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STUDY OF BIOCHEMICAL MARKERS IN PATIENTS OF CHRONIC KIDNEY DISEASE

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ABSTRACT

Aim: To study Serum Urea, Creatitine, Calcium and Phosphorus in all stages of chronic kidney disease patients. **Material And Methods**: In this study 175 of CKD patients. (35 patients of each stage) in the age group of 20-60 yrs, in which 117 Male & 58 Female will be included. will be included. Estimation of Serum Urea, Creatitine, S.Calcium and Phosphorus are done on AUTOQUANT 400i (Meril) fully Autoanalyzer. **Results**: S.Urea, S.Creatitine & S.Calcium Mean±SD values in I to V stages in CKD patients. S.Urea is significant in all stages ($p<0.00$) increase values in as compaired to I to V stages. S.Creatitine & S.Phosphorus in I to V stages is not significant. S.Calcium in I to V stages is significant ($p<0.05$). S.Urea, S.Creatitine, S.Calcium & S.Phosphorus values are compaired with standard and controls of kit. **Conclusion**: The bone mineral metabolism abnormalities start during 1st stage of CKD as renal furcation decreases long before the need for renal replacement therapy and can be positively or negatively influenced by the treatment. It is recommended that attending physicians monitor and control biochemicals parameters early in the development of CKD before the need for dialysis.

KEYWORD: S.Urea, S.Creatitine, S. Calcium, s. Phosphorous.

I. INTRODUCTION

Chronic kidney disease (CKD) has become a mjour health problem worldwide.[1] National Kidney foundation " Kidney disease outcomes quality initiative (KDOQI) defines CKD is characterized by a gradual decline kidney function which causes kidney damage.[2] Chronic kidney disease develops over months or years leads eventually to end stage renal failure. Kidney amage indicated by persistant proteinuria, haematuria & anatomical abnormality.(2) Decreased kidney function indicated by a glomerular filteration rate (GFR) of less than 60 ml/min/1.73m^2 present for 3 or more months[2], CKD induces a slow and progressive decline of kidney function enhanced by various factors including infections, auto immune diseases, diabetes and other endocrine disorders, cancer, and toxic chemicals.[3] It is usually a result of complications arising from other serious medical conditions. Unlike acute renal failure, which happens quickly and suddenly, chronic renal failure occurs gradually - over a period of weeks, months, or years - as the kidneys slowly stop working, leading to an end-stage renal disease (ESRD).[2,3]

The most important complication is cardiovascular morbidity/mortality associated with CKD. The high mortality rate among patients in initial stages of CKD who finally requires dialysis. The kidney and the skeleton have intimate biological relationships that can affect bone strength as well as renal physiological functions.[3] High blood pressure is one of the leading causes of kidney failure. It may also damage the blood vessels in the kidney affecting the secretion of waste products. Waste may secrete extra cellular fluids and further raise the blood pressure eventually leading to ESRD.[4,5,7] Dialysis improves many symptoms of kidney failure, but some problems including hypertension, anemia and itch often require additional drug treatments as well. The progression of kidney damage is marked by the rise in two important chemical substances in the blood - creatinine and urea whose evaluation in serum helps to assess Glomerular Filtration Rate (GFR) followed by renal function. Creatitine nor urea is directly toxic and they are only a measure of kidney function.[6,7] Creatitine is produced from muscles and is excreted through the kidneys along with other waste products. Creatitine concentration in serum is maintained by the balance between its generation and excretion by the kidneys. The quantity of Creatitine in serum depends on their generation, glomerular filtration and tubular secretion of serum Creatitine. Calculations based on serum Creatitine and the age groups of the patient are used to estimate more precisely the degree of kidney function. Urea is an organic compound and plays a vital role in the metabolism of nitrogen-containing compounds. It is a waste product from dietary protein and is also filtered into urine by the kidneys.[6,7] Urea nitrogen is a normal waste nitrogen product found in blood that comes from the breakdown of protein from foods. Healthy kidneys remove urea nitrogen from blood, but the level of urea in blood rises with kidney failure

occurs.[7] Calcium is essential for building new bone & keeping bones strong. In diet and supplements sometimes calcium pills are provide extra calcium levels instead of these high phosphorous foods for people with chronic kidney disease.[8] Limiting dietary phosphates is essential for control hyper phosphatemia. This can be difficult for CKD patients. Dialysis removes phosphates but routinely and it is important to know the levels in predialysis CKD patients.[9] Due to kidney failure calcium levels in blood becomes low and decreased phosphorus excreation due to kidney failure, parathyroid glands begin removing calcium from bones to get calcium for maintained in blood over months & years, as calcium is stripped from bones this can make the bone weak.[8,16] Urea, Creatitine, Calcium and phosphorus levels are important biomarkers as they play a pivotal role in diagnosis and follow-up of kidney failure.[10,11]

Aim: To study Serum Urea, Creatitine, Calcium and Phosphorus in all stages of chronic kidney disease patients.

II. MATERIAL AND METHODS

The present study was carried out at the Department of Biochemistry and Department of Nephrology Bharati Vidyapeeth (Deemed To Be University) Medical College and Hospital, Sangli., Maharashtra, India during the period 2018 to 2020 with approval of Institute of Ethical Committee (IEC/ Dissertation 2017-18/246). In period over 2 year. 175 of CKD patients. (35 patients of each stage) in the age group of 20-60 yrs, will be included. Staging of CKD and other than factors will be done by Nephrologists. Patients information was filled in proforma contains patients name, age, sex, diet (veg/non-veg/mixed), habits, clinical history, family history. study Serum Urea, Creatitine, Calcium and Phosphorus was done by Meril 400i Fully autoanalyzer.

Estimation of Serum Urea, Creatitine and S. Calcium

Estimation of Serum Urea, Creatitine, S.Calcium and Phosphorus are done on AUTOQUANT 400i (Meril) fully Autoanalyzer in Department of Biochemistry Bharati Vidyapeeth (Deemed To Be University) Medical College and Hospital, Sangli.

1) Estimation of Serum Urea:- Urease- GLDH, Fixed Time method is used.

Reference Value is 13-45mg%.[12]

2) Estimation of Serum Creatitine:- Jaffe's Method, Initial Rate method is used

Reference Value is 0.6 – 1.4 mg%[12]

3) Estimation of Serum Calcium:-Arsenazo III. End Point method is used.

Reference Value is Serum Calcium: - 8.4 -10.4 mg%.[12]

4) Estimation of Serum Phosphorus:- Ammonium MolybdateMethod, End – Point

Reference Value:- Serum Phosphorus : - 2.5 to 4.5 mg%.[12]

AUTOQUANT 400i (Meril) fully Autoanalyzer are calibrated by BIOCAL and Controls is BIOPATH and BIONORM.[13,14]

III. Statistical analysis

Statistical comparisons were performed with spreadsheet software (Excel, Microsoft). The statistical analysis was done using the ANOVA, "t" and Chi-Square test. All results were calculated as mean ± SD and a "p" value of <0.05 was considered statistically significant.

IV. RESULTS :- In this study 175 Diagnosed patients of chronic kidney disease of I to V stages (35 patients of each stage) in the age group of 20-60 yrs, in which 117 Male & 58 Female will be included.

Table no 1: CKD stages wise distribution of patients.

Sr. No	stage	Male	Female	Total
1	I	25	10	35
2	II	22	13	35
3	III	21	14	35
4	IV	21	14	35
5	V	28	7	35
	Total	**117**	**58**	**175**

Table no- 1 shows CKD stagewise distribution of patients in each 35 CKD patients. Distribution of cases in various stages of CKD were as follows There were totally 175 cases; they were divided into various stages I to V.

Table no 2: Agewise distribution of CKD patients.

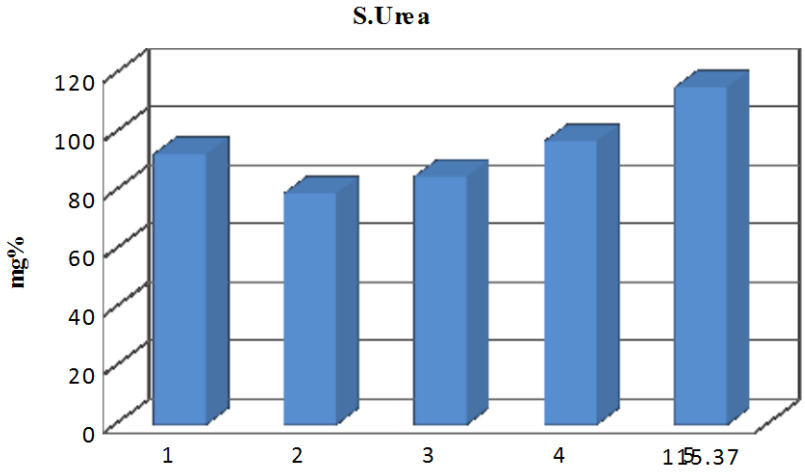
Sr. No	Age	Male	Female	Total
1	20-30yrs	14	8	22
2	31-40yrs	16	8	24
3	41-50yrs	16	11	27
4	51-60yrs	71	31	102
	Total	117	58	175

Table shows CKD Agewise distribution of patients. Age group 20-30yrs, 31-40yrs, 41-50yrs, 51-60 yrs. According to their age. Maximum number of patients, 102 in the age group of 51-60 years, Minimum number of patients followed by 22 patients in 20-30 years.

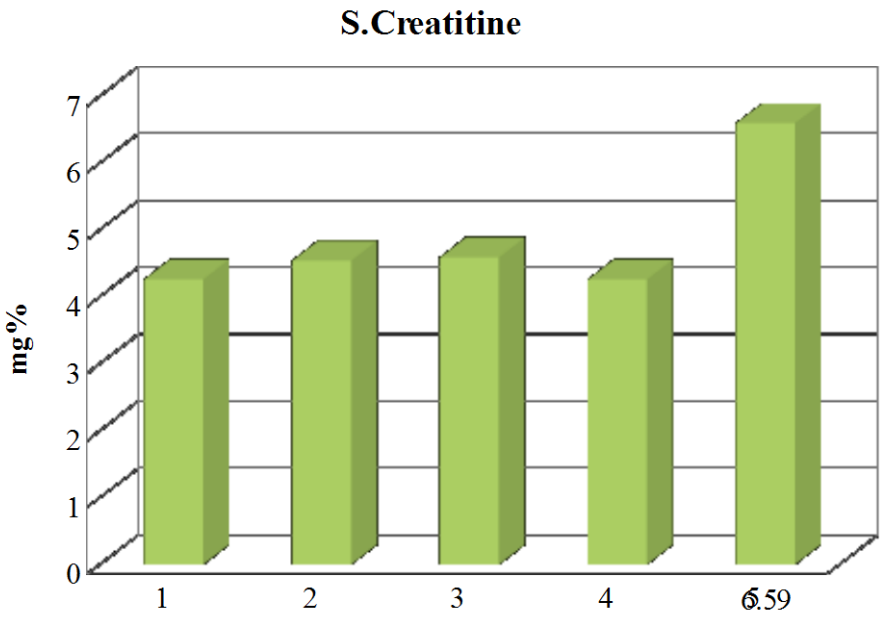
Table 3: Stagewise values of biochemical parameter in CKD patients.

Stage	S.Urea (mg%) (Mean±SD)	S.Creatitine (mg%) (Mean±SD)	S.Calcium (mg%) (Mean±SD)	S.Phosphorous (mg%) (Mean±SD)
I	92.49±38.11	4.26±2.26	8.54±0.61	4.25±1.16
II	79.06±35.11	4.54±2.79	8.84±1.06	4.34±1.16
III	84.83±36.74	4.59±2.38	9.01±0.90	4.24±1.24
IV	96.91±33.36	4.26±2.48	8.44±0.87	4.36±1.40

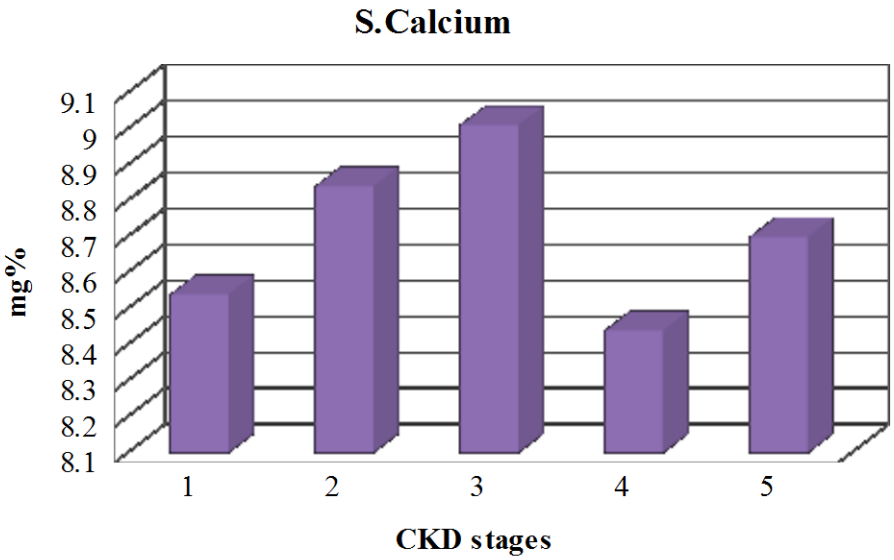
V	115.37±34.92	6.59±12.10	8.70±0.82	4.27±1.18
Total	**93.73±37.42**	**4.85±5.85**	**8.71±0.88**	**4.29±1.22**
	F= 5.322 (p=0.00)	**F=0.999 (p=0.410)**	**F=2.482 (P=0.046)**	**F= 0.074 (p=0.990)**



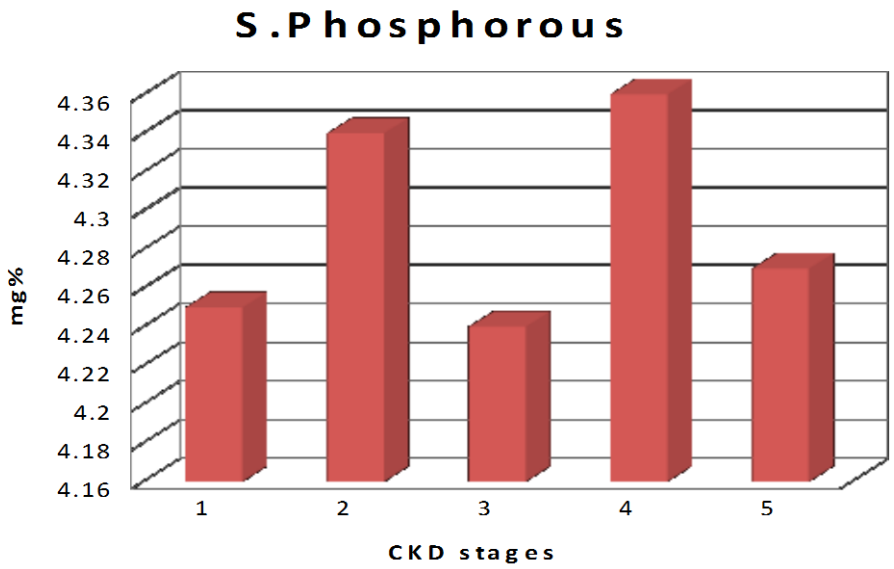
Graph no-1 shows stage wise serum urea levels in CKD patients.



Graph no-1 shows stage wise serum Creatitine levels in CKD patients.



Graph no-1 shows stage wise serum Calcium levels in CKD patients.



Graph no-1 shows stage wise serum phosphorous levels in CKD patients.

Table and graph shows S.Urea, S.Creatitine & S.Calcium Mean±SD values in I to V stages in CKD patients. S.Urea is significant in all stages ($p<0.00$) increase values in as compaired to I to V stages. S.Creatitine & S.Phosphorus in I to V stages is not significant. S.Calcium in I to V stages is significant ($p<0.05$). S.Urea, S.Creatitine, S.Calcium & S.Phosphorus values are compaired with standard and controls of kit.

V. DISCUSSION

Chronic kidney disease (CKD) refers to all 5 stages of kidney damage, from very mild damage in Stage 1 to complete kidney failure in Stage 5. Renal failure is a gradual, progressive and irreversible loss of normal functioning of kidneys. Increased levels of urea and Creatitine excretion in blood by impaired kidneys made very complication in patients. In present study total of 175 CKD patients (I to V) who were diagnosed by Nephrologist, based on their clinical history, clinical examinations and renal function tests were randomly evaluated. Biochemical markers levels of such as serum Urea, Creatitine and Calcium Mean levels are shows in Table no-1 and graph shows 35 CKD patients patients of each (I to V) stage 20-60 yrs are studied. S.Urea, S.Creatitine S.Calcium & S. Phosphorus Mean±SD values in I to V stages in CKD patients. S.Urea & S.Calcium is significant increase values in as compaired to I to V stages. S.Creatitine & & S. Phosphorus in I to V stages is not significant. S.Calcium in I to V stages is

significant ($p<0.05$). This type of studies supported by Sanjay Vikrant and Anupam Parashar(2016).[15]

The use of Urea & Creatitine measurement to assess renal function levels of both reflect glomerular filtration rate (GFR), Irrespective of its cause, kidney disease is associated with decrease in GFR, and the severity of kidney disease correlates closely but inversely with GFR.[17] Urea and Creatitine levels are used to support the diagnosis of CKD.[19] In CKD patients progressive loss of kidney functions leads to Fluctuation in extracellular calcium ion levels is sensed by the parathyroid calcium- sensing receptors and subsequently regulates the synthesis and secretion of parathyroid hormone.[21] PTH is the primary calcium- and phosphate-regulating hormone produced by chief cells in the parathyroid glands.[19] Kidney failure cause a number of homeostatic mechanisms that control serum calcium and phosphorous and normal bone metabolism.[19,20] However, our understanding of calcium & phosphorous balance throughout the stages of chronic kidney disease is important to balance mineral metabolism with a cation as complex as calcium, is known, Both negative and positive calcium balance have important implications in patients with chronic kidney disease, where negative balance may increase risk of osteoporosis and fracture and positive balance may increase risk of vascular calcification and cardiovascular events.[19]

In CKD the kidneys fail to maintain the phosphorus load so the result is skeleton disorders of the bone. Previous studies shown that phosphorus is involved in the whole process of vascular calcification leading renal osteodystrophy with CKD mineral bone disorder.[20,22] This study is designed to examine the frequency and severity of the skeletal demineralization in patients of all stages of CKD by measurements of serum levels of urea, creatitine, calcium and phosphorous.

V. CONCLUSION

The bone mineral metabolism abnormalities start during 1st stage of CKD as renal furcation decreases long before the need for renal replacement therapy and can be positively or negatively influenced by the treatment. It is recommended that attending physicians monitor and control biochemicals parameters early in the development of CKD before the need for dialysis.

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