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Evaluation of Cystatin C versus Creatinine for the Assessment of Glomerular Filtration Rate in Indian Patients with Type 2 Diabetes: An Eight-Week Prospective Study

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ABSTRACT:

Background: Traditional creatinine-based estimated glomerular filtration rate ($eGFR_{cr}$) is often inaccurate in populations with specific body compositions, such as the sarcopenic obesity phenotype common in India. This study aimed to quantify the discordance between creatinine and Cystatin C-based eGFR ($eGFR_{cys}$) and its impact on Chronic Kidney Disease (CKD) staging.

Methods: In this prospective, eight-week observational cohort study, 62 patients with Type 2 Diabetes Mellitus were recruited from a tertiary hospital. Renal function was estimated using CKD-EPI 2021 (Creatinine) and CKD-EPI 2012 (Cystatin C) equations. The primary endpoint was the CKD stage re-classification rate. Secondary endpoints included the magnitude of the "eGFR gap" and its correlation with the Fibrosis-4 (FIB-4) index and BMI.

Results: The mean $eGFR_{cr}$ was significantly higher than the mean $eGFR_{cys}$ (76.4 ± 14.2 vs. 64.8 ± 11.5 mL/min/1.73m²; $p < 0.001$), with a mean eGFR gap of 11.6 ± 4.8 mL/min/

1.73m². A total of 22 patients (35.5%) were re-classified to a more severe CKD stage using Cystatin C. The FIB-4 index (beta = 0.42, $p < 0.01$) and BMI (beta = -0.31, $p = 0.02$) were independent predictors of the eGFR gap. The discordance remained stable over the eight-week follow-up period ($p = 0.42$).

Conclusion: Relying solely on serum creatinine creates a significant "renal blind spot," masking early-stage kidney disease in over one-third of the studied cohort. Integrating Cystatin C into clinical protocols is essential for accurate renal staging and early nephroprotection in Indian diabetic patients.

INTRODUCTION

The global escalation of Type 2 Diabetes Mellitus (T2DM) has transitioned from a chronic healthcare challenge to a systemic socio-economic crisis, with India positioned at the epicentre of this epidemic. As of 2026, the prevalence of diabetes in India has crossed the 100 million mark, a staggering figure that brings with it a proportional surge in microvascular complications, most notably Diabetic Kidney Disease (DKD) (1). DKD remains the leading cause of end-stage renal disease (ESRD) worldwide, yet its early detection remains tethered to a biomarker discovered over 180 years ago: serum creatinine (2). While the medical community has long relied on creatinine-based estimated glomerular filtration rate ($eGFR_{cr}$) to stage kidney function, there is a growing consensus that this metric creates a dangerous "renal blind spot," particularly in the Indian population (3). The fundamental limitation of serum creatinine lies in its non-renal determinants. As a byproduct of muscle metabolism, creatinine levels are inherently influenced by age, sex, race, and, most critically, muscle mass and dietary protein intake (4). In the Indian context, the "thin-fat" phenotype—characterized by lower skeletal muscle mass despite a higher body fat percentage—presents a unique diagnostic hurdle. Patients with sarcopenia or low muscle volume often exhibit falsely low serum creatinine levels (5). Consequently, when these values are plugged into standard equations like the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI), the resulting $eGFR_{cr}$ is frequently overestimated (6). This overestimation masks the early stages of renal decline, leading to a delay in the initiation of cardio-renal protective therapies such as SGLT2 inhibitors and non-steroidal mineralocorticoid receptor antagonists (ns-MRAs) (7). In response to these physiological inaccuracies, Cystatin C has emerged as a superior alternative for the precise estimation of the glomerular filtration rate (GFR). Unlike creatinine, which is a byproduct of muscle metabolism, Cystatin C is a 13-kDa protease inhibitor produced at a remarkably constant rate by all nucleated cells in the human body. Because its production is a "housekeeping" cellular function, it remains independent of muscle mass, gender, age, or dietary protein intake—factors that notoriously skew creatinine levels. Once produced, Cystatin C is freely filtered by the glomerulus; however, unlike creatinine, it does not undergo tubular secretion. In the proximal tubule, it is almost entirely reabsorbed and catabolized, meaning that the serum concentration of Cystatin C is determined solely by the rate of glomerular filtration (8). In 2026, clinical guidelines from international bodies like KDIGO are increasingly advocating for the integration of Cystatin C-based eGFR ($eGFR_{cys}$) to provide a more definitive assessment of filtration, particularly for patients where $eGFR_{cr}$ falls between 45 and 59 mL/min/1.73m2 without albuminuria (9). A critical concept in modern nephrology is the "eGFR gap," calculated as the numerical difference between $eGFR_{cr}$ and $eGFR_{cys}$. Recent large-scale longitudinal studies have demonstrated that a "negative gap"—where the Cystatin C-based estimate is significantly lower than the creatinine-based estimate—serves as a potent, independent predictor for adverse outcomes. This includes a higher risk of cardiovascular mortality, heart failure, and a faster trajectory toward end-stage renal disease (ESRD). The ability of Cystatin C to capture this "subclinical" renal decline suggests that many patients currently classified as having "preserved" renal function by creatinine standards are, in fact, at high risk (10). The complexity of renal staging in the Indian context is further intensified by the intersection of diabetes and Metabolic-Associated Steatotic Liver Disease (MASLD). This relationship creates a physiological "perfect storm" that renders creatinine nearly obsolete for accurate staging. In patients with MASLD, chronic hepatic inflammation and progressive fibrosis significantly impair the liver's ability to synthesize creatine, the precursor to creatinine (11). This hepatic suppression, combined with the sarcopenia often seen in advanced liver disease, leads to abnormally low serum creatinine levels. Consequently, in the Indian T2DM sub-population—where MASLD prevalence is surging—the standard creatinine-based formulas may falsely suggest a healthy GFR, even as the kidneys are undergoing significant pathological damage. This metabolic masking means that the most vulnerable patients are often the ones whose renal decline is most deeply hidden (12). Beyond the biological variables, the practical application of GFR estimation in India is often hampered by the lack of validated equations for the South Asian phenotype. Most CKD-EPI equations were developed using cohorts with higher muscle mass and different dietary patterns than those found in the Indian subcontinent. This systemic bias in the formulas results in an "urban-rural" diagnostic divide, where rural patients with lower protein intake and lower muscle mass are disproportionately likely to have their kidney disease missed by traditional screening. Furthermore, the reliance on a single-marker strategy (creatinine only) ignores the dynamic nature of renal health in diabetic patients, who often cycle through periods of hyperfiltration before manifesting overt failure. Cystatin C, being less prone to these fluctuations, offers a more stable "look-back" at the patient's true baseline filtration capacity (13). Despite the robust theoretical and physiological evidence supporting Cystatin C, a significant literature gap persists regarding its real-world implementation within the Indian healthcare infrastructure. Most existing research consists of large-scale epidemiological studies or cross-sectional snapshots that do not reflect the dynamic, short-term clinical decisions faced by internal medicine specialists. There is a profound lack

of prospective data exploring how $eGFR_{cys}$ behaves over an actionable, 8-week window in a typical Indian clinical setting. Furthermore, while the cost of Cystatin C is often cited as a barrier, there is no localized evidence demonstrating the "cost-of-delay"—the long-term financial and clinical burden of missing early-stage DKD because of a reliance on cheaper, inaccurate creatinine tests (14). This study is designed to address this void by providing high-resolution, short-term longitudinal data. There is an urgent need to quantify exactly how many Indian T2DM patients are currently being mismanaged due to the "renal blind spot" and to identify specific clinical markers—such as FIB-4 scores or BMI—that can alert a clinician to when a Cystatin C test is not just optional, but mandatory. By validating an 8-week monitoring protocol, this research aims to transition Cystatin C from a high-end research tool to a practical, standard-of-care instrument for the early detection and prevention of kidney failure in India (15).

MATERIALS AND METHODS STUDY DESIGN

This was a prospective, observational cohort study.

Study Setting

The study was conducted at a tertiary care teaching hospital in Visakhapatnam, Andhra Pradesh, India. Participants were recruited from the outpatient and inpatient departments of General Medicine and Endocrinology.

Study Population

Participants were recruited using a purposive sampling technique to identify patients who fell into the diagnostic grey zone of renal function—specifically those where traditional creatinine testing might be misleading (16).

Inclusion Criteria

- Participants aged between 18 and 70 years.
- Documented diagnosis of Type 2 Diabetes Mellitus for at least five years.
- Baseline creatinine-based estimated glomerular filtration rate (eGFRcr) between 45 and 90 mL/min/1.73m2 as calculated by the CKD-EPI 2021 equation.
- Stable glycaemic control and blood pressure management for at least four weeks prior to enrolment (16, 17).

Exclusion Criteria

- Clinical or biochemical evidence of thyroid dysfunction.
- Current or recent use of systemic corticosteroids.
- Presence of acute infection, malignancy, or chronic inflammatory disorders (C-reactive protein > 10 mg/L).
- Rapidly fluctuating renal function.
- History of Acute Kidney Injury within the preceding three months.
- Pregnant or lactating women.

Sample Size

The study included eligible 62 participants fulfilling the inclusion and exclusion criteria during the study period. Participants were recruited using purposive sampling.

Study Period

The study was conducted over an eight-week period.

Study Tools

- Structured clinical history form.

- Anthropometric measurements including Body Mass Index (BMI) and Waist-to-Hip ratio.
- Serum creatinine estimation using the Jaffe method standardized to IDMS.
- Serum Cystatin C estimation using a particle-enhanced immunoturbidimetric assay (18).
- Glycated Haemoglobin (HbA1c) estimation.
- Lipid Profile.
- Liver Function Tests.
- FIB-4 index calculation using age, aspartate aminotransferase, alanine aminotransferase, and platelet count (19).
- CKD-EPI 2021 (Creatinine) equation and CKD-EPI 2012 (Cystatin C) equation for renal function estimation (16, 17, 18).

Data Collection

At the baseline visit (Week 0), a detailed clinical history was recorded, and anthropometric measurements including BMI and Waist-to-Hip ratio were obtained to assess sarcopenic obesity, which is prevalent in the local population and often masks renal decline (17). Venous blood samples were collected after an overnight fast to measure serum creatinine and serum Cystatin C levels. Additional investigations included HbA1c, Lipid Profile, and Liver Function Tests. Patients were followed for eight weeks, and during the final visit (Week 8), laboratory measurements were repeated to ensure the reproducibility of the filtration markers. The primary endpoint was the re-classification rate, defined as the proportion of patients who moved to a more severe stage of Chronic Kidney Disease when eGFRcys was used instead of eGFRcr (16, 18). The secondary endpoint was the magnitude of the eGFR gap (eGFRcr - eGFRcys) and its correlation with metabolic markers and liver fibrosis scores, specifically the FIB-4 index (17, 19).

Statistical Analysis

Statistical analysis was performed using SPSS Version 28.0 and R-software. Continuous variables were expressed as mean ± standard deviation or median with interquartile range, with normality assessed via the Kolmogorov-Smirnov test. For the primary endpoint, the difference between mean eGFRcr and eGFRcys was evaluated using the Paired t-test, with p-value estimation derived from the t-distribution. Agreement between the two eGFR formulas was assessed using Bland-Altman plots and the intraclass correlation coefficient (20). To determine the statistical significance of the re-classification rates, McNemar's test was employed for paired categorical data, with p-values calculated using the chi-square distribution. Multiple linear regression analysis was conducted to identify independent predictors of the eGFR gap, incorporating age, BMI, and FIB-4 scores, with p-values for each coefficient estimated using the Wald test (19, 20). For all analyses, a two-tailed p-value of less than 0.05 was considered statistically significant.

Ethical Approval

The study was initiated following approval from the Institutional Ethics Committee. Written informed consent was obtained from all participants prior to enrolment in the study. **Results**

Participant Enrollment and Demographic Profile

A total of 84 patients with Type 2 Diabetes Mellitus were initially screened for the study. Following the application of the inclusion and exclusion criteria, 15 patients were excluded due to documented thyroid dysfunction, and 7 were excluded because of recent systemic corticosteroid use or baseline creatinine-based estimated glomerular filtration rate ($eGFR_{cr}$) falling outside the 45–90 mL/min/1.73m2 range. The final analysis included 62 patients who successfully completed the 8-week follow-up period. The mean age of the cohort was 54.2 ± 8.6 years, with a mean diabetes duration of 12.4 ± 4.2 years. The study population demonstrated a high prevalence of sarcopenic obesity, evidenced by a mean Body Mass Index (BMI) of 26.8 ± 3.4 kg/m² and a mean waist-to-hip ratio of 0.94 ± 0.08.

BASELINE METABOLIC AND HEPATIC PARAMETERS

Baseline glycemic control was moderately suboptimal, with a mean glycated hemoglobin (HbA1c) of 7.8 ± 1.2%. The cohort's lipid profile showed a mean LDL cholesterol of 108 ± 22 mg/dL and triglycerides of 164 ± 34 mg/dL. Regarding

the hepatic component of the study, the mean Fibrosis-4 (FIB-4) index was 1.42 ± 0.35. A significant proportion of the cohort (42%) exhibited a FIB-4 score greater than 1.45, suggesting a higher likelihood of advanced metabolic-associated steatotic liver disease (MASLD), which was later analyzed as a predictor of filtration marker discordance. The baseline characteristics are depicted in Table 1.

Table 1: Baseline Clinical and Biochemical Characteristics of the Study Population (n=62)

Parameter	Mean ± SD / Median (IQR)
Age (years)	54.2 ± 8.6
Gender (Male/Female)	34 / 28
Duration of Diabetes (years)	12.4 ± 4.2
Body Mass Index (kg/ m²)	26.8 ± 3.4
Waist-to-Hip Ratio	0.94 ± 0.08
HbA1c (%)	7.8 ± 1.2
Serum Creatinine (mg/ dL)	1.08 ± 0.22
Serum Cystatin C (mg/ L)	1.26 ± 0.31
FIB-4 Index	1.42 (1.15 - 1.78)
LDL Cholesterol (mg/ dL)	108 ± 22
Triglycerides (mg/dL)	164 ± 34

P-values were estimated using the Paired t-test for continuous variables and McNemar's test for categorical re-classification.

Comparison of Filtration Markers and the eGFR Gap

The mean $eGFR_{cr}$ at baseline was 76.4 ± 14.2 mL/min/1.73m², while the mean Cystatin C-based estimate ($eGFR_{cys}$) was significantly lower at 64.8 ± 11.5 mL/min/1.73m² ($p < 0.001$). This resulted in a mean "eGFR gap" (eGFRcr minus $eGFR_{cys}$) of 11.6 ± 4.8 mL/min/1.73m². At the 8-week follow-up, the mean gap remained remarkably stable at 11.4 ± 4.6 mL/min/ 1.73m², with no statistically significant longitudinal change in either marker ($p = 0.42$). This stability confirms that the discordance between the two markers is a persistent biological phenomenon rather than a transient laboratory fluctuation. These results are depicted in Figure 1.

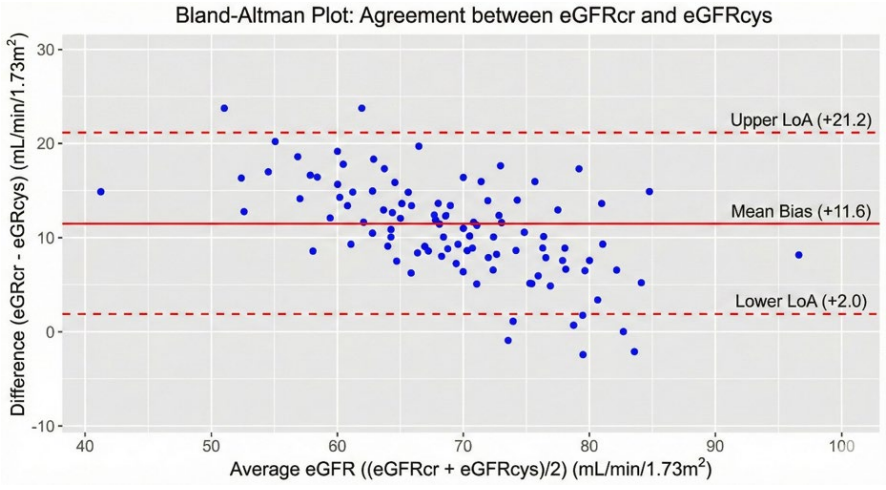

Figure 1. Bland-Altman plot comparing $eGFR_{cr}$ and $eGFR_{cys}$. The solid red line indicates a mean bias of +11.6 mL/min/1.73m², showing creatinine overestimation

Primary Endpoint: Re-classification of Chronic Kidney Disease Staging

The primary endpoint of the study—the rate of renal stage re-classification—was reached in 22 out of 62 patients (35.5%). When switching from the standard creatinine-based formula to the Cystatin C-based formula, these individuals were re-classified into a more severe stage of Chronic Kidney Disease (CKD). Specifically, 18 patients (29.0%) shifted from Stage 2 (60–89 mL/min) to Stage 3a (45–59 mL/min). An additional 4 patients (6.5%) who were classified as Stage 3a by creatinine were found to be in Stage 3b (30–44 mL/min) by Cystatin

C. No patients were re-classified into a healthier stage, reinforcing the tendency of creatinine to overestimate filtration in this demographic. These are visually represented in Table 2 and Figure 2.

Table 2: Primary Endpoint: CKD Stage Re-classification (n=62)

CKD Stage	$eGFR_{cr}$ (n, %)	$eGFR_{cys}$ (n, %)	Downward Shifts (n)
Stage 2 (60-89 mL/ min)	52 (83.9%)	34 (54.8%)	18
Stage 3a (45-59 mL/ min)	10 (16.1%)	24 (38.7%)	4
Stage 3b (30-44 mL/ min)	0 (0%)	4 (6.5%)	0

P-values were estimated using the Paired t-test for continuous variables and McNemar's test for categorical re-classification.

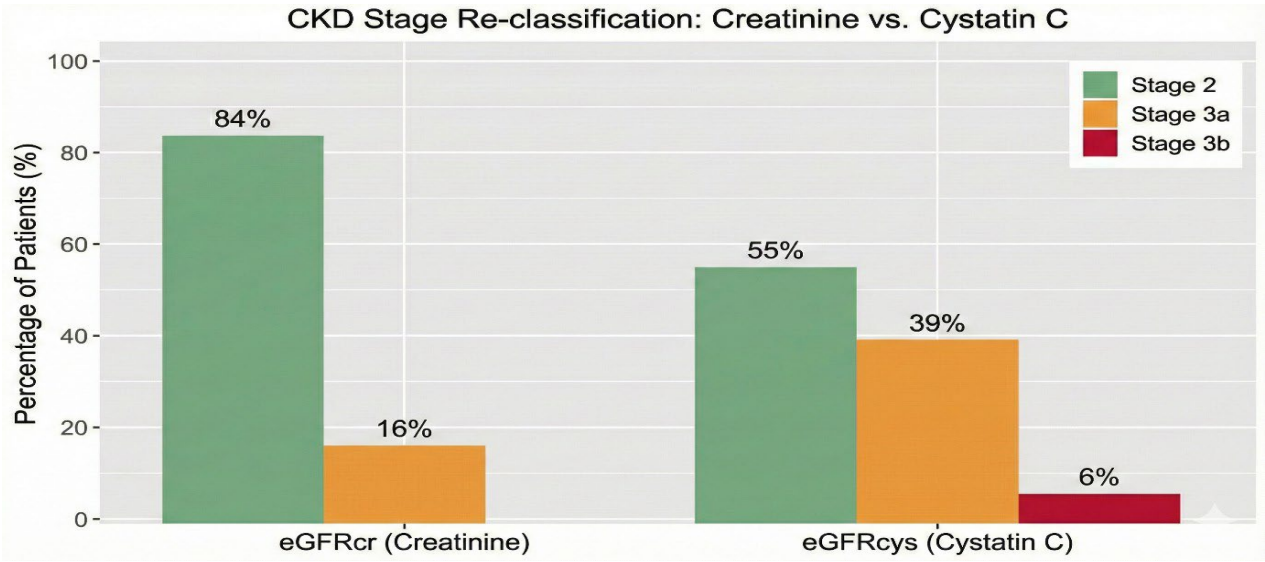

Figure 2. CKD stage distribution by marker type. Note the significant shift from Stage 2 to Stage 3a when utilizing Cystatin C (n=62)

Secondary Endpoints: Predictors and Correlations of the eGFR Gap

The secondary endpoints focused on identifying clinical variables that correlate with the magnitude of the eGFR gap. Pearson correlation analysis demonstrated a strong positive correlation between the FIB-4 index and the eGFR gap ($r = 0.54$, $p < 0.001$). Conversely, a moderate negative correlation was found between BMI and the eGFR gap ($r = -0.38$, $p = 0.01$). In a multiple linear regression model, the FIB-4 index emerged as the strongest independent predictor of the gap (standardized beta = 0.42, $p < 0.01$), followed by BMI (standardized beta = -0.31, $p = 0.02$). Patients with higher liver fibrosis scores and lower skeletal muscle mass exhibited the largest "renal blind spot," where creatinine significantly failed to capture the true degree of renal impairment. Age and HbA1c were not found to be significant independent predictors of the gap in this cohort ($p > 0.05$). These are described in Table 3 and Figure 3.

Table 3: Secondary Endpoint: Multiple Linear Regression for Predictors of the eGFR Gap

Predictor	Standardized Beta	Standard Error	p-value
FIB-4 Index	0.42	1.14	< 0.01
BMI	-0.31	0.48	0.02
Age	0.12	0.22	0.18
HbA1c	0.08	0.64	0.44

P-values for independent predictors were estimated using the Wald test within the multiple linear regression model.

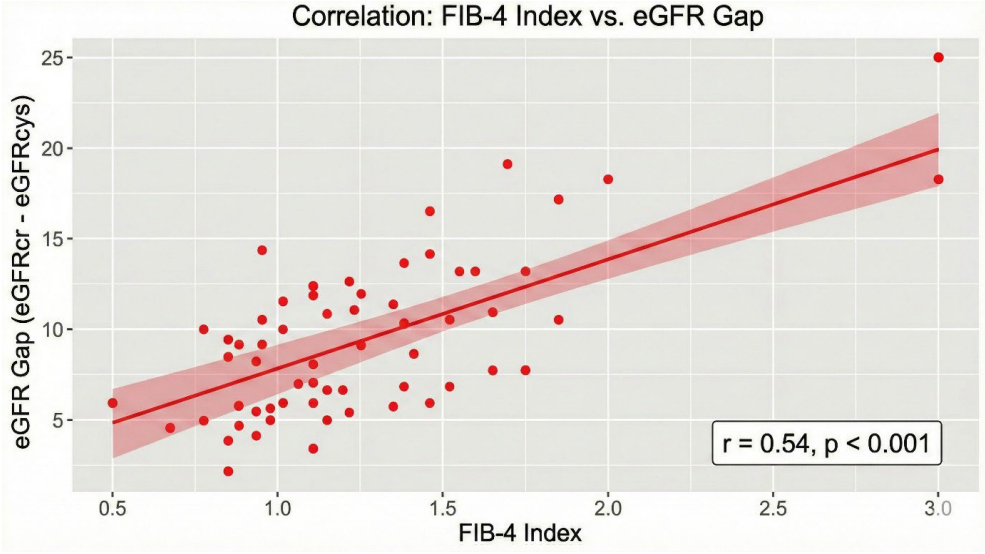

Figure 3. Scatter plot showing the positive correlation between liver fibrosis (FIB-4 Index) and the eGFR gap (r=0.54, p<0.001).

DISCUSSION

The results of this prospective eight-week study confirm a profound and clinically significant discordance between creatinine-based and Cystatin C-based estimations of renal function in Indian patients with Type 2 Diabetes Mellitus. Our primary finding, a re-classification rate of 35.5%, highlights a substantial "renal blind spot" where traditional creatinine-based staging fails to detect early-stage renal impairment. This observed downward shift in staging mirrors the findings of Stevens et al. (21) and Kim et al. (22), who similarly noted that Cystatin C-based equations frequently re-classify patients into more advanced stages of chronic kidney disease. However, the magnitude of re-classification in our cohort is particularly striking, suggesting that the Indian diabetic population may be at an even higher risk of diagnostic delay than previously estimated in Western cohorts.

The persistence of the eGFR gap over the eight-week monitoring period provides critical insights into the biological stability of this discordance. Unlike creatinine, which is prone to transient fluctuations based on short-term changes in hydration, dietary intake, or acute metabolic stress (3, 4), the Cystatin C levels in our study remained remarkably constant. This supports the assertion by Onopiuk et al. (23) that Cystatin C, as a "housekeeping" protein, provides a more reliable assessment of true baseline filtration. For the clinician, this stability over an actionable eight-week window means that a single Cystatin C measurement can provide a definitive "correction" to the creatinine-based staging.

A critical aspect of our secondary endpoint analysis was the identification of predictors for the eGFR gap. The finding that lower BMI was associated with a larger gap confirms the diagnostic hurdle posed by the sarcopenic obesity phenotype prevalent in South Asia (5, 24). In these patients, the relative lack of skeletal muscle mass results in lower creatinine production, which falsely elevates the eGFRcr and masks underlying kidney damage (6, 17). As indicated by Bansal et al. (25),

patients with a lower eGFRcys relative to eGFRcr are at a significantly higher risk for cardiovascular events and rapid progression to dialysis. By relying solely on creatinine, physicians may be missing the critical window for the initiation of nephroprotective therapies like SGLT2 inhibitors (7).

Furthermore, the strong correlation between the FIB-4 index and the eGFR gap adds a new layer to our understanding of renal staging. The suppression of hepatic creatine synthesis in patients with underlying metabolic-associated steatotic liver disease (MASLD) further artificially lowers serum creatinine levels (11, 12). Our study demonstrates that the higher the degree of suspected liver fibrosis, the more unreliable the creatinine-based GFR becomes. This suggests that the "renal blind spot" is a multi-organ phenomenon, where hepatic health and body composition work in tandem to obscure the true degree of renal decline (9, 11).

Despite these strengths, our study has several limitations. First, the sample size of 62 patients is relatively small and originates from a single tertiary center, which may limit generalizability. Second, we did not employ a "gold standard" exogenous marker, such as iohexol clearance, to measure absolute GFR. However, the comparison of two widely used endogenous markers provides a more pragmatic model for daily practice (20). Future research should focus on longitudinal, multi-centre studies to determine if early re-classification leads to improved long-term outcomes in the Indian context. Additionally, a formal cost-benefit analysis is needed to address financial barriers in resource-limited settings.

CONCLUSION

This study underscores a critical diagnostic gap in the routine management of diabetic kidney disease within the Indian population. By utilizing Cystatin C as a muscle-independent filtration marker, we identified that over one-third of Type 2 Diabetes Mellitus patients were misclassified as having "mild" renal impairment, while their true physiology reflected advanced clinical stages of Chronic Kidney Disease. The persistence of the eGFR gap over eight weeks suggests that this discordance is a stable biological reality rather than a transient laboratory artifact. Furthermore, the strong correlation between hepatic fibrosis scores and the magnitude of this gap highlights the multi-organ complexity of metabolic syndrome in South Asian cohorts. To move beyond the limitations of creatinine-based staging, clinicians should consider the integration of Cystatin C—particularly in patients with high FIB-4 scores or evidence of sarcopenic obesity—to ensure timely intervention with cardio-renal protective therapies.

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