Abstract

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Rapidly progressive paucimmune glomerulonephritis following COVID-19 infection

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Background: Ant neutrophil cytoplasmic antibody-associated vasculitis (AAV) is a systemic disease that causes vasculitis in various organs. Although the cause of the onset is unknown, infection has been reported to be a causative factor. The subsequent cytokine storm triggered by the immune response against SARS-CoV-2 infection has been reported to lead to symptoms being more severe. We report a case with the onset of AAV following COVID-19 infection.

Case report: A 75-year-old female presented to our observation for asthenia, low-grade fever and arthralgia from some months. Past medical history: at September 2020 SARS-Cov 2 infection with asymptomatic course; at July 2021 second SARS-COV2 infection complicated by interstitial pneumonia with one intercurrent dose of ChAdOx1 nCoV-19 vaccine. Diagnostic tests revealed mild leucocytosis with predominance of neutrophils, low increase of CRP, acute renal failure (serum creatinine values 3, 71 mg/dl), severe anaemia subjected to blood transfusions, COVID-19 RT-PCR negativity and p-ANCA positivity. CT thorax showed 5% lung involvement suggestive of outcomes of COVID-19. Renal needle biopsy revealed a picture compatible with rapidly progressive paucimmune glomerulonephritis; therefore she started pulse steroid therapy followed by 1 mg/kg prednisolone and first intravenous infusion of Rituximab, with the planning of close nephrological follow-up.

Discussion: In literature, ANCA-vasculitis in the setting of COVID-19 has been already reported in 5 patients. In terms of the causal relationship between SARSCoV-2 and AAV and the possible mechanism, angiotensin-converting enzyme 2 (ACE2) receptors are known to be involved in the cell invasion of SARS-CoV-2, and since SARS-CoV-2 has a high affinity for ACE2 receptors, invasion into endothelial cells is observed, which reportedly causes vasculitis. Following infection, monocyte-derived macrophages and neutrophils are recruited, which further increase the inflammatory response, leading to cytokine storm and causing fibrinoid necrosis, abnormalities in the coagulation/ fibrinolytic system, thrombotic microangiopathy, and endothelial cell damage. ANCA is produced by cytokines, activated neutrophils, and macrophages, and the induction of vasculitis by neutrophil extracellular traps could determine the onset of necrotizing crescentic glomerulonephritis. The possibility that the virus directly damages renal tissues has also been reported, although a consensus has not been reached. Rituximab is an effective alternative to cyclophosphamide in the treatment of AAV.

Conclusion: COVID-19 may be a trigger of this life-threatening autoimmune disease. More clinical and experimental investigations are necessary to further establish and confirm a causal link between these diseases.