Section: Biochemistry

Research

STATUS OF MINERALS, UREA, URIC ACID AND CREATININE IN CHRONIC KIDNEY FAILURE IN A TERTIARY CARE TEACHING HOSPITAL

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Abstract

Background: Renal failure has now become a public health problem. According to a study, the prevalence of CKD in India is 13 -15.04 percent. CRF is linked to diabetes and hypertension as risk factors. Diabetes and hypertension account for 40-60% of CRF cases in India. **Materials and Methods:** A total of (76) seventy-six subjects in the age range of 32 to 73 years were chosen at random and informed consent was obtained from all of them. Thirty-eight people served as controls, with thirty-eight clinically confirmed instances of chronic renal failure (creatinine > 7.0 mg/dl) serving as cases. **Result:** When comparing patients to controls, RBS, creatinine, urea, uric acid, magnesium, phosphorous, and potassium levels were considerably higher (p 0.05). When comparing patients to controls, calcium, iron, and salt levels were considerably lower (p 0.05). In CRF patients with hypertension, RBS, magnesium, calcium, phosphorus, iron, and potassium were lower than in CRF subjects with hypertension and DM. Creatinine, urea, and salt levels were higher in CRF patients with hypertension compared to CRF subjects with both hypertension and diabetes, although the differences were not statistically significant (p-value > 0.05). **Conclusion:** This study was effective in CRF patients who have high serum phosphate and magnesium levels but low serum calcium and iron levels. The study's scope was to expand to include serum phosphorus, magnesium, serum calcium, and iron in the renal profile in the future.

INTRODUCTION

Chronic Kidney Disease (CKD) is characterized by a progressive loss of renal function.[1] Infections, autoimmune illnesses, diabetes, hypertension, cancer, and toxic substances can all cause the kidneys to lose their normal function, notably excretory and regulatory functions.[2] Chronic renal failure is on the verge of becoming a significant public health issue and is fast gaining pandemic proportions over the world.[3,4] Renal failure has now become a public health concern. According to a study, the prevalence of CKD in India is 13 -15.04 percent. CRF is linked to diabetes and hypertension as risk factors. Diabetes and hypertension account for 40-60% of CRF cases in India.[5] A steady decline in kidney function causes end-stage renal disease (ESRD). Chronic renal failure is characterised by a progressive decrease of renal function, regardless of the underlying aetiology of the kidney illness. The primary aetiology of CRF has changed away from glomerulonephritis and interstitial nephritis and toward atherosclerosis and diabetic nephropathy. Progressive glomerulopathies cause a rapid and permanent decrease of kidney function. The constant drop in the glomerular filtration rate causes changes in renal parameters and minerals in the blood in chronic renal failure. According to Kidney Disease Improving Global Outcomes, a GFR of less than 60 ml/minute/1.73 m2 is an indication of CKD (KDIGO). CKD is classified into five phases. GFR is less than 15 ml/minute in stage five of CKD.[6] In stage five CKD, serum creatinine is greater than 5.0 mg/dl in men and greater than 4.0 mg/dl in women.[7] Albuminuria and an albumin-to-creatinine ratio of more than 30 mg/g3 can be used to diagnose renal disorders that induce kidney damage in urine samples. Hypertension damages the blood arteries in the kidney, causing waste materials to accumulate.[8] Creatinine is an anhydride derivative of creatine that is excreted. Glycine, arginine, and methionine make

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up the tripeptide creatine. Urea and creatinine are compounds made from arginine, an amino acid found in the liver (urea cycle) and the kidney, where it is predominantly eliminated through glomerular filtration. These compounds are retained as the number of working nephrons decreases, and their concentration in the blood grows.[9] The majority of kidney ailments progress proportionately free of the original disease to end-stage renal failure. Chronic renal failure causes water, electrolyte, and mineral abnormalities.[10] Minerals are required for the normal growth and function of the human organism. Minerals such as sodium, potassium, calcium, magnesium, iron, and phosphorus are all common. Patients with chronic renal failure should adjust their diets based on the amount of minerals in their bodies. When the glomerular filtration rate (GFR) falls below 20 – 30 mL/min, the amount of serum magnesium rises.[11] Magnesium homeostasis is maintained by intestine absorption and renal excretion. Filtration and reabsorption are used to determine magnesium excretion. The plasma magnesium content has a big impact on how magnesium is handled by the kidneys. In hypermagnesemia, fractional excretion of magnesium is high, while in hypomagnesemia, it is low.[12] Patients with chronic kidney disease (CKD) have CKD-mineral bone disorder (CKDMBD). where there is a significant disruption in bone and mineral metabolism for normal muscular irritability, sodium is required. Sodium aids in the maintenance of blood volume and the regulation of blood pressure. Patients with chronic renal failure have high salt levels, which not only raises blood pressure but also promotes fluid retention in the body. The purpose of this study would be to see how indispensable minerals were in chronic renal failure before dialysis.[13]

MATERIALS AND METHODS

This is a case-control study was conducted in the Department of Biochemistry, G S V M Medical College and Hospital, Kanpur in collaboration with Nephrology department during the period from September, 2020 from June,2021. After obtaining approval from the GSVM Medical College and Hospital's Institutional Ethical Committee, a total of (76) seventy-six subjects in the age range of 32 to 73 years were chosen at random and informed consent was obtained from all of them. Thirty-eight people served as controls, with thirty-eight clinically confirmed instances of chronic renal failure (creatinine > 7.0 mg/dl) serving as cases. 7 mL of venous blood was taken from the patients and left to stand for 30 minutes before being centrifuged at 3000 RPM for 5 minutes and the samples analysed. RBS, creatinine, urea, uric acid, sodium, potassium, magnesium phosphorous, iron, and calcium were measured using an Erba EM 200 Automated Chemistry Analyzer. RBS was determined using the Hexokinase method, Creatinine was determined using the Jaffe's method, Urea was determined using the GLDH method, Sodium and Potassium were determined using the ISE Direct method, Magnesium was determined using the Xylidyl blue method, Phosphorous was determined using the Molybdate UV method, Iron was determined using the TPTZ method, and Calcium was determined using the Arsenazo method. The study included hypertensive individuals, diabetic patients, and patients with chronic renal failure. Healthy controls were people who went to the GSVM hospital's outpatient department for a health checkup and were included in the study. Thyroid diseases, liver disorders, cardiac patients, and dialysis patients were all excluded from the study. The t-test was used to analyse the data. Significant was determined as a p-value of less than 0.05.

RESULTS

The patients ranged in age from 32 to 73 years old. The comparison of RBS, creatinine, urea, sodium, potassium, magnesium, phosphorous, iron, and calcium parameters between controls and cases (CRF) revealed a statistically significant difference in all parameters, p-value 0.05 [Table 1].

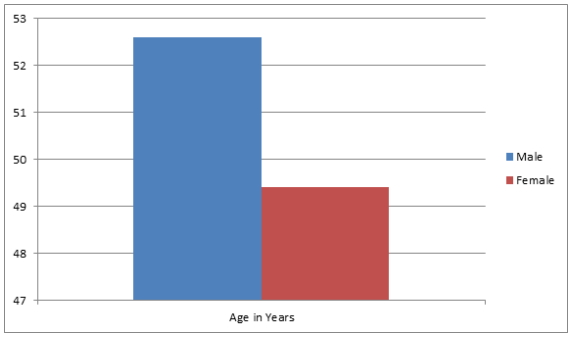

Figure 1: Shows the mean age in years a/c to Gender.

A comparison of CRF patients by gender in dialysis patients revealed no statistically significant differences in any of the parameters (p-value > 0.05) in [Table2, Figure 1]. Shows the RBS, creatinine, urea, uric acid, sodium, potassium, magnesium, phosphorus, iron, and calcium parameters in CRF with hypertension versus CRF with both hypertension and DM patients showed statistically no significant difference (p-value > 0.05).

When comparing patients to controls, RBS, creatinine, urea, uric acid, magnesium, phosphorous, and potassium levels were considerably higher (p 0.05). When comparing patients to controls, calcium, iron, and salt levels were considerably lower (p 0.05). In CRF patients with hypertension, RBS, magnesium, calcium, phosphorus, iron, and potassium were lower than in CRF subjects with both hypertension and DM. Creatinine, urea, and salt levels were higher in CRF patients with hypertension compared to CRF subjects with both

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hypertension and diabetes, although the differences were not statistically significant (p-value > 0.05).

Table 1: Biochemical markers in the controls and patients were compared.

Variables	Group A Mean ± SD	Group B Mean ± SD	P-Value <0.05
RBS (mg/dl)	86.4 ± 16.04	138.1 + 53.3 *	0.01
Creatinine (mg/dl)	0.61 ± 0.02	12.4 ±2.48 *	0.04
Urea (mg/dl)	30.5 ± 7.42	187.2 ± 29.7 *	0.001
Uric acid(mg/dl)	4.1 ± 1.25	12.27 ±3.7 *	0.02
Magnesium (mg/dl)	2.4 ± 0.06	3.24 ± 0.07 *	0.03
Calcium (mg/dl)	10.2 ± 3.2	6.24 ± 2.54*	0.05
Phosphorous (mg/dl)	2.6 ± 0.6	7.26 ± 1.04*	0.02
Iron (µg/dL)	126.6 ± 22.4	42.3 ± 12.23 *	0.04
Sodium (mEq/L)	140.6 ± 28.5	131.48 ± 24.7 *	0.001
Potassium (mEq/L)	4.5 ± 0.9	4.27 ± 0.57 *	0.04

Table 2: Comparison of biochemical variables in CRF subjects based on gender

Variables	Females (13) Mean ± SD	Males (25) Mean ± SD	P Value > 0.05
Age in years	49.4 ± 10.4	52.6 ± 11.52	0.51
RBS (mg/dl)	156.7 ± 29.41	124.7 ± 14.3	0.32
Creatinine (mg/dl)	13.2 ± 2.31	11.6 ± 2.04	0.42
Urea (mg/dl)	195.6 ± 31.05	186.3 ± 30.2	0.65
Uric acid(mg/dl)	12.07 ±2.6	12.09 ±2.67	0.53
Magnesium (mg/dl)	2.98 ± 0.34	3.27 ± 1.05	0.45
Calcium (mg/dl)	7.67 ± 1.32	8.45 ± 2.6	0.14
Phosphorous (mg/dl)	8.26 ± 2.02	7.6 ± 1.93	0.26
Iron (µg/dL)	39.7 ± 10.2	45.7 ± 11.8	0.15
Sodium (mEq/L)	133.8 ± 25.27	130.5 ± 05.79	0.54
Potassium (mEq/L)	4.84 ± 1.02	4.59 ±1.23	0.11

Table 3: Comparison of biochemical variables in CRF patients with Hypertension and both Hypertension and DM cases

Variables	CRF with HTN (13) Mean ± SD	CRF with HTN + DM (18) Mean ± SD	P Value > 0.05
RBS (mg/dl)	106.4 ± 18.4	164.5 ± 28.4	0.21
Creatinine (mg/dl)	11.2 ± 3.06	10.21 ± 34.4	0.36
Urea (mg/dl)	188.6 ± 25.7	170.6 ± 24.3	0.51
Uric acid(mg/dl)	12.7 ±2.3	12.5 ± 2.04	0.14
Magnesium (mg/dl)	2.9 ± 0.9	3.6 ± 1.02	0.22
Calcium (mg/dl)	7.68 ± 1.3	7.98 ± 0.43	0.35
Phosphorous (mg/dl)	6.1 ± 2.23	7.87 ± 2.31	0.42
Iron (µg/dL)	41.4 ± 9.6	43.2 ± 9.3	0.16
Sodium (mEq/L)	133.2 ± 24.6	131.2 ± 23.5	0.64
Potassium (mEq/L)	4.7 ± 1.02	5.1 ± 1.9	0.26

DISCUSSION

The incidence and prevalence of End Stage Renal Disease (ESRD) have steadily increased over the last three decades.[14] The fastest-growing causes of CKD patients include the rapidly rising global incidences of diabetes and hypertension.[15] According to a recent estimate, 285 million people aged 20 to 79 years old worldwide (6.6 percent) had diabetes in 2010, and 438 million people (7.8%) of the adult population are predicted to get diabetes by 2030.[16] Diabetes prevalence is estimated to rise from 31.7 million in 2000 to 79.4 million in 2030 in India alone.[17] About a third of people who are affected will see their renal function deteriorate with time.[18] According to worldwide data on the global burden of hypertension, 20.6 percent of Indian men and 20.9 percent of Indian women had hypertension in 2005, and rates of hypertension are anticipated to rise to 22.9 percent and 23.6 percent for Indian men and women, respectively, by 2025.[19] Blood pressure regulation is well acknowledged as a key element in delaying the progression of CKD and preventing its main complications, ESRD and cardiovascular disease.[20] Because the kidneys are the most commonly damaged organ in hypertension, even in normotensives, long-term exposure to elevated blood pressure might cause early renal damage.[21] Chronic renal failure is a progressive condition that causes an irreversible decrease in glomerular filtration rate, which leads to an increase in blood urea and serum creatinine levels.[22] Hypertension, diabetes, autoimmune diseases, and other factors are the most common causes of chronic renal failure. Reduced kidney function is linked to a number of biochemical problems, such as electrolytes, calcium, and phosphorus levels in the blood. When comparing cases to controls, we found a substantial increase in serum glucose, creatinine, and urea levels in patients (p-value 0.05). Other research undertaken by other authors yielded similar results.[10,23] In this study, there is a substantial increase in serum potassium, magnesium, and phosphorous levels in cases (p 0.05) when compared to controls. The findings were consistent across research undertaken by different authors.[24] In CRF,

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hyperkalemia is caused by a reduction in renal excretion, which could be owing to leakage from the intracellular space or a defective thirst mechanism. Hypermagnesemia in chronic renal failure is caused by a reduction in magnesium excretion by the kidneys, which is a precursor to renal impairment. In chronic renal failure, increased serum phosphorus levels compensate for the skeleton's loss of reservoir function.[25] When comparing cases to controls, serum sodium, calcium, and iron levels were found to be considerably lower (p 0.05) in patients. Reduced serum sodium levels in CRF due to renal dilution and concentration management issues.[26] Low plasma calcitriol (1,25 dihydroxy cholecalciferol) levels, which are generated in the kidney from 25-OH cholecalciferol by the activity of the enzyme 1-α-hydroxylase, result in decreased intestinal calcium absorption. Poor intake and reduced absorption from the gut cause low iron levels in advanced chronic renal failure, resulting in a negative iron balance. Anemia is frequent in chronic renal failure because erythropoietin (EPO), which aids in the synthesis of red blood cells from the bone marrow, is not produced in sufficient amounts due to kidney impairment. Chronic renal failure patients have higher serum urea and creatinine levels, which can lead to a variety of other serious conditions,[23] such as heart and blood vessel disease. Mineral bone disease causes high serum phosphate and decreased serum calcium in chronic renal failure.[27] In contrast to female CRF participants, we found a rise in blood calcium mean value 8.45 2.6 (mg/dl) and a decrease in serum potassium mean value 4.59.23 (mEq/L) in male CRF subjects, with no statistical significance. In research conducted by various authors, the same results in serum calcium mean value 8.26 1.18 (mg/dl) and serum potassium mean value 4.73 1.04 (mEq/L) were discovered.[28] We found higher serum creatinine 11.2 3.06 (mg/dl) in CRF patients with hypertension compared to CRF patients with both hypertension and DM serum creatinine 10.21 34.4 (mg/dl) in our study, although there was no statistical significance. The same findings were observed in a number of studies by different authors.[29]

CONCLUSION

In conclusion, this research is effective in CRF patients who have high serum phosphate and magnesium levels but low serum calcium and iron levels. The study's scope was to expand to include serum phosphorus, magnesium, serum calcium, and iron in the renal profile in the future.

REFERENCES

1. Pandya D, Nagrajappa AK, Ravi KS. Assessment and Correlation of Urea and Creatinine Levels in Saliva and Serum of Patients with Chronic Kidney Disease, Diabetes and Hypertension- A Research Study. J Clin Diagn Res. 2016;10(10):ZC58-ZC62. doi: 10.7860/JCDR/2016/20294.8651.
2. Abdulla HI, Al-Kotany MY, Mahdi KA. Assessment of oral manifestations of patients with renal failure undergoing hemodialysis by serum and salivary biomarkers. MDJ. 2012;9(1):118-29.
3. Narula AS. Chronic Kidney Disease: The Looming Threat. Med J Armed Forces India. 2008;64(1):2-3. doi: 10.1016/S0377-1237(08)80134-7.
4. Anupama YJ, Uma G. Prevalence of chronic kidney disease among adults in a rural community in South India: Results from the kidney disease screening (KIDS) project. Indian J Nephrol. 2014;24(4):214-21. doi: 10.4103/0971-4065.132990.
5. Varma PP. Prevalence of chronic kidney disease in India - Where are we heading? Indian J Nephrol. 2015;25(3):133-5.
6. Levey AS, Eckardt KU, Tsukamoto Y, Levin A, Coresh J, Rossert J, et al. Definition and classification of chronic kidney disease: a position statement from Kidney Disease: Improving Global Outcomes (KDIGO). Kidney Int. 2005;67(6):2089-100. doi: 10.1111/j.1523-1755.2005.00365.x.
7. Couchoud C, Pozet N, Labeeuw M, Pouteil-Noble C. Screening early renal failure: cut-off values for serum creatinine as an indicator of renal impairment. Kidney Int. 1999;55(5):1878-84. doi: 10.1046/j.1523-1755.1999.00411.x.
8. Santulli G, Trimarco B, Iaccarino G. G-protein-coupled receptor kinase 2 and hypertension: molecular insights and pathophysiological mechanisms. High Blood Press Cardiovasc Prev. 2013;20(1):5-12. doi: 10.1007/s40292-013-0001-8.
9. Kurzer F, Senderson PM. Urea in the history of organic chemistry. J Chemi Educ. 1956;33(9):452–459.
10. Johnson RJ, Nakagawa T, Jalal D, Sánchez-Lozada LG, Kang DH, Ritz E. Uric acid and chronic kidney disease: which is chasing which? Nephrol Dial Transplant. 2013;28(9):2221-8. doi: 10.1093/ndt/gft029.
11. Saha HH, Harmoinen AP, Pasternack AI. Measurement of serum ionized magnesium in CAPD patients. Perit Dial Int. 1997;17:347– 352.
12. Navarro-González JF, Mora-Fernández C, García-Pérez J. Clinical implications of disordered magnesium homeostasis in chronic renal failure and dialysis. Semin Dial. 2009;22(1):37-44. doi: 10.1111/j.1525-139X.2008.00530.x.
13. Hill Gallant KM, Spiegel DM. Calcium Balance in Chronic Kidney Disease. Curr Osteoporos Rep. 2017;15(3):214-221. doi: 10.1007/s11914-017-0368-x.
14. Schoolwerth AC, Engelgau MM, Hostetter TH, Rufo KH, Chianchiano D, McClellan WM, et al. Chronic kidney disease: a public health problem that needs a public health action plan. Prev Chronic Dis. 2006;3(2):A57.
15. 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. doi: 10.1186/1471-2369-14-114.
16. Ramchandran A, Das AK, Joshi SR, Yajnik CS, Shah S, Kumar KMP. Current status of diabetes in India and need for novel therapeutic agents. JAPI. 2010;58:7-9.
17. Olokoba AB, Obateru OA, Olokoba LB. Type 2 diabetes mellitus: a review of current trends. Oman Med J. 2012;27(4):269-73. doi: 10.5001/omj.2012.68.
18. Mittal A, Sathian B, Kumar A, Chandrasekharan N, Sunka A. Diabetes mellitus as a potential risk factor for renal disease among napalese: A hospital based case control study. Niger J Eng. 2010;1(1):22-25.
19. Anchala R, Kannuri NK, Pant H, Khan H, Franco OH, Di Angelantonio E, et al. Hypertension in India: a systematic review and meta-analysis of prevalence, awareness, and control of hypertension. J Hypertens. 2014;32(6):1170-7. doi: 10.1097/HJH.0000000000000146.
20. Coresh J, Wei GL, McQuillan G, Brancati FL, Levey AS, Jones C, et al. Prevalence of high blood pressure and elevated serum creatinine level in the United States: findings from the third National Health and Nutrition Examination

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Survey (1988-1994). Arch Intern Med. 2001;161(9):1207-16. doi: 10.1001/archinte.161.9.1207.
21. Schillaci G, Reboldi G, Verdecchia P. High-normal serum creatinine concentration is a predictor of cardiovascular risk in essential hypertension. Arch Intern Med. 2001;161(6):886-91. doi: 10.1001/archinte.161.6.886.
22. Tomás I, Marinho JS, Limeres J, Santos MJ, Araújo L, Diz P. Changes in salivary composition in patients with renal failure. Arch Oral Biol. 2008;53(6):528-32. doi: 10.1016/j.archoralbio.2008.01.006.
23. ul Amin N, Mahmood RT, Asad MJ, Zafar M, Raja AM. Evaluating Urea and Creatinine Levels in Chronic Renal Failure Pre and Post Dialysis: A Prospective Study. J Cardiovasc Dis. 2014;2(2):1-4.
24. Cunningham J, Rodríguez M, Messa P. Magnesium in chronic kidney disease Stages 3 and 4 and in dialysis patients. Clin Kidney J. 2012;5(Suppl 1):i39-i51. doi: 10.1093/ndtplus/sfr166.
25. Macdougall IC, Bircher AJ, Eckardt KU, Obrador GT, Pollock CA, Stenvinkel P, et al. Iron management in chronic kidney disease: conclusions from a "Kidney Disease: Improving Global Outcomes" (KDIGO) Controversies Conference. Kidney Int. 2016;89(1):28-39. doi: 10.1016/j.kint.2015.10.002.
26. Lim LM, Tsai NC, Lin MY, Hwang DY, Lin HY, Lee JJ, et al. Hyponatremia is Associated with Fluid Imbalance and Adverse Renal Outcome in Chronic Kidney Disease Patients Treated with Diuretics. Sci Rep. 2016;6:36817. doi: 10.1038/srep36817.
27. Hruska KA, Mathew S, Lund R, Qiu P, Pratt R. Hyperphosphatemia of chronic kidney disease. Kidney Int. 2008;74(2):148-57. doi: 10.1038/ki.2008.130.
28. Paudel YP, Dahal S, Acharya T. Biochemical profile of chronic kidney disease patients in various age and gender group subjects visiting KIST medical college & teaching hospital Kathmandu. J Chitwan Med Coll. 2013;3(4):36–39.
29. Verma A, Vyas S, Agarwal A, Abbas S, Agarwal DP, Kumar R. Diabetic Kidney Disease and Hypertension: A True Love Story. J Clin Diagn Res. 2016;10(3):OC11-3. doi: 10.7860/JCDR/2016/18806.7511.

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