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Complement System is Overactivated in Patients with IgA Nephropathy after COVID-19

Weiyi Guo, Guoqin Wang, Lingqiang Kong, Lijun Sun, Xiaoyi Xu, Wenrong Cheng, Hongrui Dong, Hong Cheng
[1]Department of Department of Nephrology, Beijing Anzhen Hospital, Capital Medical University, China

Objectives : Since the coronavirus disease of 2019 (COVID-19) pandemic swept across the world, the flare of immune-mediated disease following SARS-CoV-2 infection has emerged rapidly. IgA nephropathy (IgAN), which has been confirmed as a complement mediated autoimmune disease, is also one of the common glomerulonephritis associated with COVID-19. Here, we aim to investigate the clinical and immunological characteristics of patients with IgAN after COVID-19.
Methods : 33 patients with renal biopsy-proven IgAN (Group CoV) who had experienced COVID-19 before renal biopsy were enrolled. Meanwhile, 44 patients with IgAN (Group non-CoV) without COVID-19 were enrolled as control. Complement proteins and Gd-IgA1 were detected by enzyme-linked immunosorbent assay and immunofluorescence. SARS-CoV-2 nucleocapsidin was detected by immunohistochemistry. Clinicopathological and immunological features between the two groups were analyzed.
Results : Compared with Group non-CoV, the level of eGFR was significantly lower in Group CoV ($p=0.010$). Histologically, compared with Group non-CoV, Group CoV presented with more percentages of segmental glomerulosclerosis/adhesion ($p=0.003$) and less percentages of mesangial hypercellularity ($p=0.017$). The level of plasma level of C5a ($p<0.001$), soluble C5b-9 ($p=0.018$), FHR5 ($p<0.001$) were all significantly higher in Group CoV compared with Group non-CoV, respectively. Compared with Group non-CoV, the intensity of glomerular MAC deposition was much stronger in Group CoV ($p=0.246$). There were no significantly different in serum levels of IgA, Gd-IgA1, IgA immune complex between two Groups. However, a greater intensity of Gd-IgA1 deposition in glomerular mesangial and capillary areas has been exhibited in Group CoV than those in Group non-CoV ($p=0.005$).
Conclusions : For IgAN after COVID-19, mucosal immune responses to SARS-CoV-2 infection may result in the overactivation of systemic and renal local complement system, and increased glomerular deposition of Gd-IgA1, which may lead to renal dysfunction and promote renal progression in IgAN patients.

Figure 1.jpg

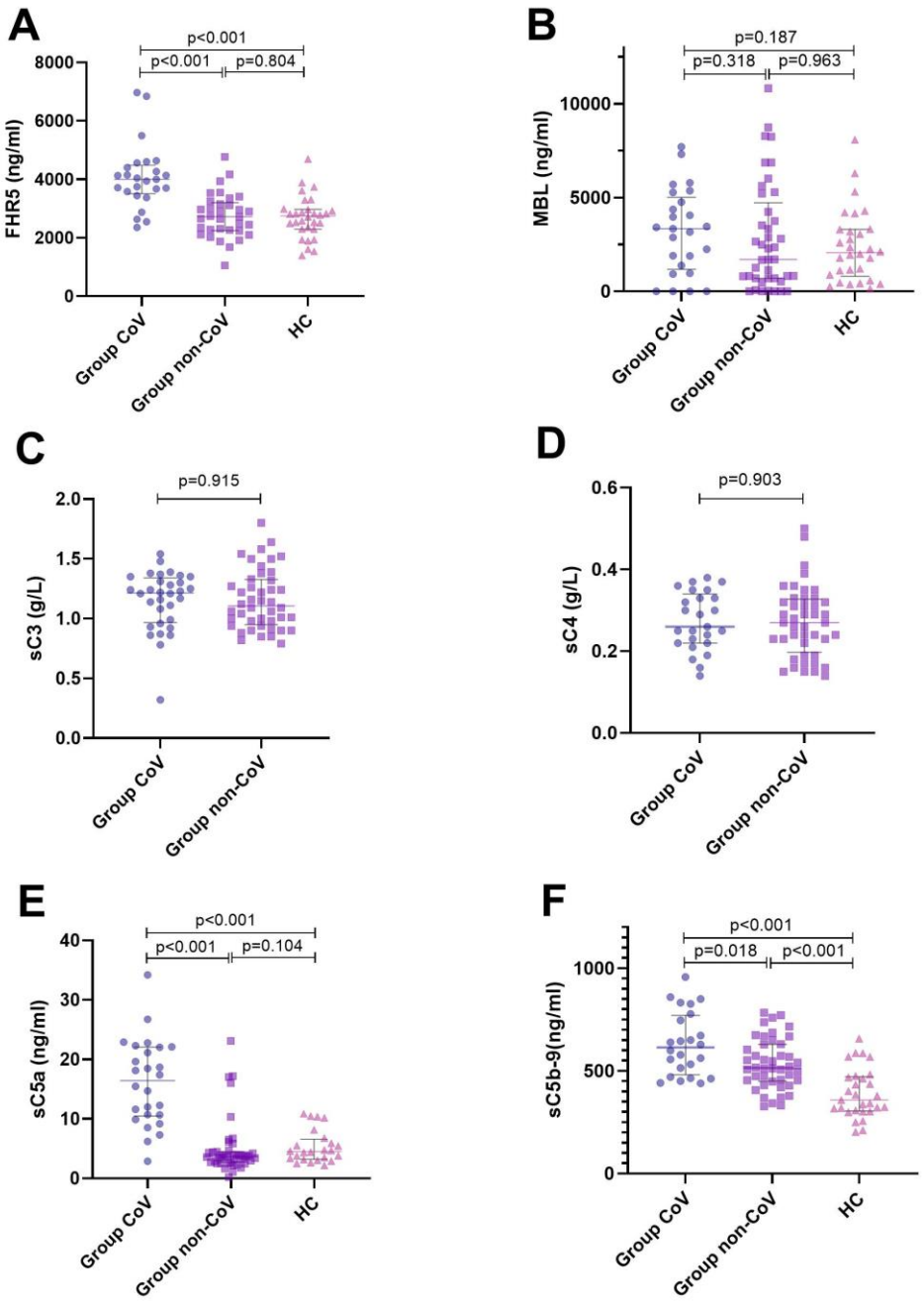
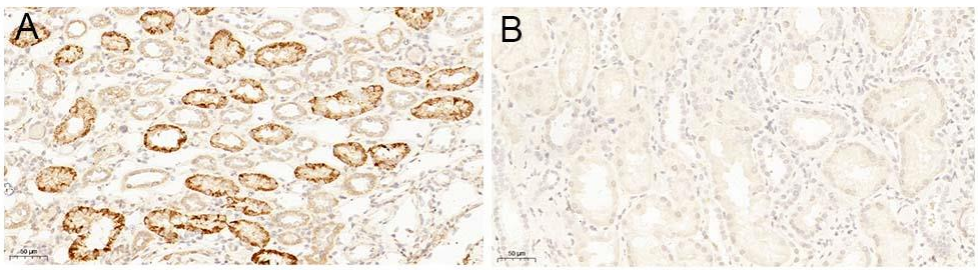

A
FHR5 (ng/ml)
p<0.001
p<0.001
p=0.804
Group CoV
Group non-CoV
HC
B
MBL (ng/ml)
p=0.187
p=0.318
p=0.963
C
sC3 (g/L)
p=0.915
D
sC4 (g/L)
p=0.903
E
sC5a (ng/ml)
p<0.001
p<0.001
p=0.104
F
sC5b-9(ng/ml)
p<0.001
p=0.018
p<0.001

Figure 1.jpg


A
B