

To Find Out Any Correlation Between The Measured Biochemical Parameters in Patients With CRF

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Abstract:

Background&Method: This study is done with an aim to find out any correlation between the measured biochemical parameters in patients with CRF. In this study, 200 people over the age of 20 including both sexes who had chronic renal failure were selected. These patients (subjects) were attending OPD and admitted to the ward of general medicine. In addition, 200 normal healthy individuals (control) over the age of 20, staff members, patient attendants, and volunteers were included as controls. The patients were diagnosed as chronic renal failure by the department of general medicine at index medical college hospital and research centre indore (MP) and Amaltas institute of medical sciences, Dewas (MP).

Result:Correlation between Urea and HS-CRP. We have found a significant positive correlation between both parameters. Correlation between Urea and Total Cholesterol. we have found a significant positive correlation between both parameters. Correlation between Urea and Homocysteine. we have found a significant positive correlation between both parameters.

Conclusion:The mean serum concentration of IL-6 of CRF patients was high (25.38 ± 17.20) in comparison of healthy control mean (2.15 ± 1.03). It has been found that the difference between them was also statistically significant p-value is <0.001 . The mean serum concentration Hs-CRP of CRF patients was high (12.09 ± 3.78) in comparison of healthy control mean (1.03 ± 0.12). It has been found that the difference between them was also statistically significant p-value is <0.001 . It should be enrolled a large number of patients. It should be cohort study with follow up of patients' clinical conditions for better establishment of these markers.

Keywords:correlation, biochemical & CRF.

Study Designed:Observational Study.

1. INTRODUCTION

Chronic kidney disease has become a global public health problem affecting the world nowadays, which increases the chances of mortality due to cardiovascular diseases. CKD is a disease resulting from the complexity of various physiological and metabolic functions of the

body, in which various complex harmful substances accumulate, which are very harmful to the body such as decreased functioning and lead to renal failure. Increased urea in blood is called 'uremia'. The main causes of uremia are due to impaired metabolism of amino acid lipid and Hcy. In addition, there is also malnutrition, inflammation and oxidative stress, anemia, vitamin D deficiency, muscle dysfunction, and lean body mass (LBM). Several other factors such as hypertension, diabetes mellitus, higher level of TC&TG, which are primarily responsible for CKD, result in persistent deterioration of kidney function, increasing the risk of CVD and mortality. Several researchers have also referred to 'cardiorenal syndrome' which suggests that kidney and cardiac system dysfunction can lead to progressive failure of both systems. (Adrian D Slee, 2012)[1]

Kidney function is used for the assessment of serum creatinine level in clinical medicine. GFR is assessment through creatinine clearance by serum creatinine levels and a timed urine sample additional creatinine is generally used to assessment creatinine clearance during CKD, rather serum creatinine has been weak predictor of GFR as its generation is and is influenced by multiple factors such as age, sex, BMI, bodyweight, muscle mass, diet, & drugs (Walser, 1998)[2]. GFR is a gold standard for assessment of kidney activity. The routinely applied equation is the Cockcroft Gault (CG) equation (Cockcroft and Gault, 1976)[3] and MDRD equations. Mostly medical practitioner utilizes MDRD for its ease of use on the internet, where one can just add values for age, weight, race, and sex to get a predictable GFR (Rule et al. 2006, Stevens et al. 2006) [3]. This method is time consuming and burdened with errors. There are number of equations present for the measurement of eGFR as listed above but following two equations are most commonly used for the estimation of eGFR.

Chronic renal failure (CKD) is characterized by developing destruction of renal parenchyma and the reduction of working nephrons, which are responsible for chronic kidney disease. Predictable universal burden of this illness is reported that due to kidney and urinary tract there are about 830,000 deceased annual, making them the 12th highest cause of deceased (1.4% of all deaths) (Kepler, 2010) [4]. This position is comparable across world bank regions, but among developing areas. It is notice that it suffers 1 in every 10 adults in India (Powe, 2003) [5]. In India pervasiveness of CRF find out around 17% along with 6% stage-3 CKD. As discussed in study the pervasiveness of CRF observed highest in Visakhapatnam and Andhra Pradesh about 46.8%, in Kanpur and Uttar Pradesh the pervasiveness of CRF was 41.7% and in Delhi it was 41% (Singh et al. 2013)[6]

2. MATERIAL & METHOD

The study was carried out at the department of Biochemistry at Index Medical College Hospital & Research Centre, Indore (MP) and Amaltas Institute Medical Sciences, Dewas (MP) after obtaining Institutional Ethics Committee permission. The present study was an observational, analytical with case control study. The selection of sample was done by convenient sampling.

STUDY GROUPS

In this study, 200 people over the age of 20 including both sexes who had chronic renal failure were selected. These patients (subjects) were attending OPD and admitted to the ward of general medicine. In addition, 200 normal healthy individuals (control) over the age of 20, staff members, patient attendants, and volunteers were included as controls. The patients were diagnosed as chronic renal failure by the department of general medicine at index medical college hospital and research centre indore (MP) and Amaltas institute of medical sciences, Dewas (MP).

The Participants were divided into two separate groups:

1. Group 1 (Subjects): Chronic Renal Failure Patients
2. Group 2 (Control): Healthy individuals

Inclusion Criteria

The study was included patients diagnosed by clinician, with known history of chronic renal failure, on dialysis, diabetes, hypertension, etc.

1. Chronic renal failure
2. Patients on Dialysis

Exclusion criteria

The study was excluded patient with known history of cardiac diseases, pregnancy, any infectious diseases, any surgical history, etc. Terminally ill patients, non-co-operative and non-willing patients.

Biochemical Investigation

Only blood sample collected in clinical biochemistry were used investigations. plasma use for glucose investigation, HbA1c were assessed in whole blood collected with EDTA, whereas Urea, creatinine, uric acid, albumin, lipid profile, CKMB, Homocysteine, Hs-CRP, IL-6 were assessing in serum as described below in detail.

Data Analysis

Determine the mean absorbance separately for duplicate samples, standards, and controls in which the duplicate average was within 20%, as well as the amount of IL-6 for all samples against their IL-6 standard concentration using a standard curve. The OD is determined by extrapolating the values.

3. RESULTS

Table-01: Gender wise Distribution of subjects and control

Sex	Subjects	Control
Male	110	111
female	90	89

Table shows distribution of subjects and controls as per Sex. We have more male population instead of female so, we have register more male for this study.

Table-02: Religion wise distribution of subjects and control

Religion	Subjects	Controls
Hindu	169	157
Muslim	26	33
Other	05	10

Table shows religion wise distribution of subjects and controls, we are having more Hindu for the study.

Table-03: Correlations between UREA and HbA1c in Subjects

Subjects	Parameters	r-value
	UREA 157.60±55.02	0.18046
	HbA1c 9.04±2.83	

Table shows correlation between Urea and HbA1c. we have found a significant positive correlation between both parameters.

Table-04: Correlations between Urea and CPK-MB in Subjects

Subjects	Parameters	r-value
	UREA 157.60±55.02	0.065884
	CPK-MB 41.91±16.80	

Table shows correlation between Urea and CPK-MB. we have found a significant positive correlation between both parameters.

Table-05: Correlations between Urea and IL-6 in Subjects

Subjects	Parameters	r-value
	UREA 157.60 ±55.02	0.082926
	IL-6 25.38±21.90	

Table shows correlation between Urea and IL-6. we have found a significant positive correlation between both parameters.

Table-06: Correlations between Urea and Hs-CRP in Subjects

Subjects	Parameters	r-value
	UREA 157.60±55.02	0.062816
	Hs-CRP 6.68 ± 3.9	

Table shows correlation between Urea and HS-CRP. We have found a significant positive correlation between both parameters.

Table-07: Correlations between Urea and HDL in Subjects

Subjects	Parameters	r-value
	UREA 157.60 ± 55.02	-0.18528
	HDL 29.82 ± 7.3	

Table shows correlation between Urea and HDL. we have found a significant negative correlation between both parameters.

Table-08: Correlations between Urea and Total Cholesterol in Subjects

Subjects	Parameters	r-value
	UREA 157.60±55.02	0.070189
	Total Cholesterol 331.73±71.80	

Table shows correlation between Urea and Total Cholesterol. we have found a significant positive correlation between both parameters.

Table-09: Correlations between Urea and Homocysteine in Subjects

Subjects	Parameters	r-value
	Urea 157.60±55.02	0.246588
	Homocysteine	

Table shows correlation between Urea and Homocysteine. we have found a significant positive correlation between both parameters.

4. DISCUSSION

IL-6 -: This study shows IL-6 level in subjects and controls. The mean value was 78.20±10.78 and 2.15±1.03 in subjects and controls. IL-6 level of male subjects and controls was 21.86±13.88 and 1.1±0.54, respectively. Serum IL-6 mean and SD in female subjects. The mean was 29.67±8.36 and 1.01±0.53, respectively for subjects and controls. During comparisons of this parameter in MS- excel for t-test we have found a significant p- value for the same. It was < 0.001. We have found a positive correlation between Urea and IL-6. (r=0.082926)

our study is also correlate with the other studies; they have found high level of IL-6. Our study shows similarity with Maura IC, et al. 2005 [8] that higher level of IL-6 levels were mostly found in CKD due to oxidative stress, chronic inflammation and as a result of fluid overload. IL-6 accumulation due to renal impairment because it is not properly excreted, especially HD & PD stimulate more inflammatory responses that increase the synthesis of IL-6. Not only does it exacerbate kidney injury, it also accelerates the progression of CKD by initiating its complications, especially chronic vascular disease. IL-6 primarily exhibits endothelial nitric oxide synthase and adiponectin expression that increases the risk of atherosclerosis. contributes to the increasing incidence of Elevated IL-6 level not only serves lead to progression of CKD.

Homocysteine -:It shows mean level of homocysteine 29.13 ± 5.14 and 6.03±1.19, in subjects and controls. There was observed statistically significant difference between the two groups. (p<0.001). Mean SD for serum homocysteine in male subjects was 20.74±9.16 and 9.51±2.80 in controls, respectively. SerumHomocysteine mean and SD in female subjects. The mean was 22.45 ±11.1 and 8.40 ± 2.90, respectively for subjects and controls. During comparisons of this parameter in MS- excel for t-test we have found a significant p- value for the same. It was < 0.001. We have found a positive correlation between Urea and Homocysteine(r=0.246588).

Our study was confirming the finding of van Guldener C, et al. 2000)[8]In patients with kidney failure, hyperhomocysteinemia is a common phenomenon. The underlying pathophysiological activity of which is unknown. In addition, proposed mechanisms include reduced renal elimination and impaired non-disposal of hcy, possibly due to inhibition of enzymes important in methioninehcy metabolism by the uremic milieu. Adeficiency of folate, pyridoxine or cyanocobalamins may also play an important role.

Other studies have a lot in common with our study. Several previous studies have found an increase in homocysteine levels. These studies were conducted onpre-dialysis and ESRD, and to a lesser extent atherothrombotic disease. In addition, the level of total homocysteine increases with the severity of the disease(Boston AG, et al. 1995, Robinson K, et al. 1996, Bachman J, et al. 1995) [9&10].

Two reports have documented dialysis patients with less to intermediated hyperhomocysteinemia age-, sex- and race-matched population-based. Controls who are free of clinical kidney disease, and whose serum creatinine levels were 1.5 mg/dL. Thereby it's highly related with hyperhomocysteinemia occurred in 83% of the patients, with a 105-timelhigher risk to controls. (Hultberg B, et al. 1993)[11].

5. CONCLUSION

The mean serum concentration of IL-6 of CRF patients was high (25.38 ± 17.20) in comparison of healthy control mean (2.15 ± 1.03). It has been found that the difference between them was also statistically significant p-value is <0.001 .

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6. REFERENCES

- [1] Adrian D Slee: Exploring metabolic dysfunction in chronic kidney disease. *Nutrition & Metabolism* 2012 9:36.
- [2] Walser, M. et al. Assessing renal function from creatinine measurements in adults with chronic renal failure. *American journal of kidney diseases*, 1998, 32(1), 23-31.
- [3] 156. Rule, A. D., Rodeheffer, R. J., Larson, T. S., Burnett Jr, J. C., Cosio, F. G., Turner, S. T., & Jacobsen, S. J. et al. Limitations of estimating glomerular filtration rate from serum creatinine in the general population. In *Mayo Clinic Proceedings* (2006, November) (Vol. 81, No. 11, pp. 1427-1434). Elsevier.
- [4] Kepler, J. et al. international comparisons. United States Renal Data System. 2010 Annual Data Report: atlas of chronic kidney disease and end-stage renal disease in the United States, (2010) 2.
- [5] Powe, N. R. et al. To have and have not: health and health care disparities in chronic kidney disease. *Kidney international*, (2003) 64(2), 763- 772.
- [6] 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.
- [7] Moura IC, Arcos-Fajardo M, Gdoura A, Leroy V, Sadaka C, Mahlaoui N, et al. Engagement of transferrin receptor by polymeric IgA1: evidence for a positive feedback loop involving increased receptor expression and mesangial cell proliferation in IgA nephropathy. *J Am SocNephrol* (2005) 16:2667–76.
- [8] Van Guldener C, Robinson K. Homocysteine and renal disease. *SeminThrombHemost*. 2000;26(3):313-24.
- [9] Bostom AG, Shemin D, Lapane KL, miller JW, Sutherland P, Nadeau M, Seyoum E, Hartman W, Prior R, wilson PW: Hyperhomocysteinemia and traditional cardiovascular disease risk factors in end-stage renal disease patients on dialysis: A case-control study. *Atherosclerosis* 1995, 114: 93-103.
- [10] Robinson K, Gupta A, Dennis V, Arheart K, Chaudhary D, Green R, Vigo P, Mayer EL, Selhub J, Kutner M, Jacobsen DW: Hyperhomocysteinemia confers an independent increased risk of atherosclerosis in end-stage renal disease and is closely linked to plasma folate and pyridoxine concentrations. *Circulation* 1996, 94:2743 -2748.

- [11] Hultberg B, Andersson A, Sterner G: Plasma homocysteine in renal failure. ClinNephrol 1993, 40:230 -235.