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EFFECT OF PROBIOTICS IN SLOWING THE PROGRESSION OF CHRONIC KIDNEY DISEASE OVER SIX MONTHS

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ABSTRACT

Chronic kidney disease (CKD) is widely prevalent and increasingly common globally, currently standing as the seventh most significant contributor to mortality worldwide. Probiotics have become a safe and affordable treatment option for many chronic illnesses. Probiotic supplementation has been shown to alter the microbiota composition in the context of chronic kidney disease (CKD),[9] lowering inflammation and the production of uremic toxins. The aim and objective of the study to evaluate the impact of probiotics on progression of CKD by monitoring changes in Serum Creatinine and Urea over six months. The study was designed a prospective observational single centre study by the conduct of 6 months a period with100 subjects of population size. The result of the study on effect of probiotics on slowing the progression of chronic kidney disease about the highest number of patients fall in the age 70-79 i.e., (33%), male patients make up majority (61%) and The highest Patients were under stage 1 (61%), It presents both mean ± standard deviation (SD) and median values for each time point. Creatinine levels decreased over time, from initial to final, both in terms of mean and median. eGFR values increased progressively from the initial stage to the final stage. Standard deviation is quite high at all stages, indicating a wide variation in uric acid levels among subjects. The study concludes that provides compelling preliminary evidence that probiotics may play a valuable role in slowing the progression of Chronic Kidney Disease by improving renal biomarkers and offering a safe, well-tolerated adjunct to conventional therapy. While further high-quality research is necessary to establish clinical guidelines, this study sets the groundwork for the incorporation of gut microbiota-targeted therapies into standard nephrology practice. The evolving understanding of the gut-kidney axis opens up new horizons for managing CKD more effectively and holistically.

KEYWORDS: Stages, Chronic kidney disease, probiotics, eGFR, urea, microbiota and Creatinine levels.

INTRODUCTION

Chronic kidney disease (CKD) is widely prevalent and increasingly common globally, currently standing as the seventh most significant contributor to mortality worldwide.[1] Patients with CKD face heightened oxidative stress, persistent low-grade inflammation, and the build-up of uremic toxins.[2,3] These alterations lead to endothelial dysfunction, cellular aging, and vascular calcification (VC), ultimately resulting in cardiovascular diseases (CVDs) and a heightened risk of morbidity and mortality.[4]

EPIDEMIOLOGY of CKD on Globally, in 2012, chronic kidney disease (CKD) was responsible for 2,968,600 (1%) of disability-adjusted life-years and 2,546,700 (1%-3%) life-years lost. In 2016, an estimated

26 million individuals in the United States were affected by CKD.[5] The Kidney Disease Outcomes Quality Initiative (KDOQI) advises that to confirm the presence of chronicity and CKD, patients should undergo testing on three different occasions within a three-month period, ensuring that at least two out of the three results are positive.[6]

The **Etiological** causes are the Non-modifiable and Modifiable CKD causes. The Non-modifiable CKD etiological factors are The advancement of CKD is negatively impacted by older age, male gender, and non-White ethnicity, including Black Americans, Afro-Caribbean people, Hispanics, and Asians (South Asians and Pacific Asians).Systemic hypertension, proteinuria, and metabolic variables are examples of modifiable CKD etiological factors.[7]

Pathophysiology

A complex interaction of molecular pathways characterizes both ESKD and CKD. Complications associated with CKD and ESKD are largely caused by inflammation, elevated oxidative and metabolic stress, endothelial dysfunction, vascular calcification from inadequate calcium and phosphate metabolism, and coagulation issues. Moreover, medications and chemicals that are normally broken down or removed by the kidneys build up as a result of the loss in GFR in advanced stages of CKD. This build-up accelerates the course of the disease and worsens renal impairment. Exogenous and endogenous chemicals, cell damage, and genetic-linked pathways connected to CKD and ESKD will all be covered in this article. The available treatments will also be covered in detail in each segment.[8]

Probiotics have become a safe and affordable treatment option for many chronic illnesses. Probiotic supplementation has been shown to alter the microbiota composition in the context of chronic kidney disease (CKD),[9] lowering inflammation and the production of uremic toxins. Furthermore, it has been shown that certain probiotics can alter P absorption and lower serum P levels via altering the gut microbiota.[10] It is yet unknown, nevertheless, if taking probiotic supplements could slow the progression of VC. As a result, there is increasing interest in assessing the possible role of probiotics and gut flora to lessen the negative effects of high P and slow down the progression of CKD.[11, 12]

Complications of chronic kidney disease are salt/fluid balance, Hypertension, Hyperkalemia, Metabolic acidosis, Hyperphosphatemia, Anemia, Treatment of Complications of End-Stage Renal Disease, Cardiovascular disease and Insulin resistance.[13, 14]

Treatment management of hypertension in people with CKD the Presence and severity of albuminuria should be evaluated. Blockade of the renin-angiotensin-aldosterone system with either an angiotensin-converting enzyme inhibitor (ACE-I) or an angiotensin II receptor blocker (ARB) is recommended for diabetes and a urine ACR.[15] Dual therapy with an ACE-I and an ARB is generally avoided, given associated risks of hyperkalemia and acute kidney injury. Aldosterone receptor antagonists may also be considered in patients with albuminuria.[16] Optimal management of diabetes is also important First, glycemic control, Second, dose adjustments in oral hypoglycemic agents whereas drugs metabolized by the liver and/or partially excreted by the kidneys (eg, metformin and some dipeptidyl peptidase 4 [DPP-4] and sodium-glucose cotransporter-2 [SGLT-2] inhibitors) and Third, use of specific medication classes such as SGLT-2 inhibitors in those with severely increased albuminuria should be considered. The Canagliflozin, ACE-I or ARB therapy.[17, 18]

All patients with CKD should be counseled to avoid nephrotoxins. Routine administration of NSAIDs in CKD is not recommended, especially among individuals who are taking ACE-I or ARB therapy. Adjustments in drug dosing are frequently required in patients with CKD. Common medications that require dose reductions include most antibiotics, direct oral anticoagulants, gabapentin and pregabalin, oral hypoglycemic agents, insulin, chemotherapeutic agents, and opiates, among others.

Dietary Management to possible benefits of dietary protein restriction must be balanced with the concern of precipitating malnutrition and/or protein wasting syndrome. Lower dietary acid loads (eg, more fruits and vegetables and less meats, eggs, and cheeses) may also help protect against kidney injury.[19]

Diagnosis of ckd In many cases, CKD is only found when a routine blood or urine test you have for another problem shows that your kidneys may not be working normally. The regular tests are recommended such as high blood pressure, diabetes, acute kidney injury, cardiovascular disease such as coronary heart disease or heart failure, other conditions that can affect the kidneys – such as kidney stones, an enlarged prostate or lupus. People taking long-term medicines that can affect the kidneys, such as lithium, omeprazole or non-steroidal anti-inflammatory drugs (NSAIDs), should also be tested regularly. The main tests of chronic kidney disease there are blood test, urine test and other tests includes ultrasound scan, MRI scan or CT scan and biopsy.[20]

AIM AND OBJECTIVE

The aim and objective of the study to evaluate the impact of probiotics on progression of CKD by monitoring changes in Serum Creatinine and Urea over six months.

METHODOLOGY

This study was conducted at Medicover Hospitals (Tertiary care hospital), Hi-Tech City, Hyderabad in study period of 6 months with the sample size 100

subjects. The study was designed a prospective observational single centre study. Subjects in the study are enrolled based on criterias The inclusion criteria such as patients with moderately decreased GFR, serum potassium level less than 3.5mmol/L and who can tolerate with a maximum dose of an ACE inhibitor or ARB and exclusion criteria are patients high serum potassium levels above a specified threshold, Pregnancy or lactating intention, with uncontrolled hypertension and dual ACE inhibitor/ARB therapy. The study collected data and obtained informed consent from participants in compliance with the Declaration of Helsinki. Approval for conducting the study was obtained from the Institutional Ethics Committee. Before taking part in the study, all patients were given detailed information and provided written consent. The statistical analysis done by using SPSS software, V.22.(1)1. SPSSI. IBMSPSS Statistics Version 22 Statistical Software: Core SystemUsers'Guide. SPSS Inc.2014.

RESULTS

In a tertiary care hospital, a prospective observational study was carried out to assess The Effect of probiotics on slowing the progression of chronic kidney disease, a total of 100 Patients were included in our investigation.

The age analysis of the patients who participated in the study is shown in table 1 and fig 1. according to the report. The highest number of patients fall in the age 70-79 i.e., (33%). And the youngest two age groups (20-29 and 30-39) have fewest patients, only 4% each. There is a gradual increase in the patient count with age, peaking in the 70-79 group. It shows that 70-79 age people are more prevalent than others.

Table 1: Descriptive analysis of age in study population (N=100).

AGE	PATIENTS	PERCENTAGE
20-29	4	4%
30-39	4	4%
40-49	14	14%
50-59	19	19%
60-69	26	26%
70-79	33	33%
TOTAL	**100**	**100%**

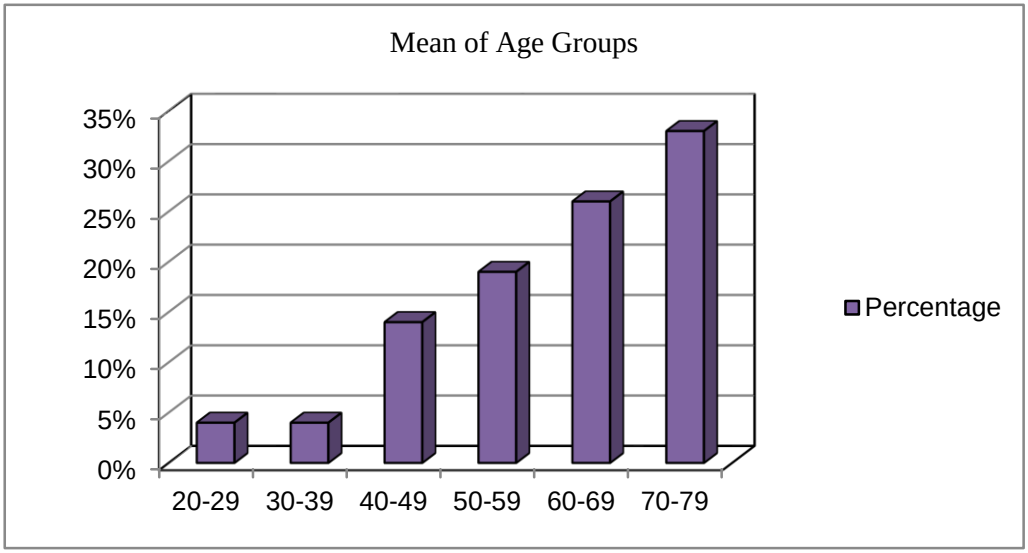

Fig. 1: Mean of Age Groups in study population.

Table 2: Descriptive analysis of Gender in study population (N=100).

GENDER	NO.OF PATIENTS	PERCENTAGES
MALE	61	61%
FEMALE	39	39%
TOTAL	100	100%

The gender analysis of the patients who participated in the study is shown in table 2 and Fig 2.

According to the report.the male patients make up majority (61%) of the total sample and female patients constitute the remaining (39%)**.** The total number of patients is 100, so each percentage value directly reflects

the number of patients. This data is helpful for understanding the gender distribution among the patients, which could be relevant for statistical analysis.

Fig 2: Pie Chart of Gender in the study population.

Table 3: Descriptive analysis of CKD stages in the initial study population (N=100).

CKD	FREQUENCY	PERCENTAGE
2	34	34%
3	44	44%
4	22	22%
TOTAL	100	100%

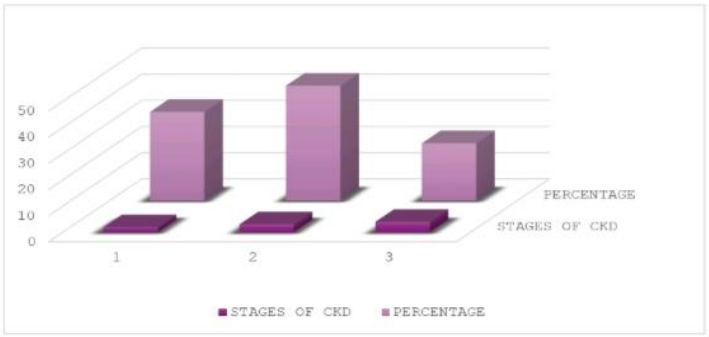

Figure 3: Bar chart of CKD stages in the study population (N=100).

The distribution of the CKD stage over the total population is shown in Table 3 and Figure 3.

The vast number of Patients during admission were under stage 3 (44%), whereas lower in stage 4 (22%). After follow-up, as it is depicted in the table 3 and fig 3. The highest Patients were under stage 1 (61%), whereas stage 2, 3, and 4 were the fewest Patients (30, 6.9 and 0.9% respectively). By this table 1 and 2 we can conclude that before the administration of probiotics the stages of ckd were greater and after administration of probiotic, the stages of ckd were lower.

Table 4: Descriptive analysis of CKD stages in the final study population (N=100)

CKD STAGE	FREQUENCY	PERCENTAGE
1	61	60.396040%
2	31	30.693069%
3	7	6.930693%
4	1	0.99099%
TOTAL	100	100%

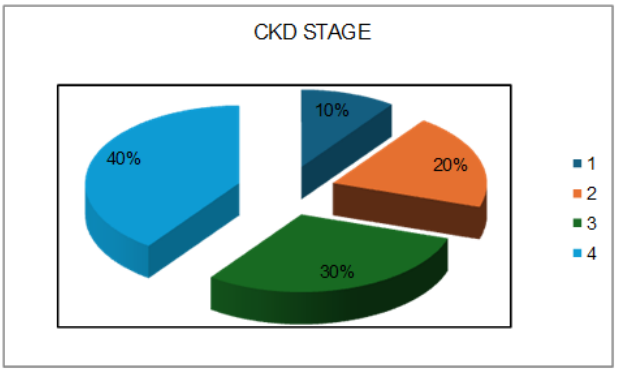

Fig. 4: Stages of ckd in final study population.

Table 5: Descriptive analysis of anthropometric parameters in study population (N=100).

PARAMETER	MEAN	MEDIAN
Height	161.43	165.0
Weight	66.57	64.85

The descriptive analysis of anthropometric parameters in the table 5 tells that the average height is 161.43cm, indicating the moderate variation in height of 17.46cm. The median of the height is 165.0 cm, which is slightly higher than the mean and the average weight is 66.57kg, with the variation of 19.54kg, indicating a wider spread in weight. The median weight is 64.85 kg, slightly lower than the mean**.**

Table 6: Descriptive analysis of investigation parameters in study population (N=100).

PARAMETER	MEAN	MEDIAN
BP	143.32	140
PULSE	83.22	84
RR	20.16	20
GRBS	103.47	98
TEMPERATURE	108.46	98
SPO2	97.81	98

Table 7: Camparision of mean Sr. creatinine in pre-operative, 3rd month and final (N=100).

Parameter	MEAN± SD
Sr. Creatinine in initial time period	2.32± 1.02
Sr. Creatinine in 3rd month	1.98 ± 0.98
Sr. Creatinine during final	1.62± 0.9

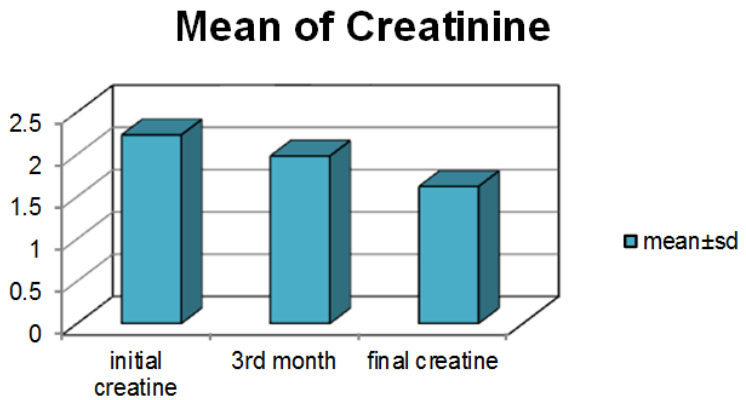

Fig. 5: Bar chart of Sr. Creatinine in pre-operative, 3rd month and final (N=100).

The analysis of the table 7 and figure 5 provides the analysis of serum creatinine levels over three time points: initial, 3rd month, and final. It presents both mean ± standard deviation (SD) and median values for each time point. Creatinine levels decreased over time, from initial to final, both in terms of mean and median. This suggests an improvement in renal function or effectiveness of treatment (probiotic) over the study duration. The standard deviation values indicate some variability in the data, but the overall trend is downward,

which is generally favourable in clinical contexts involving kidney health.

Table 8: Camparision of mean eGFR in pre-operative, 3rd month and final (N=100).

PARAMETER	MEAN± SD
eGFR at initial time period	39.8 ± 43.43
eGFR in 3rd month	43.19 ± 15
eGFR during final	50.31 ± 17.56

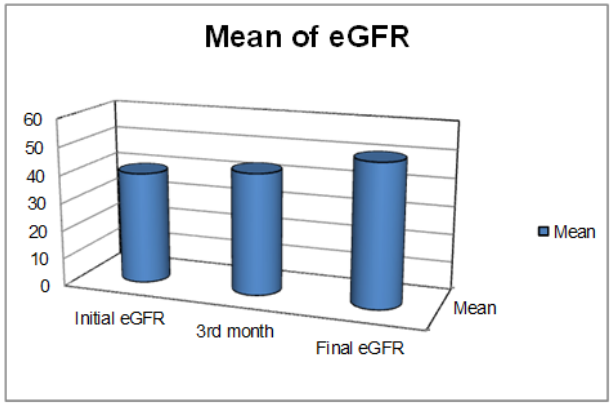

Fig. 6: Bar chart of eGFR in pre-operative, 3rd month and final (N=100).

The table 8 and figure 6 provides an analysis of eGFR (estimated Glomerular Filtration Rate) over three points: Initial, 3rd month, and Final. It includes both Mean ± SD (Standard Deviation). eGFR values increased progressively from the initial stage to the final stage. This indicates an improvement in kidney function over time. The mean values also reflect a consistent upward trend, suggesting that the treatment (probiotic) may have had a positive impact. Standard deviation values show some variability but are within a reasonable range.

Table 9: Camparision of mean uric acid in pre-operative, 3rd month and final(N=100)

PARAMETER	MEAN±SD
Uric acid at initial period of time	8.16±11.17
Uric acid in 3rd month	8.22±9.59
Uric acid during finals	8.5±9.74

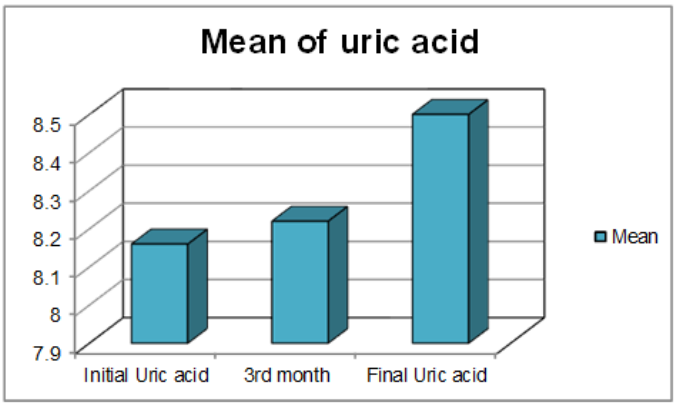

Fig. 7: Bar chart of uric acid in pre-operative, 3rd month and final (N=100).

The table 9 and figure 7 provides an analysis of uric acid levels measured over time, with mean ± standard deviation (SD) and at three different time points: Mean values of uric acid remain relatively stable over time: around 8.15–8.22 mg/dl. Standard deviation is quite high at all stages, indicating a wide variation in uric acid levels among subjects. This data could suggest increasing uric acid levels in the patients who has reduced uric acid majority of participants over time, even though the mean remains nearly constant fir the patients who has normal uric acid levels.

Table 10: Camparision of urea in pre-operative, 3rd month and final(N=100).

Parameter	MEAN±SD
Urea at initial period of time	43.43±22.26
Urea in 3rd month	38.17±19.31
Urea during finals	32.95±17.11

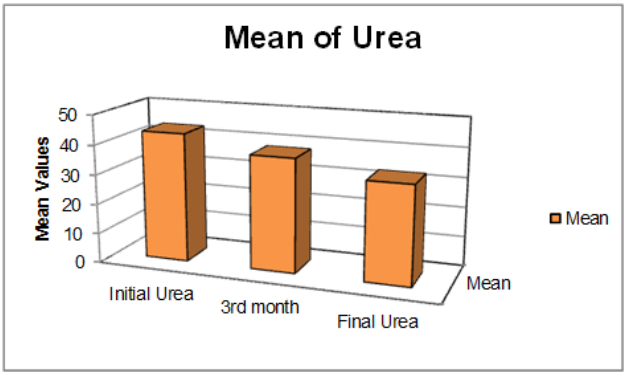

Fig 8: Bar chart of uric acid in pre-operative, 3rd month and final (N=100).

The table 10 and the figure 8 provide an analysis of urea levels at three different time points: Initial, 3rd month, and Final. The values are expressed as Mean ± Standard Deviation (SD). There is a gradual decrease in mean urea levels over time from 43.43 initially to 32.95 at the final stage. Standard deviation also decreases over time (from 22.26 to 17.11), suggesting reduced variability in urea levels among the participants. This trend indicates a progressive improvement or normalization of urea levels, possibly due to treatment (probiotic).

DISCUSSION

Serum Creatinine, GFR, Serum Uric Acid, urea were considered as outcome variable. Descriptive analysis was carried out by mean and standard deviation for quantitative variables, frequency and proportion for categorical variables. Non normally distributed quantitative variables were summarized by median and interquartile range (IQR). Data was also represented using appropriate diagrams like bar diagram, pie diagram. All Quantitative variables were checked for normal distribution within each category of explanatory variable by using visual inspection of histograms and normality Q-Q plots. A prospective, observational study was organized and carried out for 6 months to evaluate the effect of probiotics on slowing the progression of chronic kidney disease A total of 100 Chronic Kidney Disease Patients with ckd 2,3,4 data was collected and they were followed up.

CONCLUSION

The conclusion of the study provides compelling preliminary evidence that probiotics may play a valuable role in slowing the progression of Chronic Kidney Disease by improving renal biomarkers and offering a safe, well-tolerated adjunct to conventional therapy. While further high-quality research is necessary to establish clinical guidelines, this study sets the groundwork for the incorporation of gut microbiota-targeted therapies into standard nephrology practice.The evolving understanding of the gut-kidney axis opens up new horizons for managing CKD more effectively and holistically.

CONFLICT OF INTEREST

The author were no the conflict of interest.

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