A Study on Renal Parameters among Patients with CKD

Swetha Sutrave¹, Sreedhar Dayapule², T. Sankar Narayana³, Eadala Suresh⁴, Neeraja Kunireddy⁵, Addanki yohoshuva⁶

¹Post Graduate, Department of Biochemistry, Siddhartha Medical College, Vijayawada, ²Associate Professor, Department of Urology and Renal Transplantation, Dr Pinnamaneni Siddhartha Institute of Medical sciences and Research Foundation, Gannavaram, Krishna(D), Andhra Pradesh, ³Assistant Professor, Department of Biochemistry, Siddhartha Medical College, Vijayawada, ⁴Senior Resident, Department of Biochemistry, Siddhartha Medical College, Vijayawada, ⁶Professor & HOD, Department of Biochemistry, Siddhartha Medical College, Vijayawada, ⁶Professor & HOD, Department of Biochemistry, Siddhartha Medical College, Vijayawada, ⁶Professor & HOD, Department of Biochemistry, Siddhartha Medical College, Vijayawada, ⁶Professor & HOD, Department of Biochemistry, Siddhartha Medical College, Vijayawada, ⁶Professor & HOD, Department of Biochemistry, Siddhartha Medical College, Vijayawada, ⁶Professor & HOD, Department of Biochemistry, Siddhartha Medical College, Vijayawada, ⁶Professor & HOD, Department of Biochemistry, Siddhartha Medical College, Vijayawada, ⁶Professor & HOD, Department of Biochemistry, Siddhartha Medical College, Vijayawada, ⁶Professor & HOD, Department of Biochemistry, Siddhartha Medical College, Vijayawada, ⁶Professor & HOD, Department of Biochemistry, Siddhartha Medical College, Vijayawada, ⁶Professor & HOD, Department of Biochemistry, Siddhartha Medical College, Vijayawada, ⁶Professor & HOD, Department of Biochemistry, Siddhartha Medical College, Vijayawada, ⁶Professor & HOD, Department of Biochemistry, Siddhartha Medical College, Vijayawada, ⁶Professor & HOD, Department of Biochemistry, Siddhartha Medical College, Vijayawada, ⁶Professor & HOD, Department of Biochemistry, Siddhartha Medical College, Vijayawada, ⁶Professor & HOD, Department of Biochemistry, Siddhartha Medical College, Vijayawada, ⁶Professor & HOD, Department of Biochemistry, Siddhartha Medical College, Vijayawada, ⁶Professor & HOD, Department of Biochemistry, Siddhartha Medical College, Vijayawada, ⁶

Abstract

Background: Chronic renal failure usually occurs over many years as the internal structures of the kidney are slowly damaged. In the early stages, there may be no symptoms. Progression may be so gradual that symptoms do not occur until kidney function is less than one-tenth of the normal. Uremia also causes hypothermia, which is believed to be due, in part to inhibition of the Na+ pump by some retained toxins. Dialysis usually returns body temperature to normal. It is often accompanied by impairment of carbohydrates, fats and proteins and defective utilization of energy. **Subjects and Methods:** A group of 72 individuals with marginally raised serum creatinine irrespective of their gender between 20 to 65 yrs were included in the study. Renal dysfunction was suspected in all of them. It's a prospective cross-sectional study conducted at the department of biochemistry. **Results:** Blood Urea levels were 12-102 mg/dl with mean 33.2+/-18.12 SD. the Total Proteins among the subjects varied in range of 4.5- 8gm/dl levels with mean of 6.58+/-0.69 SD. The Serum Albumin levels were 1.7 - 5gm/dl with mean of 3.70+/-0.71 SD, the blood glucose levels range among the subject were between 59 - 256 mg/dl where mean was about 108.59 +/-33.5 SD. **Conclusion:** The various parametric distribution among the subject where the creatine levels ranged from 0.6-1.6 mg/dl with mean of 1.03+/-0.23 SD. Blood Urea levels were 12-102 mg/dl with mean 33.2+/-18.12 SD.

Keywords: Chronic renal failure, Total Proteins, Albumin levels.

Corresponding Author: Dr. Sreedhar Dayapule, Associate Professor, Department of Urology and Renal Transplantation, Dr Pinnamaneni Siddhartha Institute of Medical sciences and Research Foundation, Gannavaram, Krishna(D), Andhra Pradesh.

Received: February 2019 Accepted: February 2019

Introduction

Chronic renal failure is a gradual and progressive loss of ability of the kidneys to excrete wastes, concentrate urine and conserve electrolytes.

Unlike the acute renal failure, with its sudden reversible failure of kidney function, chronic renal failure is slowly progressive. It often results from any disease that causes loss of kidney function. It can range from mild dysfunction to severe kidney failure. Progression may continue to end-stage renal disease.^[1]

Chronic renal failure usually occurs over many years as the internal structures of the kidney are slowly damaged. In the early stages, there may be no symptoms. Progression may be so gradual that symptoms do not occur until kidney function is less than one-tenth of the normal.

Chronic renal failure (CRF) and End Stage Renal Disease (ESRD) affects 2 out of 1000 people in the United States (USA). Diabetes and Hypertension are the two most frequent causes accounting for approximately two third of the cases of CRF and ESRD (10). Azotemia is elevated blood urea nitrogen (BUN > 28 mg/dl) and creatinine (Cr>1.5mg/dl). Uremia is azotemia with symptoms and signs of renal failure.^[2]

It is the term generally applied to the clinical syndrome that results from profound loss of renal function. Although the cause (s) of the syndrome remains unknown, the term uremia was adopted originally because of a presumption that the abnormalities result from retention in the blood of urea and other end products of metabolism typically excreted in the urine. Uremia involves more than renal excretory failure alone.^[3]

Impairments of the excretory function of the kidney result in an elevation in blood urea nitrogen (BUN), creatinine and various protein metabolic products. Impairment in the synthetic function results in the decrease in the production of erythropoietin (causing anemia) and active vitamin D3 (causing hypocalcemia secondary to hyperparathyroidism, hyperphosphatemia, and renal osteodystrophy.^[4]

Impairment in synthetic function also results in a reduction in acid, potassium, salt and water excretion (causing acidosis, hyperkalemia, hypertension, and edema). Elevated levels of plasma guanidosuccinic acid, by interfering with activation of platelet factor III by adenosine diphosphate (ADP), contribute to the impaired platelet function in CRF.^[5]

Uremia also causes hypothermia, which is believed to be due, in part to inhibition of the Na+ pump by some retained toxins. Dialysis usually returns body temperature to normal. It is often accompanied by impairment of carbohydrates, fats and proteins and defective utilization of energy. Ability to metabolize glucose is impaired in most patients with

Sutrave et al; Renal Farameters among Fatients with CKD

CRF. Fasting blood sugar levels are usually normal or slightly elevated. Because insulin is removed from the plasma largely by renal cells, which degrade it intracellularly, circulating insulin levels in plasma are slight to moderately increased in most fasting uremic subjects — the glucose intolerance of uremia results largely from peripheral resistance to the action of insulin.

The altered state of metabolism in uremia patients leads to abnormal amino acid profiles. In addition to the hypercatabolism seen in uremia, the capacity to eliminate nitrogenous end products of protein catabolism is reduced so that CRF may be regarded as a state of protein intolerance.

Hypertriglyceridemia decreased plasma levels of highdensity lipoprotein cholesterol, and increased plasma levels of lipoprotein-a antigen (LP (a) are common in uremia, where as cholesterol levels in plasma are usually normal. The high incidence of premature atherosclerosis in patients on chronic dialysis may be related in part to these abnormalities in lipid metabolism.^[6]

Hypertension is the most common complication of endstage renal failure. Since fluid overload is the major cause of hypertension in uremia, the normotensive state can usually be restored by aggressive ultrafiltration with dialysis. Nevertheless, because of hyperreninemia, some patients may remain hypertensive despite rigorous salt and water restriction and ultrafiltration. Rarely, patients develop accelerated or malignant hypertension, manifested by marked by elevated systolic and diastolic pressures, extreme encephalopathy hyperreninemia, seizures, retinal hemorrhages, and papilloedema - a high percentage of patients with left ventricular hypertrophy or dilated cardiomyopathy. These changes are thought to be related to prolonged hypertension. Uremia may develop pericarditis due to retention of metabolic toxins.

Subjects and Methods

A group of 72 individuals with marginally raised serum creatinine irrespective of their gender between 20 to 65 yrs were included in the study. Renal dysfunction was suspected in all of them. It's a prospective cross-sectional study conducted at the department of biochemistry. The exclusion criteria being patients with diabetes mellitus of any type, malignancy, infectious diseases, nephrotic syndrome, organ transplantation on dialysis, patients on immune suppression, contrast-induced nephropathy, and pregnancy.

Inclusion Criteria:

Most frequent causes of chronic kidney disease like:-

- Glomerulonephritis.
- Adult polycystic kidney disease.
- H/O hypertension.
- Interstitial nephritis
- Obstructive uropathy.

Exclusion Criteria:

- diabetic mellitus of any type,
- malignancy

- infectious diseases
- nephritic syndrome
- organ transplantation and on dialysis
- patients on immune suppression
- contrast induced nephropathy and
- pregnancy.
- who are not willing to enrol in the study.
- Patients with trauma.

Sample Collection:

After obtaining written and informed consent from the patients, serum samples were collected, under aseptic precautions from each person enrolled in this study.

The patients are classified into the CKD stage according to the American National Kidney Foundation guidelines using eGFR from MDRD formula; CKD stages 1–5 correspond to GFR.

Results

Table 1: Gender Distribution						
Gender	No., of Study Subjects					
Genuer	No.,	%				
Male	57	79.17%				
Female	15	20.83%				
Total	72	100.00%				

In this study total 72 subjects were included among them 57 were male and 15 were female, where 49 subjects are less than 50 yrs and rest are above 50 yrs.

	Table 2: Age and Gender Distribution							
ſ	Age	Male		Female		Total		
	(Yrs)	No.	%	No.	%	No.	%	
	<= 50	37	64.91%	12	80.00%	49	68.06%	
	> 51	20	35.09%	3	20.00%	23	31.94%	
ſ	Total	57	100.00%	15	100.00%	72	100.00%	
	Mean +/- SD	46.19	+/- 14.70	42.13	+/- 11.95	45.34	+/- 14.19	

Table 3: Renal parameters.						
Item	Range	Mean +/- SD				
Creatinine mg/dl	0.6 - 1.6	1.03 +/- 0.23				
B. Urea mg/dl	12 -102	33.29 +/- 18.12				
T.protein gm/dl	4.5 - 8	6.58 +/- 0.69				
S.Albumin gm/dl	1.7 - 5	3.70 +/- 0.71				
B.glucose mg/dl	59 - 256	108.59 +/- 33.55				
BTP mg/lt	0.09 - 1.61	0.53 +/- 0.34				
GFR(BTP) ml/min/1.73	39 - 161	67.26 +/- 20.54				
GFR (Cre) ml/min/1.73	41 - 150	80.94 +/- 24.16				

The various parametric distribution among the subject where the creatinine levels ranged from 0.6-1.6 mg/dl with mean of 1.03+/- 0.23 SD. Blood Urea levels were 12-102 mg/dl with mean 33.2+/- 18.12 SD. the Total Proteins among the subjects varied in range of 4.5- 8gm/dl levels with mean of 6.58+/- 0.69 SD. The Serum Albumin levels were 1.7 – 5gm/dl with mean of 3.70+/- 0.71 SD, the blood glucose levels range among the subjects were between 59 –

256 mg/dl where mean was about 108.59 +/- 33.5 SD . The GFR (creatinine) in ml/min/1.73 ranged from 41- 150 ml / min / 1.73 with mean of 80.94 +/- 24.16 ml/ min /1.73 SD .the range of BTP levels was about 0.09 – 1.61 mg / Lt with mean of 0.53+/- 0.34 SD. The GFR(BTP) ranged from 39 – 161 ml/ min with a mean of 67.26 +/- 20.54 SD.

Discussion

In this study total 72 subjects were included among them 57 were male, and 15 were female, where 49 subjects are less than 50 yrs, and rest are above 50 yrs.

The various renal functional parametric distribution among the subject was assessed, in which the creatinine levels ranged from 0.6-1.6 mg/dl with a mean of 1.03 ± 0.23 SD, The normal standard creatinine range as per Tietz textbook of clinical chemistry ranges from 0.9-1.3 mg/dl for male subjects and females 0.6-1.1 mg/dl .in this study group 57 males 11 male subjects had high normal levels of serum creatinine and six female subjects out of total 15 female subjects. In the study group, the blood urea levels were 12-102 mg/dl with mean 33.2+/- 18.12 SD The normal blood urea levels as per Tietz textbook of clinical chemistry ranges from 15-40 mg/dl. The total proteins among the subjects varied in the range of 4.5- 8gm/dl levels with the mean of 6.58+/- 0.69 SD and the normal range as per Tietz textbook of clinical chemistry ranges from 6.5-8.0 gm/dl. The Serum Albumin levels were 1.7 - 5 gm/dl with a mean of 3.70+/- 0.71 SD, and normal range as per Tietz textbook of clinical chemistry ranges from 3.5-5.2 mg/dl. The blood glucose levels range among the subjects was between 59 -256 mg/dl where mean was about 108.59 +/- 33.5 SD and normal range as per Tietz textbook of clinical chemistry range from 74-100mg/dl. The GFR (creatinine) in ml/min/1.73 ranged from 41- 150 ml / min / 1.73 with mean of 80.94 +/- 24.16 ml/ min /1.73 SD and the normal normal range as per Tietz textbook of clinical chemistry ranges from 90-130 ml / min / 1.73.

Creatinine is the most widely used endogenous marker of GFR in routine practice. Creatinine whose mol wt is a 113 Da is freely filtered at the glomerulus and also secreted in

urine by the proximal tubule.^[7,8] The serum creatinine alone fails to identify patients in the early stage of CKD, therefore NKF-K/DOQI guidelines recommend reporting an estimated GFR (eGFR), calculated from prediction formulas, in addition to the serum creatinine value.^[9,10]

Conclusion

The various parametric distribution among the subject where the creatine levels ranged from 0.6-1.6 mg/dl with mean of 1.03+/-0.23 SD. Blood Urea levels were 12-102 mg/dl with mean 33.2+/-18.12 SD.

References

- Kanaoka Y. et al, Cloning and crystal structure of hematopoietic prostaglandin D synthase. Cell 90: 1085–1095, 1997.
- Han F, et al; Induction of lipocalin-type prostaglandin D synthase in mouse heart under hypoxemia. Biochem Biophys Res Commun 385: 449–453, 2009.
- Eguchi Y,et al; Expression of lipocalin-type prostaglandin D synthase (beta-trace) in the human heart and its accumulation in the coronary circulation of angina patients. Proc Natl Acad Sci U S A 94: 14689–14694, 1997.
- Nagata N,:et al; De novo synthesis, uptake and proteolytic processing of lipocalin-type prostaglandin D synthase, beta-trace, in the kidneys. FEBS J 276: 7146–7158, 2009.
- 5. Matsuoka T,et al; Prostaglandin D2 as a mediator of allergic asthma.Science 287: 2013–2017, 2000.
- Beckmann CT, et al; Binding of biliverdin, bilirubin, and thyroid hormones to lipocalin-type prostaglandin D synthase. Biochemistry 38: 8006–8013, 1999).
- Gerhardt T, Pöge U, Stoffel-Wagner B, Klein B, Klehr HU, Sauerbruch T, Woitas RP.: Serum levels of beta-trace protein and its association to diuresis in hemodialysis patients. Nephrol Dial Transplant 23: 309–314, 2008.
- Lindström V, Grubb A, Alquist Hegbrant M, Christenson et al;. Different elimination patterns of beta-trace protein, beta2-microglobulin and cystatin C in hemodialysis, haemodiafiltration, and haemofiltration. Scand J Clin Lab Invest 68: 685–691, 2008.
- Tanaka T, Urade Y, Kimura H, Eguchi N, Nishikawa A; et al ; Lipocalin type prostaglandin D Synthase (beta –trace) is a newly recognized type of retinoid transporter. J Biol Chem. 272(25):15789-95; 1997.
- Zhou Y, Shaw N, Li Y, Zhao Y, Zhang R, Liu ZJ et al.: Structure-function analysis of human l-prostaglandin D synthase bound with fatty acid molecules. FASEB J 24: 4668–4677, 2010.

Copyright: © the author(s), 2019. It is an open-access article distributed under the terms of the Creative Commons Attribution License (CC BY 4.0), which permits authors to retain ownership of the copyright for their content, and allow anyone to download, reuse, reprint, modify, distribute and/or copy the content as long as the original authors and source are cited.

How to cite this article: Sutrave S, Dayapule S, Narayana TS, Suresh E, Kunireddy N, Yohoshuva A. A Study on Renal Parameters among Patients with CKD. Asian J. Med. Res. 2019;8(1):BC04-BC06. DOI: dx.doi.org/10.21276/ajmr.2019.8.1.BC2

Source of Support: Nil, Conflict of Interest: None declared.