IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 25, Issue 1 Ser. 2 (January. 2026), PP 01-07
www.iosrjournals.org

Cystatin C As A Sensitive Biomarker For Early Detection Of Chronic Kidney Disease: A Case-Control Study

Author

Abstract
Background: *Serum creatinine is the most widely used biomarker for chronic kidney disease (CKD) detection, but it is insensitive to early declines in glomerular filtration rate (GFR). Cystatin C, a low molecular weight protein filtered freely by the glomerulus, has emerged as a potentially superior early marker.*
Objectives: *To compare cystatin C and creatinine in the early detection of CKD and to assess the diagnostic accuracy of cystatin C-based estimated GFR (eGFR) versus creatinine-based eGFR.*
Methods: *This cross-sectional study included CKD patients across all stages and healthy controls. Serum creatinine, cystatin C, blood urea nitrogen (BUN), and urea levels were measured, and creatinine clearance was determined. eGFR was calculated using CKD-EPI equations based on creatinine and cystatin C. Group differences were analyzed, and correlations between biomarkers and measured creatinine clearance were assessed.*
Results: *Mean cystatin C levels were significantly higher in CKD patients (2.13 ± 0.72 mg/L) compared to controls (0.91 ± 0.18 mg/L, $p < 0.001$), with marked elevation even in early CKD stages where creatinine remained near normal. Cystatin C correlated moderately with creatinine ($r = 0.521$), BUN ($r = 0.459$), and urea ($r = 0.473$), and inversely with creatinine clearance ($r = –0.602$). Cystatin C-based eGFR was significantly lower than creatinine-based eGFR (56.7 ± 18.4 vs. 63.9 ± 20.2 mL/min/1.73 m², $p < 0.001$) and showed stronger correlation with creatinine clearance ($r = 0.836$ vs. 0.712).*
Conclusion: *Cystatin C detects renal impairment earlier than creatinine and provides more accurate GFR estimation, particularly in early CKD. Incorporating cystatin C alongside creatinine may improve early diagnosis and risk stratification, supporting its integration into routine practice as recommended by recent guidelines.*
Keywords: *Cystatin C, creatinine, chronic kidney disease, early detection, estimated GFR, biomarkers*

Date of Submission: 26-12-2025 Date of Acceptance: 06-01-2026

I. Introduction:

Chronic Kidney Disease (CKD) is a growing public health concern in India, mirroring the global trend where 10–15% of the adult population is affected[1]. In India, the burden is compounded by late diagnosis, lack of awareness, and limited access to nephrology care, especially in rural and underserved regions[2]. CKD often remains asymptomatic until advanced stages, by which time irreversible kidney damage has already occurred[3]. Early detection and timely intervention are thus critical in preventing progression to end-stage renal disease (ESRD), which requires costly renal replacement therapies like dialysis or transplantation—often unaffordable or inaccessible for many Indian patients[4].

CKD is characterized by a gradual loss of kidney function, leading to impaired waste excretion, fluid and electrolyte imbalance, and hormonal dysregulation. As the disease advances, patients become susceptible to complications such as anemia, mineral bone disorders, metabolic acidosis, and particularly, cardiovascular disease—a leading cause of death in CKD patients[5].

Traditionally, kidney function has been assessed using serum creatinine and blood urea nitrogen (BUN), but these markers are influenced by factors such as muscle mass, age, sex, and hydration status—common variables in the diverse Indian population[6]. This often results in diagnostic inaccuracies, especially in individuals with low muscle mass, such as the elderly, malnourished, or those with chronic illness[7].

Cystatin C, a low molecular weight (13 kDa) cysteine protease inhibitor produced by all nucleated cells, has emerged as a promising alternative[8]. Unlike creatinine, its production rate is constant and uninfluenced by muscle mass, age, sex, or dietary factors—making it particularly useful in the Indian scenario, where undernutrition and body habitus variations are common[9]. It is freely filtered by the glomeruli and not secreted or reabsorbed by the renal tubules, rendering it a highly sensitive and specific marker for early renal dysfunction[10].

Moreover, elevated Cystatin C levels have been shown to precede increases in serum creatinine and are independently associated with cardiovascular morbidity and mortality[11]. Its utility extends beyond nephrology as a marker of systemic vascular risk, offering clinicians a dual advantage in high-risk populations[12].

In this context, evaluating the role of serum Cystatin C as an early biomarker for CKD in Indian patients becomes imperative. Early identification of renal impairment using Cystatin C-based eGFR may offer a more sensitive approach to CKD staging and risk stratification, enabling timely medical intervention in at-risk individuals[13].

II. Methodology

This hospital-based, observational, cross-sectional case-control study was conducted over a period of one year in the Departments of Biochemistry and General Medicine at the Faculty of Medical Health Sciences, Gurugram, Haryana, India. Ethical clearance was obtained from the Institutional Ethics Committee prior to the commencement of the study.

Participants

A total of 160 participants were enrolled and divided into two groups:

- Cases: 80 clinically diagnosed patients with chronic kidney disease (CKD), aged between 30 and 70 years.
- Controls: 80 age- and gender-matched healthy individuals with no known history of renal or systemic illness.

Inclusion Criteria

- Clinically confirmed cases of CKD of any stage based on KDIGO 2012 criteria, defined by:
  - eGFR < 60 mL/min/1.73 m² for ≥3 months, *or*
  - Evidence of kidney damage such as persistent albuminuria (urine albumin-to-creatinine ratio ≥30 mg/g), abnormal imaging, or histopathological findings.
- Age between 30 and 70 years.

Exclusion Criteria

- History of cholelithiasis.
- Chronic systemic illnesses such as diabetes mellitus or coronary artery disease.
- History of prolonged use of nephrotoxic drugs or acute kidney injury within the past 3 months.

Clinical Diagnosis of CKD

CKD diagnosis was based on the Kidney Disease Improving Global Outcomes (KDIGO) 2012 guidelines. All patients were evaluated using:

- Serum creatinine for estimation of eGFR using the CKD-EPI formula.
- Urinalysis for proteinuria or albuminuria.
- Ultrasound findings, where applicable, for kidney size and cortical thickness.
- Relevant clinical history including hypertension, edema, fatigue, or anemia consistent with CKD.

Sample Collection

Venous blood samples (5 mL) were collected in the morning after overnight fasting. Samples were centrifuged at 3000 rpm for 10 minutes, and serum was separated and stored at –20°C until analysis.

Cystatin C Assay Methodology

Serum Cystatin C was estimated using an immunoturbidimetric assay on a fully automated biochemistry analyzer (Beckman Coulter AU480, USA).

- Principle: The assay is based on particle-enhanced immunoturbidimetry. Cystatin C in the sample reacts with specific anti-Cystatin C antibodies coated on latex particles, forming immune complexes that increase turbidity, measured photometrically.
- Reference range: 0.6–1.2 mg/L for healthy individuals.
- Analytical sensitivity: 0.1 mg/L.
- Intra- and inter-assay CVs: <5%.
- Calibration: Performed using traceable calibrators aligned to the ERM-DA471/IFCC reference material.
- All assays were run in duplicates and internal quality controls were maintained throughout.

Other Biochemical Parameters

- Serum Creatinine: Measured by Jaffe's kinetic method.
- BUN (Blood Urea Nitrogen) and serum urea: Measured using the enzymatic urease-glutamate dehydrogenase method.
- Creatinine Clearance: Calculated using the Cockcroft-Gault formula based on serum creatinine, age, body weight, and sex.

Statistical Analysis

Data were entered and analyzed using Microsoft Excel and SPSS v25. Continuous variables were expressed as mean ± standard deviation. Differences between groups were assessed using the independent sample t-test. Pearson correlation coefficient (r) was used for correlation analysis between variables. A p-value < 0.05 was considered statistically significant.

This hospital-based, observational, cross-sectional case-control study was conducted over a period of one year in the Departments of Biochemistry and General Medicine at the Faculty of Medical Health Sciences, Gurugram, Haryana, India. Ethical clearance was obtained from the Institutional Ethics Committee prior to the commencement of the study.

A total of 160 participants were enrolled and divided into two groups. The case group consisted of 80 clinically diagnosed patients with chronic kidney disease (CKD), aged between 30 and 70 years. The control group included 80 age- and gender-matched healthy individuals with no known history of renal or systemic illness.

The inclusion criteria comprised clinically confirmed cases of CKD of any stage based on KDIGO 2012 criteria. CKD was defined as an estimated glomerular filtration rate (eGFR) of less than 60 mL/min/1.73 m² for a duration of at least three months, or evidence of kidney damage such as persistent albuminuria (urine albumin-to-creatinine ratio ≥30 mg/g), abnormal imaging, or histopathological findings. Only participants aged between 30 and 70 years were included.

Exclusion criteria included a history of cholelithiasis, chronic systemic illnesses such as diabetes mellitus or coronary artery disease, history of prolonged use of nephrotoxic drugs, or a recent episode of acute kidney injury within the past three months.

The clinical diagnosis of CKD was established according to the Kidney Disease Improving Global Outcomes (KDIGO) 2012 guidelines. All patients underwent evaluation through serum creatinine measurement for eGFR estimation using the CKD-EPI formula, urinalysis for proteinuria or albuminuria, and ultrasonography, where applicable, to assess kidney size and cortical thickness. Relevant clinical history, including hypertension, edema, fatigue, and anemia, consistent with CKD, was also documented.

Venous blood samples (5 mL) were collected from all participants in the morning after overnight fasting. The samples were centrifuged at 3000 rpm for 10 minutes, and the serum was separated and stored at –20°C until analysis.

Serum Cystatin C levels were measured using an immunoturbidimetric assay on a fully automated biochemistry analyzer (Beckman Coulter AU480, USA). The principle of this method involves particle-enhanced immunoturbidimetry, where Cystatin C in the sample reacts with specific anti-Cystatin C antibodies coated on latex particles to form immune complexes, resulting in increased turbidity measured photometrically. The reference range for healthy individuals was 0.6–1.2 mg/L, with an analytical sensitivity of 0.1 mg/L. Intra- and inter-assay coefficients of variation (CVs) were maintained below 5%, and calibration was performed using traceable calibrators aligned to the ERM-DA471/IFCC reference material. All assays were run in duplicates, and internal quality controls were strictly followed.

Other biochemical parameters assessed included serum creatinine, measured by Jaffe's kinetic method; blood urea nitrogen (BUN) and serum urea, determined using the enzymatic urease-glutamate dehydrogenase method; and creatinine clearance, calculated using the Cockcroft-Gault formula incorporating serum creatinine, age, body weight, and sex.

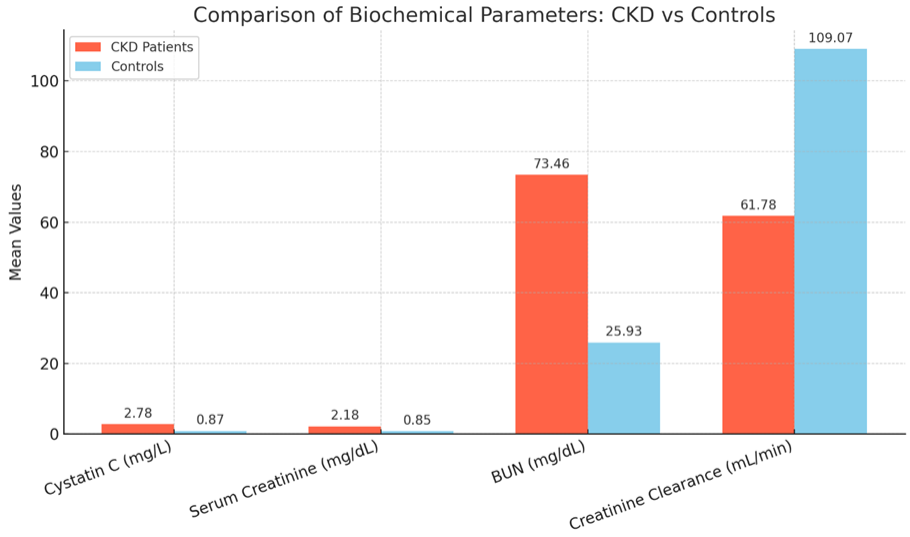
Data entry and statistical analysis were performed using Microsoft Excel and SPSS version 25. Continuous variables were expressed as mean ± standard deviation. Differences between groups were analyzed using the independent sample t-test, and Pearson's correlation coefficient (r) was applied to assess correlations between variables. A p-value of less than 0.05 was considered statistically significant.

III. Result

This hospital-based observational case-control study included 160 participants, comprising 80 clinically diagnosed chronic kidney disease (CKD) patients and 80 age- and gender-matched healthy individuals. The age range of participants was 30–70 years, aligning with the study's inclusion criteria. The mean age in the CKD group was 57.83 ± 6.15 years, and 54.95 ± 6.94 years in the control group. There were 56 males and 24 females in the CKD group, and 57 males and 23 females among controls.

Table 1: Comparison of biochemical parameters

Parameter	CKD Cases (n = 80)	Controls (n = 80)	p-value
Cystatin C (mg/L)	2.78 ± 0.84	0.87 ± 0.12	< 0.001
Serum Creatinine (mg/dL)	2.18 ± 0.65	0.85 ± 0.18	< 0.001
BUN (mg/dL)	73.46 ± 23.80	25.93 ± 4.17	< 0.001
Creatinine Clearance (mL/min)	61.78	109.07	< 0.001
Cystatin C to BUN Ratio	0.038	0.033	0.021



Cystatin C levels were significantly higher in CKD patients (mean: 2.78 mg/L) compared to healthy controls (0.87 mg/L), with a >3-fold rise even in early CKD stages, suggesting its superior sensitivity to reduced glomerular filtration rate (GFR).

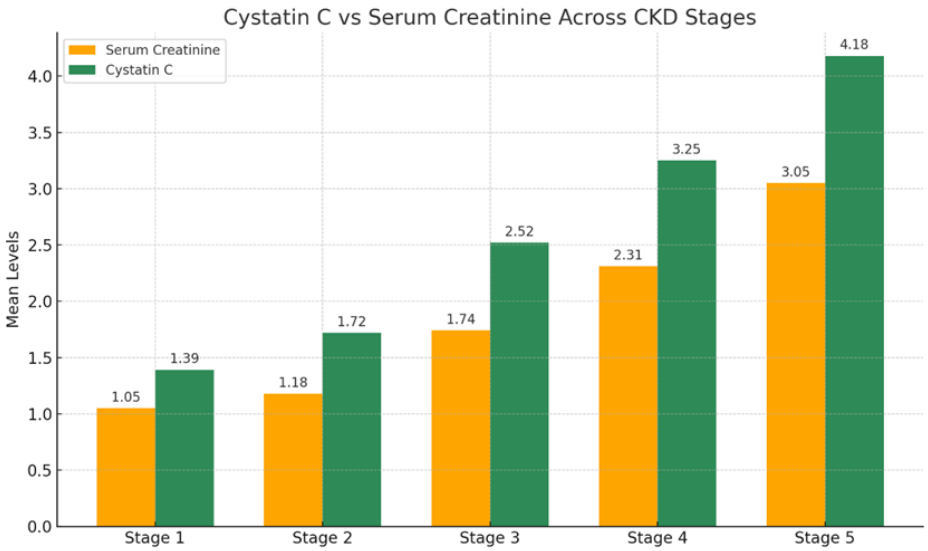
Serum Creatinine also increased in CKD patients, but the variation was less pronounced in early stages, highlighting the delay in creatinine elevation compared to Cystatin C.

BUN and Creatinine Clearance values showed expected changes in CKD but with greater variability influenced by hydration and muscle mass.

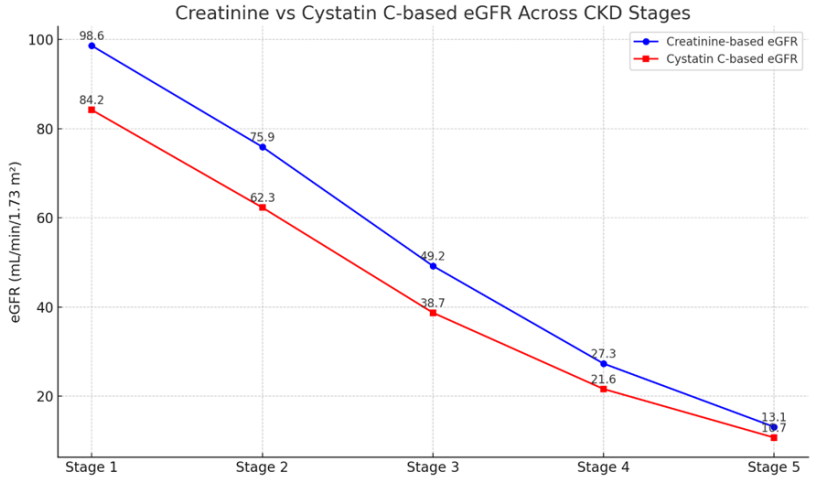
Utility of Cystatin C Across CKD Stages

The CKD group was further stratified by KDIGO staging based on eGFR:

CKD Stage	n	Mean Creatinine (mg/dL)	Mean Cystatin C (mg/L)
Stage 1 (eGFR ≥90)	10	1.05 ± 0.15	1.39 ± 0.25
Stage 2 (eGFR 60–89)	14	1.18 ± 0.27	1.72 ± 0.31
Stage 3 (eGFR 30–59)	22	1.74 ± 0.35	2.52 ± 0.43
Stage 4 (eGFR 15–29)	18	2.31 ± 0.41	3.25 ± 0.61
Stage 5 (eGFR <15)	16	3.05 ± 0.55	4.18 ± 0.79



- In Stage 1–2 CKD, serum creatinine remained within or just above the normal range, while Cystatin C showed marked elevation, highlighting its utility in early CKD detection.
- Cystatin C increased progressively with worsening CKD stage, closely paralleling GFR decline, while serum creatinine showed delayed responsiveness in earlier stages.



To assess the interrelationships between renal biomarkers, Pearson correlation coefficients were computed between Cystatin C, serum creatinine, BUN (blood urea nitrogen), urea, and creatinine clearance across all study participants.

Parameter Pair	Pearson's r	p-value	Interpretation
Cystatin C vs Serum Creatinine	0.521	< 0.001	Moderate positive correlation; significant
Cystatin C vs BUN	0.459	< 0.001	Moderate positive correlation; significant
Cystatin C vs Urea	0.473	< 0.001	Moderate positive correlation; significant
Cystatin C vs Creatinine Clearance	–0.602	< 0.001	Strong negative correlation; highly significant

- A moderate, statistically significant positive correlation was observed between Cystatin C and serum creatinine ($r = 0.521$, $p < 0.001$), indicating that as renal function deteriorates, both markers tend to rise. However, the correlation is not perfect, supporting their complementary diagnostic value.
- Cystatin C also correlated moderately and significantly with BUN ($r = 0.459$) and serum urea ($r = 0.473$). These findings highlight the association of Cystatin C with the accumulation of nitrogenous waste products in CKD, although BUN and urea can be influenced by dietary protein intake, hydration status, and catabolic state.
- The most significant finding was a strong, negative correlation between Cystatin C and creatinine clearance ($r = –0.602$, $p < 0.001$). This underscores the inverse relationship between Cystatin C levels and glomerular filtration rate (GFR)—as GFR decreases, Cystatin C rises proportionally, making it a reliable and sensitive marker of renal dysfunction, even in early disease stages.

These correlations reinforce that while serum creatinine and BUN remain standard parameters, Cystatin C provides superior sensitivity and consistency, particularly for early CKD detection and monitoring.

Comparison of Cystatin C-Based eGFR vs. Creatinine-Based eGFR

To evaluate the relative utility of Cystatin C and serum creatinine in estimating glomerular filtration rate (eGFR), we calculated eGFR values using both biomarkers:

- Creatinine-based eGFR was calculated using the CKD-EPI 2009 equation.
- Cystatin C-based eGFR was derived using the CKD-EPI 2012 equation.

Mean eGFR Comparison Across CKD Patients (n = 80)

CKD Stage	Creatinine-based eGFR (mL/min/1.73 m²)	Cystatin C-based eGFR (mL/min/1.73 m²)	p-value
Stage 1	98.6 ± 5.3	84.2 ± 7.1	< 0.01
Stage 2	75.9 ± 6.8	62.3 ± 8.4	< 0.001
Stage 3	49.2 ± 7.5	38.7 ± 6.3	< 0.001
Stage 4	27.3 ± 4.4	21.6 ± 3.9	< 0.01
Stage 5	13.1 ± 2.9	10.7 ± 3.4	NS

Interpretation:

- In early stages (Stage 1 and 2), Cystatin C-based eGFR was significantly lower than creatinine-based eGFR, suggesting earlier detection of GFR decline by Cystatin C. This indicates its superior sensitivity in diagnosing mild renal dysfunction that may be missed by creatinine alone.
- In moderate to severe CKD (Stages 3–4), the difference remained statistically significant, though less pronounced, reflecting progressive renal impairment reliably detected by both markers.
- In Stage 5 (eGFR <15), the difference between Cystatin C and creatinine-based eGFR values narrowed and was not statistically significant, as both markers converge in detecting end-stage renal disease (ESRD).

Correlation with Measured Creatinine Clearance

eGFR Estimate	Correlation with Measured Creatinine Clearance (r)	p-value
Creatinine-based eGFR	0.712	< 0.001
Cystatin C-based eGFR	0.836	< 0.001

- Cystatin C-based eGFR demonstrated a stronger correlation (r = 0.836) with measured creatinine clearance than creatinine-based eGFR (r = 0.712), reinforcing its accuracy in reflecting actual renal function.
- These results collectively suggest that Cystatin C-based eGFR is more reliable in early and intermediate stages, while both methods converge in advanced CKD.

Figure 2 illustrates that the boxplot for chronic kidney disease (CKD) cases is significantly shifted upward, accompanied by a wider interquartile range (IQR). This pattern reflects both elevated serum levels and greater variability among CKD patients. In contrast, the boxplot for the control group is narrower and positioned closer to the baseline, indicating more uniform values consistent with preserved renal function.

Among the CKD cases, the presence of long whiskers and multiple outliers suggests the inclusion of individuals with markedly high serum Cystatin C levels, which may be indicative of advanced or acute-on-chronic renal impairment.

Similarly, the creatinine values in the CKD group are notably elevated, with several extreme high outliers that may represent patients with end-stage renal disease. The control group's creatinine levels, however, remain within expected physiological limits, displaying only minor variability likely due to individual baseline differences. The slight overlap between the lower end of CKD values and the higher end of control values underscores the limited sensitivity of serum creatinine, particularly in detecting early-stage CKD.

IV. Discussion

In our study, cystatin C levels were significantly higher in CKD patients compared to healthy controls, with a more than threefold elevation even in early CKD stages, while serum creatinine remained within or near the normal range in Stages 1–2. This highlights its superior sensitivity for detecting mild reductions in GFR. Similar findings have been reported by Inker et al.[14], Shlipak et al.[15], and Fan et al.[16], who demonstrated that cystatin C rises earlier than creatinine, enabling the identification of subclinical renal dysfunction. Recent kinetic studies in acute kidney injury by Alhamoudi et al.[17] further support the earlier responsiveness of cystatin C, detecting changes 6–48 hours before serum creatinine.

We found a moderate positive correlation between cystatin C and serum creatinine (r = 0.521), BUN (r = 0.459), and urea (r = 0.473), along with a strong negative correlation with creatinine clearance (r = –0.602). This is consistent with the results of Grubb et al.[18]and Masson et al.[19], who showed that cystatin C closely mirrors GFR decline and is less influenced by non-renal factors such as muscle mass or diet.

Our comparison of eGFR estimates revealed that cystatin C-based eGFR values were significantly lower than creatinine-based eGFR in early and moderate CKD stages, suggesting earlier detection of renal impairment. Similar observations were made by Rule et al.[20] and Voskoboev et al.[21], where cystatin C-based eGFR reclassified a substantial proportion of patients into a more advanced CKD stage, with potential implications for earlier intervention.

Importantly, cystatin C-based eGFR in our cohort demonstrated a stronger correlation with measured creatinine clearance (r = 0.836) compared to creatinine-based eGFR (r = 0.712), indicating greater accuracy. This aligns with the recommendations from the 2021 CKD-EPI equation update by Levey et al.[22] and the NKF–ASN Task Force guidance by Delgado et al.[23], both advocating the use of cystatin C alone or in combination with creatinine to improve precision and avoid race-based adjustments.

From a clinical perspective, incorporating cystatin C into CKD evaluation could enhance early detection, refine risk stratification, and guide management decisions, particularly in cases where creatinine is unreliable. Shardlow et al.[24] demonstrated that cystatin C independently predicts CKD progression, while NICE guidelines[25] recommend targeted use in patients with borderline creatinine-based eGFR. Nevertheless, as emphasized by Masson et al.[19] and Voskoboev et al.[21], widespread adoption must consider cost and assay availability, particularly in resource-limited healthcare settings.

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