Analysis of Clinical and Pathological Features of Omicron Variants of SARS-CoV-2 associated Kidney Injury

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Abstract

Kidney injury is common in patients with Coronavirus Disease-19 (COVID-19). In this study, 49 patients with Omicron associated kidney injury were included, 38 of whomperformed renal biopsy. Patients were divided into 2 groups: Group A for patients developing kidney injury afterSARS-CoV-2 infection and Group B for patients with aggravated renal insufficiency after SARS-CoV-2 infection. The clinical, pathological and prognostic characteristicsof the patients and theirC3 levels were observed.In our center, the clinical diagnoses of patients with COVID-19 associated kidney injury were mainly acute kidney injury(AKI), chronic kidney disease(CKD) and nephrotic syndrome(NS); while the pathological diagnoses were mainly IgA nephropathy(IgAN)、focal segmental glomerulosclerosis(FSGS) and membranous nephritis(MN).80% of COVID-19 associated nephropathy (COVAN) patients had normal serum C3 complement level, and a few patients had increased or decreased C3 level. In renal tissue, C3 deposits were observed in 68.4% of patients.29% of patients experienced deterioration of renal function after treatment, but no patients developed to end-stage renal disease (ESRD). Among all of them, one case presenting with thrombotic microangiopathy (TMA) had a more severe renal pathological lesion and poorer prognosis. We observed differences of clinical and pathological features of patients with COVID-19associated kidney injury between races, regions and virus variants. Asian patients with Omicron associated kidney injury have milder kidney injury and a better renal prognosis.

Introduction:

Since December 2019, a new respiratory infectious disease 'COVID-19' has become a global epidemic. Kidney is one of the most frequently involved extrapulmonary organs. The renal injury caused by SARS CoV-2 is called COVID-19 associated nephropathy (COVAN), which is related to the poor prognosis of patients. Renal injury usually manifests as nephritis, recurrence or aggravation of kidney disease, transplant rejectionand acute kidney injury (AKI). The mortality rate of COVID-19 cases with AKI is significantly higher than those without kidney disease[1]. According to current reports, COVID-19 associated kidney injury has multiple pathological manifestations, including collapsed glomerulonephritis (CG), acute interstitial nephritis (ATI), focal segmental glomerulosclerosis(FSGS) and thrombotic microvascular disease (TMA). CG is reported to be the most characteristic manifestation after SARS-CoV-2 infection[2].

Since the first pandemic of wild-type SARS-CoV-2in 2019, the strains have gradually mutated into Alpha, Beta, gamma, Delta and other strains. By 2022, COVID-19 virus has been compiled into the omicron variant. There have been plenty of reports on kidney injury caused by the early pandemic delta strain, while at present, the reports on the clinical and pathological characteristics of kidney injury caused by omicron

infection are relatively lacking. Although a large number of studies on COVID-19 associated kidney injury have been reported abroad, the clinicopathological characteristics of COVID-19 associated kidney injury in China are different from those in foreign countries due to strain variation and ethnic differences. This study retrospectively included 49 COVID-19 associated kidney injury patients from the Second Xiangya Hospital of Central South University from December 2022 to March 2023, 38 of whomperformed kidney biopsy, and analyzed their clinical and pathological features to explore the characteristics and prognosis of COVID-19 associated kidney injury.

Methods:

This single center study included 49 cases of onset or aggravation of kidney injury associated with COVID-19 infection between December 2022 and March 2023 in Nephrology Institute of the Second Xiangya Hospital ofCentral South University. 38 of them performed kidney biopsy.Inclusion criteria included cases with a confirmed COVID-19 infection by RNA PCR or antigen detection within one week before or after kidney injury. Patients were divided into 2 groups: Group A for patients with onset of kidney injury after SARS CoV-2 infection; Group B for patients with pre-existing kidney disease who experienced aggravation of renal insufficiency after SARS-CoV-2 infection.

Kidney biopsy sections were stained with hematoxylin and eosin(H&E), Periodic acid-Schiff(PAS),Masson-Trichrome, Periodic-acid silver methenamine (PASM), and Congo Red and examined by light microscopy. Immunofluorescence(IF) staining was performed for IgG, IgA, IgM, C3, C1q, Kappa light chain and Lambda light chain.All cases were diagnosed by nephropathologist.

Results:

Clinical features

In group A, acute renal injury was the most common manifestation (35%), followed by nephrotic syndrome (27%), characterized by macroalbuminuria, hypoproteinemia, and edema, and also included simple hematuria and/or microalbuminuria (19%), chronic nephritis (15%)and lupus nephritis (4%). (Figure 1A.) Most patients in group B presented with CKD(55%) and NS(40%); Only one patient presented with lupus nephritis(LN) (5%).(Figure 1B.)

Pathological features

A total of 38 patients completed kidney biopsy, including 18 patients in group A and 20 patients in group B. IgAN(39%) accounted for the largest proportion of pathological types in Group A, followed by MN(17%), FSGS(11%) and Endocapillary proliferative glomerulonephritis(EPGN) (11%). (Figure 2A.) Among 20 patients in Group B, the most common renal pathologic type was IgAN(45%), followed by FSGS(25%) and MN (15%).(Figure 2B)(Figure 3.)

Serum C3 and the deposition of C3in kidney

The serum C3 level of 25 patients with COVID-19 associated kidney injury was measured. 80% of them had normal C3 level. Among 38 patients with COVID-19 associated kidney injury who performed kidney biopsy, 31.6% presented with C3 (-), 7.9% presented with C3 (+), 28.9% presented with C3 (++), and 31.6% presented with C3(+++).(Figure 3.)

Prognosis

Follow-up visits were conducted in 13 patients of Group A and 14 patients of Group B.Among the 13 patients in group A, 7 patients had partial remission of proteinuria, 5 patients had no remission, and 1 patient had increased proteinuria. Hematuria was partially remission in 5 patients, stable in 4 patients, and increased in 4 patients. Four patients had stable creatinine, three had increased creatinine, and one had decreased creatinine.Among 14 patients in group B, 6 patients had partial remission of proteinuria, 6 patients had no remission of proteinuria, and 1 patient had remission of proteinuria. Hematuria was partially alleviated in

6 patients, increased in 4 patients, and stable in 4 patients. Five patients had reduced creatinine and four had stable creatinine.(Table 1.)

Discussion:

In this study, 46 patients who developed kidney injury after COVID-19 infection or had an aggravation of the original kidney injury were included.The patients with new-onset renal damage mainly present with AKI and NS, while the patients withaggravated renal damage mainly presents with CKD and NS. 20% of the patients presented with impaired renal function.A previous international meta-analysis involving 49,048 patients found that the incidence of AKI among hospitalized patients with COVID-19 in the United States and Europe was as high as 28.6%, while the incidence in Asia was 5.5%[3]. In our study, 19.6% of patients developed AKI, with a significantly lower incidence and milder clinical symptoms. The reason might be that compared with the Delta variant in the pandemic waves before 2022, the Omicron variantis more transmissible but lessintrinsic virulent[4].

Among 38 patients with renal biopsy in this study, IgAN was the most common pathological type, other pathological diagnoses included MN, FSGS, acute interstitial nephritis(AIN), mesangial proliferative glomerulonephritis(MsGPN), EPGN, henoch-schonlein purpura nephritis(HSPN), TMA, and LN.Currently, GC has been reported as the most common pathological manifestation of COVID-19 associated nephropathy, which is mostly seen in patients with African ancestry and is associated with a high-risk APOL1 genotype[5]. The differences of pathological type of COVID-19 associated kidney injury may be related to race, region and strain variation. Compared with European and American populations, the frequency and percentage of population attributable risk (PAR%)of the risk alleles of IgAN-susceptible genes were significantly higher in the Chinese population[6]. Compared with the database of kidney biopsies over a 10-year period prior to the pandemic in our center, IgAN accounted for a higher proportion of COVAN patients. Ma Y et al. speculated that SARS-CoV-2 invaded respiratory tract and intestinal mucosa through the encoded S protein, activated antigen presenting cells, stimulated B cells to transform into Gd-IgA1λB cells and secreted Gd-IGa1through T-cell dependent and independent pathways, and then formed immune complexes deposited in the mesangium of the glomeruli, ultimately leading to the occurrence of IgAN[7]. Among other pathological types, a patient diagnosed with TMAhada more severe renal pathological lesion and poorer prognosis.

80% of patients with COVID-19 associated kidney injury had normal serum C3 complement level, a few patients had increased or decreased C3 level. In renal tissues, C3 deposits were observed in 68.4% of patients. Some scholars believe that low serum C3 level indicates overactivation and depletion of the complement system, which is related to the severity of the disease and poor prognosis[8-10]. It is currently believed that complement is involved in the indirect mechanism of COVID-19 kidney injury: SARS-CoV-2 binds to endothelial angiotensin converting enzyme 2 (ACE2), activates the lectin pathway and classical pathway of complement, recruits neutrophils and monocytes/macrophages, releases proteases,cytokinesand reactive oxygen species, leads to inflammation, subcutaneous matrix destructionand vasculitis-like lesions[11].

Among the 17 patients of follow-up, 29% of them experienced deterioration of renal function after treatment, but no patients developed to ESRD. However, in a cohort study of AKI in hospitalized patients with COVID-19 in 2020, 46% of hospitalized patients experienced AKI, 19% of the AKI patients entered dialysis and 50% died[12]. At present, there are few international reports on the prognosis of patients with Omicron infection. The possible reasons for the favorable prognosis of patients in this study are as follows: Firstly, international guidelines for treatment of SARS-CoV-2 infection have been developed. Antiviral drugs such as Nematovir, Azfudine and Monoravir as well as neutralizing antibody drugs such as ambavir/Romisivirwere available for targeted antiviral therapy. Early detection and treatment can be achieved in most patients. Secondly, the involved patients were mainly infected with Omicron variant. Compared to the initial strain reported internationally, Omicron variant was less virulent and less likely to cause severe cases.

Limitations:

There are some limitations to this study. Due to the short follow-up period, the long-term prognosis of patients could not be assessed. Besides, this study failed to find direct evidence of SARS-CoV-2 infec-

tion in the patients' renal biopsy specimens. In previous studies, SARS-CoV-2 was detected by electron microscopy (TEM)[13], immunohistochemistry (IHC)[14], RNA in situ hybridization (RNA-ISH)[15] and reverse transcription-polymerase chain reaction(RT-PCR)[16], but the above positive results were all from autopsy. SARS-CoV-2 has rarely been detected in renal biopsy specimens. The kidney injury associated with SARS-CoV-2 infection in non-critical patients may mainly be caused through indirect mechanisms.

Conclusion:

In conclusion, we present a series of native kidney biopsy in association with the COVID-19 pandemic. Of patients in our center, the main clinical manifestations were AKI、CKD and NS, while the main pathological manifestation were IgAN、FSGS and MN. Generally, patients in our center had a favorable prognosis. We observed that the clinical and pathological features of COVID-19 associated with kidney injury vary by race、region and virus variant.Asian patients with the Omicron infection have milder kidney injury and a better prognosis.

Abbreviations

COVID-19: Coronavirus Disease-19; COVAN: COVID-19 associated nephropathy ; AKI: acute kidney injury; CKD: chronic kidney disease; ESRD: end-stage renal disease; NS: nephrotic syndrome; IgAN: IgA nephropathy; FSGS: focal segmental glomerulosclerosis; MN: membranous nephritis;TMA: thrombotic microangiopathy ; CG: collapsed glomerulonephritis ;LN: lupus nephritis; ATI: acute interstitial nephritis; EPGN: endocapillary proliferative glomerulonephritis; TMA: thrombotic microvascular disease; AIN: acute interstitial nephritis; MsGPN: mesangial proliferative glomerulonephritis; HSPN: henoch-schonlein purpura nephritis; H&E: hematoxylin and eosin; PAS: Periodic acid-Schiff; PASM: Periodic-acid silver methenamine; IF: Immunofluorescence; TEM: electron microscopy; IHC: immunohistochemistry; RNA-ISH: RNA in situ hybridization; RT-PCR: reverse transcription-polymerase chain reaction

Author's contributions

Data curation, formal analysis, investigation, methodology, writing: ZWQ. Conception or design: XJZ. Acquisition, analysis, or interpretation of data: JYW, SYX, YCH, ZL.Provided guidance: YFL, SGY, LS, HL. All authors contributed tomanuscript revision and read and approved the submitted version.

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Declaration of competing interest

No potential conflict of interest relevant to this article was reported.

Consent for publication

All patients signed approved informed consent forms.

Data availability statement

Not applicable.

Ethics approval and consent to participate

This study has passed the ethical review of the Second Xiangya Hospital (LYF2020148).

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Figure legends:

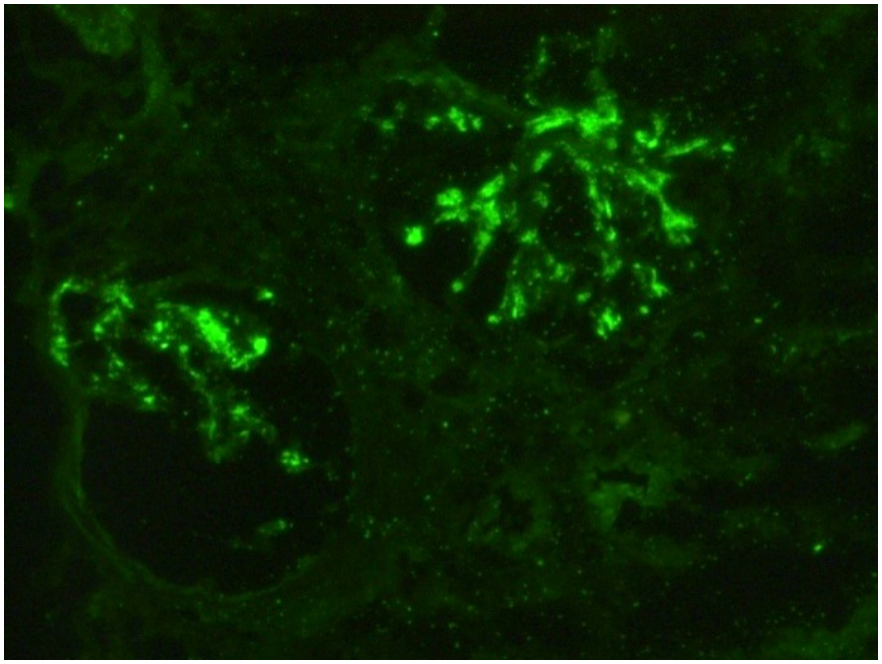
Figure 1: Clinical characteristics of COVID-19 associated kidney injury. (A)Patients developing kidney injury after SARS CoV-2 infection; (B)Patients with aggravated renal insufficiency after SARS CoV-2 infection.

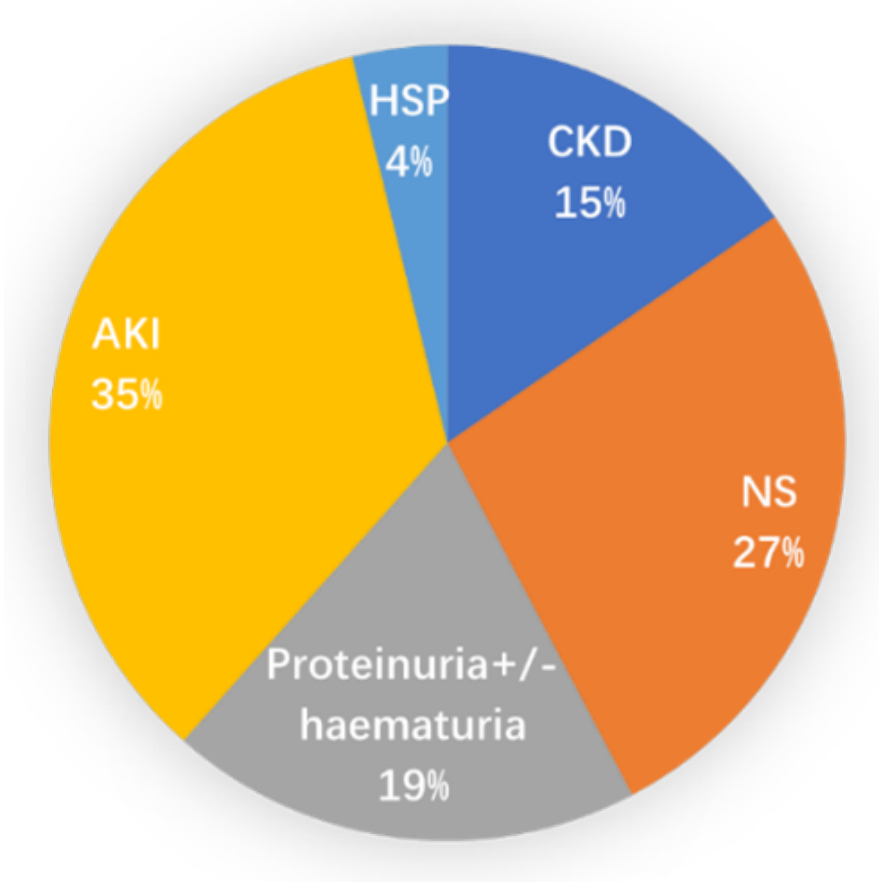
Figure 2: Pathological features of COVID-19 associated kidney injury. (A)Patients developing kidney injury after SARS CoV-2 infection; (B)Patients with aggravated renal insufficiency after SARS CoV-2 infection.

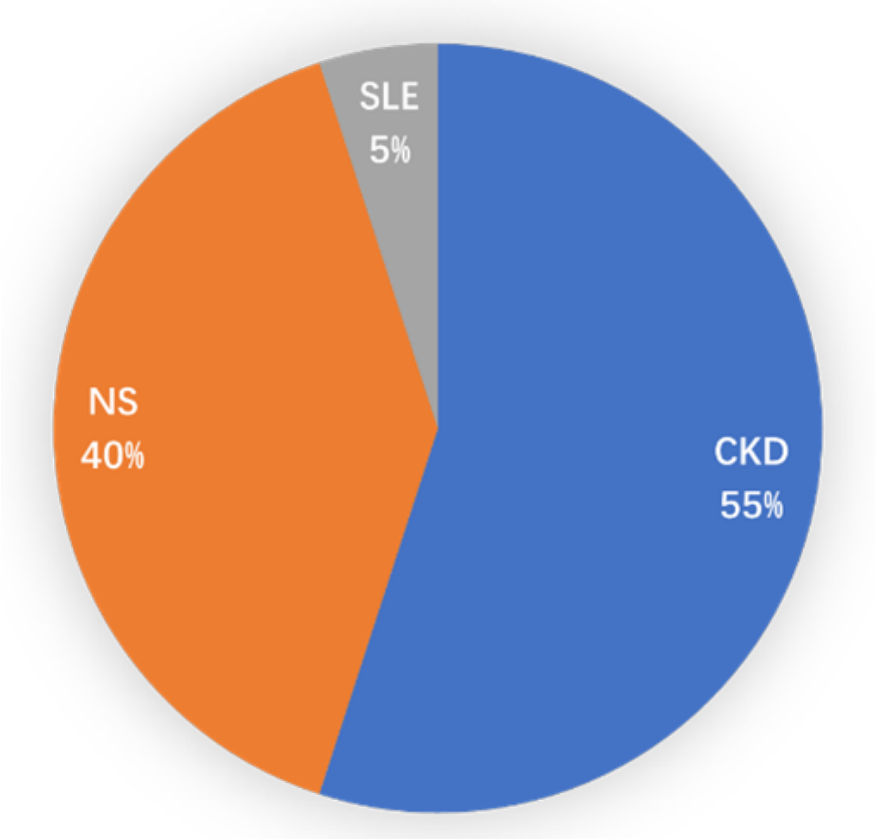
Figure 3: Renal Pathological changes in patients with COVID-19 associated kidney injury: (A) Patient with IgAN shows IgA+++ depositing in glomerulas (IF ×100); (B) Patient with IgAN shows segmental sclerosis and mild mesangial proliferation (PAS×200); (C): Patient with interstitial nephritis shows Interstitial inflammatory cell infiltration (HE ×200); (D) Patient with TMA shows arteriole intima edema and myxoid degeneration (HE ×200).

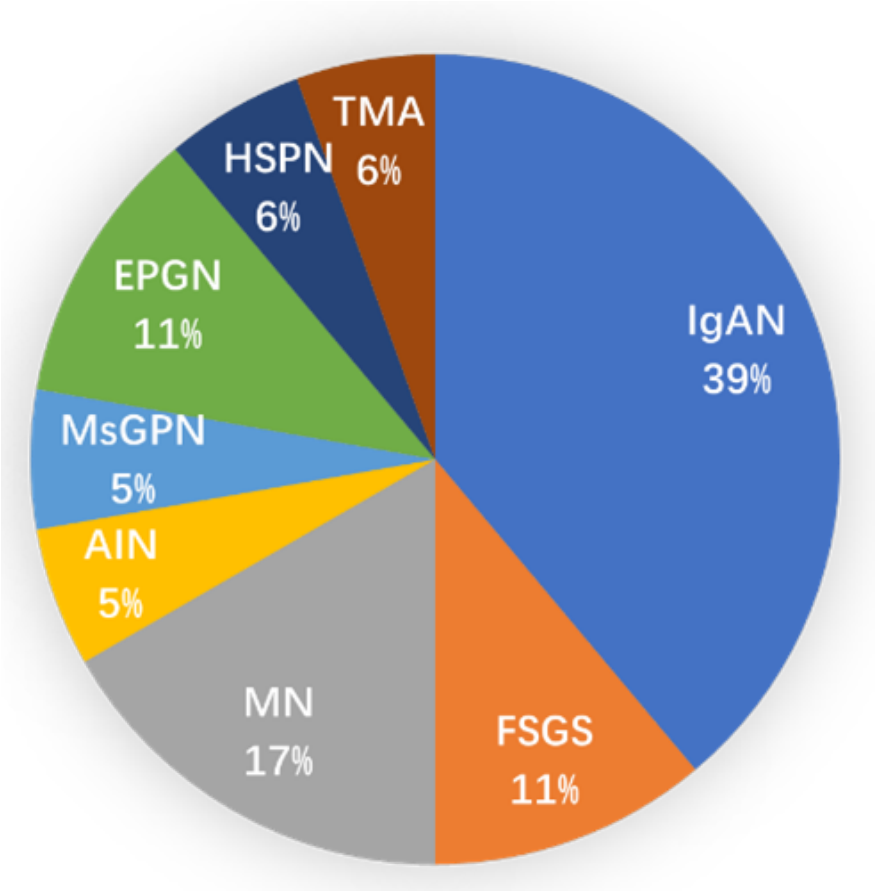
Figure4: Serum C3 and deposition of C3 in patients with COVID-19 associated kidney disease. (A) Serum C3 levels: normal: 0.70˜1.40g/L; increase: >1.40g/L; decrease: <0.70g/L**;** (B) Intensity of C3 deposition by Immunofluorescence staining in renal tissue.

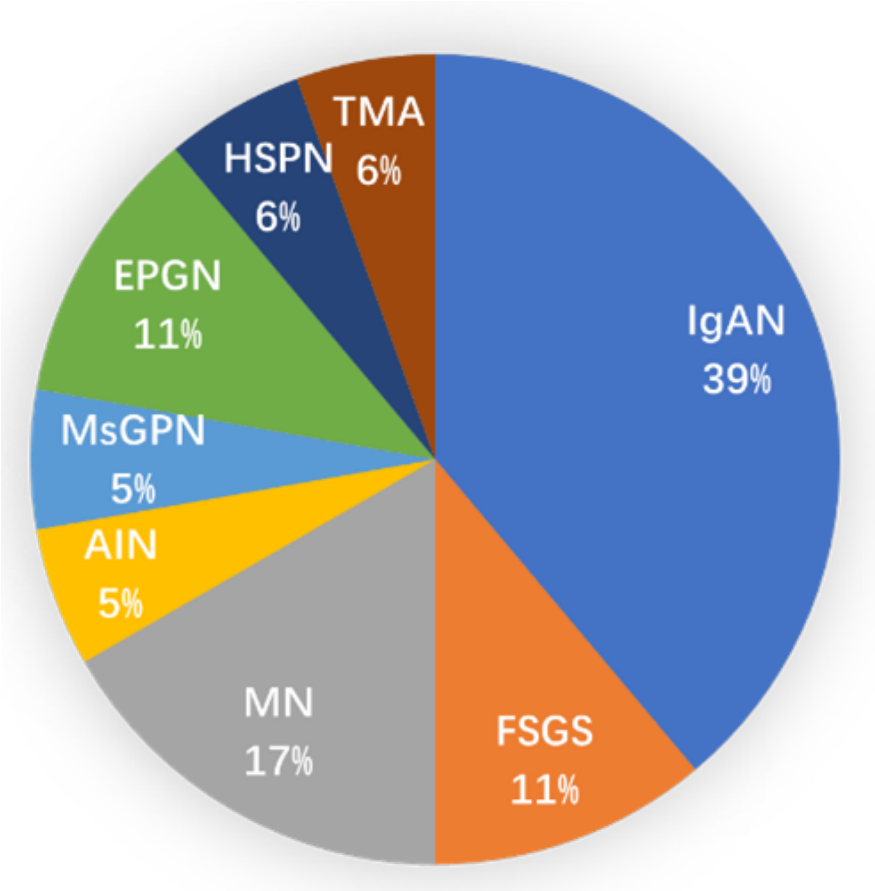
Table 1: (A) Renal Pathology Findings and Clinical Presentation of Patients in Group A; (B) Renal Pathology Findings and Clinical Presentation of Patients in Group B.

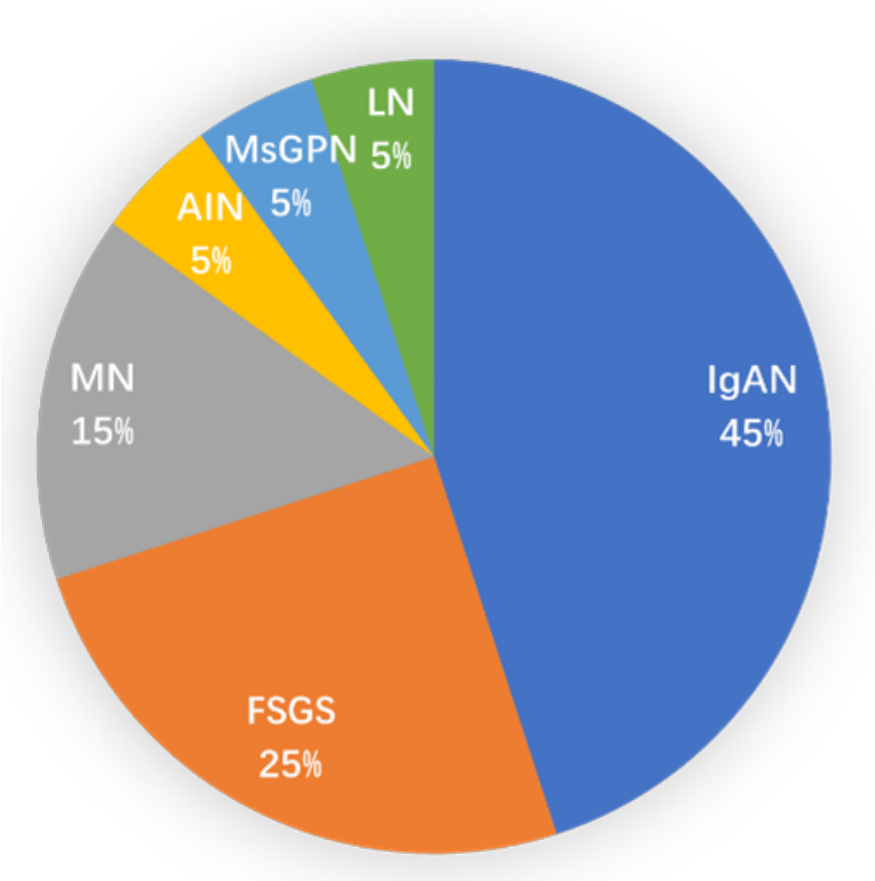


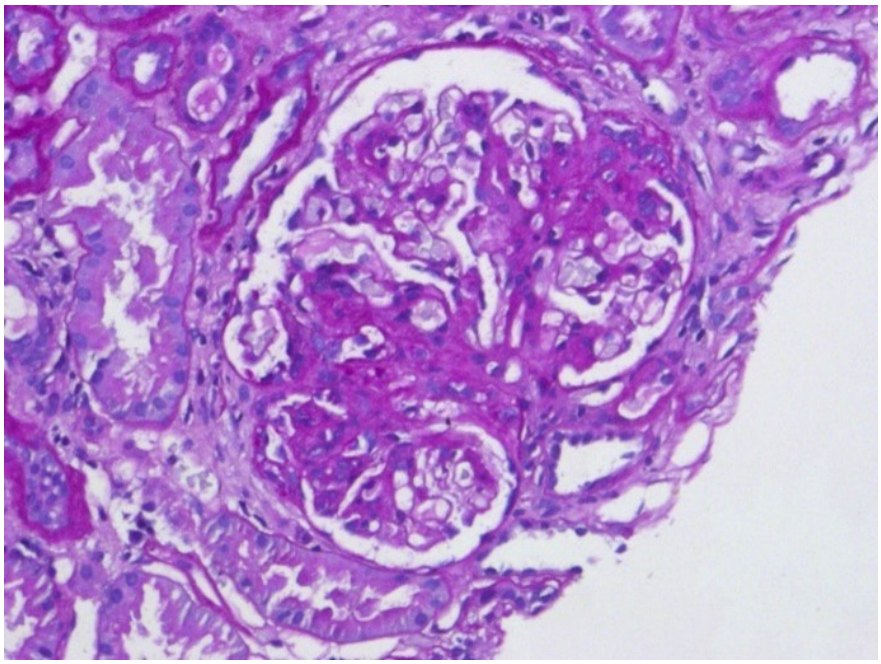


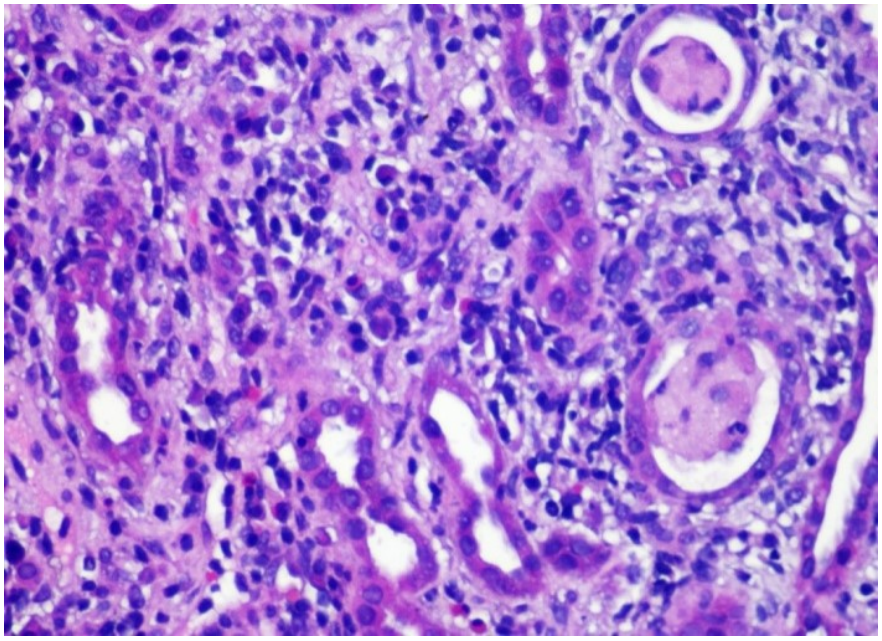


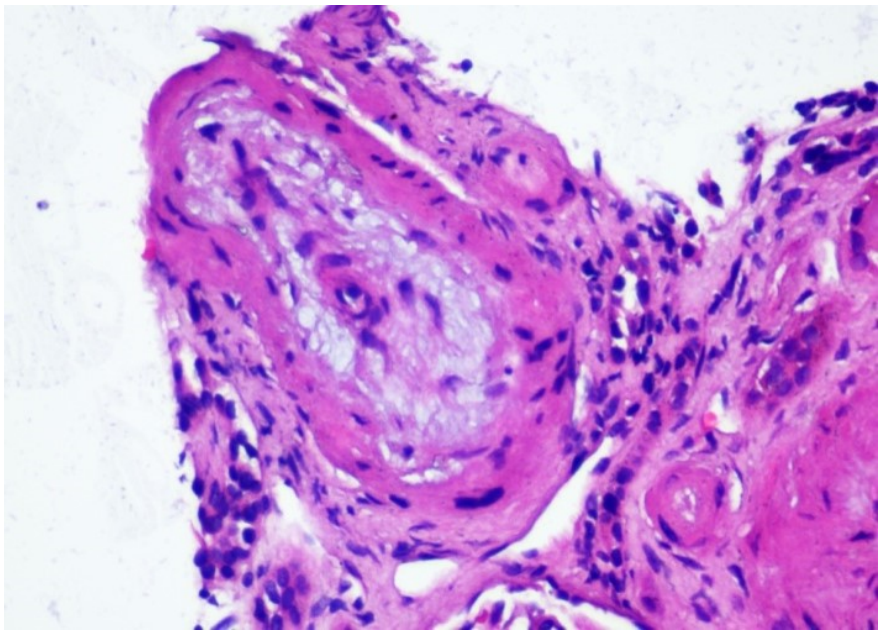


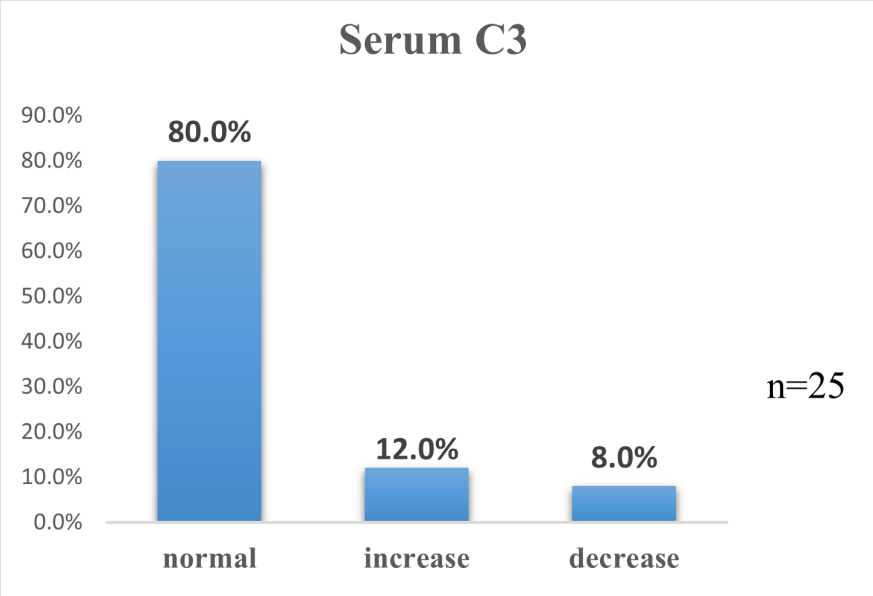


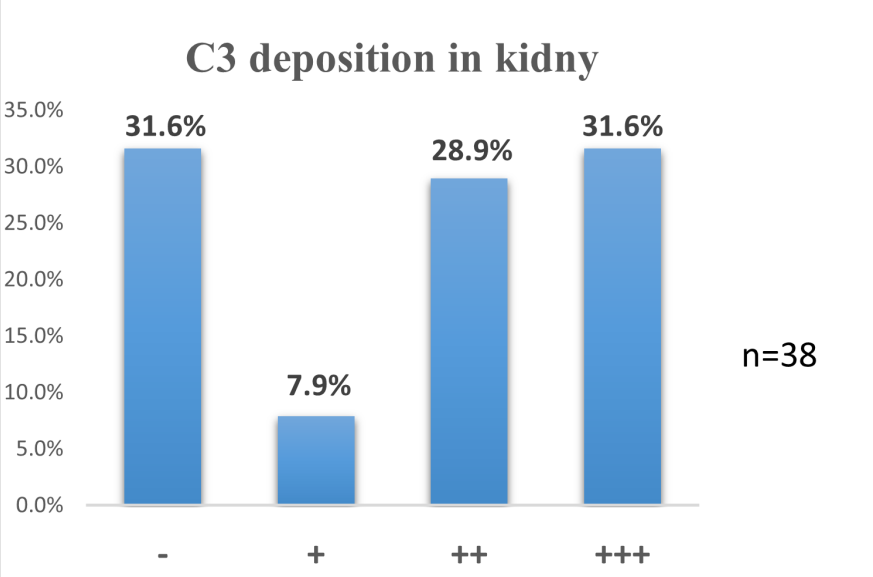












Case	Pathology	Age	Sex	Kidney presentation	Scr（umol/L）	Proteinuria（mg/d）	SN-RBC	Alb（g/L）	Hb（g/L）	C3（g/L）	C4（g/L）	Outcome				
												Proteinuria	Hematuria	Scr	C3	C4
1	IgAN	28	F	CKD	78.0	677.55	725000	46.7	117	1.25	0.34	PR	PR	Stable	-	-
2	IgAN	56	M	CKD	154.0	2842.85	< 8000	17.6	143	-	-	PR	Increase	Increased	-	-
3	IgAN	42	F	CKD	182.3	1023.00	72000	39.8	92	-	-	PR	PR	Increased	-	-
4	IgAN	35	F	Proteinuria	48.0	3.20	9000	39.4	131	0.89	0.21	-	-	-	-	-
5	IgAN	36	F	NS	55.7	-	108000	38.0	130	1.19	0.41	-	-	-	-	-
6	IgAN	37	M	NS	270.8	9354.00	9000	38.0	156	1.16	0.24	NR	Stable	Decreased	-	-
7	IgAN	41	F	CKD	66.0	642.90	45000	45.4	122	1.24	0.25	PR	Increased	-	-	-
8	MN	33	F	Hematuria + Proteinuria	66.8	2829.00	275000	15.4	134	1.13	0.3	-	-	-	-	-
9	MN	46	M	NS	83.8	12456.80	326000	20.2	160	-	-	NR	Stable	Stable	-	-
10	MN	60	F	NS	72.0	12059.60	105000	30.6	125	1.46	0.51	-	-	-	-	-
11	FSGS	18	M	NS	82.0	25568.00	10000	20.5	165	1.16	0.33	-	-	-	-	-
12	FSGS	17	M	NS	75.0	4776.50	< 8000	21.1	176	-	-	NR	Stable	Stable	-	-
13	EPGN	47	F	Hematuria + Proteinuria	45.7	272.30	300000	26.0	118	2.59	0.63	PR	PR	-	2.14	0.62
14	EPGN	18	M	Proteinuria	55.0	-	189000	24.9	121	0.28	0.18	NR	PR	Increased	1.03	0.21
15	HSPN	59	M	HSP	84.0	2084.80	45000	35.1	147	1.06	0.26	NR	Increased	-	-	-
16	MsPGN	44	F	NS	72.0	-	1800000	30.2	122	1.15	0.2	Increased	PR	-	-	-
17	TMA	39	M	AKI	306.9	457.30	< 8000	40.5	124	1.06	0.28	PR	Increased	Increased	-	-
18	AIN	20	M	Proteinuria	141.9	-	<8000	37.8	102	1.80	0.41	PR	stable	Stable	1.70	0.41

Case	Pathology	Age	Sex	Kidney presentation	Scr（umol/L）	Proteinuria（mg/d）	SN-RBC	Alb（g/L）	Hb（g/L）	C3（g/L）	C4（g/L）	Outcome				
												Proteinuria	Hematuria	Scr	C3	C4
1	IgAN	34	M	CKD	44.5	908.30	45000	45.3	155	1.21	0.29	PR	Increased	-	-	-
2	IgAN	37	F	CKD	82.0	6254.40	15000	37.6	125	-	-	PR	Increased	-	-	-
3	IgAN	29	F	CKD	113.0	555.00	18000	42.8	110	-	-	NR	Stable	Stable	-	-
4	IgAN	24	M	CKD	89.0	1017.00	225000	37.6	139	-	-	NR	PR	-	-	-
5	IgAN	38	M	CKD	69.0	-	826000	38.4	136	-	-	PR	PR	Stable	-	-
6	IgAN	49	M	CKD	309.9	2360.50	27000	227.5	112	0.99	0.20	NR	Increased	Decreased	-	-
7	IgAN	30	M	CKD	127.0	343.30	306000	43.7	136	1.06	0.18	-	-	-	-	-
8	IgAN	63	F	NS	254.2	6408.00	65000	17.9	88	0.95	0.21	PR	PR	Decreased	-	-
9	IgAN	42	F	CKD	69.2	364.80	125000	41.8	122	1.02	0.26	PR	PR	Stable	-	-
10	FSGS	16	M	NS	85.8	6717.81	< 8000	17.6	143	-	-	-	-	-	-	-
11	FSGS	27	F	CKD	71.0	727.90	225000	41.8	1120	1.08	0.29	-	-	-	-	-
12	FSGS	29	M	NS	-	2577.90	< 8000	26.3	137	1.07	0.23	R	Stable	-	-	-
13	FSGS	50	M	NS	69.6	4614.00	32000	17.0	125	-	-	-	-	-	-	-
14	FSGS	51	F	CKD	155.0	-	495000	41.2	128	1.18	0.21	-	PR	Stable	-	-
15	MN	60	M	NS	219.0	4773.61	< 8000	20.5	65	-	-	NR	Increased	Decreased	-	-
16	MN	26	M	NS	186.0	9498.00	< 8000	27.9	92	0.80	0.20	NR	Stable	Decreased	-	-
17	MN	50	M	NS	62.0	3274.10	315000	27.2	126	-	-	-	-	-	-	-
18	AIN	65	M	CKD	248.0	511.00	< 8000	41.5	117	0.54	0.03	PR	Stable	Decreased	0.79	0.25
19	MsPGN	53	M	NS	608	7148.2	-	18	108	1.25	0.54	-	-	-	-	-
20	LN	28	M	SLE	80.0	779.00	90000	36.0	151	-	-	NR	PR	-	-	-

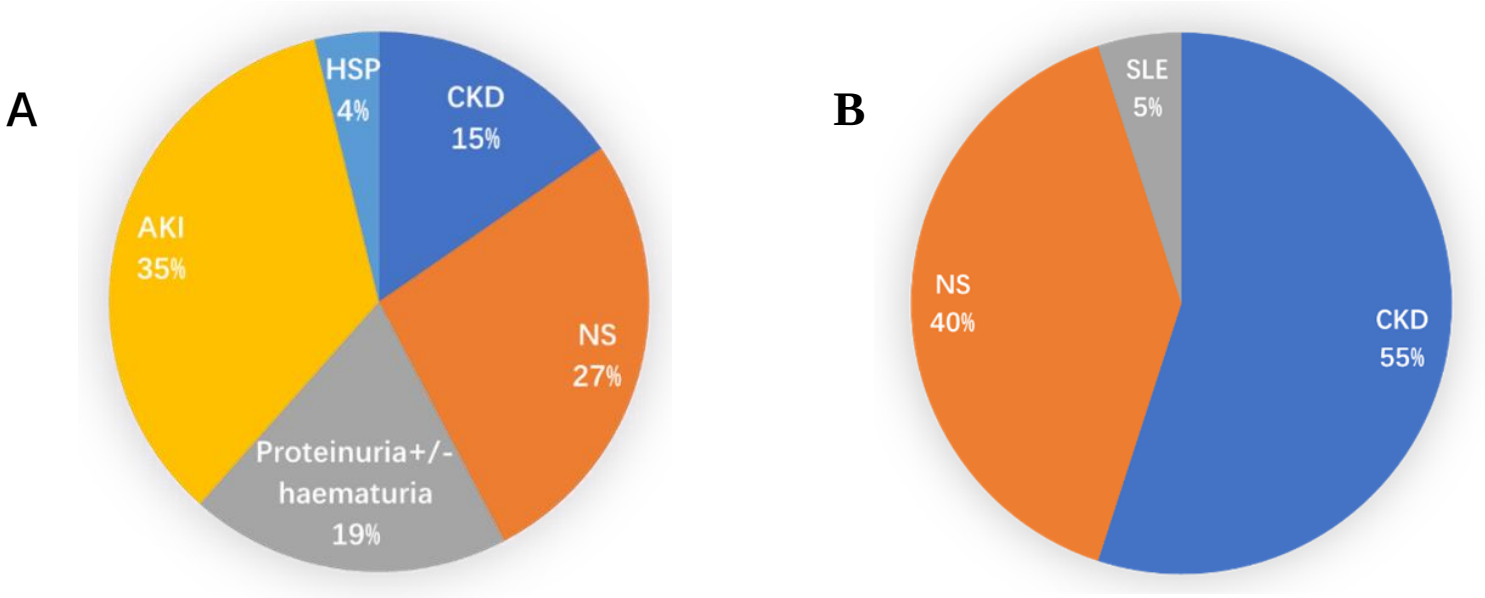


Figure 1: Clinical characteristics of COVID-19 associated kidney injury.
(A)Patients developing kidney injury after SARS CoV-2 infection;
(B)Patients with aggravated renal insufficiency after SARS CoV-2 infection.

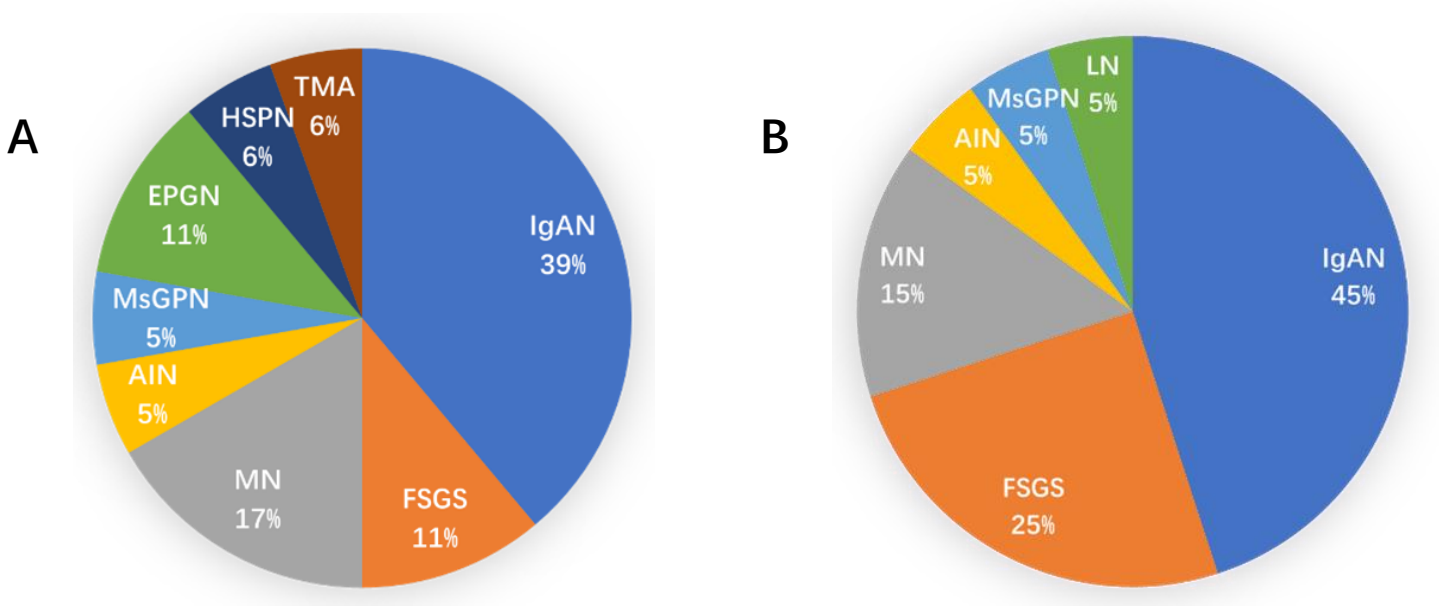


Figure 2: Pathological features of COVID-19 associated kidney injury
(A)patients developing kidney injury after SARS CoV-2 infection
(B)patients with aggravated renal insufficiency after SARS CoV-2 infection

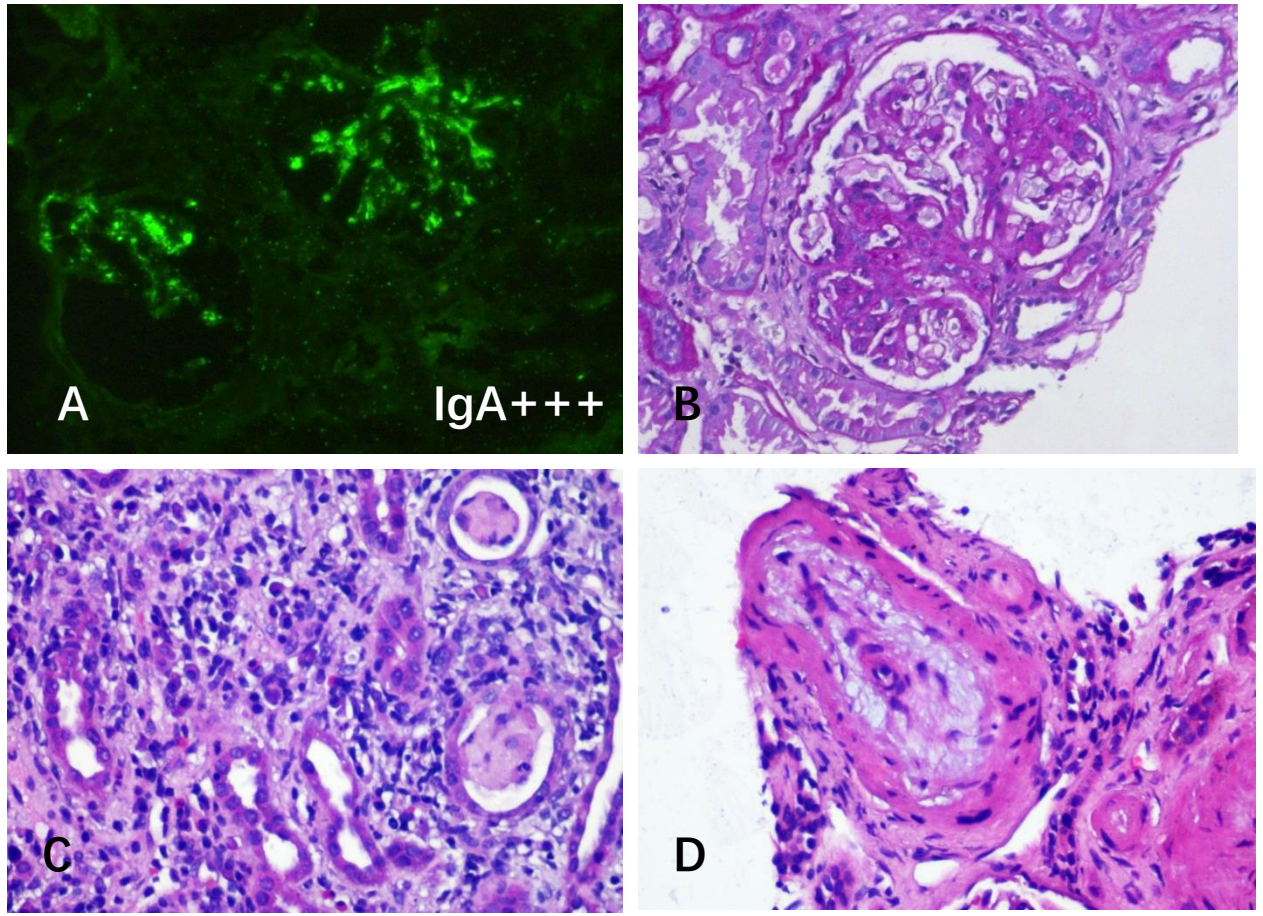
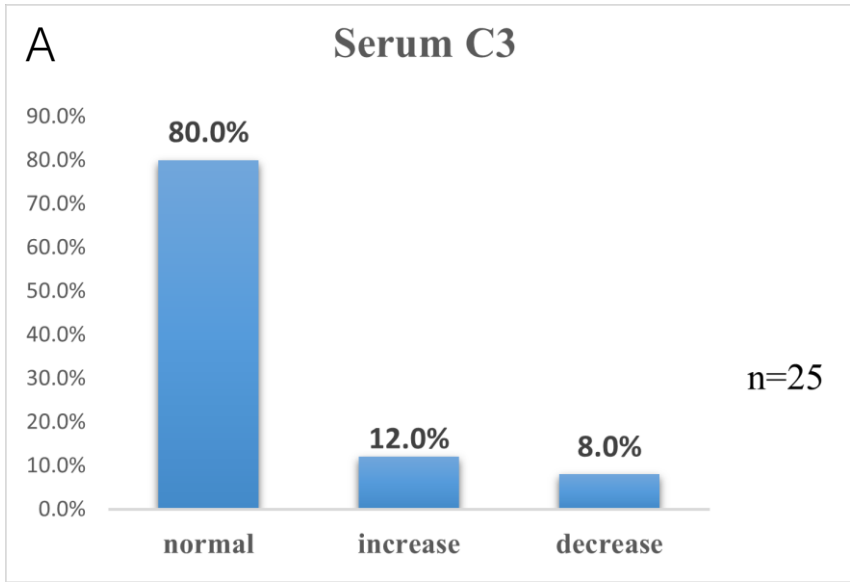


Figure 3:Renal Pathological changes in patients with COVID-19 associated kidney injury
(A)Patient with IgAN shows IgA+++ deposite in glomerulas (IF ×100),
(B)Patient with IgAN shows segmetal sclerosis and mild mesangial proliferation (PAS×200),
(C)Patient with interstitial nephritis shows Interstitial inflammatory cell infiltration (HE ×200),
(D)Patient with TMA shows arteriole intima edema and myxoid degeneration (HE ×200).



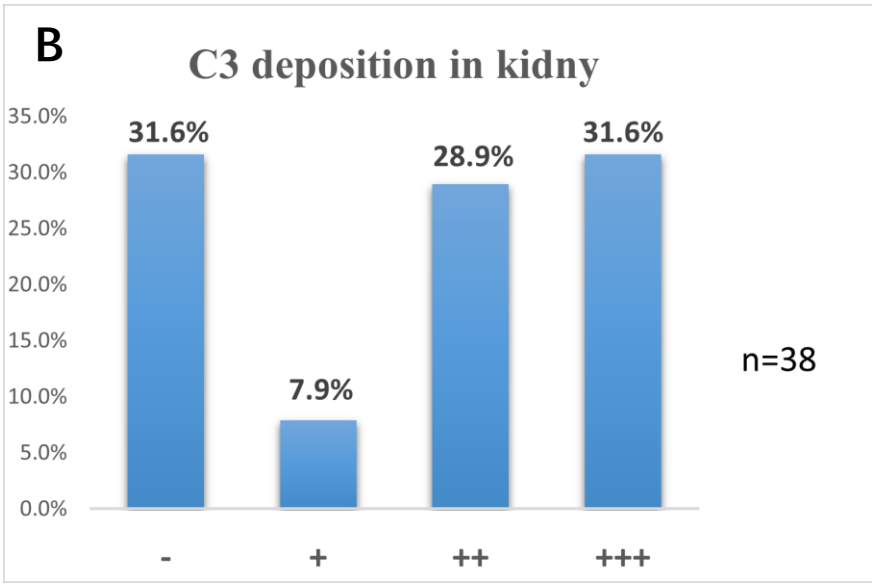


Figure 4. Serum C3 and deposition of C3 in patients with COVID-19 associated kidney disease.
(A) Serum C3 levels: normal: 0.70~1.40g/L; increase: >1.40g/L; decrease: <0.70g/L**;**
(B) Intensity of C3 deposition by Immunofluorescence staining in renal tissue.

Table 1A: Kidney Pathology Findings and Clinical Presentation of Patients in Group A

Case	Pathology	Age	Sex	Kidney presentation	Scr (umol/L)	Proteinuria (mg/d)	SN-RBC	Alb (g/L)	Hb (g/L)	C3 (g/L)	C4 (g/L)	Outcome				
												Proteinuria	Hematuria	Scr	C3	C4
1	IgAN	28	F	CKD	78.0	677.55	725000	46.7	117	1.25	0.34	PR	PR	Stable	-	-
2	IgAN	56	M	CKD	154.0	2842.85	< 8000	17.6	143	-	-	PR	Increase	Increased	-	-
3	IgAN	42	F	CKD	182.3	1023.00	72000	39.8	92	-	-	PR	PR	Increased	-	-
4	IgAN	35	F	Proteinuria	48.0	3.20	9000	39.4	131	0.89	0.21	-	-	-	-	-
5	IgAN	36	F	NS	55.7	-	108000	38.0	130	1.19	0.41	-	-	-	-	-
6	IgAN	37	M	NS	270.8	9354.00	9000	38.0	156	1.16	0.24	NR	Stable	Decreased	-	-
7	IgAN	41	F	CKD	66.0	642.90	45000	45.4	122	1.24	0.25	PR	Increased	-	-	-
8	MN	33	F	Hematuria + Proteinuria	66.8	2829.00	275000	15.4	134	1.13	0.3	-	-	-	-	-
9	MN	46	M	NS	83.8	12456.80	326000	20.2	160	-	-	NR	Stable	Stable	-	-
10	MN	60	F	NS	72.0	12059.60	105000	30.6	125	1.46	0.51	-	-	-	-	-
11	FSGS	18	M	NS	82.0	25568.00	10000	20.5	165	1.16	0.33	-	-	-	-	-
12	FSGS	17	M	NS	75.0	4776.50	< 8000	21.1	176	-	-	NR	Stable	Stable	-	-
13	EPGN	47	F	Hematuria + Proteinuria	45.7	272.30	300000	26.0	118	2.59	0.63	PR	PR	-	2.14	0.62
14	EPGN	18	M	Proteinuria	55.0	-	189000	24.9	121	0.28	0.18	NR	PR	Increased	1.03	0.21
15	HSPN	59	M	HSP	84.0	2084.80	45000	35.1	147	1.06	0.26	NR	Increased	-	-	-
16	MsPGN	44	F	NS	72.0	-	1800000	30.2	122	1.15	0.2	Increased	PR	-	-	-
17	TMA	39	M	AKI	306.9	457.30	< 8000	40.5	124	1.06	0.28	PR	Increased	Increased	-	-
18	AIN	20	M	Proteinuria	141.9	-	<8000	37.8	102	1.80	0.41	PR	stable	Stable	1.70	0.41

PR: Partial Remission; NR: No Remission; R: Remission

Table 1B: Kidney Pathology Findings and Clinical Presentation of Patients in Group B

Case	Pathology	Age	Sex	Kidney presentation	Scr (umol/L)	Proteinuria (mg/d)	SN-RBC	Alb (g/L)	Hb (g/L)	C3 (g/L)	C4 (g/L)	Outcome				
												Proteinuria	Hematuria	Scr	C3	C4
1	IgAN	34	M	CKD	44.5	908.30	45000	45.3	155	1.21	0.29	PR	Increased	-	-	-
2	IgAN	37	F	CKD	82.0	6254.40	15000	37.6	125	-	-	PR	Increased	-	-	-
3	IgAN	29	F	CKD	113.0	555.00	18000	42.8	110	-	-	NR	Stable	Stable	-	-
4	IgAN	24	M	CKD	89.0	1017.00	225000	37.6	139	-	-	NR	PR	-	-	-
5	IgAN	38	M	CKD	69.0	-	826000	38.4	136	-	-	PR	PR	Stable	-	-
6	IgAN	49	M	CKD	309.9	2360.50	27000	227.5	112	0.99	0.20	NR	Increased	Decreased	-	-
7	IgAN	30	M	CKD	127.0	343.30	306000	43.7	136	1.06	0.18	-	-	-	-	-
8	IgAN	63	F	NS	254.2	6408.00	65000	17.9	88	0.95	0.21	PR	PR	Decreased	-	-
9	IgAN	42	F	CKD	69.2	364.80	125000	41.8	122	1.02	0.26	PR	PR	Stable	-	-
10	FSGS	16	M	NS	85.8	6717.81	< 8000	17.6	143	-	-	-	-	-	-	-
11	FSGS	27	F	CKD	71.0	727.90	225000	41.8	1120	1.08	0.29	-	-	-	-	-
12	FSGS	29	M	NS	-	2577.90	< 8000	26.3	137	1.07	0.23	R	Stable	-	-	-
13	FSGS	50	M	NS	69.6	4614.00	32000	17.0	125	-	-	-	-	-	-	-
14	FSGS	51	F	CKD	155.0	-	495000	41.2	128	1.18	0.21	-	PR	Stable	-	-
15	MN	60	M	NS	219.0	4773.61	< 8000	20.5	65	-	-	NR	Increased	Decreased	-	-
16	MN	26	M	NS	186.0	9498.00	< 8000	27.9	92	0.80	0.20	NR	Stable	Decreased	-	-
17	MN	50	M	NS	62.0	3274.10	315000	27.2	126	-	-	-	-	-	-	-
18	AIN	65	M	CKD	248.0	511.00	< 8000	41.5	117	0.54	0.03	PR	Stable	Decreased	0.79	0.25
19	MsPGN	53	M	NS	608	7148.2	-	18	108	1.25	0.54	-	-	-	-	-
20	LN	28	M	SLE	80.0	779.00	90000	36.0	151	-	-	NR	PR	-	-	-