***Original Research Article***

**The Rural Renal Equation: Age, BMI, and Biochemical Insights**

**ABSTRACT**

**Background:** Renal function is a critical marker of systemic health, influenced by both physiological aging and metabolic stress. However, limited data exist on how demographic and biochemical variables jointly impact kidney function in rural Indian populations. **AIM:** To assess the influence of age, body mass index (BMI), and key biochemical indices on renal function within a rural Indian cohort. **Methods:** A cross-sectional study of 200 individuals was conducted, analysing estimated glomerular filtration rate (eGFR), serum creatinine, and blood urea nitrogen (BUN) across age groups. Pearson’s correlation and multivariate linear regression were used to explore associations between renal function and demographic/biochemical predictors. **Results:** The mean age was 59.7 ± 17.2 years, and mean BMI was 24.5 ± 4.2 kg/m². A significant age-dependent decline in GFR was observed: from 131.2 mL/min in individuals <40 years to 62.1 mL/min in those >60 years. Strong negative correlations were found between age and GFR (r = -0.71, p < 0.001) and between BMI and GFR (r = -0.35, p = 0.002), while BMI positively correlated with serum creatinine (r = 0.29, p = 0.006). Age also showed a strong positive correlation with BUN (r = 0.64, p < 0.001). In multivariate analysis, age (β = -0.56, p < 0.001), BMI (β = -0.28, p = 0.004), and bicarbonate levels (β = +0.31, p = 0.002) independently predicted GFR. **Conclusion:** Age and BMI are strong, independent predictors of renal function decline in rural populations. Elevated bicarbonate levels appear to exert a protective effect. These findings underscore the need for age- and weight-adjusted screening protocols in low-resource settings to enable earlier intervention.

**Key Words:** BMI ((Body Mass Index), Ageing, Biochemical Indices, Cohort, Blood Urea Nitrogen (BUN)

**INTRODUCTION**

Chronic kidney disease (CKD) represents a significant and growing global health concern, affecting approximately 9.1% of the world's population, with substantial variation across regions and populations [1]. In India, recent studies estimate that nearly 15–17% of the adult population may be affected by some form of kidney dysfunction, with rural populations facing particular challenges due to limited healthcare access and late-stage presentation [2,3]. Age is a major non-modifiable risk factor for the progressive decline in renal function. Studies have consistently shown that glomerular filtration rate (GFR) declines by approximately 0.75–1.0 mL/min/year after the age of 40 years, largely due to cumulative nephron loss, glomerulosclerosis, and microvascular changes [4]. In the Indian context, the Indian Chronic Kidney Disease (ICKD) study has highlighted that age-related decline in GFR is compounded by a high burden of comorbidities such as hypertension and diabetes, even in semi-urban and rural settings [5].

Body mass index (BMI) is another critical factor influencing renal outcomes. Higher BMI has been associated with increased risk of incident CKD, independent of traditional risk factors [6]. Obesity may contribute to renal injury through mechanisms including glomerular hyperfiltration, activation of the renin-angiotensin-aldosterone system (RAAS), and chronic low-grade inflammation [7]. A study also demonstrated that obesity-related glomerulopathy is an emerging cause of CKD, emphasizing the urgent need to monitor BMI as a modifiable risk factor [8]. Biochemical indices such as serum creatinine, blood urea nitrogen (BUN), and serum bicarbonate offer important insights into renal and metabolic health. Elevated serum creatinine and BUN are established markers of declining GFR, whereas low serum bicarbonate levels have been associated with faster CKD progression and increased mortality [9,10]. Another study reported that metabolic acidosis, indicated by low serum bicarbonate, significantly accelerates loss of kidney function in CKD patients [11].

Despite these well-established relationships, there remains a paucity of data specific to rural Indian populations. Rural communities often exhibit unique risk factor profiles, including differences in diet, physical activity, healthcare access, and awareness, making it critical to generate localized evidence. Understanding how demographic and biochemical factors influence renal function in these populations is essential for designing effective prevention and intervention strategies. Thus, the present study seeks to evaluate the influence of age, BMI, and biochemical parameters on renal function in a rural Indian cohort, contributing valuable insights to the field of community nephrology.

**MATERIALS AND METHODS**

**Study Setting and Participants**

This community-based cross-sectional observational study was conducted over a six-month period in a rural district of India to evaluate the effects of age, body mass index (BMI), and select biochemical markers on renal function. A total of 200 individuals, aged 14 to 93 years, were recruited through voluntary participation in health camps and outpatient departments of local primary healthcare centres. Ethical approval was obtained from the Institutional Ethics Committee, and the study was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants or their legal guardians in the case of minors. Inclusion criteria were: individuals aged 14 years and above, permanent residents of the selected rural region, and availability of complete anthropometric and biochemical data. Participants were excluded if they had a known diagnosis of advanced chronic kidney disease (Stage 4 or 5), as severely impaired renal function may overshadow more subtle physiological variations and hinder the evaluation of associations with demographic and metabolic factors. Individuals who had experienced an acute kidney injury (AKI) within the preceding three months were also excluded, since such events may cause temporary and potentially misleading fluctuations in serum creatinine and glomerular filtration rate (GFR). Additionally, participants with active systemic infections or inflammatory conditions were excluded to avoid the confounding effects of acute-phase responses on renal and biochemical indices. Those who had been recently hospitalized (within the past 30 days) were not considered, given that recent medical interventions or acute illness could transiently alter renal parameters and biochemical profiles. Lastly, individuals on medications known to significantly influence renal function—such as corticosteroids, nephrotoxic agents, or diuretics—were excluded, as these drugs may independently affect GFR, electrolyte balance, or serum creatinine, thereby compromising the reliability of renal function assessment. These criteria were meticulously applied to minimize confounding variables and enhance the internal validity of the study findings.

**Data Collection and Measurements**

Data collection was performed using a structured, pre-tested questionnaire to obtain demographic details, medical history, and lifestyle factors. Anthropometric measurements were conducted using standardized tools and techniques. Height was measured to the nearest 0.1 cm using a Seca 213 Portable Stadiometer (Seca GmbH & Co. KG, Hamburg, Germany), and weight was recorded with a HealthSense Ultra-Lite PS 126 Digital Weighing Scale (HealthSense, India), accurate to 100 grams. BMI was calculated as weight (kg) divided by the square of height (m²). Fasting venous blood samples were collected from participants in the morning hours following an overnight fast. Blood was drawn under aseptic conditions using Vacutainer tubes and immediately transported under cold chain to central laboratory for biochemical analysis. Serum creatinine, blood urea nitrogen (BUN), and bicarbonate (HCO₃⁻) levels were measured using the VITROS 4600 Chemistry Analyzer. This system utilizes MicroSlide dry chemistry technology, with enzymatic methods for all three analytes to ensure high specificity and minimal interference.

Serum creatinine was assessed using a dry-slide enzymatic method involving creatinine amidohydrolase, which enables accurate detection with reduced susceptibility to interfering substances such as bilirubin or glucose. BUN was measured enzymatically via the urease method, where urea is hydrolysed to ammonia and carbon dioxide, followed by a secondary reaction generating a quantifiable colour change. Bicarbonate levels were determined through an enzymatic reaction catalysed by phosphoenolpyruvate carboxylase and malate dehydrogenase, which measures NADH consumption proportional to bicarbonate concentration. These validated methodologies, integral to the VITROS 4600 platform, ensure robust, reliable, and reproducible results. Renal function was evaluated using estimated glomerular filtration rate (eGFR), which was automatically calculated by the clinical autoanalyzer based on serum creatinine levels and patient demographics (age and sex). Unlike traditional formula-based estimation methods such as CKD-EPI or MDRD, this approach provided standardized automated eGFR values for all participants

**Statistical Analysis**

Statistical analysis was performed using IBM SPSS Statistics Version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were presented as mean ± standard deviation (SD). The Shapiro-Wilk test was used to assess normality of the data distribution. Bivariate relationships between renal function markers and independent variables (age, BMI, biochemical indices) were assessed using Pearson’s correlation coefficient. To identify independent predictors of reduced eGFR, a multivariate linear regression model was applied, adjusting for age, BMI, and bicarbonate levels. A p-value of <0.05 was considered statistically significant for all tests. Data quality and integrity were maintained through double data entry, cross-verification, and anonymized record storage.

**RESULT**

The present study analyzed the demographic, anthropometric, and biochemical characteristics of 200 participants to evaluate their association with renal function.

**Table 1. Demographics and Anthropometric Characteristics (N = 200)**

|  |  |  |  |
| --- | --- | --- | --- |
| **Parameter** | **Mean ± SD** | **Min** | **Max** |
| Age (Years) | 59.7 ± 17.2 | 14 | 93 |
| BMI (kg/m²) | 24.5 ± 4.2 | 16.1 | 36.8 |

The study cohort had a mean age of 59.7 years with a standard deviation of 17.2, with an age range of 14–93 years. The mean BMI was 24.5 kg/m², with values ranging from 16.1 to 36.8 kg/m².

**Chart 1: Showing Renal Function Distribution by Age Groups**

In individuals <40 years, the mean GFR is 131.2 mL/min and creatinine are 0.67 mg/dL. For those aged 40–60, the mean GFR is 105.6 mL/min and creatinine are 0.89 mg/dL. In individuals over 60 years, the mean GFR is 62.1 mL/min and creatinine are 1.52 mg/dL.

**Table 2. Correlation Analysis of Renal Function with Demographic and Biochemical Parameters**

|  |  |  |
| --- | --- | --- |
| **Variables** | **Pearson’s r** | **p-value** |
| Age vs. GFR | -0.71 | <0.001 |
| BMI vs. GFR | -0.35 | 0.002 |
| BMI vs. Creatinine | 0.29 | 0.006 |
| Age vs. BUN | 0.64 | <0.001 |

A strong negative correlation was observed between age and GFR (r = -0.71, p < 0.001). BMI and GFR showed a moderate negative correlation (r = -0.35, p = 0.002). A mild positive correlation was found between BMI and creatinine (r = 0.29, p = 0.006). Age and BUN showed a strong positive correlation (r = 0.64, p < 0.001).

**Table 3. Multivariate Regression for Predictors of Low GFR**

|  |  |  |
| --- | --- | --- |
| **Predictor** | **Beta Coefficient** | **p-value** |
| Age | -0.56 | <0.001 |
| BMI | -0.28 | 0.004 |
| HCO₃⁻ | +0.31 | 0.002 |

Multivariate regression analysis identified age, BMI, and serum bicarbonate (HCO₃⁻) as significant predictors of GFR. Age showed a strong negative association with GFR (β = -0.56, p < 0.001). BMI had a significant negative association with GFR (β = -0.28, p = 0.004). Serum bicarbonate (HCO₃⁻) showed a positive association with GFR (β = +0.31, p = 0.002).

**DISCUSSION**

This study aimed to assess the impact of age, body mass index (BMI), and key biochemical parameters on renal function in a rural Indian population. Using estimated glomerular filtration rate (eGFR) and serum creatinine as indicators of renal health, the analysis sought to delineate how demographic and metabolic factors influence kidney function. The cross-sectional design and relatively diverse age range (14–93 years) provided a comprehensive perspective on renal health across the lifespan in this community-based cohort. Our findings reveal a significant and progressive decline in renal function with advancing age. Line chart 1 shows a marked reduction in mean GFR from 131.2 mL/min in participants aged <40 years to 62.1 mL/min in those over 60 years, while serum creatinine increased from 0.67 mg/dL to 1.52 mg/dL. Correlation analysis in Table 3 further confirms a strong inverse relationship between age and GFR (r = -0.71, p < 0.001) and a significant positive correlation between age and blood urea nitrogen (BUN) (r = 0.64, p < 0.001). BMI also emerged as an independent risk factor: Table 3 reveals a moderate negative correlation with GFR (r = -0.35, p = 0.002) and a mild positive correlation with creatinine (r = 0.29, p = 0.006). Multivariate regression (Table 4) identified age (β = -0.56, p < 0.001) and BMI (β = -0.28, p = 0.004) as significant negative predictors of GFR, while bicarbonate (HCO₃⁻) showed a protective association (β = +0.31, p = 0.002), suggesting that metabolic acidosis may play a role in renal impairment.

These observations are consistent with findings from prior epidemiological and clinical studies. Age-related renal decline is well-documented, with studies such as Coresh et al. (12) and Weinstein & Anderson et al. 13) reporting reductions in nephron number and renal blood flow with age, contributing to lower GFR and increased creatinine. Similarly, the link between obesity and impaired renal function has been established in multiple studies. Hall et al. (14) and Kovesdy et al. (15) highlighted that elevated BMI is associated with increased glomerular pressure, hyperfiltration, and subsequent glomerulosclerosis, leading to reduced renal function. Our finding that bicarbonate levels correlate positively with GFR aligns with work by Raphael et al. (16), which emphasized the role of metabolic acidosis in accelerating chronic kidney disease (CKD) progression and the potential renoprotective effects of maintaining serum bicarbonate within the normal range. The pathophysiological mechanisms underlying our findings are multifactorial. Aging leads to structural changes in the kidney, including glomerulosclerosis, tubular atrophy, and interstitial fibrosis Denic et al. (17) These changes reduce the filtration surface and functional nephron number, lowering GFR. In obesity, adipose tissue contributes to systemic inflammation and insulin resistance, which in turn increase intraglomerular pressure and promote renal injury Tsuboi et al. (18) Moreover, chronic low-grade inflammation in obese individuals enhances oxidative stress and fibrotic signalling within renal tissues. The observed positive association between bicarbonate and GFR may be due to the role of acidosis in stimulating ammonia production, promoting complement activation, and contributing to tubulointerstitial damage, as supported by Wesson et al. (19) Maintaining higher bicarbonate levels may help neutralize acid load and mitigate this injury, thereby preserving renal function. Hence our study demonstrates that increasing age and higher BMI are strongly associated with declining renal function, while higher serum bicarbonate levels may offer protective benefits. These results underscore the importance of early screening for renal dysfunction, especially in aging and overweight populations, even in rural settings. Limitations of this study include its cross-sectional nature, which precludes causal inference, and potential confounders such as dietary habits and undiagnosed comorbidities that were not accounted for. Future longitudinal studies with larger samples and inclusion of inflammatory markers are warranted to validate and extend these findings.

**CONCLUSION**

The study highlights that advancing age and higher BMI are significant risk factors for declining renal function in a rural Indian population, while higher serum bicarbonate levels are associated with better kidney health. These findings underscore the need for early screening and lifestyle interventions to preserve renal function, particularly among aging and overweight individuals in underserved communities.

**REFERENCES**

1. Bikbov B, Purcell CA, Levey AS, Smith M, Abdoli A, Abebe M, et al. Global, regional, and national burden of chronic kidney disease, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017. *Lancet*. 2020;395(10225):709–33.
2. Rajapurkar MM, John GT, Kirpalani AL, Abraham G, Agarwal SK, Almeida AF, et al. What do we know about chronic kidney disease in India: first report of the Indian CKD registry. *BMC Nephrol*. 2012; 13:10.
3. Varma PP. Prevalence of chronic kidney disease in India—Where are we heading? *Indian J Nephrol*. 2015;25(3):133–5.
4. Denic A, Glassock RJ, Rule AD. Structural and functional changes with the aging kidney. *Adv Chronic Kidney Dis*. 2016;23(1):19–28.
5. ICKD Study Investigators. Risk factors and outcomes associated with CKD in India: The ICKD Study. *Kidney Int Rep*. 2020;5(6):973–82.
6. Kramer H, Luke A, Bidani A, Cao G, Cooper R, McGee D. Obesity and prevalent and incident CKD: The Hypertension Detection and Follow-Up Program. *Am J Kidney Dis*. 2005;46(4):587–94.
7. Eknoyan G. Obesity and chronic kidney disease. *Nefrologia*. 2011;31(4):397–403.
8. Hall JE, do Carmo JM, da Silva AA, Wang Z, Hall ME. Obesity-induced hypertension: Interaction of neurohumoral and renal mechanisms. *Circ Res*. 2015;116(6):991–1006.
9. Levey AS, Coresh J. Chronic kidney disease. *Lancet*. 2012;379(9811):165–80.
10. Tangri N, Stevens LA, Griffith J, Tighiouart H, Djurdjev O, Naimark D, et al. A predictive model for progression of chronic kidney disease to kidney failure. *JAMA*. 2011;305(15):1553–9.
11. Raphael KL, Zhang Y, Ying J, Greene T. Prevalence of and risk factors for reduced serum bicarbonate in CKD in the Chronic Renal Insufficiency Cohort (CRIC) study. *Clin J Am Soc Nephrol*. 2014;9(5):710–9.
12. Coresh J, Selvin E, Stevens LA, Manzi J, Kusek JW, Eggers P, et al. Prevalence of chronic kidney disease in the United States. *JAMA*. 2007;298(17):2038–47.
13. Weinstein JR, Anderson S. The aging kidney: physiological changes. *Adv Chronic Kidney Dis*. 2010;17(4):302–7.
14. Hall JE, do Carmo JM, da Silva AA, Wang Z, Hall ME. Obesity-induced hypertension: interaction of neurohumoral and renal mechanisms. *Circ Res*. 2015;116(6):991–1006.
15. Kovesdy CP, Furth SL, Zoccali C. Obesity and kidney disease: hidden consequences of the epidemic. *Can J Kidney Health Dis*. 2017; 4:2054358117698669.
16. Raphael KL. Metabolic acidosis and subclinical kidney injury. *Curr Opin Nephrol Hypertens*. 2018;27(2):94–9.
17. Denic A, Glassock RJ, Rule AD. Structural and functional changes with the aging kidney. *Adv Chronic Kidney Dis*. 2016;23(1):19–28.
18. Tsuboi N, Utsunomiya Y, Kanzaki G. Obesity-related glomerulopathy and the nephron complement. *Kidney Int Rep*. 2017;2(2):229–34.
19. Wesson DE, Simoni J, Broglio K, Sheather S. Acid retention accompanies reduced GFR in humans and increases plasma levels of endothelin and aldosterone. *Am J Physiol Renal Physiol*. 2010;298(6): F1254–62.