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Case Report

Post-COVID ANCA-associated Vasculitis: A Case Report

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ABSTRACT

Although it has been reported rarely in the literature in patients who develop acute kidney injury after COVID-19 disease, ANCA-related vasculitis should also be kept in mind. Thus, it is possible to reduce mortality and morbidity. We presented a middle-aged male patient who was diagnosed with post-COVID ANCA-associated vasculitis.

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Introduction

The new coronavirus disease 2019 (COVID-19), which severe acute respiratory syndrome coronavirus (severe acute respiratory syndrome coronavirus 2, SARS-CoV-2) is responsible for, is a highly contagious respiratory infection disease. This viral syndrome affects many organs and systems, including the kidneys, apart from the lungs.[1] Anti-neutrophil cytoplasmic antibody (ANCA)-associated vasculitis (AAV) are life-threatening autoimmune diseases frequently accompanied by necrotizing rapidly progressive crescentic glomerulonephritis, which include findings of glomerulonephritis in mononuclear cell infiltration of small and medium-sized vessels and kidney biopsy. In the literature, various complications and symptoms that spread over a long period of time have been described in cases with a history of COVID-19 disease.[2] Anti-glomerular basement membrane disease, de novo ANCA-associated vasculitis with glomerulonephritis and IgA vasculitis with nephritis (Henoch-Schönlein purpura) have been reported in cases with COVID-19 infection.[3-5] We presented a case of ANCA-associated vasculitis after COVID-19.

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Case Report

A 51-year-old male patient diagnosed with chronic obstructive pulmonary disease and diabetes mellitus was followed up in the chest diseases clinic after the diagnosis of post-COVID pneumonia with the complaint of shortness of breath about one month after the COVID-19 treatment was completed. In the patient's physical examination, there was ral in the bilateral bases in the lungs. There was no feature in the cardiovascular system and abdominal examination. Pretibial oedema was negative. There was no history kidney disease (previous laboratory results showed a serum creatinine of 0.90 mg/dL 3 months before). When he was consulted to the nephrology department due to high creatinine (5.4 mg/dL) and urea (134 mg/dL) levels during his hospitalization, he was intermittently taken to hemodialysis considering acute kidney injury (AKI). Other blood tests were: leukocytes $17.2x10^9$/L, hemoglobin 8.3 g/dL, platelets $271x10^3$/L, potassium 4.4 mmol/L, sodium 135 mmol/L, albumin 2.9 g/dL, and CRP 132 mg/L. In arterial blood gas analysis, pH was 7.32, HCO_3 was 15.3 mmol/L, lactate was normal, and anion gap was 10, consistent with metabolic acidosis. There was microscopic hematuria and protein positivity (++) in the urinalysis. Daily protein loss in urine was 2 g.

Anti-nuclear antibodies (ANA), ANA profile, proteinase-3 (PR3), anti-glomerular basement membrane (anti-GBM) and hepatitis markers were negative. Cytoplasmic-antineutrophil cytoplasmic antibodies (C-ANCA), perinuclear-antineutrophil cytoplasmic antibodies (P-ANCA) and myeloperoxidase (MPO) were positive at 1/10 titer (end-point). Serum protein electrophoresis complements C3 and C4, immunoglobin G, A, and M were within normal limits. Chest computed tomography revealed bilateral pulmonary infiltration and nodular cavitary lesions (Image 1). We diagnosed the patient with ANCA-associated vasculitis, administered 500 mg of cyclophosphamide treatment, and continued methylprednisolone at a continuous dose of 0.5 mg/kg. A permanent jugular venous dialysis catheter was inserted because the patient's need for hemodialysis continued. We discharged the patient with two sessions of dialysis per week.

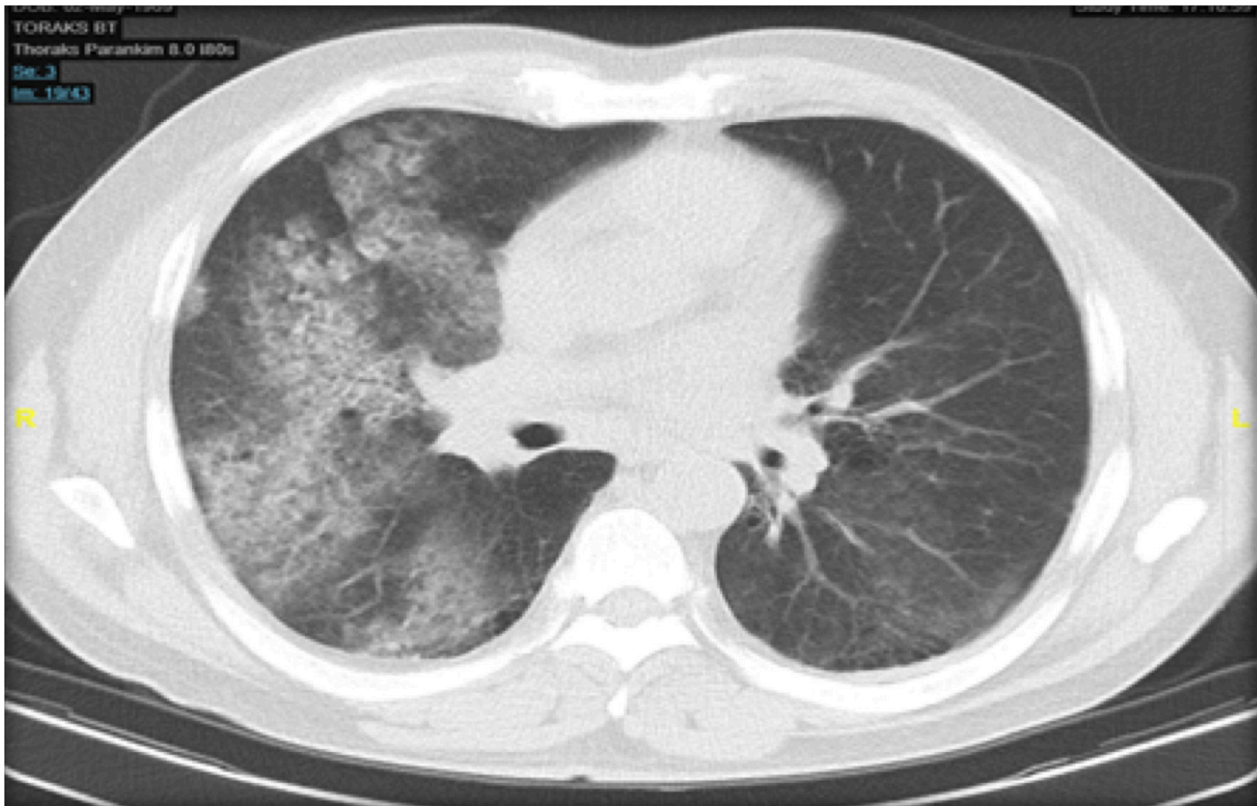

Image 1. The appearance of the lesions in the chest computed tomography of the case.

Discussion

COVID-19 has been a significant global economic and health burden. COVID-19 primarily affects the respiratory system, but kidney involvement is not uncommon. The relationship between COVID-19 disease and acute kidney injury is well known, but the number of cases in which COVID-19 and ANCA-associated vasculitis coexist is rare in the literature. In a systematic review, Wong et al.[2] analyzed the relationship between COVID-19 and vasculitis. Among 9 cases of vasculitis, 4 had Kawasaki disease, 3 had leukocytoclastic vasculitis, and 2 had IgA vasculitis. One of the cases did not respond to treatment and died.[2]

Morris et al.[6] reported that a patient who developed ANCA-associated vasculitis after COVID-19 had lung involvement due to vasculitis and the need for hemodialysis. Because of the worsening of the coagulopathy, they diagnosed vasculitis without performing a kidney biopsy. After the clinical condition of our patient stabilized, we continued intermittent dialysis. We also diagnosed ANCA-associated vasculitis in our case without renal biopsy based on clinical and laboratory findings and radiological imaging, and applied immunosuppressive treatment. One required hemodialysis in the other two COVID-19-related cases, and the other did not.[5] In some instances of ANCA-associated vasculitis developed after COVID-19, renal replacement therapy was not required, although severe kidney damage was present at the time of diagnosis.[7,8]

Duran et al.[9] applied glucocorticoid and cyclophosphamide treatment in patients with ANCA-associated vasculitis developing after COVID-19. Unlike our case, Uppal et al.[5] preferred to use rituximab with glucocorticoids to treat ANCA-associated vasculitis after COVID-19.

Conclusion

ANCA-associated vasculitis should also be considered in the differential diagnosis of COVID-19 cases presenting acute kidney injury. Diagnostic kidney biopsy may not always be possible in patients with COVID-19 who have renal involvement due to the risk of hypercoagulation and venous thromboembolism. Therefore, in selected cases, anamnesis, physical examination, laboratory and imaging findings should be evaluated together, and treatment should not be delayed. In conclusion, the possibility of ANCA-related vasculitis should be considered in the aetiology of acute kidney injury.

Acknowledgment

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Conflict of interest

The authors declared that there are no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Authors' Contribution

Study Conception: KO, MS; Study Design: MS; Supervision: AY, AO, MY, MG, KD; Materials: KO, MS, SEGB; Data Collection and/or Processing: KO, MS, SEGB; Statistical Analysis and/or Data Interpretation: KO, MS, SEGB; Literature Review: KO, MS; Manuscript Preparation: KO, MS, AE; Critical Review: AE.

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