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Successful Treatment of Post-Covid-19 Severe Anca-Associated Vasculitis: Case Report

Tratamiento Exitoso de Vasculitis Grave Asociada a Anca Post-Covid-19: Informe de un Caso

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RESUMEN

Antecedentes: La pandemia de SARS-CoV-2 se ha convertido en un importante problema de salud mundial. La afectación renal en pacientes con COVID-19 es común y se asocia con una alta mortalidad. Si bien la necrosis tubular aguda por inestabilidad hemodinámica es la causa más frecuente, también se han descrito otros mecanismos complejos y destructivos relacionados con la tormenta de citocinas y la activación del sistema inmunitario. **Presentación del caso:** Se informa de un caso de vasculitis asociada a anticuerpos anticitoplasma de neutrófilos (PR3-ANCA) que se presentó con síndrome pulmonar-renal grave, una complicación rara y potencialmente mortal que se manifestó aproximadamente dos meses después de la infección por COVID-19. **Conclusiones:** Si bien no se puede establecer una relación causal definitiva, la desregulación inmunitaria desencadenada por la COVID-19 podría haber contribuido al desarrollo de vasculitis asociada a ANCA (VAA). La VAA es una enfermedad grave, y el tratamiento inmunosupresor inmediato condujo a la recuperación completa.

Palabras Clave: ANCA, COVID-19; informe de caso; glomerulonefritis con secreciones; síndrome pulmonar-renal.

ABSTRACT

Background: The SARS-CoV-2 pandemic has become a major global health concern. Kidney involvement in COVID-19 patients is common and associated with high mortality. Although acute tubular necrosis due to hemodynamic instability is the most frequent cause, other complex and destructive mechanisms related to cytokine storm and immune system activation have also been reported. **Case presentation:** We report a case of proteinase-3 anti-neutrophil cytoplasmic antibody (PR3-ANCA)-associated vasculitis presenting with severe pulmonary-renal syndrome, a rare and potentially fatal complication that occurred approximately two months after COVID-19 infection. **Conclusions:** While a causal relationship cannot be definitively established, immune dysregulation triggered by COVID-19 may have contributed to the development of ANCA-associated vasculitis (AAV). AAV is a serious disease, and prompt immunosuppressive therapy led to full recovery.

Keywords: ANCA, COVID-19, case

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report; crescentic glomerulonephritis; pulmonary-renal syndrome.

BACKGROUND

Coronavirus disease 2019 (COVID-19), caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has led to significant morbidity and mortality worldwide since its emergence in late 2019. While most cases are asymptomatic or present with mild influenza-like symptoms, a substantial proportion develop severe pneumonia, acute respiratory distress syndrome, multi-organ failure, or death [(1)]. COVID-19 can trigger an exaggerated immune response, particularly in susceptible individuals[(2)]. Epitope expansion and antigen mimicry are the first triggers for antibody production [(3)]. Antineutrophil cytoplasmic antibody (ANCA)-associated vasculitis (AAV) is a systemic disease affecting organs such as the kidneys, lungs, skin, and gastrointestinal tract. Although its exact pathogenesis remains unclear, infections have been implicated as triggers, particularly via cytokine storms[(4,5)]. AAV requires rapid diagnosis and treatment; any delay increases morbidity and mortality [(6,7)]. Severe organ involvement often complicates timely diagnosis and treatment. We present a previously healthy 51-year-old male who developed severe ANCA-associated vasculitis following mild COVID-19, presenting with both acute kidney injury (AKI) and diffuse alveolar hemorrhage (DAH), and successfully treated prior to ANCA confirmation.

CASE PRESENTATION

A 51-year-old man without prior comorbidities presented with AKI, anuria, and hemoptysis. He had received two doses of the Biontech mRNA vaccine approximately 8 months earlier and had tested positive for COVID-19 via PCR approximately 2 months earlier due to symptoms of a runny nose, fever, and fatigue. He required neither hospitalization nor medical treatment during his COVID-19 infection and became PCR-negative.

However, fifteen days after recovery, he developed weakness, anorexia, joint pain, nausea, and daily hemoptysis. Over the following ten days, his urine output progressively declined. At a local hospital, chest computed tomography (CT) revealed a cavitary lesion with central opacification in the right pulmonary apex and bilateral ground-glass opacities, suggestive of diffuse alveolar hemorrhage. (**Figure 1**) Bacterial and fungal lung infections were ruled out. Sputum tuberculosis cultures, acid-fast bacilli (AFB) stains, and immunological tests, including PR3-ANCA, MPO-ANCA, ANA, and anti-GBM, were initially negative.

With worsening renal function and clinical deterioration, he was referred to our hospital for evaluation of pulmonary-renal syndrome and potential hemodialysis. Upon admission, laboratory tests showed serum creatinine 12.02 mg/dL, eGFR 4 mL/min/1.73 m², BUN 118.65 mg/dL, albumin 2.7 g/dL, and C-reactive protein 292 mg/dL. Urinalysis demonstrated proteinuria and hematuria. Severe anemia, leukocytosis, and normal platelet counts were noted. The patient was tachypneic and hypoxic at presentation. Within two hours, he developed a massive pulmonary hemorrhage requiring urgent intubation.

Due to the critical presentation, empirical treatment was initiated immediately with 1000 mg intravenous pulse methylprednisolone and plasmapheresis, despite the absence of histological or serological confirmation. Subsequent serological re-evaluation revealed positive PR3-ANCA. Cyclophosphamide 500 mg IV was administered, and plasmapheresis continued for five sessions. Following three days of pulse steroids, maintenance therapy with 80 mg IV prednisolone was initiated. Hemodialysis was started due to uremia and anuria. Tracheostomy was performed on day 26 of intensive care. Gradual clinical improvement was observed, with increasing urine output and resolution of hypoxia. After 42 days, the tracheostomy was closed. The renal function recovered, and the patient was discharged after 50 days in intensive care. **(Figure 2b, Figure 3**). Maintenance therapy included 60 mg oral prednisolone daily and cyclophosphamide 500 mg IV every 15 days.

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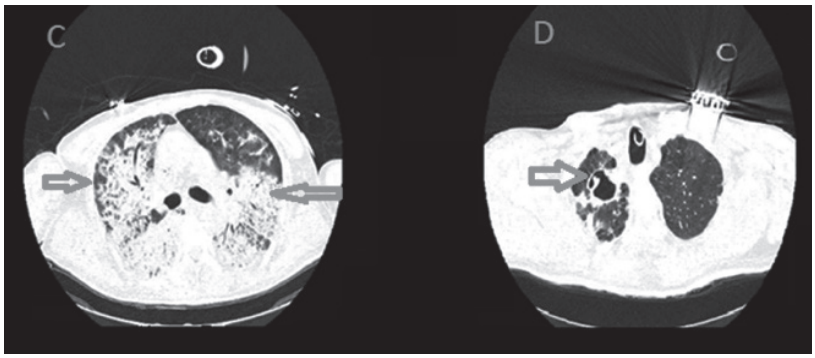


Figure 1: Thoracic CT showing widespread bilateral ground-glass opacities consistent with diffuse alveolar hemorrhage (C) and a cavitary lesion in the right pulmonary apex (D).

Figure 2: Chest X-ray (PA view) at presentation (A) and after 8 weeks (B).

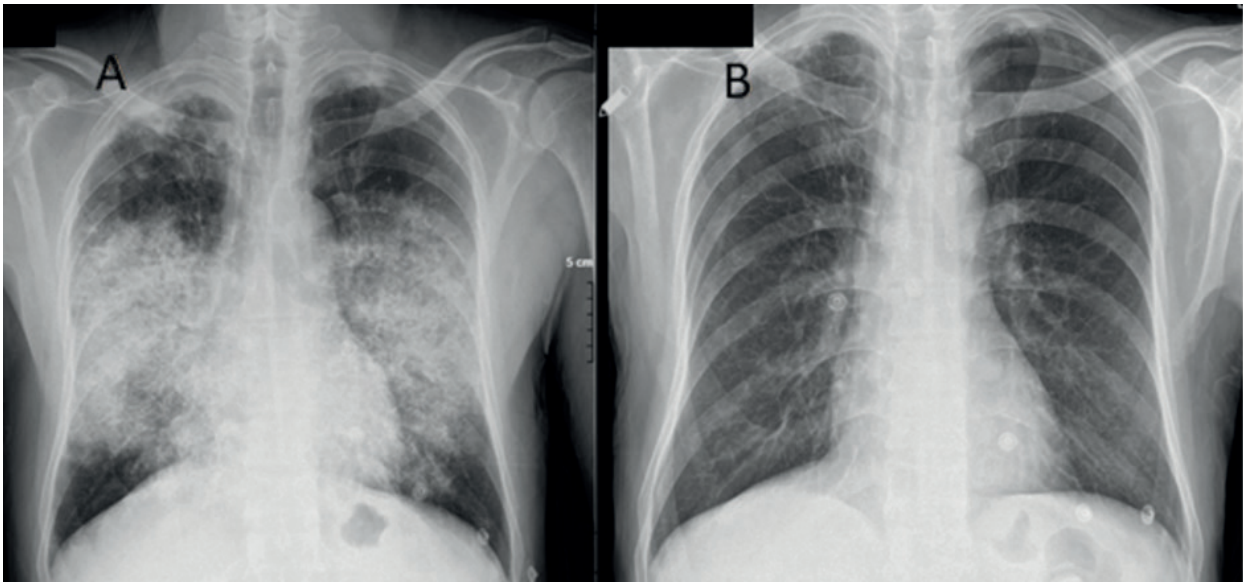
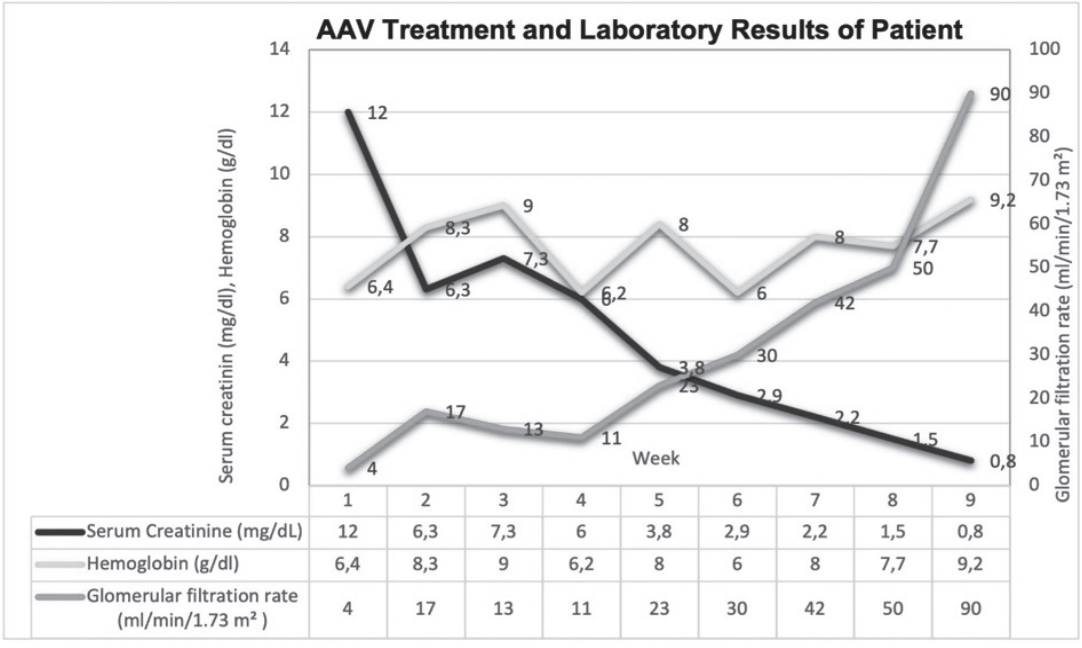


Figure 3: Treatment timeline showing initiation of pulse steroids and plasmapheresis, followed by cyclophosphamide, gradual renal recovery, and discontinuation of hemodialysis



DISCUSSION

Herein, we report a case of severe PR3-ANCA-associated vasculitis presenting with dialysis-requiring AKI and DAH following SARS-CoV-2 infection. Granulomatosis with polyangiitis, a small vessel vasculitis, manifests across a spectrum from mild disease to life-threatening massive alveolar hemorrhage. Immunosuppression remains the cornerstone of treatment; untreated cases carry high mortality [7]. Although causality cannot be definitively proven, accumulating evidence suggests that SARS-CoV-2 may trigger autoimmune vasculitis through mechanisms of immune dysregulation, molecular mimicry, and endothelial injury [3,5].

COVID-19 induces a hyperinflammatory state characterized by elevated cytokines (IL-6, IL-8, TNF-α) and activation of neutrophil extracellular traps (NETs), both of which have been implicated in the pathogenesis of AAV. NETs provide autoantigens, such as PR3 and MPO, thereby promoting ANCA formation in genetically predisposed individuals. Additionally, molecular mimicry between viral antigens and host neutrophil proteins may facilitate autoimmunity [15,18].

Several case reports describe de novo ANCA-associated vasculitis following COVID-19 [8,14]. Among these, Hussein et al. described a fatal case of PR3-ANCA vasculitis with massive hemoptysis in a COVID-positive patient. Maritati et al. and Morris et al. reported severe post-COVID AAV requiring dialysis; both had poor outcomes [9,11,12]. In contrast, our case represents a rare instance of full recovery despite dialysis-requiring renal failure and respiratory failure.

Although a direct causal link between SARS-CoV-2 infection and subsequent ANCA-associated vasculitis cannot be confirmed, several reports describe the delayed onset of autoimmune vasculitis weeks to months after infection, suggesting that SARS-CoV-2 infection may unmask latent autoimmunity [15,16]. In our patient, ANCA seroconversion occurred after recovery from mild COVID-19, suggesting a delayed post-infectious immune process rather than direct viral injury. Similar delayed vasculitic phenomena have been observed after influenza and hepatitis C infections, supporting the possibility that persistent immune dysregulation following COVID-19 may precipitate autoimmunity [19,20].

CLINICAL IMPLICATIONS

Clinicians should maintain a high index of suspicion for AAV in patients presenting with pulmonary-renal syndromes after COVID-19, even several weeks post-infection. Early initiation of immunosuppressive therapy before histological confirmation can be life-saving, as demonstrated in our case. Further research is needed to determine the true incidence and pathophysiological link between SARS-CoV-2 and AAV.

CONCLUSIONS

To our knowledge, this is the first reported case of post-COVID PR3-ANCA-positive vasculitis presenting with both massive hemoptysis and dialysis-dependent renal failure, who fully recovered. The key factor contributing to survival was the immediate initiation of immunosuppressive therapy, without waiting for histological confirmation.

Although the direct causal relationship between COVID-19 and ANCA-associated vasculitis remains unproven, our case supports the hypothesis that immune dysregulation triggered by SARS-CoV-2 infection may contribute to the onset of vasculitis. Awareness of this potential association is crucial for timely diagnosis and treatment. Our patient's full recovery underscores the importance of early aggressive immunosuppressive therapy.

ABBREVIATIONS

COVID-19: Coronavirus disease 2019.
PR3-ANCA: Proteinase-3 anti-neutrophil cytoplasmic antibodies.
ANCA: Antineutrophil cytoplasmic antibody.
AAV: Antineutrophil cytoplasmic antibody-associated vasculitis.
DAH: Diffuse alveolar hemorrhage.
AFB: Acid-fast bacilli.
MPO-ANCA: Myeloperoxidase-Antineutrophil cytoplasmic antibody.
ANA: Antinuclear Antibody.
anti-GBM: Anti-glomerular basement membrane.
eGFR: Estimated glomerular filtration rate.
BUN: Blood urea nitrogen.

DECLARATIONS

Ethics Approval and Consent to Participate

The patient provided written consent to participate in the study.

Consent for Publication

Written informed consent was obtained from the patient for the publication of this case report.

availability of Data and Materials

Data are available from the corresponding author upon request.

Competing Interests

The authors declare that they have no competing interests.

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