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ORIGINAL ARTICLE

Pattern of Kidney Injury Molecule-1 in Different Stages of Chronic Kidney Disease

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Abstract

In clinical practice, the evaluation of renal function is performed by measuring serum creatinine to calculate glomerular filtration rate (eGFR) and urinary albumin (uACR). Both indices mainly reflect glomerular damage in the kidney. But tubular injury is also important in the pathogenesis of renal disease and appears to precede glomerular injury. Kidney injury molecule-1 (KIM-1), a transmembrane glycoprotein, is considered as a potential biomarker of renal tubular injury. The aim of the study was to find out the correlation of urinary KIM-1 levels with the stages of chronic kidney disease (CKD) and its association with the progression of the disease. In this cross-sectional study, total 100 CKD patients from all stages and 28 healthy controls were included. Urinary KIM-1 was measured through enzyme-linked immunosorbent assay. Urinary KIM-1 was indexed to urine creatinine (KIM-1/Cr ratio) by dividing urinary concentration of KIM-1 by urinary creatinine concentration to account for the concentration of urine. Among the case group 13% (n=13) had stage 1and stage 2, 15% (n=15) had stage 3a, 22% (n=22) had stage 3b, 22% (n=22) had stage 4 and 28% had stage 5 CKD. The mean age of the CKD patients were 53 years. Among them 64% were male. The mean urinary KIM-1/Cr level was significantly higher in case group than in control group (940.96±192.98 vs. 497.20± 39.60 pg/mg; P<0.001). Urinary KIM-1/Cr was negatively associated with eGFR (r= -0.53; p=0.001) and positively with uACR (r= 0.31; p=0.002). Urinary KIM/Cr concentrations were significantly varies among the different stages of CKD (P< 0.001) with trends of higher values with the advancement of stages of CKD. Urinary KIM-1 levels correlated with traditional markers of renal damage and had the potential to predict the progression of CKD which could be explored further in longitudinal studies.

Keywords: Chronic Kidney Disease (CKD), Kidney Injury Molecule-1 (KIM-1), Markers, Renal Damage

Introduction

Chronic kidney disease (CKD) is a progressive condition with significant morbidly and mortality[1]. It affects more than 850 million patients worldwide. It is predicted that by 2040, CKD will be the fifth leading cause of death[2]. Clinical management of CKD is mainly supportive and targeted to prevent complications[3]. Considering the huge burden of the disease and its association with cardiovascular events,

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early diagnosis of the condition and identification of the patients who are at increased risk of progression to develop end stage kidney disease is essential[4].

Since 2012, kidney disease improving global outcome (KDIGO) guidelines recommends glomerular filtration rate (GFR) and albuminuria for assessment and risk stratification of patients with CKD[5]. But both markers have several limitations which pose a major challenge in clinical practice to predict the adverse outcomes associated with the disease. In early stages of CKD, nephrons can develop compensatory hypertrophy and hyperfiltration which may reflect a normal GFR[6]. The decline in GFR is evident only when substantial kidney damage has been occurred and resulting inflammation and fibrosis leading to nephron loss[7,8]. On the other hand, the trajectory of GFR decline is nonlinear with the progression of the disease as there is higher rate of kidney function deterioration in advanced stages of CKD which precludes accurate prediction of the timing of renal replacement therapy and adequate patient preparation[9]. Furthermore, serum creatinine is used for estimation of GFR in most clinical settings as it is inexpensive and widely available. But the levels of serum creatinine may vary with age, sex, muscle mass, dietary habits and medications. Due to these compounding factors, the recognition of disease progression from GFR estimation is more challenging[10]. Urinary albumin-creatinine ratio (ACR) is a late marker of CKD which may not appear until the kidney has endured prolonged inflammation but may absent in non-proteinuric causes of the disease[7,8]. Intra-person variability of albuminuria is also high. Even a 50% decline or increment in albuminuria in an individual may not reflect disease remission or progression[11].

In recent years, several biomarkers reflecting disturbances in different biologic pathways implicated in CKD development have been emerged[12]. Traditional markers e.g. GFR and albuminuria mainly reflect glomerular function and injury whereas tubular cell atrophy and interstitial fibrosis are important pathologic factors for CKD progression[13]. The newly emerged biomarkers expand the evaluation kidney dysfunction beyond the glomerular axis exploring tubulointerstitial damage and inflammation[12]. These urinary markers of renal tubular cell injury can predict the risk of subsequent loss of kidney function, beyond the eGFR and albuminuria[13].

Kidney injury molecule-1 (KIM-1) is a transmembrane glycoprotein expressed in proximal tubular cells and immune cells of the kidney and released upon tissue injury or ischemia[14]. KIM-1 is now increasingly considered as a potential biomarker and is correlated well with proteinuria, foot process effacement and tubular injury.[15] KIM-1 expression is undetectable in healthy kidney tissue but elevated expression is demonstrated in the setting of chronic injury at both glomerular and tubular level and associated with glomerulosclerosis, tubular atrophy and interstitial fibrosis[15,16].

Although plasma and urine levels of KIM-1 are elevated in CKD but the role of urinary KIM-1 levels in predicting decline of GFR remains inconclusive as most studies were done using plasma KIM-1 levels. In this study we sought to determine the association of urinary KIM-1 levels with the progression of CKD.

Methods

This cross-sectional study was conducted in the department of Nephrology at National Institute of Kidney Disease and Urology (NIKDU), Dhaka, Bangladesh from March 2020 to February 2021. Adult CKD patients of all stages (stage: 1-5) and 28 healthy controls were included in this study. CKD patients with any unstable conditions (e.g. acute kidney insult due any reason, acute myocardial infarction, and malignancy), patients on immunosuppressive medications, and history of requiring dialysis were excluded. The study protocol was approved by ethical committee of NIKDU (memo No.: NIKDU/ERC/2020/59; date: 08/12/2020). All participants provided written informed consent.

Study procedure

A pretested data collection sheet consists of demographics, clinical presentations, physical examination findings and investigations was used. At initial visit, after taking relevant history participants were selected according to the inclusion and exclusion criteria. Anthropometric parameters and physical findings were recorded. Blood pressure was measured using a digital blood pressure machine (Omron JPN-2). After explaining procedures and objectives of the study, participants were asked to come with at least 12 hours fast on a particular date. At the final visit blood sample and a spot second morning urine sample were collected. The serum and urine samples were stored in ultra–deep freezer at “80 C until analysis.

Laboratory measurements

Serum measurements include creatinine, calcium, phosphate, uric acid, C- reactive protein (CRP), total cholesterol; HDL, LDL, triglyceride, albumin, hemoglobin and urinary measurements include creatinine, albumin, KIM-1 were measured in local laboratories using standard clinical laboratory methods. In order to calculate eGFR, Modification of Diet in Renal Disease (MDRD) equation was used. CKD was defined according to KDIGO criteria.

Statistical analysis

Statistical analysis of the data was done by using statistical software SPSS (IBM version 20). Categorical variables were expressed as frequencies with percentages and continuous variables were expressed as mean ± standard deviation (SD). To compare the continuous variables, Independent t test was done. Chi-square test was done to compare between categorical variables. Pearson correlation and Spearman´s correlation tests were done to observe correlation between different variables as appropriate. The urinary biomarker was indexed to urine creatinine by dividing urinary KIM-1 level by urine creatinine level (urinary KIM-1-creatinine ratio) to account for the concentration of urine. For analysis, patients were stratified based on their eGFR. Comparison of urinary KIM-1-creatinine ratios between different stages of CKD was done by ANOVA test. A p value d"0.05was considered as statistically significant.

Results

Initially, 110 previously diagnosed CKD patients (stage 1–5) were enrolled. Due to unwillingness to give sample, 10 patients were excluded from the study. Finally, 100 patients and 28 healthy adults were included in the study. Among the case group 13% (n=13) had stage 1-2, 15% (n=15) had stage 3a, 22% (n=22) had stage 3b, 22% (n=22) had stage 4 and 28% had stage 5 CKD (Figure- 1).

Baseline characteristics of the participants were shown in table-1. CKD patients and healthy individuals were BMI matched. The mean age of the CKD patients were 52.76±11.64 years and 64% of the CKD patients were male. Urinary kidney injury molecule-1 (uKIM-1) level was significantly higher among CKD patients than control group (748.76 ±288.78 vs. 431.858 ±27.09 pg/ml; p<0.001). Also, urinary KIM-1/Cr ratio was significantly higher in CKD patients than healthy controls (940.96 ±192.28 vs. 497.20 ±39.60 pg/mg; p<0.001). Urine ACR, serum uric acid, C-reactive proteins (CRP) were significantly higher in CKD patients compared to healthy controls. Serum calcium, serum albumin and HDL were significantly lower among the CKD patients' group in comparison to the healthy control group (Table 1).

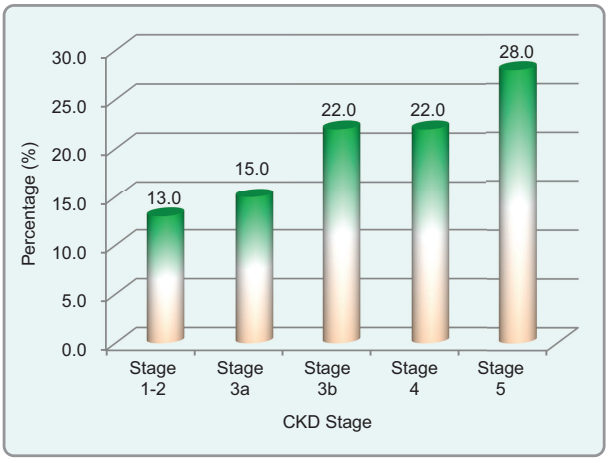

Figure-1: *Distribution of CKD patients according to different CKD stages (n= 100)*

Table-I

Baseline characteristics of the study population (N= 128)

Characteristics	Case group (n=100)	Control group (n=28)	p-value
Male (%)	64 (64%)	21 (75%)	0.276*
Age (years)	52.76±11.64	38.89±5.91	0.001**
BMI (kg/m^2)	24.12±3.89	24.69±1.02	0.445**
SBP (mmHg)	137.6±26.4	120.36±3.86	0.001**
DBP (mmHg)	83.58±11.3	78.68±3.15	0.025**
KIM-1 (pg/ml)	748.76 ±288.78	431.858 ±27.09	<0.001**
KIM-1/cr (pg/mg)	940.96±192.98	497.20 ±39.60	<0.001**
Spot urine ACR (mg/ml)	413.29±678.12	29.90±13.60	0.003**
CRP (mg/l)	6.47±4.74	2.22±1.24	0.001**
Serum calcium (mg/dl)	7.98±1.88	8.87±0.45	0.014**
Serum phosphate (mg/dl)	3.81±2.17	3.69±0.67	0.773**
Serum uric acid (mg/dl)	7.42±2.86	4.82±1.04	0.001**
Serum albumin (g/dl)	3.86±0.89	4.76±0.34	0.001**
Serum triglyceride (mg/dl)	171.35±102.24	141.89±34.44	0.136**
Serum cholesterol (mg/dl)	161.24±56.93	160.73±21.26	0.963**
Serum HDL (mg/dl)	34.63±11.58	45.15±10.85	0.001**

*Chi-square test and ** Independent t test were done

In most cases; the cause of CKD remained undetermined (38%) followed by diabetes (33%) and hypertension (24%) (Figure2).

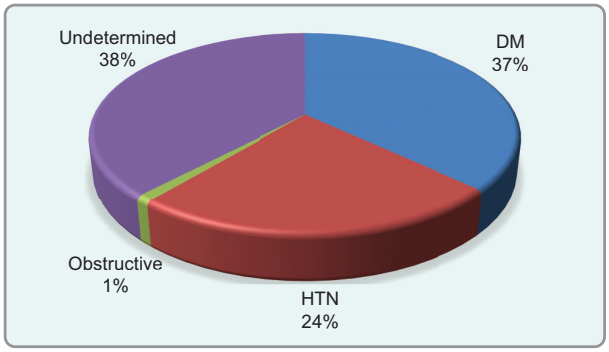

Figure 2: *Causes of CKD among cases (n= 100)*

A significant negative correlation between urinary KIM-1-creatinine ratio and eGFR (rŒ -0.532; p< 0.001) was observed (figure 3).

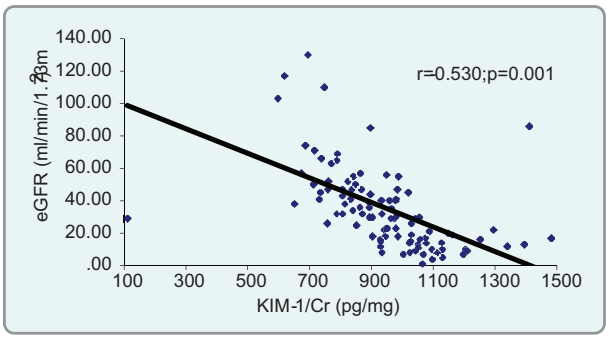

Figure 3: *Scatter diagram showing negative association of urinary KIM-1/Cr and eGFR*

It was observed that, urine KIM-1-creatinine ratios were significantly varies among the different stages of CKD. There was a tendency of increasing levels of KIM-1-creatinine ratio with advancement of CKD stages (Table 2).

Table-II

Levels of urinary KIM-1/Cr ratios in different stages of CKD

CKD stages(n=100)	Urinary KIM-1/ Cr ratio (pg/mg) Mean±SD	p-value
Stage 1+2 (n=13)	783.87±203.29	<0.001
Stage 3a (n=15)	813.03±86.30	
Stage 3b (n=22)	888.72±86.37	
Stage 4 (n=22)	951.99±215.67	
Stage 5 (n=28)	1114.83±132.99	

p value reached from ANOVA test.

The correlation analysis showed that urinary KIM-1 levels significantly correlated with albuminuria. A significant positive association between urinary KIM-1-creatinine and urine ACR (r=0.309, p=0.002) was observed (Figure 4).

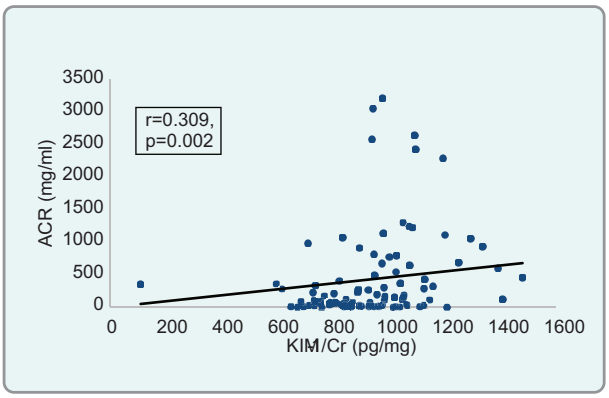

Figure 4: *Scatter diagram showing positive association of urinary KIM-1/Cr and ACR*

Discussion

KIM-1 presents at very low concentration in healthy kidneys[17]. It is expressed in proliferating epithelial cells at a regenerating state of proximal tubules after injury and is considering as a biomarker for detection of kidney tubular damage[15]. This study compared urinary KIM-1 concentrations (uKIM-1) in CKD patients of all stages and healthy adults and evaluate an association of urinary concentrations of tubular injury marker KIM-1 with glomerular filtration rate and urinary albumin excretion.

In this study, lower level of uKIM-1 was found in early stages (CKD stage 1 &2) and a higher level was observed in advanced stage. Higher uKIM-1 levels were associated with the risk of rapid decline of renal function and could be a predictor of more advanced stages as documented in previous studies[18-20]. GR Lobato et al. compared CKD patients not on dialysis who progressed to worse stage with those who did not progress and found a trend of higher uKIM-1 levels among the progressors[21]. M M Van Timmmeren et al. examined KIM-1 expression in renal biopsies and its correlation with urinary KIM-1 levels which included biopsies from renal disease patients and controls. uKIM-1 was increased in renal patients versus controls and correlated negatively with renal function and positively tissue KIM-1 expression. Tissue KIM-1 was up regulated in renal disease and associated with dedifferentiation of proximal tubule cells, inflammation and fibrosis[22]. So, the findings of the present study were in line with those studies mentioned earlier suggested that uKIM-1 could predict CKD progression.

In the current study, a positive correlation between urinary KIM-1 and albuminuria was observed. The increased levels of urinary KIM-1 had been observed to correlate with proteinuria in diabetic patients[23]. Aslan O et al. found a positive correlation between urinay KIM-1 and urine albumin-creatinine ratio ($r = 0.400$, $P < 0.001$) in type 2 diabetes patients[24]. Siddiqui K et al. observed that kidney injury molecule-1-to-creatinine ratio was varied according to different albuminuria statuses in diabetic patients.[25] Significant and positive correlations were observed between uACR and uKIM-1 by de Carvalho JA et al.[26] Urinary KIM-1 levels were found to be increased in proteinuric non diabetic CKD patients also. Reduction of proteinuria with anti-proteinuric treatment resulted in a parallel decreased in urinary KIM-1 level[27]. A positive correlation between urinary KIM-1-creatinine ratio and proteinuria was noted among patients with nephrotic syndrome and active lupus nephritis[28,29]. These studies indicate that urinary KIM-1 concentration is increased with the degree of proteinuria, a key finding that was observed in the present study.

Our study has several limitations. First, this was a cross-sectional. Although urinary KIM-1 levels were significantly differed among the stages of CKD but to demonstrate robust correlations between the changes of urinary biomarker with disease progression, a longitudinal follow up study design would be more appropriate. Second, small size of the control group (n=28). Third, the prolonged sample storage time. However, the stability of urinary KIM-1 was not significantly affected when samples stored in ‹ 80! as demonstrated in previous study[30].

Conclusion

This study detected a trend towards increased levels of urinary KIM-1 levels from early to advanced stages of CKD. The association of urine KIM-1 levels with traditional markers of renal damage e.g. eGFR and uACR was also evident. The potential clinical implications of urinary KIM-1 level should be explored further in a longitudinal follow up study with larger population.

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