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ORIGINAL ARTICE

Clinical Features of COVID-19 Hospitalized Patients with different degree of Proteinuria

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ABSTRACT

Background: Coronavirus disease-19 (COVID-19) is a lung disease that also negatively affects several organ systems. Patients with COVID-19 frequently have kidney dysfunction. The exact impact of proteinuria and its degree on the clinical outcome of patients with COVID-19 is unclear. This study aimed to assess the effect of different degrees of proteinuria on the clinical outcome of hospitalized patients with COVID-19. **Methods:** This study included 100 patients with COVID-19 at Isolation Department, Zagazig University and Al-Ahrar Teaching Hospitals who presented with proteinuria on admission. Patients were divided into two groups based on their urine protein creatinine ratio (UPCR): those with UPCR less than 1gm and those with UPCR greater than 1gm. **Results:** There is a statistically significant relation between the two groups regarding neutrophil count (p=0.027) (higher in patients with UPCR > 1gm), and lymphocytes (p=0.003) (lower in patients with UPCR > 1gm). There is a statically significant relation between the degree of proteinuria and the incidence of acute kidney injury (p<0.001) (proteinuria higher in those who developed AKI). Also, there is a significant difference between the two groups regarding the conscious level (p=0.019) (disturbed conscious level more in patients with UPCR >1gm). **Conclusions:** Proteinuria in COVID-19 hospitalized patients is associated with poor clinical outcomes and could lead to further renal deterioration and AKI, so it is important to screen for it.
Key Words: COVID 19; Proteinuria; Acute kidney injury; Clinical Outcome.

INTRODUCTION

Acute respiratory syndrome is caused by coronavirus disease (COVID-19). Acute cardiac injury, coagulopathy, and thromboembolic consequences are some of its clinical and systematic manifestations, even though respiratory symptoms predominate. Kidney involvement is one of COVID-19's side effects [1].

Proteinuria, which is defined as the daily excretion of urine protein greater than 150 mg and may be transitory or persistent, is an indicator of underlying renal disorder [2]. The clinical effects of proteinuria are numerous. It is utilized to identify, categorize, monitor the progression, and assess the efficacy of a treatment for kidney affection by different aetiologies [3]. Patients who were admitted to Intensive care units (ICUs) and those with a fatal outcome have a higher incidence of renal involvement up to 65 % [4].

More recently a retrospective study revealed that acute kidney injury (AKI) occurred in 46% of COVID-19 patients, and 19% of those patients underwent dialysis [5]. Other data showed that on admission, 26.7% of patients had haematuria and 43.9% of patients had proteinuria [6].

Although not all biopsy samples had viral genetic material and cytopathogenic effects, a direct viral infection of the renal epithelium may be one factor contributing to kidney damage [7].

Pro-inflammatory cytokines may influence the pathophysiology of proteinuria, a condition that is also caused by proximal tubular resorption abnormalities and reduced glomerular permeability [8]. Therefore, this study aimed to assess the impact of proteinuria and its level among hospitalized COVID-19 patients on clinical outcomes.

METHODS

This comparative cross-sectional study was conducted on a total of 100 patients who were found to have proteinuria on admission with covid-19 that was confirmed by urinary protein/creatinine ration at the Isolation Department Zagazig University Hospital and Al-Ahrar Teaching Hospital between April 2021 and April 2022. Inclusion criteria were Patients of both sexes who were admitted to the hospital by COVID-19 infection, with an age more than 18 years old. Exclusion criteria were patients with past history of renal failure requiring long term haemodialysis as a replacement therapy, age <18 years old and, patients who was known to have a proteinuria from any cause in history.

An approval of the study was obtained from Zagazig University Academic and Ethical Committee (ZU-IRB #9340/15-3-2022) before the start of the study. The current study methods were adhered to the Helsinki Declaration's current revision. All participants were given information about the various aspects of the study, and they were only enrolled after signing a consent form.

Patients included in the study were subjected to full history taking and clinical examination. All patients were diagnosed with COVID-19 after RT-PCR tests on nasopharyngeal swabs yielded a positive result, as suggested by worldwide guidelines. Measurements included complete blood count, C-reactive protein, D dimer, ferritin, inflammatory markers (IL-6, pro calcitonin), urea, creatinine, protein creatinine ratio, ALT, AST, electrolytes, PH, and albumin.

Urinary protein to creatinine ratio (PCR) is reported as mg protein per gram creatinine (mg protein/g creatinine) in urine after measuring protein and creatinine separately in spot urine sample. Urinary PCR ≥ 150 mg/g characterize presence of proteinuria.

We divided our patients according to the degree of proteinuria in two subgroups either more than one gram/ gm creatinine or below. We chose the level of one gram that usually is considered according to different studies as indicator of significant kidney affection and related to the survival [9].

Before a patient can be discharged, the following requirements must be satisfied: A considerable improvement in respiratory symptoms; improved chest CT imaging revealing reduced inflammation; and normal body temperature for more than three days [10].

The data during the hospital admission, clinical outcomes, hospital stay, the need for mechanical ventilation (MV), and in-hospital death were obtained.

Statistical analysis: The statistical package for the social sciences, SPSS, version 26, was used to analyse the data. Using the chi-square test and fisher exact, categorical variables were described. Means, standard deviations, and median and range were all used to define quantitative variables. The ROC curve, Mann-Whitney test, Shapiro-Wilk test, and independent sample t-test were applied. The level of statistical significance was set at $P<0.05$. A highly significant difference was present if $p\leq0.001$.

RESULTS

This study included 100 hospitalized patients with COVID-19 infection, sixty males (60%) and 40 females (40%) which was statistically significant (p=0.04). There is a non-significant difference between groups regarding age **(Table 1).**

There is a statistically significant relation between groups (according to levels of proteinuria either below or above 1 gm) regarding patients who develop AKI that need hemodialysis and disturbed consciousness level (P<0.005). On the other hand, there is non-significant relation between proteinuria and either presenting symptoms, comorbid diabetes, hypertension, use of ACEI/ARB or diuretics **(Table 1).**

There is no statistically significant relation between groups as regard systolic, diastolic blood pressure, pulse, or temperature **(Table 2).**

There is a statistically significant relation between the two groups regarding neutrophil count (p=0.027) (higher in patients with UPCR > 1gm), and lymphocytes (p=0.003) (lower in patients with UPCR > 1gm). There is no statistically significant relation between groups as regard other laboratory parameters **(Table 3)**.

There is statistically significant correlation between urine protein creatinine ratio and both lymphocytic count (r= 0.252, p= 0.012) and IL-6 (r= 0.451, p= 0.024), while there is statistically significant negative correlation between urine protein creatinine ratio and neutrophil count (r= -0.256, p= 0.01) **(Figure 1)**.

There is a statistically significant relation between groups as regards the incidence of AKI (proteinuria was higher in those who developed AKI). There is a statistically significant difference between the two groups as regards outcome in form of increasing survivors in the group of UPCR <1gm and decreased survivors in the group of UPCR >1gm. There is non-significant relation between groups as regards need for ventilation or length of hospital stay **(Table 4)**.

Relation between overall survival and timing of proteinuria was assessed using appropriate survival analysis, there is no statistically significant association between them (p=0.1) **(Table 5) (Figure 2)**.

Table 1: Relation between proteinuria and clinical data in the two groups.

	Group of UPCR <1gm	**Group of UPCR > 1gm**	**χ^2/t**	**P**
	N=45 (%)	**N=55 (%)**		
Gender: **Female** **Male**	 23 (41.1%) 32 (58.9%)	 17 (37.9%) 28 (62.2%)	4.209	0.04*
Age (year)	60.29 ± 10.61	59.47 ± 13.59	0.329	0.743
Diabetes	16 (35.6%)	17 (30.9%)	0.242	0.643
Hypertension	26 (57.8%)	29 (52.7%)	0.255	0.614
Non proteinuric CKD	7 (15.6%)	5 (9.1%)	0.979	0.322
AKI being on dialysis	14 (31.1%)	5 (9.1%)	7.798	0.005*
ACEI/ARBs	16 (35.6%)	14 (25.5%)	1.203	0.273
Diuretics	7 (15.6%)	14 (25.5%)	1.462	0.227
Cough	38 (84.4%)	45 (81.8%)	0.121	0.728
Fever	43 (95.6%)	53 (96.4%)	0.042	0.837
Disturbed conscious level	34 (75.6%)	29 (52.7%)	5.533	0.019*
χ^2chi square test *p<0.05 is statistically significant, UPCR: urinary protein to creatinine ratio, CKD: chronic kidney disease, AKI: acute kidney injury. ACEI/ARBs : angiotensin converting enzyme inhibitors/angiotensin receptor blockers.				

Table 2: Relation between proteinuria and vital data in the two groups.

	Group of UPCR <1gm	**Group of UPCR > 1gm**	**t**	**P**
	Mean ± SD	**Mean ± SD**		
SBP	132.89 ± 28.47	133.91 ± 30.04	-0.173	0.863
DBP	86.11 ± 18.92	86.09 ± 19.21	0.005	0.996
Pulse	101.73 ± 17.41	97.04 ± 20.79	1.208	0.23
Temperature	38.29 ± 0.75	38.29 ± 0.63	1.004	0.996
t independent sample t test, SBP systolic blood pressure, DBP diastolic blood pressure				

Table 3: Relation between proteinuria and laboratory data in the two groups.

	Group of UPCR <1gm	Group of UPCR > 1gm	t	P
	Mean ± SD	Mean ± SD		
Hemoglobin	11.29 ± 2.31	11.04 ± 2.3	0.545	0.587
Platelet count	249.24 ± 45.95	263.27 ± 60.69	-1.279	0.204
Neutrophil	6.84 ± 1.13	7.35 ± 1.13	-2.239	0.027*
Lymphocyte	1.65 ± 0.4	1.38 ± 0.47	3.078	0.003*
Sodium	136.04 ± 6.65	136.65 ± 7.14	-0.438	0.662
Potassium	4.42 ± 1	4.31 ± 0.78	0.582	0.562
PH	7.32 ± 0.15	7.33 ± 0.13	-0.492	0.624
Bicarbonate	19.68 ± 6.89	21.93 ± 6.23	-1.302	0.196
PCO2	39.29 ± 7.06	41.27 ± 7.51	-1.35	0.18
	Median (IQR)	**Median (IQR)**	**MW**	**P**
WBCs	9.6(7.62 – 12.05)	9(7 – 13.3)	-0.173	0.862
Creatinine	1.1 (0.84 – 1.32)	2.5 (1.54 – 3.79)	-4.224	0.0001
ALT	31(20.5 – 41.5)	34(24 – 51)	-1.408	0.159
AST	46 (35 – 63)	46 (35.5 – 64)	-1.28	0.201
INR	1.4 (1.2 – 1.5)	1.3 (1.1 – 1.4)	-0.855	0.393
IL-6	6.5 (5 – 11.25)	12 (8.5 – 20.1)	-1.152	0.249
Procalcitonin	0.83 (0.53 – 1.8)	2 (0.74 – 4.6)	-0.568	0.57
D dimer	0.8 (0.5 – 1.5)	0.8 (0.43 – 1.5)	-0.187	0.852
CRP	49 (41.5 – 59)	50 (44.2 – 64)	-0.417	0.677

t independent sample t test MW Mann Whitney test IQR interquartile range *p<0.05 is statistically significant. ALT alanine transaminase, AST aspartate aminotransferase, INR international normalized ratio, IL-6 interleukin 6, D-dimer disseminated Intravascular Coagulation, CRP c reactive protein.

Table 4: Relation between proteinuria and in-hospital events in the two groups.

	Group of UPCR <1gm	Group of UPCR > 1gm	χ^2	P
	N=55 (%)	N=45 (%)		
Ventilator	30 (54.5%)	31 (68.9%)	0.032	0.856
AKI	14 (25.5%)	32 (71.1%)	20.77	<0.001**
Outcome: **Survivors** **Non-survivors**	 42 (76.4%) 13 (23.6%)	 24 (53.3%) 21 (46.7%)	11.55	<0.001**
	Median (IQR)	Median (IQR)	MW	P
Hospital stays	9 (8 – 11)	9 (7 – 12)	-0.765	0.444

χ^2chi square test, *p<0.05 is statistically significant, MW Mann Whitney test IQR interquartile range. **p≤0.001 is statistically highly significant, AKI acute kidney injury

Table 5: Relation between overall survival and timing of proteinuria.

		Total N	N of Events	Censored		Survival time, Months		P
				N	%	Mean		
						Estimate ±SD	95% CI	
Proteinuria	**Group of UPCR <1gm**	55	13	42	(76.4%)	12.19 ± 0.63	10.97 – 13.42	0.1
	Group of UPCR > 1gm	45	21	24	(53.3%)	13.49 ± 0.6	12.4 – 16	
Overall		100	34	66	66.0%	12.82 ± 0.43	11.97 – 13.66	

UPCR urinary protein to creatinine ratio
*p<0.05 is statistically significant

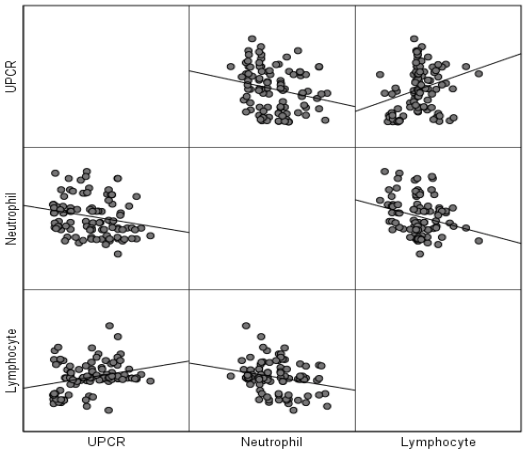

Figure 1: Scatter matrix showing significant correlation between UPCR and both neutrophil and lymphocyte.

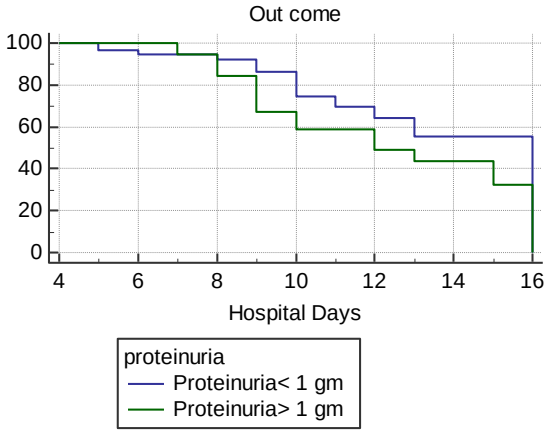

Figure 2: Kaplan Meier plot showing relation between overall survival and proteinuria among studied patients.

DISCUSSION

The novel severe acute respiratory syndrome coronavirus 2, which is the cause of the coronavirus disease 2019 (COVID-19), has quickly spread throughout the world and is linked to terrible morbidity and mortality in hospitalized patients. Patients with COVID-19 had renal problems, according to recent reports [11-13].

Acute kidney involvement was an independent risk factor for mortality, according to a recent Chinese study [6]. On admission, they discovered that 26.7% of patients had hematuria and 43.9% of patients had proteinuria. Moreover, Chan et al. revealed that of the 435 individuals with AKI who underwent available urine tests, 84% had proteinuria, 81% had hematuria, and 60% had leukocyturia [5].

Therefore, our aim of the study was not only to record proteinuria as an incident finding in COVID-19 patients, but also to assess the effect of different degrees of it on the clinical outcome.

In our study, patients were divided based on their initial UPCR level (<1 versus ≥1 g protein/g creatinine) to compare baseline characteristics; as there were 55% of the studied patients had UPCR≥1 g/g; while in a retrospective analysis of a cohort of 153 hospitalized patients with COVID-19, Huart et al. [14] found almost 80% of people have abnormal proteinuria, 43% of them have category 3 proteinuria (over 500 mg/g). Hirsch et al. [15] found 646 patients in New York City had a prevalence of 42.1%, according to dipstick data.

The significant amount of renal tubular protein in the urine results is consistent with the AKI in COVID-19, which is related to acute tubular necrosis. The postmortem data show that ATN appears to be the most common cause [7]. Although proteinuria in COVID-19 has been observed in the absence of AKI and may indicate a subclinical insult, it is important to understand the disease pathways causing AKI in COVID-19 [16].

In the current study, 46 patients (46%) developed AKI that was more evident in the proteinuria > 1 gm patients' group, and this was relatively near to the results in the study done by Karras et al. (10) who found AKI in 88 (44%) patients. Karras et al. showed that the demand for AKI and renal replacement treatment might be predicted by the presence of low molecular weight proteinuria, which may occur before an increase in serum creatinine [10].

The prevalence of AKI across 20 cohorts was 17% in a comprehensive review and meta-analysis of COVID-19, with a range of 0.5-80% [17]. This indicates that kidney involvement is frequent in COVID-19 individuals who are hospitalized. In COVID-19, AKI has previously been linked to in-hospital mortality [6] and renal abnormalities in COVID-19 continue to be linked to a poor outcome [18].

As regards the current study, it was found that there is a statistically significant relation between the degree of proteinuria and neutrophil (higher in the group of UPCR > 1gm patients) that agree with Chen et al. findings [6]. Our explanation is that higher degrees of proteinuria is associated with a higher incidence of secondary bacterial infection (evidenced by high neutrophil count) that may represent immune system dysfunction due to protein loss.

According to our study, the lymphocytic count was correlated significantly with urinary protein to creatinine ratio that coincide with Huart et al. [14] who reported similar findings in patients with severe infection compared to patients with non-severe infections. These results may be explained by the fact that individuals with severe COVID-19 infections had an enhanced inflammatory response, including greater white blood cell counts, lower lymphocyte and platelet counts, and higher C-reactive protein levels.

In our study, there is a statistically significant relation between the degree of proteinuria and sex (males represented 69.1% of patients with proteinuria). Other studies suggest that men take less care of their health and frequently present to the healthcare system with more advanced sickness, the association between the predominance of males in AKI cases in patients by COVID-19 is unclear [12].

In the present study, there is non-significant relation between the degree of proteinuria and each age and comorbidity by hypertension which disagrees with Karras et al. [10] who found older patients with a history of hypertension have higher rates of proteinuria; this difference may be attributed to racial differences.

Also, another study by Nlandu et al. [19] reported that patients with proteinuria were older and more likely to have high blood pressure.

In the current study, there was non-significant relation between the degree of proteinuria and either presenting symptoms, or comorbid diabetes, that was similar to the findings mentioned by Karras et al. [10]. In this study, there is non-significant relation between the degree of proteinuria and the use of ACEI that coincide with Chaudhri et al. [20] who had the same results.

In our study, there was no statistically significant relation between the degree of proteinuria and D dimer which disagrees with Ouahmi et al. [16] who found that D-dimers were correlated with the level of proteinuria.

In the current study, there was statistically significant relation between degree of proteinuria and incidence of AKI (higher in those who developed AKI). There is statistically significant relation between proteinuria and outcome (proteinuria was associated with poor outcome in COVID 19 infection) that agree with Chaudhri et al. [20] who found that individuals with proteinuria had a greater incidence of in-hospital AKI, ICU hospitalization, and mortality.

In the current study, there was statically high significant relation between two groups according to UPCR and survival (higher survivors more with UPCR <1gm).

Also, in another study by Ouahmi et al. [13] showed that proteinuria during the beginning of the infection with COVID-19 was linked to a poor outcome. Pei et al. [7] found that in 333 Chinese patients with COVID-19, it was revealed that those who had renal involvement (hematuria, proteinuria, or AKI) had a greater inpatient death rate than those who did not (11.2 vs. 1.2%).

In another study of 701 Chinese patients hospitalized with COVID-19, Cheng et al. [6] reported that after controlling for age, sex, illness severity, comorbidity, and leukocyte count, the proteinuria and hematuria were linked to a higher risk of in-hospital mortality. Nlandu et al. [19] revealed that higher degrees of proteinuria in patients with COVID-19 was considered as a predictor of mortality.

In the present study, there is non-significant relation between degree of proteinuria and length of hospital stay that disagree with Ouahmi et al. [16] who found that the length of hospital stay was predicted by proteinuria more than 0.3 g/g, probably as a result of the patients' varied baseline characteristics (our cohort was younger and with a larger sample size).

Our study has some limitations: First, the study is retrospective in nature. Second some baseline labs on admission were not available. Lastly, important variables like weight and height were not available for some patients.

Although there are strength points: the relatively adequate sample size, it was done in two separate hospitals, and we use confirmative analysis between patients as regard different levels of proteinuria in relation to wide range of parameters.

Conclusions: Proteinuria in COVID-19 hospitalized patients is associated with poor clinical outcome. Additional renal deterioration and development of AKI in those who develop proteinuria could herald further worse consequences. That may necessitate that hospitalized patients with COVID-19 be screened for proteinuria as underlying subclinical renal involvement and closely monitor them.

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