urea	67.13±22.12	27.41±5.04
Serum creatinine	3.18±1.60	1.09±0.31
Serum sodium	137.69±8.59	138.13±2.98
Serum potassium	4.42±0.97	4.14±0.34
Serum chloride	102.71±11.01	103.88±4.16

Table 2:- Comparision of Laboratory Tests

Serum blood urea levels at the beginning of the study was 67.13± 22.12. At the study end, blood urea levels were reduced to 27.41± 5.04.

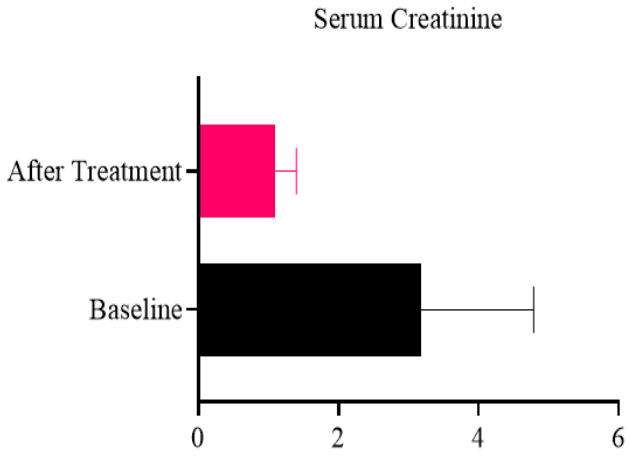

Fig 2:- Comparision of Serum Creatinine

The mean changes in the serum creatinine level from baseline to after treatment is 2.09± 1.29.

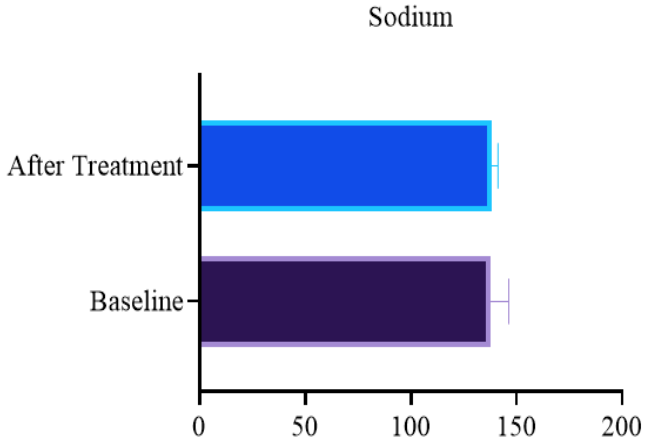

Fig 3:- Comparision of Serum Sodium

Serum sodium levels before treatment were 137.69±8.59 and there was a marginal increase in the sodium levels in CKD patients after treatment to 138.13±2.98.

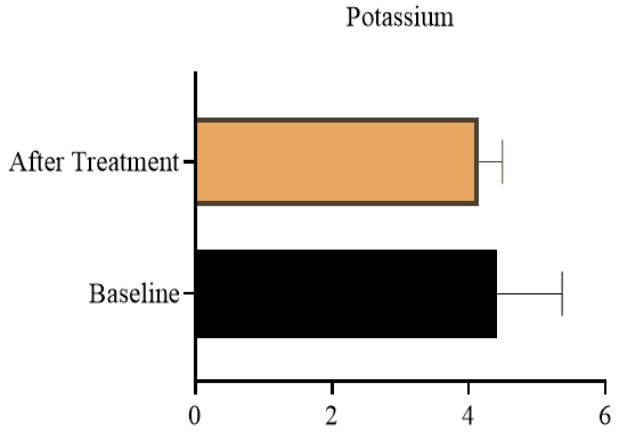

Fig 4:- Comparision of Serum Potassium

Serum Potassium levels before treatment were 4.42±0.97 and there was a slight decrease in the potassium levels in CKD patients after treatment to 4.14±0.34.

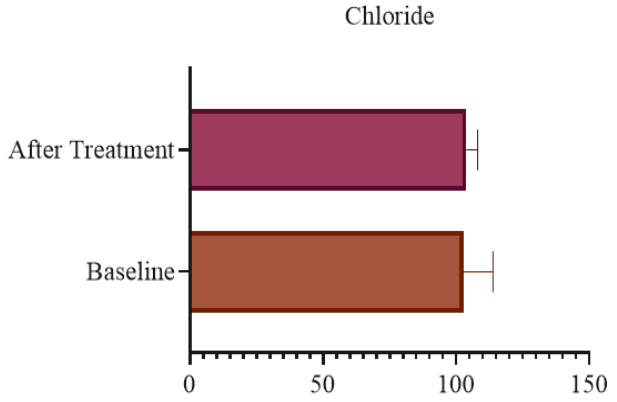

Fig 5:- Comparision of Serum Chloride

Serum chloride levels at the beginning of the study was 102.71±11.01 . At the study end, chloride levels were increased to 103.88±4.16.

V. DISCUSSION

The current study is prospective observational study including 105 diseased subjects .Diseased subjects are the patients with Chronic renal disease.All the subjects were tested for the serum electrolytes , serum creatinine and blood urea. Out of 105 diseased subjects, 66 (62.85%) were Males and 39 (37.14%) were Females with various renal diseases. In our study male patients are more prone to the renal disorders with a prevalence of 62.85%. Studies done by **R. Freethi *et al*** showed male patients are more prone to renal diseases with a prevalence of 68.33%. In the diseased subjects**,** 6 (5.71%) were under 18-27, 11 (10.47%) were under 28-37, 17 (16.19%) were under 38-47, 26 (24.76%) were under 48-57, 28 (26.66%) were under 58-67, 10 (9.52%) were under 68-77 and 7 (6.66%) were under 78-87.[8]

Serum sodium in the diseased subjects was 134.76 ± 4.93 with p <0.0001, which is statistically significant. In 105 diseased subjects 37 subjects are with hyponatremia i.e. <135mmol/l with a prevalence of 35.23% and 8 subjects are with hypernatremia i.e. >145mmol/l with a prevalence of 7.61%. This study was contrary to ***Rashmi Rekha Phukan et al.*** showed that Serum sodium in patients was 138.36±8.12 and in controls was 141.43±3.94 with p 0.015, which is statistically significant. Among the 71 CKD patients, 20 patients were hyponatremic with serum sodium value <135 mmol/L having prevalence of 28.1%. Whereas 9 patients were hypernatremic having serum sodium value >145 mmol/L with prevalence of 12.6%.[9]

Serum potassium in the diseased subjects was 4.5 ± 0.95 with p <0.0001 which is statistically significant. Among 105 diseased subjects 10 are hypokalemic i.e. <3.5mmol/l with prevalence of 9.5% and and 15 are hyperkalemic i.e >5.0 with prevalence of 14.28% . This study given in accordance wtih ***Pritee Pendkar et al.*** which showed that Serum potassium in the diseased subjects was 5.16 ± 0.65 and in control subjects was 3.2 ± 0.38 with p < 0.05.[10]

Serum chloride in the diseased subjects was 104.83 ± 7.02 and with p <0.0001 which is statistically significant.

Serum creatinine in the diseased subjects was 5.82 ± 3.32 with p <0.0001 that is statistically significant. Similar study was performed by ***Suresh M et al.*** states Serum creatinine was highly increased in CKD patients 4.76±2.29 as compared to control subjects 0.95±0.22 with p <0.0001 that was statistically significant (p<0.05).[11]

Singh S et.al have found that Serum Urea (mg/dl) in diseased subjects was 123.15±49.68 and in control subjects was 24.40±8.46 with p <0.001 which is statistically

significant. In our study Serum urea in the diseased subjects was 130.39 ± 65.46 with p<0.0001 which is statistically significant.[12]

VI. CONCLUSION

In the present study the changes in the serum levels of sodium, potassium, chloride, creatinine and blood urea were monitored. Management of renal profile was done in order to monitor treatment in CKD patients.Biochemical parameters are deranged in patients with chronic kidney disease.Thus routine investigation of biochemical parameters of CKD patients especially serum creatinine to prevent complications of chronic kidney disease like cardiovascular diseases, dyslipidaemia, hyperkalaemia, metabolic acidosis, anaemia, and bone and mineral disorders is recommended. Correction of these abnormalities help to reduce the morbidity and mortality related to CKD. Chronic kidney disease should be considered a cardiovascular risk equivalent. It is imperative not only to aggressively employ therapeutic interventions to slow the progression of CKD, but also to identify those patients with the greatest need for aggressive CVD risk-factor reduction.

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