Proteinuria and Clinical Outcomes in Hospitalized COVID-19 Patients
A Retrospective Single-Center Study

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Abstract
Background and objectives Kidney involvement is frequent among patients with coronavirus disease 2019 (COVID-19), and occurrence of AKI is associated with higher mortality in this population. The objective of this study was to describe occurrence and significance of proteinuria in this setting.

Design, setting, participants & measurements We conducted a single-center retrospective study to describe the characteristic features of proteinuria measured within 48 hours following admission among patients with COVID-19 admitted in a tertiary care hospital in France, and to evaluate its association with initiation of dialysis, intensive care unit admission, and death.

Results Among 200 patients with available data, urine protein-creatinine ratio at admission was ≥1 g/g for 84 (42%), although kidney function was normal in most patients, with a median serum creatinine of 0.94 mg/dl (interquartile range, 0.75–1.21). Median urine albumin-creatinine ratio was 110 mg/g (interquartile range, 50–410), with a urine albumin-protein ratio <50% in 92% of patients. Urine retinol binding protein concentrations, available for 85 patients, were ≥0.03 mg/mmol in 62% of patients. Urine protein-creatinine ratio ≥1 g/g was associated with initiation of dialysis (odds ratio, 4.87; 95% confidence interval, 1.90 to 6.54; \( P < 0.001 \)), admission to the intensive care unit (odds ratio, 3.55; 95% confidence interval, 1.93 to 6.71; \( P < 0.001 \)), and death (odds ratio, 3.56; 95% confidence interval, 1.90 to 6.54; \( P < 0.001 \)).

Conclusions Proteinuria is very frequent among patients admitted for COVID-19 and may precede AKI. Low levels of albuminuria suggest a predominant tubular origin, confirmed by the elevated levels of urine retinol binding protein. Urine protein-creatinine ratio ≥1 g/g at admission is strongly associated with poor kidney and patient outcome.

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Introduction
Infection with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) virus is responsible for coronavirus disease 2019 (COVID-19). Despite predominant respiratory manifestations, COVID-19 has a broad clinical spectrum encompassing asymptomatic infection, mild upper airways illness with fever, and severe pneumonia with respiratory failure, as well as thromboembolic complications, coagulopathy, acute cardiac injury, and neurologic symptoms (1,2). Patients with critical presentations require admission to intensive care units (ICUs) either for acute respiratory distress syndrome or for cytokine storm syndrome leading to multiple organ dysfunction (3). Mortality is particularly important among older patients as well as populations with comorbidities such as obesity, diabetes, hypertension, chronic respiratory disease, or previous immunosuppression (4).

Several teams have reported kidney involvement in SARS-CoV-2–infected patients. Although initial reports suggested that acute kidney failure was rare (<5%) in the entire infected population (1,5), the incidence of this complication is much higher, up to 65%, among patients with severe disease who were admitted to the ICU (6) or those with a fatal outcome (2,7–9). More recently, a retrospective study of 3993 patients with COVID-19 hospitalized in New York revealed that AKI occurred in 46% of patients and that 19% of patients with AKI required dialysis (10). An early report from a large (n=701) cohort in China revealed that 43.9% of patients had proteinuria and 26.7% had hematuria on admission (11). The same authors reported that among 333 patients hospitalized in Wuhan with COVID-19 pneumonia, 75.4% had transient abnormal urine dipstick tests (12). Among the 435 patients with AKI and available urine tests reported by Chan et al. (10), 84% had proteinuria, 81% had hematuria, and 60% had leucocyturia.

In the first published autopsy series (13), postmortem examination of kidney pathology was reported in...
patients during an outpatient clinic visit in the 2-month period following hospital discharge.

**Laboratory Measures**

Urine biochemical parameters were obtained prospectively on spot urine collection within 48 hours after admission. Proteinuria was expressed as urine protein-creatinine ratio (UPCR), and albuminuria was expressed as urine albumin-creatinine ratio (UACR). Tubular dysfunction was evaluated in a subset of patients by assessment of urine retinol binding protein concentration, expressed as urine retinol binding protein-creatinine ratio.

The total urinary protein concentration was quantified by measuring pyrogallol red at 600-/800-nm absorbance (urinary protein assay; Beckman Coulter), and the albumin concentration was measured by immunoturbidimetry analysis (DIAGAM assay; Beckman Coulter) using a Beckman Coulter AU680 analyzer. The urinary levels of retinol binding protein were measured using a Siemens BN II Nephelometer Analyzer II. Urine protein concentrations were corrected for creatinine levels, which were measured using a colorimetric assay (modified kinetic Jaffe method) on a Beckman Coulter DXC analyzer (serum) or a Beckman Coulter AU680 analyzer (urine).

**Definitions**

AKI was defined by the Kidney Disease Improving Global Outcomes (KDIGO) AKI criteria (17). The KDIGO criteria for AKI were applied using only serum creatinine variations, as urine output data were not available for patients developing AKI before entering ICU. Baseline serum creatinine value was defined by the most recent value found in the centralized electronic medical records of the Assistance Publique-Hôpitaux de Paris network preceding the current admission. Whenever data were not available, we used as baseline value the initial serum creatinine level measured at the time of admission. eGFR was calculated using the Chronic Kidney Disease Epidemiology Collaboration formula (18). CKD was defined as a previously known eGFR below 60 ml/min per 1.73 m² preceding actual hospitalization or a former history of kidney transplantation.

Indication for ICU admission was on the basis of the presence of acute respiratory distress syndrome requiring high-flow nasal oxygen therapy, noninvasive mechanical ventilation, or invasive mechanical ventilation. Hospitalization in non-ICU departments was decided when patients required only nasal low-flow oxygen administration and standard medical monitoring.

Comorbidities, including diabetes mellitus, hypertension, or CKD, were retrospectively collected in individual medical files of the included patients.

**Statistical Analyses**

Categorical variables were summarized as percentages, and continuous variables were expressed as the mean ± SD or median with interquartile range (IQR). Characteristics of subgroups were compared with the use of a t test or a Wilcoxon rank-sum test for continuous measures and a chi-squared test or a Fisher exact test for categorical variables. The effect of initial proteinuria on the occurrence of death.
was examined by Kaplan–Meier analysis with the use of log-rank test. Multivariable logistic regression was performed to evaluate the relationship between UPCR, retinol binding protein-creatinine ratio, and outcomes adjusted for age, sex, body mass index (BMI), hypertension, diabetes, and serum creatinine at admission. Adjusted analysis excluded patients with missing data for one or more covariates. Results are reported as odds ratios (ORs) with 95% confidence intervals (95% CIs). Statistical significance was set at two-sided \( P < 0.05 \). Analyses were performed with either version 8.0.2 of JMP software (SAS Institute) or version 1.2.5033 of the R Studio software (R Project for Statistical Computing; R Foundation).

**Results**

**Study Population**

Among the 322 patients admitted by April 15, 2020, 122 patients were excluded from analysis due to lack of data on proteinuria or to presence of preexisting kidney failure requiring long-term dialysis therapy. Figure 1 shows the flow diagram of the study. The characteristics of patients without available proteinuria are detailed in Supplemental Table 1, revealing a population with fewer comorbidities and lower risk of admission to ICU or death.

Overall, 200 patients—143 men and 57 women—were included in this study (Table 1). Median age was 63 years (IQR, 54–73), with 32 of 200 (16%) patients aged above 80 years. BMI was available for 171 patients, with a median value of 26.7 kg/m\(^2\) (IQR, 24.3–30.1). Of note, 66 (39%) patients had a BMI between 25 and 30 kg/m\(^2\), and 44 (26%) patients had a value above 30 kg/m\(^2\). Former history of hypertension was reported in 103 patients (51%), and diabetes mellitus was reported in 51 (25%) patients. Previously known CKD was reported for 18 patients, of whom seven were kidney transplant recipients. Previous use of antihypertensive therapy with angiotensin-converting enzyme inhibitor (ACEI) or angiotensin II receptor blocker (ARB) was reported for 60 (30%) patients, whereas 42 (21%) patients were receiving diuretics. The median delay between self-reported first signs of viral infection and admission was 7 days (IQR, 5–9).

**Proteinuria**

Median UPCR at admission was 0.86 g/g (IQR, 0.38–1.50). Proteinuria was <0.2 g/g in 24 (12%) patients, <0.5 g/g in 72 (36%) patients, between 0.5 and 1 g/g in 44 (22%) patients, between 1 and 3 g/g in 68 (34%) patients, and above 3 g/g in only 16 (8%) patients. Despite the fact that urine specimens were collected within 48 hours of admission, 45 patients had these urine tests performed after the AKI diagnosis was made on the basis of serum creatinine elevation.

Baseline UACR was available for only 152 patients (76% of the entire study population). Median UACR was 0.11 g/g (IQR, 0.05–0.41). The median UACR-UPCR ratio was 18% (IQR, 9.6–33.4). UACR-UPCR was above 50% for only 8% of patients.

Baseline urine retinol binding protein was measured in 85 patients. All but two of them had UPCR and UACR measured on the same urine specimen. The urine retinol binding protein-creatinine ratio was below the detection threshold (0.03 mg/mmol) in 32 (37%) patients. For the 53 (63%) patients with detectable urine retinol binding protein, the median value of urine retinol binding protein-creatinine ratio was 1.35 (IQR, 0.63–4.81) mg/mmol. Among the 85 patients with available urine retinol binding protein assessment (Table 1), those with urine retinol binding protein-creatinine ratio above the detection

![Figure 1. Flow diagram. Depicts included and excluded patients, as well as numbers of patients available for each analysis. COVID-19, coronavirus disease 2019; RBP, retinol binding protein; UACR, urine albumin-creatinine ratio; UPCR, urine protein-creatinine ratio.](image-url)
Association of Proteinuria with Outcomes

Table 1. Baseline characteristics for patients admitted for coronavirus disease 2019 according to proteinuria level at admission

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>All Patients, n=200</th>
<th>Urine Protein-Creatinine Ratio, g/g</th>
<th>Urine Retinol Binding-Protein-Creatinine Ratio, mg/mmol</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;1, n=116 ≥1, n=84</td>
<td>&lt;0.03, n=32 ≥0.03, n=53</td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>143 (71)</td>
<td>78 (67)</td>
<td>65 (77)</td>
</tr>
<tr>
<td>Age, yr</td>
<td>63 (54-73)</td>
<td>60 (53-69)</td>
<td>67 (55-79)</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>26.7 (24.3-30.1)</td>
<td>26.3 (23.9-30.3)</td>
<td>27.4 (24.6-30.1)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>103 (51)</td>
<td>53 (46)</td>
<td>50 (59)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>51 (25)</td>
<td>25 (21)</td>
<td>26 (31)</td>
</tr>
<tr>
<td>CKD</td>
<td>18 (9)</td>
<td>10 (9)</td>
<td>8 (9)</td>
</tr>
<tr>
<td>Use of ACEI or ARB</td>
<td>60 (30)</td>
<td>25 (21)</td>
<td>35 (41)</td>
</tr>
<tr>
<td>Use of diuretics</td>
<td>42 (21)</td>
<td>19 (16)</td>
<td>23 (27)</td>
</tr>
<tr>
<td>First symptoms to admission, d</td>
<td>7 (5-9)</td>
<td>7 (6-9.7)</td>
<td>7 (4-8)</td>
</tr>
<tr>
<td>UPCR, g/g</td>
<td>0.86 (0.38–1.50)</td>
<td>0.41 (0.25–0.72)</td>
<td>1.6 (1.3–2.1)</td>
</tr>
<tr>
<td>UACR, mg/g, %a</td>
<td>110 (50–410)</td>
<td>60 (20–100)</td>
<td>480 (260–830)</td>
</tr>
<tr>
<td>UACR-UPCR, %</td>
<td>18 (9.6–33.4)</td>
<td>13 (6.7–20.3)</td>
<td>32 (20.9–43.3)</td>
</tr>
<tr>
<td>URBPCR, mg/mmol</td>
<td>0.31 (0–2.76)</td>
<td>0 (0–0.14)</td>
<td>2.8 (1–5.8)</td>
</tr>
<tr>
<td>Serum creatinine, mg/dl</td>
<td>0.94 (0.73–1.21)</td>
<td>0.85 (0.69–1.12)</td>
<td>1.03 (0.85–1.36)</td>
</tr>
</tbody>
</table>
| Quantitative values are given as medians (interquartile ranges), and qualitative values are given as number of patients/percentage. BMI, body mass index; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin II receptor blocker; UPCR, urine protein-creatinine ratio; UACR, urine albumin-creatinine ratio; URBPCR, urine retinol binding protein-creatinine ratio.
| aData available for 171 patients. | bData available for 152 patients. | cData available for 85 patients. |

Outcomes

Median serum creatinine level at admission was 0.94 mg/dl (IQR, 0.75–1.21). Only 29 (14%) patients had initial serum creatinine >1.5 mg/dl at admission. Median peak serum creatinine was 1.19 mg/dl (IQR, 0.84–2.16), with 79 (39%) patients having at least one value above 1.5 mg/dl during their hospitalization.

AKI was diagnosed in 88 (44%) patients, with 28 (14%), 24 (12%), and 36 (18%) patients presenting stage 1, 2, or 3 AKI, respectively. KRT was required for 27 (13%) patients.

During hospitalization, 118 (59%) patients were admitted to the ICU for severe COVID-19 pneumonia. At last follow-up, 58 (29%) had died after a median delay of 10 days (IQR, 6–20) from admission; 142 (71%) were discharged, with a median length of stay of 11 days (IQR, 7–26) from admission.

Long-term follow-up data after hospitalization for COVID-19 were available for 135 of 142 surviving patients. After a median follow-up period of 36 (IQR, 27–54) days, the median serum creatinine was 0.82 (IQR, 0.67–1) mg/dl. Among the ten surviving patients with last known serum creatinine >1.5 mg/dl, eight had preexisting CKD.
Kaplan–Meier unadjusted survival curves according to baseline proteinuria (<1 or ≥1 g/g) are shown in Figure 2, with a significant difference between groups as concerning occurrence of death (P=0.006).

A multivariable analysis was performed, including UPCR≥1 g/g, age, sex, BMI, hypertension, diabetes, and serum creatinine at admission (Supplemental Table 2). The criterion UPCR≥1 g/g remained associated with need for KRT (OR, 4.84; 95% CI, 1.72 to 8.20; P=0.001), admission to the ICU (OR, 10.25; 95% CI, 3.82 to 30.23; P=0.001), occurrence of death (OR, 3.03; 95% CI, 1.46 to 6.39; P=0.003), and the composite outcome of death or need for KRT (OR, 4.24; 95% CI, 2.08 to 8.89; P=0.001).

The presence of a urine retinol binding protein-creatinine ratio ≥0.03 mg/mmol at admission was associated with a higher incidence of AKI (OR, 5.49; 95% CI, 1.95 to 15.98; P=0.002), admission to the ICU (OR, 10.25; 95% CI, 3.82 to 30.23; P<0.001), and death (OR, 22; 95% CI, 4.21 to 406; P<0.001) in unadjusted analysis (Table 2). The multivariable analysis performed using the urine retinol binding protein revealed that a urine retinol binding protein-creatinine ratio >0.03 mg/mmol was associated with occurrence of AKI (OR, 5.18; 95% CI, 1.18 to 29.9; P=0.04), admission to the ICU (OR, 15.5; 95% CI, 4.35 to 69.7; P<0.001), and death (OR, 20.9; 95% CI, 3.79 to 394; P=0.005) (Supplemental Table 3).

**Table 2. Study outcomes (unadjusted analysis)**

<table>
<thead>
<tr>
<th>Outcomes</th>
<th>Urine Protein-Creatinine Ratio</th>
<th>Urine Retinol Binding Protein-Creatinine Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;1 g/g, n=116</td>
<td>≥1 g/g, n=84</td>
</tr>
<tr>
<td></td>
<td>Odds Ratio (95% Confidence Interval)</td>
<td>Odds Ratio (95% Confidence Interval)</td>
</tr>
<tr>
<td></td>
<td>P Value</td>
<td>P Value</td>
</tr>
<tr>
<td>Peak serum creatinine, mg/dl</td>
<td>1.06 (0.77–1.54)</td>
<td>0.92 (0.77–1.19)</td>
</tr>
<tr>
<td>AKI, all stages</td>
<td>1.79 (1.13–3.79)</td>
<td>1.44 (0.99–1.44)</td>
</tr>
<tr>
<td>Stage 1</td>
<td>36 (31)</td>
<td>6 (19)</td>
</tr>
<tr>
<td>Stage 2</td>
<td>15 (13)</td>
<td>4 (12)</td>
</tr>
<tr>
<td>Stage 3</td>
<td>10 (9)</td>
<td>1 (3)</td>
</tr>
<tr>
<td>KRT</td>
<td>11 (9)</td>
<td>1 (0)</td>
</tr>
<tr>
<td>ICU required</td>
<td>7 (6)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Death</td>
<td>55 (47)</td>
<td>5 (3)</td>
</tr>
<tr>
<td></td>
<td>64 (76)</td>
<td>8 (25)</td>
</tr>
<tr>
<td></td>
<td>20 (24)</td>
<td>1 (3)</td>
</tr>
<tr>
<td></td>
<td>3.55 (2.03 to 13.00)</td>
<td>8 (25)</td>
</tr>
<tr>
<td></td>
<td>(1.93 to 6.71)</td>
<td>41 (77)</td>
</tr>
<tr>
<td></td>
<td>(1.90 to 6.54)</td>
<td>10.25 (3.82 to 30.23)</td>
</tr>
</tbody>
</table>

Quantitative values are given as medians (interquartile ranges), and qualitative values are given as number of patients/percentage. Odds ratios and P values are given for unadjusted analysis. ICU, intensive care unit.

**Discussion**

This study confirms that proteinuria is very frequent among patients with symptomatic SARS-CoV-2 infection admitted to the hospital, with a UPCR above 1 g/g at admission for 43% of them. Moreover, our results indicate that this proteinuria contains very small amounts of albuminuria, as the urine albumin-protein ratio is >50% for only 8% of patients, even among patients with UPCR≥1 g/g. In line with this, the urine levels of retinol binding protein are elevated in >50% of tested patients, an observation that strengthens the hypothesis that COVID-19–associated proteinuria reflects low-molecular weight proteins, which cannot be reabsorbed by the proximal kidney tubule in this condition due to acute tubular damage. Critically, in accordance with previous reports (19,20), we demonstrate that low-molecular weight proteinuria reflects early tubular dysfunction sometimes preceding AKI as suggested by the frequent normal-range serum creatinine level at admission.

The kidney histopathologic data published to date have indeed described diffuse injury of proximal tubular epithelial cells with loss of brush border and vacuolar degeneration followed by ATN (13,21). Causes of ATN are multiple in the setting of severe infection (22), as systemic hypoxia, local hypoxia due to microvascular damage, drug toxicity, or sepsis-induced rhabdomyolysis can all promote tissue injury. Nevertheless, electron microscopic examination of kidney biopsy from patients with COVID-19 has revealed virus-like particles within the tubular epithelium, suggesting direct evidence of tubular cells invasion by SARS-CoV-2 (14), and viral RNA has been found in kidney tissue from patients with COVID-19–associated AKI (16). Interestingly, it has been shown that proximal tubule...
kidney cells express the surface angiotensin-converting enzyme 2 (ACE2), which is instrumental for the coronavirus to bind and penetrate target cells (13,23). More recently, proximal tubule dysfunction has been demonstrated in patients with COVID-19 attested by the presence of low-molecular weight proteinuria (70%–80%), neutral aminoaciduria (46%), and defective handling of both uric acid (46%) and phosphate (19%), corresponding to a partial Fanconi syndrome (24).

Of note, viral-like particles have also been observed inside podocytes and endothelial cells, a finding that may promote glomerular damage. Although two recent cases of collapsing glomerulopathy associated with COVID-19 have been published (25,26), glomerulopathy due to SARS-CoV-2 infection seems to be rare in Asian and European populations, an idea supported by the findings of this study. Nevertheless, this complication may be especially prevalent among patients of African ancestry, in particular owing to a specific genetic background and the presence of APOL1 genetic variants, which predispose to the occurrence of collapsing glomerulopathy (25). This severe form of FSGS could be secondary to a direct effect of the virus on podocytes or could be the consequence of the SARS-CoV-2-induced cytokine storm, a condition that has been associated with glomerular lesions (27).

Our data confirm that overt proteinuria is more frequent among older patients with history of hypertension. The use of renin-angiotensin-aldosterone system inhibitors, such as ACEi and ARB, was more frequent among patients with high-grade proteinuria. Early reports had suggested that these drugs could modify cellular mRNA expression and protein level of ACE2, the SARS-CoV-2 receptor (28). Nevertheless, their use has not been associated with greater severity of disease, and recent studies have not confirmed ACE2 overexpression in the kidney after renin-angiotensin-aldosterone system inhibitor exposure (29).

In addition, we demonstrate that the presence of low-molecular weight proteinuria may precede elevation of serum creatinine and predict both AKI and KRT requirement, as it has been shown in non–COVID-19–associated ATN and AKI associated with sepsis (20,30). Although we have no control non–COVID-19 group, we show that UPCR≥1 g/g and retinol binding protein-creatinine ratio >0.03 mg/mmol are associated with a higher risk of severe COVID-19 that requires admission in the ICU and leads to higher mortality, even among patients with no significant elevation of serum creatinine at admission.

The prognostic value of kidney function impairment during COVID-19 has already been suggested in other series (2,10,11,31), but our study is the first large series to show that the outcome is associated with simple urine tests, such as UPCR and UACR, used in daily practice in contrast with new biomarkers, such as neutrophil gelatinase–associated lipocalin or kidney injury marker-1 (22,32), that have been evaluated in other situations with high risk of AKI but not yet in early phases of COVID-19.

This study has some limitations. First, the study population probably included patients who have the most severe forms of COVID-19, with a high proportion of patients requiring intensive care. In consequence, our results cannot be generalized to nonhospitalized patients with COVID-19. Second, some clinical data are missing, notably regarding level of inflammatory markers, severity of respiratory disease, and incidence of other complications, such as coagulopathy or cardiovascular events. These parameters strongly affect patient survival in this setting, and therefore, we cannot affirm that proteinuria is indeed an independent predictor of patient outcome. Third, the use of the UPCR should be interpreted with caution in AKI, as urinary creatinine excretion is variably decreased in this setting. Nevertheless, the aim of this study was not to estimate the 24-hour proteinuria by this ratio, as it is proposed in CKD, but was only to validate this simple test as a prognostic marker during COVID-19. Fourth, urine retinol binding protein—which was used as a marker for proximal tubular dysfunction in our study—was available for only 42% of the study population, and we do not have data about other tubular biomarkers, such as glycosuria, phosphaturia, or neutrophil gelatinase–associated lipocalin, or kidney pathology data to confirm the hypothesis of a proximal tubule injury. However, both proximal tubular lesions and presence of other features of Fanconi syndrome have recently been reported among patients with COVID-19 (24). To our knowledge, this study is the first large series to report on the composition of proteinuria during COVID-19—in contrast with previous publications based on dipstick analysis (10,11)—and to demonstrate that proteinuria in this setting contains mostly low-molecular weight proteins rather than albumin.

In conclusion, this study reveals that COVID-19 is associated with early and frequent tubular proteinuria, which is associated with poor kidney outcome and higher mortality among patients with symptomatic COVID-19. The comprehension of the precise mechanisms underlying this tubular injury requires further investigations.

Disclosures
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Supplemental Material

This article contains the following supplemental material online at http://cjASN.asnjournals.org/lookup/suppl/doi:10.2215/CJN.09130620/-/DCSupplemental.

Supplemental Table 1. Characteristics and outcome of admitted patients with and without proteinuria data available at admission.

Supplemental Table 2. Multivariable logistic regression analysis of outcomes odds according to UPCR ≥1 g/g, age, sex, BMI, hypertension, diabetes, and initial serum creatinine.

Supplemental Table 3. Multivariable logistic regression analysis of outcomes odds according to urine retinol binding protein-creatinine ratio ≥0.03 mg/ml, age, sex, BMI, hypertension, diabetes, and initial serum creatinine.

Supplemental Table 4. Characteristics of proteinuria in patients with and without diabetes and with and without CKD.

Supplemental Figure 1. Correlation between urine retinol binding protein-creatinine ratio and UACR-UPCR, UACR, and UPCR.

References


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