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SARS-COV-2 deposition enhances renal complement activation and aggravates kidney injury in MN after COVID-19

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Objectives : COVID-19 has been reported to be associated with the occurrence and recurrence of membranous nephropathy (MN). Only few kidney biopsies from COVID-19 patients demonstrate the presence of SARS-CoV-2. The clinicopathological characteristics and complement system activation of MN after COVID-19 are unclear.
Methods : We collected 38 MN patients who developed new-onset proteinuria after COVID-19, and 100 patients with primary MN diagnosed before COVID-19 pandemic as control. Clinicopathological and immunological features between MN patients with and without SARS-COV2 infection were analyzed. We performed immunofluorescence and in situ hybridization assays for detection of the SARS-COV2. Glomerular staining for factor H, FHR-5 and MAC were detected by immunohistochemical. Serum C5b-9 were detected by enzyme-linked immunosorbent assay.
Results : Thirteen of 38 patients had positive staining for SARS-COV-2 in renal tissue. Compared with control-patients, the clinical manifestations were more severe in patients after COVID-19. Patients with positive SARS-COV-2 staining had a higher proportion of nephrotic syndrome, lower level of serum albumin, and greater severity of renal interstitial fibrosis than those of patients with negative SARS-COV-2 staining. The level of serum MAC and the staining intensity of MAC in renal tissue of MN after COVID-19 were significantly higher than those of control-patients, although no differences in staining intensity of C1q, MBL, factor H and factor H-related protein 5 were observed. However, MAC expression in MN patients with positive SARS-CoV-2 staining was stronger than that in both control-patients and MN after COVID-19 with negative SARS-CoV-2 staining. Meanwhile, the expression trend of factor H was consistent with that of MAC. Thus, excessive activation of the complement system aggravated symptoms in MN after COVID-19.
Conclusions : The presence of SARS-CoV-2 in renal tissue further enhanced activation of complement alternative pathway, and increased the expression of MAC in podocytes. Therapeutic strategy targeting the complement system may need to be considered.