Usefulness of biochemical markers in monitoring renal function in chronic kidney disease

ABSTRACT

Aims: Chronic kidney disease (CKD) is a common, progressive, and dynamic disease. It often regresses over time despite the fact that treatment has been shown to slow the progression of the disease. Biomarkers allow monitoring of disease activity in the early stages of the disease. Biochemical markers, such as serum creatinine, blood urea nitrogen, and estimated glomerular filtration rate, are routinely used to assess kidney function.

Study design: Samples were collected from different hospitals in Baghdad Governorate, Iraq (Iraq Dialysis centre - Al-Zohour Dialysis centre - Al-Yarmouk Hospital Dialysis Center - Shallal Al-Sadrain Dialysis centre), during the period from November 2023 to May 2024.

Methodology: The study included 75 patients with CKD, aged (35 to 75 years), and the ratio of males to females according to disease group (Group 1 15/10 - Group 2 18/12 - Group 3 12/8). And who were receiving treatment in the nephrology unit. The disease groups were divided according to the severity of the disease:

Stage 1: early onset of disease

Stage 2: moderate renal impairment

Stage 3: severe renal impairment and end-stage renal failure

Results: After the proposed treatment period (6 months of treatment), all groups showed improvement in key biochemical markers, with differential improvement in half of them based on the extent of chronic kidney disease. For example, group 2 (stage 3 chronic kidney disease) had blood urea nitrogen, serum creatinine, and estimated glomerular filtration rate of 35 ± 3 to 28 ± 2 mg/dL, serum creatinine of 2.8 ± 0.3 to 2.3 ± 0.2 mg/dL, and glomerular filtration rates ranging from 45 ± 8 to 55 ± 6 ml/min/1.73 m . The results showed that patients with early CKD responded better to treatment, mainly due to relative preservation of kidney function.

Conclusion: This study investigates the usefulness of these indicators in monitoring CKD activity and evaluating the feasibility of therapeutic interventions.

***Keywords**:Chronic kidney disease (CKD), biochemical markers, renal function, serum creatinine, blood urea nitrogen, eGFR*.

1. INTRODUCTION

Chronic kidney disease (CKD) involves a progressive loss of kidney function, resulting in metabolic waste [1],resulting in physical disabilities and increasing the risk of cardiovascular other issues and complications are likely to occur [2]. The disease usually progresses slowly, which is why it is important to recognize early signs of intervention in a timely manner [3]. The CKD approach is essential for the treatment and early prevention of end-stage renal disease ESRD [4]. , often with biochemical markers of the need for dialysis or alternative concepts, such as serum creatinine urea nitrogen BUN and glomerular filtration rate EGFR measurements are used in order to monitor kidney function [5]. These markers reflect the ability of the kidneys to filter and remove waste products from the blood and are important for the diagnosis and staging of CK[6]. The aim of this study was to evaluate the utility of these biochemical markers in monitoring CKD progression, especially in patients receiving treatment regimens [7], and factors such as age, comorbidities, and treatments are of increasing interest to determine whether these traditional markers can reliably predict disease outcome and guide long-term timing of treatment decisions [8]. The aim of this study was to evaluate the utility of these biochemical markers in monitoring CKD progression, especially in patients receiving treatment regimens [9]. We sought to evaluate the efficacy using these markers and by comparing treatments in different stages of CKD. We expect that these markers will provide valuable insights into renal function, leading to timely intervention and optimal therapeutic strategies to slow the potential progression of ESRD Furthermore, that understanding the limitations of these markers may also highlight the need for further development and potential improvement of new biomarkers [10].

2. MATERIAL AND METHODS

2.1. Study Population

The study involved 75 patients with CKD, ranging in age from (35 to 75 years), who were undergoing treatment in a nephrology unit. The patients were divided into three groups based on CKD stage:

- Group 1: CKD Stage 1–2 (mild renal impairment).
- Group 2: CKD Stage 3 (moderate renal impairment).

- Group 3: CKD Stage 4–5 (severe renal impairment and ESRD).

2.2. Sample Collection

Blood samples: were collected from each patient during routine clinical visits. Serum creatinine, BUN, and eGFR have been measured. The Modification of Diet in Renal Disease (MDRD) equation was used to calculate eGFR [11] [12].

Some important points need to be considered when collecting samples:

1- Fasting requirements: In some cases, patients may need to fast for a certain period of time (e.g. 8-12 hours) before sampling, especially when glucose or lipid levels are tested in addition to renal function [13].

2- Medication review: Patients should be asked about any medications they are currently taking, as certain medications (eg, diuretics and ACE inhibitors) may affect the results of kidney function tests [14]. Depending on which test or test is being performed, medications time may have to be considered or controlled [15].

2.3. Treatment Protocols

Patients received various treatment interventions, including:

- Antihypertensive therapy (ACE inhibitors, ARBs)
- Diuretics
- Phosphate binders
- Dietary interventions (protein restriction)
- Hemodialysis (for patients with ESRD)

The study looked at changes in biochemical markers in response to these treatments over a 6-month period.

2.4. Statistical Analysis

Descriptive statistics (mean, standard deviation) were used to summarize biochemical marker values. A paired t-test was conducted to assess the significance of changes in marker levels before and after treatment interventions [16].

3. RESULTS

3.1. Baseline Characteristics:

Table 1, summarizes the baseline characteristics of the study population. The patients were evenly distributed across CKD stages, with most patients in Group 2 (Stage 3).

Characteristic	Group 1 (Stage 1–2)	Group 2 (Stage 3)	Group 3 (Stage 4–5)
Number of Patients	25	30	20
Mean Age (years)	50 ± 5	60 ± 7	65 ± 8
Male/Female Ratio	15/10	18/12	12/8
Mean BUN (mg/dL)	18 ± 2	35 ± 3	65 ± 5
Mean Serum Creatinine (mg/dL)	1.2 ± 0.2	2.8 ± 0.3	5.6 ± 0.4
Mean eGFR (mL/min/1.73 m^2)	85 ± 10	45 ± 8	20 ± 5

Table 1: The baseline characteristics of the study population.

3.2. Changes in Biochemical Markers Post-Treatment

After the treatment period (6 months of treatment), patients showed crucial role improvement in their biochemical markers, especially those in group 2 (stage 3 CKD). Table 2 shows the biochemical marker values in each group before and after treatment.

Biochemical Marker	Group 1 (Stage 1–2)	Group 2 (Stage 3)	Group 3 (Stage 4–5)
BUN (mg/dL)	18 ± 2 → 16 ± 1	35 ± 3 → 28 ± 2	65 ± 5 → 60 ± 4
Serum Creatinine (mg/dL)	1.2 ± 0.2 → 1.1 ± 0.2	2.8 ± 0.3 → 2.3 ± 0.2	5.6 ± 0.4 → 5.2 ± 0.4
eGFR (mL/min/1.73 m^2)	85 ± 10 → 90 ± 9	45 ± 8 → 55 ± 6	20 ± 5 → 22 ± 4

Table 2: Post-treatment changes in biochemical markers (mean ± SD).

The results showed that the largest and best improvements in serum creatinine and urea levels occurred in group 2 (stage 3 CKD), indicating the effectiveness of early intervention. Patients in group 3 (stage 4-5) showed less crucial improvements, but renal function was stabilized in most cases.

3.3. Impact of Specific Treatment Protocols

Table 3 highlights the impact of different treatment protocols on serum creatinine levels across all CKD stages. Antihypertensive drugs (mainly ACE inhibitors and ARBs) were associated with significant improvements in serum creatinine levels, especially in group 2 patients.

Treatment Protocol	Group 1 (Stage 1–2)	Group 2 (Stage 3)	Group 3 (Stage 4–5)
Antihypertensive Therapy	1.2 → 1.1	2.8 → 2.2	5.6 → 5.1
Diuretics	1.2 → 1.1	2.8 → 2.3	5.6 → 5.3
Phosphate Binders	1.2 → 1.2	2.8 → 2.4	5.6 → 5.4
Hemodialysis (ESRD patients)	N/A	N/A	5.6 → 5.2

Table 3: Effect of treatment regimen on serum creatinine (mg/dL).

Discussion:

Baseline Characteristics

The baseline characteristics in Table (1) provide an overview of the study population, divided into three groups based on CKD stage. The distribution across CKD stages was fairly even, with the majority of patients falling into group 2 (CKD stage 3).

Age and gender: The mean age increase with the severity of CKD, which is consistent with the progressive nature of CKD being among the most common in older individuals [17]. The study also showed a slightly higher proportion of males than females in all groups, consistent with the overall patient population distribution [18].

Biochemical markers: As expected, baseline levels of blood urea nitrogen (BUN), serum creatinine, and estimated glomerular filtration rate (eGFR) deteriorated with progression of CKD group 1 (CKD stage 1-2), showed a slight increase in normal mental function, while group 3 (stage 4-5 CKD) showed significantly impaired renal function with significantly increased serum creatinine, serum creatinine, and estimated glomerular filtration rate.

Changes in biochemical markers after treatment

After the proposed treatment period (6 months of treatment), all groups showed improvement in key biochemical markers (blood nitrogen, serum creatinine, and estimates of glomerular filtration rate), with half having differential improvement it is based on CKD.

Group 2 (stage 3 CKD) blood urea nitrogen, serum creatinine, estimated glomerular filtration rate 35 ± 3 to 28 ± 2 mg/dl and serum creatinine 2.8 ± 0.3 to 2. 3 ± 0.2 mg/dL, calculated as glomerular filtration rates ranging from 45 ± 8 to 55 ± 6 ml/minute/1.73 m. The results showed that patients with early CKD respond better to treatment, mainly due to relative preservation of renal function.

Group 1 (stage 1-2 CKD) showed slight improvements, and these patients would be expected to have near-normal kidney function initially [19]. However, the slight decrease in serum urea and creatinine and the slight increase in estimated glomerular filtration rate suggest that treatment helps maintain kidney function even in the early stages. Group 3 (stage 4-5 CKD) showed less significant improvements. Although serum creatinine and blood urea levels decreased slightly, these patients are likely to have advanced, irreversible kidney damage, limiting the effect of treatment [20]. However, stable kidney function (small increase in estimated glomerular filtration rate) suggests that even with advanced CKD treatment, further damage can be delayed [21]. Small changes, such as blood urea a decrease from 65 ± 5 to 60 ± 4 mg/dL and a serum creatinine of decreased from 5.6± 0.4 mg/dL to 5.2 ± 0.4 mg/dL, illustrating the challenges of managing late stage CKD.

Effect of specific treatment protocols

Table 3 shows the effect of different treatment protocols on serum creatinine in all stages of CKD. Treatments inclusive (antihypertensive therapy, diuretics, phosphate stabilizers) and, in some cases for patients with end-stage renal failure, dialysis[22].

Antihypertensive therapy: was the most effective treatment in all groups, with the greatest improvement seen in group 2 (CKD stage 3). Serum creatinine decreased from 2.8 mg/dL to 2.2 mg/dL in this group, suggesting that ACE angiotensin receptor blockers and blockers are

particularly beneficial these drugs prevent hypertension by reducing proteinuria and lowering intraglomerular pressure and slows the progression of CKD It is known.

Diuretics: There has also been significant progress in diuretics, although somewhat less pronounced than antihypertensive. In group 2, serum creatinine decreased from 2.8 to 2.3 mg/dL. Diuretics help manage excess fluid and help reduce blood pressure which can have a positive effect on kidney function, especially in the middle of CKD.

Phosphate binders: These agents, which were used primarily to prevent hypophosphatemia in patients with CKD, had little effect on serum creatinine [23]. The small changes in serum creatinine in all groups (e.g., 2.8 → 2.4 mg/dL in the group) suggest that although phosphate binders are important for mineral and osteoporosis maintenance, their direct effects on renal filtration activity upstream is limited [24].

Dialysis: For patients in group 3 (stage 4–5), dialysis was used as a last resort. The results showed that it did not significantly improve serum creatinine levels (5.6 → 5.2 mg/dL), but it did manage waste removal in patients with severely impaired renal function. The slight decrease in serum creatinine may reflect some residual renal function or the effectiveness of dialysis in managing toxic build-up. This is expected, as dialysis is primarily a supportive treatment that replaces rather than restores renal function [25]. However, stabilizing these markers is vital in managing symptoms and preventing complications such as uraemia, electrolyte imbalance, and fluid overload in patients with end-stage CKD.

Limitations and considerations for future research

Although studies support the use of serum creatinine, blood urea, and estimates of glomerular filtration rate in the treatment of CKD, it is important to acknowledge some limitations that must be considered [26]. These markers, although valuable, is not always predictive of kidney damage, especially CKD]. Early CKD may have subclinical damage and may not be accompanied by significant changes in biochemical markers[27] , so future studies should examine emerging biomarkers such as cystatin C, urinary albumin, inflammatory markers, which may provide new insights for slower CKD progression and disease progression to help identify at-risk patients.

4. Conclusion:

Biochemical markers including serum creatinine, BUN, and eGFR play an important role in the monitoring of renal function in CKD patients. Early recognition and management of the aforementioned symptoms with timely intervention can slow disease progression and improve patient outcomes clearly, antihypertensive drugs significantly affect renal function, especially in patients with moderate CKD.

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