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Clinical and pathological renal outcomes of COVID-19 patients: an Egyptian retrospective multi-center pooled analysis

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Abstract

Background Kidneys have been one of the different organs affected by **s**evere acute respiratory syndrome coronavirus 2 (SARS-CoV-2) since its discovery, Acute kidney injury was the most common presentation. A pooled data from different kidney centers or hospitals in Egypt who sent their renal biopsy specimens from patients with renal trouble, up to 4 months after catching SARS-CoV-2, to PATH LAB for diagnosis, were analyzed.

Results Beside acute kidney injury, a variety of different presentations was found, such as accidentally discovered impaired kidney function, varying degrees of proteinuria, and nephrotic syndrome. Not only acute tubular injury, acute tubulointerstitial nephritis, or thrombotic microangiopathy, but the extent of observation for 4 months revealed, unexpected pathologies, such as podocytopathies, membranous glomerulonephritis, proliferative and necrotizing glomerulonephritis, and lupus nephritis.

Conclusion This virus has been incriminated in a chain of different kidney disease presentations and pathologies, although, a causal relationship is difficult to prove.

Keywords COVID-19, Patients, Renal, Clinical, Pathology

Introduction

Coronavirus disease 19 (COVID-19) due to **s**evere acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has been a global pandemic since 2019 [1]. The beginning was fever and lung injury which left victims everywhere it spread (6,714,775 deaths as of 9 Jan 23) [2]. Unfortunately, the lung wasn't the sole target for this lethal virus, affliction of other body organs was discovered sequentially, due to persistence of this pandemic for a considerate time thereafter [3]. Many markers predicted severity and mortality, such as mean platelet volume (MPV) [4], and Red cell distribution width (RDW) [5]. The involvement of Kidney has been common and resulted in poor prognosis [6]. The potential routes for kidney injury are ischemia in the context of hemodynamic instability, coagulation, lung injury and hypoxemia, cytokine storm or possible kidney viral invasion [7]. The more time passed, the more knowledge obtained in the different kidney pathology in the era of COVID-19. Of course, isolation status and bad general condition cancelled many biopsies, so, many of the initial case series was based on autopsy [8]. Acute kidney injury (AKI) in hospitalized patients was the most apparent manifestation, it was reported in many studies [9, 10]; it reached 46% in one study [11]. It caused higher mortality [12]. Acute tubular injury (ATI), collapsing glomerulopathy (CG), and endothelial injury or thrombotic microangiopathy (TMA), were the most common histological Findings [13–15]. There is shortage of data regarding kidney involvement in COVID-19 patients from Egypt, so this study aims to address it. Unfortunately, our study lacked

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assessment of many factors that affect COVID-19 severity and prognosis like HLA type [16], PDL1 [17], TGF-β [18], and IL10 [19].

Methods

Renal biopsies from living patients with recent or remote COVID-19 within 4 months, with renal affliction, from July 2020 to January 2022, were sent sequentially from many kidney centers and hospitals in Cairo, Giza, and other Egyptian cities, to PATH LAB for analysis. Case identification was done through clinical symptoms such as fever or chest symptoms and confirmed by positive nasopharyngeal swabs reverse-transcriptase polymerase chain reaction (RT-PCR) testing. COVID-19 severity was estimated based on NIH clinical spectrum classification [20]. Full medical history and workup until biopsy were sent from the referring center/hospital. Cases, who have ever received any type of COVID-19 vaccines, and renal transplant recipients, were excluded from the study.

A total of 104 cases, examination of Core needle biopsy was done using light microscopy (LM) for all cases, after staining with hematoxylin eosin, periodic acid–Schiff, Jones methylamine silver, and trichrome. Immunofluorescence microscopy was done in 89 cases based on LM finding; immunohistochemistry for SARS-CoV-2 viral particles was done for eleven cases that were presented with AKI. Electron microscopy was performed in only 29 cases.

Indications for biopsy according to the main patients' presentations were:

AKI, defined as per Kidney Disease Improving Global Outcomes (KDIGO) guidelines. [21]

Acute on top of chronic kidney disease (AKI on CKD), defined as per KDIGO guidelines. [21]

Nephrotic syndrome (NS): it is typically defined as proteinuria ≥ 3.5 g/day or spot urine protein to creatinine ratio > 3,000 mg/g (300 mg/mmol) plus hypoalbuminemia and edema. [22]

Nephrotic range proteinuria (NRP): nephrotic range is defined as proteinuria ≥ 3.5 g/day or spot urine protein to creatinine ratio > 3,000 mg/g (300 mg/mmol) in the absence of clinically overt NS. [22]

Subnephrotic proteinuria (SP): proteinuria is defined as less than 3.5 g and > 0.2 g of protein excreted in the urine over 24 hours or spot urine protein to creatinine ratio < 3,000 mg/g and > 0.2 . [22]

Impaired kidney function newly discovered: defined as per (KDIGO) guidelines [23]

Statistical analysis

Data analysis was performed using the IBM SPSS Statistics for Windows, Version 26.0 (SSPS Inc., Armonk, New York, USA). Data are reported as medians (interquartile ranges); being non-parametric, or frequencies (percentages).

Results

15 patients out of 104 (14.4%) presented with AKI (Table 1), median age was 43 years (28–55), 10 males (M), 5 females (F), 3 patients had history of hypertension (HTN), and only 1 patient had history of diabetes mellitus (DM). 9 patients had severe COVID-19 and the others had mild to moderate disease. Median duration from COVID-19 diagnosis to biopsy was 10 days

Table 1 Clinical and laboratory findings in different clinical presentations

	AKI	AKI on CKD	newly discovered impaired kidney function	nephrotic range proteinuria	nephrotic syndrome	subnephrotic range proteinuria
No	15	25	25	8	18	13
Age (years)	43 (28–55)	51 (37–60)	43 (33–55.5)	40.5 (22.25–55)	36 (25.25- 52.25)	35 (22–45)
sex	10 M, 5 F	14 M, 11 F	14 M, 11 F	6 M, 2 F	9 M, 9 F	8 M, 5 F
Severe COVID-19	9 (60%)	13 (46%)	8 (32%)	2 (25%)	1 (5.6%)	2 (15%)
Duraion from covid-19 diagnosis to biopsy (days)	10 (7–12)	30 (21.5–92.5)	34 (30–71)	80.5 (41.25–118.25)	63 (44–91)	50 (32–101)
sCr	7.1 (4.6–13)	4.6 (3.2–6.15)	4.5 (2.5–8.45)	1.24 (1–1.48)	1.2 (1.1–1.8)	0.93 (0.8–1.2)
eGFR (ml/min/1.73 m2)	7.52 (4.57–11.16)	14.3 (8.21–21.87)	15.18 (6.14–32.11)	71.62 (55.68- 80.81)	63.09 (41.05–84.97)	97.31 (71.76–124.6)
Urine RBC	15 (4–30)	4 (3–13)	3 (2–5)	4(2–9)	4 (3–8)	4 (3–40)
UPCR (gm/gm)	2 (0.3–2.9)	1 (0.57–2.46)	1.46 (0.36–2.63)	3.95 (3.536- 4.93)	6.5 (4.54–13)	1.5 (0.95–2.5)
Need for RRT	7 (46.7%)	5 (20%)	7 (28%)	none	none	none

Y Yes, *N* No, *M* Male, *F* Female, *SCr* Serum creatinine, *eGFR* Estimated glomerular filtration rate: mL/min/1.73 m^2, *RBC* Red blood cell (per high-power field), *UPCR* Urine protein-creatinine ratio, *RRT* Renal replacement therapy

(7–12), median estimated glomerular filtration rate (eGFR) at time of biopsy was 7.52 ml/min/1.73 m^2 (4.57–11.16), median urine protein-creatinine ratio (UPCR) was 2 gm/gm (0.3–2.9), and median urine RBCS/high power field(HPF) was 15 (4–30). 7 patients needed renal replacement therapy (RRT) at time of biopsy. Pathology showed 4 cases with ATI, (there were 1 with pigment, 1 with oxalate, and 1 with acute tubulointerstitial nephritis (ATIN)), 1 case showed ATIN, 1 case showed CG, 2 cases showed infection-related glomerulonephritis (IRGN), 2 cases showed proliferative/necrotizing glomerulonephritis (prolif/necr GN), 3 cases showed TMA; 1 of them complicated with renal infarction, and accidentally discovered 2 cases with Light chain nephropathy (LCN). Of the 11 cases tested, none showed SARS-CoV-2 viral particles with immunohistochemistry.

25 cases out of 104 (24%) presented with AKI on CKD (Table 1), median age was 51 years (37–60), (14 M, 11 F), all of them had documented CKD before having COVID-19, 11 patients with combined DM and HTN, 4 patients with HTN, 3 with Systemic lupus erythematosus (SLE), 1 with Focal segmental glomerulosclerosis (FSGS), 1 with primary Membranous glomerulonephritis (MGN), 1 with rheumatoid arthritis (RA), 1 with systemic sclerosis and 3 without history of co morbid conditions. 13 patients had severe COVID and the others (12) had mild to moderate disease. Median duration from COVID-19 diagnosis to biopsy was 30 days (21.5–92.5), median eGFR at time of biopsy was 14.3 ml/min/1.73 m^2 (8.21–21.87), median UPCR ratio was 1 gm/gm (0.57–2.46), and median urine RBCS/HPF was 4 (3–13). 5 patients needed RRT at time of biopsy.

In those with history of DM or HTN or both (15 patients), pathology showed chronic changes related to HTN, DM or both in most cases, and besides, in Case 2 LCN, Case 4 IRGN, Cases 8, 11, 15 FSGS, Case 12 prolif/necr GN, Cases 13, 17 TMA, and cases 18, 21, 23 ATIN. 1 case with history of FSGS showed only FSGS, 1 case with history of MGN showed only MGN, 1 case with history of scleroderma showed TMA, 1 case with history of lupus nephritis (LN) showed findings of ESRD, and 1 case with history of rheumatoid arthritis showed prolif/necr GN. There were 3 cases with CKD of unknown etiology, 1 showed chronic tubulointerstitial nephritis (CTIN), 1 showed FSGS and the 3rd showed ATIN.

25 cases out of 104 (24%) were presented with impaired kidney function discovered during regular checkup or after any medical concern (Table 1), median age was 43 years (33–55.5), (14 M, 11 F), 6 patients had history of HTN, 1 patient had history of DM plus HTN, 3 patients had history of SLE and the others (15) had no history of medical illnesses. 8 patients had severe COVID-19 and the others (17) had mild to moderate disease. Median duration from COVID-19 diagnosis to biopsy was 34 days (30–71), median eGFR at time of biopsy was 15.18 ml/min/1.73 m^2 (6.14–32.11), median UPCR was 1.46 (0.36–2.63), and median urine RBCS/HPF was 3 (2–5). 7 patients needed RRT at time of biopsy.

Pathology showed 3 cases with ATI (1 with pigment), 5 cases with ATIN (1 case showed Granulomatous interstitial nephritis), and 1 case showed IgG4), 2 cases showed amyloidosis (1 with amyloid A), 1 case showed CG, 1 showed IRGN, 1 with glomerulopathy and organized deposits, 9 cases showed FSGS, and 3 cases showed findings of end stage renal disease.

8 patients out of 104 (7.7%) presented with NRP (Table 1), median age was 40.5 years (22.25–55), (6 M, 2 F), 1 patient had history of HTN, 2 patients had history of DM and HTN (1 of them had CKD), 1 patient had SLE, 1 patient had history of FSGS and 3 patients had no history of medical illness. 2 patients had severe COVID-19 and the others (6) had mild to moderate disease. Median duration from COVID-19 diagnosis to biopsy was 80.5 days (41.25–118.25), median eGFR at time of biopsy was 71.62 ml/min/1.73 m^2 (55.68- 80.81), median UPCR was 3.95 gm/gm (3.536- 4.93), and median urine RBCS/HPF was 4(2–9). None needed RRT at time of biopsy.

The patient with history of SLE showed LN class II-V and the patient with history of FSGS showed also FSGS. The other patients with history of DM, hypertension, both or none showed 2 cases with minimal change disease (MCD), 2 cases with MGN (one showed positive tissue staining for phospholipase A2 receptor [PLA2R]), 1 case showed FSGS, and 1 case showed Cryoglobulinemic glomerulonephritis.

18 patients out of 104 (17.3%) presented with NS (Table 1), median age was 36 years (25.25- 52.25), (9 M, 9 F), 5 patients had history of HTN, 1 patient had history of DM, 1 patient had history of SLE, and the other 11 patients had no history of medical illness. Only 1 patient had severe COVID-19 and the others (17) had mild to moderate disease. Median duration from COVID-19 diagnosis to biopsy was 63 days (44–91), median eGFR at time of biopsy was 63.09 ml/min/1.73 m^2 (41.05–84.97), median UPCR was 6.5 (4.54–13), and median urine RBCS/HPF was 4 (IQR, 3–8). None needed RRT at time of biopsy. Pathology showed 3 cases with LN class IV; only one of them had history of SLE, 2 patients showed amyloidosis (1 of them was AL), 4 patients showed MCD, 5 patients showed MGN, (3 with positive tissue staining for PLA2R), 1 patient showed FSGS, 1 patient showed ATIN (with pigment), 1 patient showed IRGN, and 1 patient showed TMA.

13 patients out of 104 (12.5%) presented with SP (Table 1), median age was 35 years (22–45), (8 M, 5 F), 1 patient had history of HTN, 2 patients had history of HTN and DM;

1 of them had CKD as well, 2 patients had history of SLE; 1 of them had LN, and 8 patients had no history of medical illness. 2 patients had severe COVID-19 and the others (11) had mild to moderate disease. Median duration from COVID-19 diagnosis to biopsy was 50 days (32–101), median eGFR at time of biopsy was 97.31 ml/min/1.73 m^2 (71.76–124.6), median UPCR was 1.5 gm/gm (0.95–2.5), and median urine RBCS/HPF was 4 (3–40). None needed RRT at time of biopsy. Light microscopy showed 1 case with AA amyloidosis, 2 cases with MCD, 2 cases with MGN (one with positive tissue staining for PLA2R), 1 case showed prolif/necr GN, 3 cases showed LN (1 with class III, 2 with class IV); but 2 cases had history of SLE, 2 cases showed FSGS, 1 case showed IRGN, and 1 patient with history of DM, HTN and CKD showed diabetic nodular glomerulosclerosis.

Independent-Samples Kruskal–Wallis Test was used for comparison between different diagnoses

Those who had SLE flare were significantly younger ($p < 0.05$) with median (IQR, 25–75) of 22 (18.5–29.5) years, in comparison to those with ATI 44.5 (37.8–60.5), ATIN 50.5 (36.3–60.3), podocytopathy 37 (29.5–51.5), MGN 38 (25.3–58), prolif/necr GN 38 (29.5–55) or IRGN 56 (23.8–70.5).

Those who had ATI, ATIN or TMA showed significantly reduced eGFR ($p < 0.05$) with median (IQR, 25–75) of 7.04 (5.22–11.26), 13.85 (6.79–27.25), and 6.64 (4.74–31.68) ml/min/1.73 m^2 respectively, in comparison to those with podocytopathy 38.04 (22.35–71.66), MGN 72.17 (42.28–101.75), and SLE flare 83.26 (60.29–124.55).

Those who had SLE flare, podocytopathy or MGN showed significantly higher UPCR ($p < 0.05$), with median (IQR, 25–75) of 3.14 (1.55–8.88), 3 (2.4–5), and 4.93 (2.81–12.95) gm/gm respectively, in comparison to those with ATI 0.31 (0.16–0.89), ATIN 0.56 (0.24–1.12), TMA 0.76 (0.07–2.28), and prolif/necr GN 0.9 (0.7–2.58).

Comparisons of durations between different possible related pathologies diagnoses and COVID-19 diagnosis using independent samples Median test (Fig. 1).

Fisher's exact test was used to determine if there was an association between different diagnoses and sex. There was a statistically significant association (two-tailed $p = 0.017$). (Table 3). It is noted female predominance

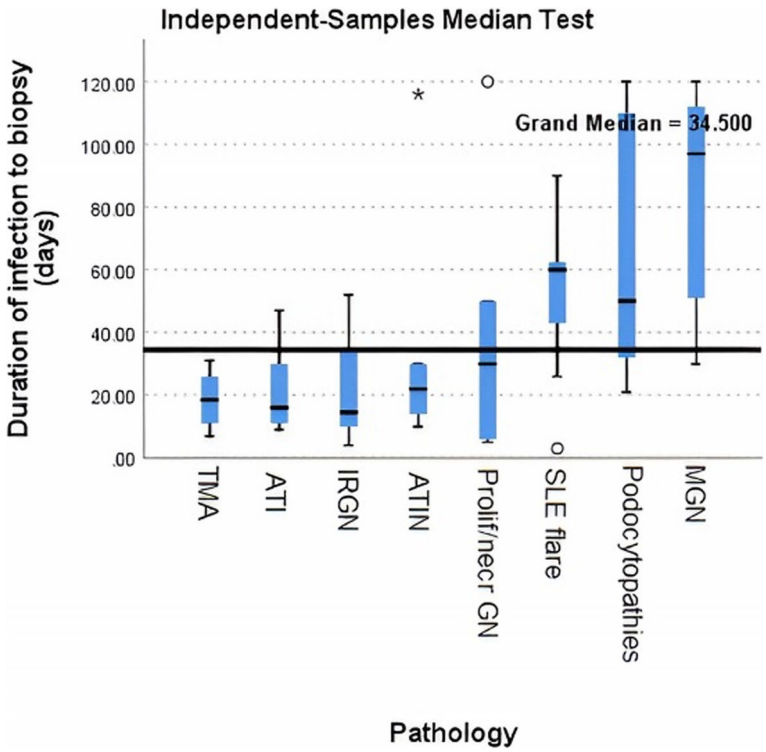

Fig. 1 ATI, ATIN, IRGN or TMA occurred early after catching COVID-19, with median (IQR, 25–75) of 16 (11–34), 22 (13–52), 15 (9–39), and 19 (11–28) days respectively. On the other side, MGN, podocytopathies (CG, MCD, FSGS), prolif/necr GN or SLE flare occurred late with median (IQR 25–75) of 97 (46–114), 50 (32–111), 30 (6–85), and 60 (35–64) days respectively (Table 2). There is significant difference between ATI and either podocytopathy (P 0.035), SLE flare (P 0.02), or MGN (P 0.007). There is significant difference between TMA and either podocytopathy (P 0.003), SLE flare (P 0.046), or MGN (P 0.04). There is significant difference between IRGN and either SLE flare (P 0.031), or MGN (P 0.039). There is significant difference between ATIN and either SLE flare (P 0.031), or MGN (P 0.039)

Table 2 Clinical and laboratory findings in different pathological presentations

	ATI	ATIN	IRGN	MGN	Podocytopathy	Prolif/necr GN	SLE flare	TMA/Renal inf
No	10	6	6	10	29	5	8	8
Age (years)	44.5 (37.8–60.5)	50.5 (36.3–60.3)	56 (32.8–70.5)	38 (25.3–58)	37 (29.5–51.5)	38 (29.5–55)	22 (18.5–29.5)	35 (24.8–44.5)
sex	5 M, 5F	2 M, 4F	6 M	7 M, 3F	19 M, 10F	4 M, 1F	1 M, 7F	3 M, 5F
Severe COVID-19	8 (80%)	5 (83.3%)	1 (16.7%)	2 (20%)	6 (20.7%)	1 (20%)	none	3 (37.5%)
Duraion from covid-19 diagnosis to biopsy (days)	16.00 (10.8–33.8)	22 (13–51.5)	14.5 (8.5–38.5)	97 (45.8–113.8)	50 (32–111.5)	30 (5.50–85)	60 (34.50–63.8)	18.5 (10.5–28)
sCr	7.80 (5.43–9.73	5.05 (2.63–6.55)	7.90 (1.18–13.75)	1.26 (.84–2.55)	2.06 (1.14–2.85)	4.50 (2.55–4.60)	.99 (.75–1.20)	8.55 (2.97–10.30)
eGFR (ml/min/1.73 m2)	7.04 (5.22–11.26)	13.85 (6.79–27.25)	9.55 (4.20–78.22)	72.17 (42.28–101.75)	38.04 (22.35–71.66)	17.27 (12.48–49.52)	83.26 (60.29–124.56)	6.64 (4.74–31.68)
Urine RBC	4 (3–20)	4 (2–10)	35 (12–45)	3 (2–4)	3 (2–4)	80 (40–93)	15 (6–36)	7 (3–53)
UPCR (gm/gm)	.31 (0.16–0.89)	.56 (.24–1.12	1.93 (1.02–5.58)	4.93 (2.81–12.95)	3 (2.40–5)	.90 (.70–2.58)	3.14 (1.55–8.88)	.76 (.07–2.28)
Need for RRT	5 (50%)	1 (16.7%)	2 (33.3%)	none	none	1 (20%)	none	4 (50%)

Y Yes, *N* No, *M* Male, *F* Female, *SCr* Serum creatinine, *eGFR* estimated glomerular filtration rate: mL/min/1.73 m^2, *RBC* Red blood cell (per high-power field), *UPCR* Urine protein-creatinine ratio, *RRT* Renal replacement therapy, *ATI* Acute tubular injury, *ATIN* Acute tubulointerstitial nephritis, *IRGN* Infection-related glomerulonephritis, *MGN* Membranous glomerulonephritis, *prolif/necr GN* proliferative and necrotizing glomerulonephritis, *TMA* thrombotic microangiopathy, *Renal inf* Renal infarction

Table 3 True and expected number of each sex in each diagnosis

			SEX		Total
			F	M	
Category	ATI	Count	5	5	10
		Expected Count	4.3	5.7	10.0
	ATIN	Count	4	2	6
		Expected Count	2.6	3.4	6.0
	IRGN	Count	0	6	6
		Expected Count	2.6	3.4	6.0
	MGN	Count	3	7	10
		Expected Count	4.3	5.7	10.0
	Podocytopathy	Count	10	19	29
		Expected Count	12.4	16.6	29.0
	Prolif/necr GN	Count	1	4	5
		Expected Count	2.1	2.9	5.0
	SLE Flare	Count	7	1	8
		Expected Count	3.4	4.6	8.0
	TMA/Renal inf	Count	5	3	8
		Expected Count	3.4	4.6	8.0
Total		Count	35	47	82
		Expected Count	35.0	47.0	82.0

ATI, acute tubular injury; ATIN, acute tubulointerstitial nephritis, *IRGN* infection-related glomerulonephritis, *MGN* Membranous glomerulonephritis, prolif/necr *GN* proliferative and necrotizing glomerulonephritis, *TMA* thrombotic microangiopathy, *Renal inf* Renal infarction

in those who had SLE flare, and male predominance in those who had IRGN.

Discussion

In our study, we tried to focus on the possible near or remote renal complications of COVID-19. Those who ever received any type of COVID-19 vaccine were excluded for the possible confounding reported complications such as MCD, MGN, IgA nephropathy, and ATIN [24–26]

104 native kidney biopsies and their respective cases were described, and analyzed. Patients biopsied had kidney affection soon or late after documented SARS-CoV-2 infection. The most common indications for biopsy were Impaired kidney function newly discovered ($N=25$), AKI on top of CKD ($N=25$), NS ($n=18$), and AKI ($n=15$).

These indications were different in other study [13] that included 17 patients, acute kidney injury and proteinuria were the most common 2 indications but biopsy was taken within 1 week of catching SARS-CoV-2, unlike our study which extended up to 4 months.

The main renal pathology in both AKI and AKI on top of CKD cases were ATI 5 cases, TMA 5 cases, beside another variable pathologies as shown above. In another study [27] on 10 patients presented e AKI after COVID-19, renal pathology showed ATI, two patients showed TMA, one had pauci-immune crescentic GN, and another had features of healed collapsing glomerulopathy; which was near to our study.

In our study, kidney biopsy showed only 2 cases with CG after having mild COVID-19. The first was female patient 28 years old that presented with AKI (eGFR 20.81 ml/min/1.73 m^2) and severe proteinuria 2.9 gm/gm, biopsy was taken 27 days after diagnosis, the other was male patient 17 years old that presented with impaired kidney function (GFR 18.52 ml/min/1.73 m^2) and severe proteinuria 2.8 gm/gm, biopsy was taken 29 days after diagnosis. Genetic testing for high-risk APOL1 genotypes was not done. Nevertheless, it is common in Persons of African descent.

A case series discussed 5 cases of CG in native kidneys, 3 men and 2 women, all were of recent African ancestry. Their mean age was 57.4 yrs, the average days of discovery of renal affliction after COVID-19 was 8.2 days. [28]. Genetic testing performed in three patients confirmed high-risk APOL1 genotypes.

In our study, we had 5 cases presented with acute necrotizing glomerulonephritis, 4 M, 1 F, age was 38 (29.5–55).

Only 1 had severe COVID. They were presented mainly with AKI in 4 cases; presentation was after median (IQR, 25–75) of 30 (5.5–80) days. Only 1 case needed RRT at time of biopsy. A similar presentation in 2 pediatric cases, after 5 and 17 days respectively was reported, both needed RRT and steroids but renal recovery happened in one case; it is noticeable that it occurred later in our study [29].

In our study we had 8 cases presented with TMA, (5 F, 3 M), median age was 35 (25–44.5) years, and presentation was after 19 days (11–28); only 3 of them had severe COVID. 7/8 of them were presented with AKI, 4/8 needed RRT at time of biopsy, and 1 was associated with renal infarction. It is apparent the severity of this affliction.

In our study, there were 8 cases presented with lupus nephritis (7F, 1 M), only 5 of them had history of SLE before. All cases experienced mild to moderate COVID-19, renal affliction was after median of 60 days (35–64). None needed RRT. There were case reports of new onset SLE [30] and lupus nephritis, [31].

It was remarkable in our study, the large number of podocytopathies, 27 cases, apart from 2 collapsing GN mentioned above. Only 6/27 had severe COVID. We had 8 cases with MCD and 19 cases with FSGS. 17/27 had no history of medical illness. Presentation was after 50 (32–111) days. Proteinuria was the main presentation; median UPCR was 3 gm/ gm (2.4–5). These pathologies were

reported mainly after COVID-19 vaccine as mentioned above, and rarely after the disease itself [32].

We had also 10 cases of membranous nephropathy, median age was 38 (25–58), (7 M, 3F), only 2 had history of severe COVID. Presentation was late, after 97 days (46–114). 5/10 cases showed positive tissue staining for PLA2R. Proteinuria was the main presentation 4.93 gm/gm (2.81–12.95) with mildly reduced eGFR 72.17 (42.27–101.75), and none needed RRT; it was rarely reported after COVID-19 itself [33].

A possible autoimmune process could be involved in the latest 2 presentation as a diversity of other autoimmune diseases has been reported also [34, 35].

Conclusion

In this study, patterns of kidney injuries were reported from different centers or hospitals, after documented COVID-19, with exclusion of renal transplant patients and those who has been vaccinated. It was noted that beside early patterns related to sepsis, procoagulant effects or drugs given, such as ATI, ATIN or TMA, there were more patterns possibly related to immune dysregulation, such as IRGN, prolif/necr GN, LN, MGN or podocytopathies; they were mainly reported later, up to 4 month post infection.

Abbreviations

AKI	Acute kidney injury
AKI on CKD	Acute on top of chronic kidney disease
ATI	Acute tubular injury
ATIN	Acute tubulointerstitial nephritis
COVID-19	Coronavirus disease 2019
CG	Collapsing glomerulopathy
CTIN	Chronic tubulointerstitial nephritis
DM	Diabetes mellitus
eGFR	Estimated glomerular filtration rate
FSGS	Focal segmental glomerulosclerosis
HTN	Hypertension
HPF	High power field
IRGN	Infection-related glomerulonephritis
LCN	Light chain nephropathy
LN	Lupus nephritis
MCD	Minimal change disease
MGN	Membranous glomerulonephritis
NS	Nephrotic syndrome
NRP	Nephrotic range proteinuria
prolif/necr GN	Proliferative and necrotizing glomerulonephritis
RRT	Renal replacement therapy
SARS-CoV-2	Severe acute respiratory syndrome coronavirus 2
SLE	Systemic lupus erythematosus
SP	Subnephrotic proteinuria
TMA	Thrombotic microangiopathy
UPCR	Urine protein-creatinine ratio

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Not applicable.

Authors' contributions

EW made the design, literature search, data analysis, and manuscript preparation. RS reviewed the manuscript and and shared in the design. WI set the concept of the research and performed the histological examination of all kidney biopsies. All authors read and approved the final manuscript.

Availability of data and materials

The data that support the findings of this study are available on request from the corresponding author.

Declarations

Ethics approval and consent to participate

All procedures performed were in accordance with the ethical standards of the National Research Centre, Medical Research Ethics committee, Egypt, approval number 321012023, dated 5 Jan 2023.

Competing interests

Authors declare that they have no conflict of interest.

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