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CASE REPORT

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New-onset ANCA-associated vasculitis presenting with neuropathy after COVID-19 infection: A case report and literature review

Aida Mohamadi[1] | **Somayeh Soroureddin**[1] | **Sepehr Nayebirad**[2] | **Zahra Tamartash**[1] | **Maryam Mohebbi**[1] | **Hoda Kavosi**[1]

[1]Rheumatology Research Center, Tehran University of Medical Sciences, Tehran, Iran

[2]Tehran Heart Center, Cardiovascular Diseases Research Institute, Tehran University of Medical Sciences, Tehran, Iran

Correspondence
Hoda Kavosi, Rheumatology Research Center, Tehran University of Medical Sciences, Shariati Hospital, North Kargar Ave, Tehran, Iran.
Email: h-kavosi@sina.tums.ac.ir

Key Clinical Message

Coronavirus Disease 2019 (COVID-19) is a viral infection caused by SARS-CoV-2, which can trigger autoimmune diseases such as antineutrophilic cytoplasmic antibody (ANCA) associated vasculitis (AAV) that affect small and medium-sized blood vessels in multiple organs. This study discusses a case with neuropathy and positive ANCA after COVID-19 infection and reviews the literature on AAV following COVID-19 infection. A 59-year-old man is presented that was referred to Shariati Hospital for evaluation of neurologic problems after a COVID-19 infection. Initially, he had flu-like symptoms. A few days later, he developed right distal upper and lower limb paresthesia. His electromyography (EMG) and nerve conduction velocity (NCV) results were consistent with polyneuropathy. Lumbar puncture (LP) was normal except for positive COVID-19 polymerase chain reaction (PCR). The patient's paresthesia worsened. Laboratory data showed leukocytosis, anemia, thrombocytosis, high erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP). Perinuclear anti-neutrophil cytoplasmic antibody (MPO-ANCA) was positive. According to the results, vasculitis was the main differential diagnosis. The sural nerve biopsy was performed, and the result was consistent with small to medium-sized vessel vasculitis. The patient was diagnosed with COVID-induced AAV. He was prescribed methylprednisolone and cyclophosphamide and was discharged with prednisolone and cotrimoxazole. In this study, a unique case of AAV induced by COVID-19 infection confirmed by nerve biopsy is presented. A review of the literature found 48 cases of new-onset AAV in adults and pediatrics after COVID-19 infection. Further research is needed to completely understand the relationship between COVID-19.

KEYWORDS

ANCA-associated, vasculitis, case report, COVID-19, neuropathy, vasculitis

1 | INTRODUCTION

Coronavirus Disease 2019 (COVID-19) is a viral infection caused by SARS-CoV-2, which can trigger autoimmunity in children and adults.[1] Different autoimmune diseases including rheumatoid arthritis, type 1 diabetes, psoriatic arthritis, and antineutrophilic cytoplasmic antibody (ANCA) associated vasculitis (AAV) have been reported after COVID-19 infection.[2] AAV is a rare autoimmune disease that affects small and medium-sized blood vessels in multiple organs. Glomerulonephritis (GN) and diffuse alveolar hemorrhage (DAH) are common presentations in post-COVID AAV patients while neurologic symptoms are very rare and have been reported in only three patients until now.[3–7] This study discusses a case with neuropathy and positive ANCA after COVID-19 infection.

2 | CASE PRESENTATION

A 59-year-old man without previous medical history was referred to Shariati Hospital for evaluation of neurologic problems after a COVID-19 infection. Initially, symptoms had started 3 weeks ago with fever, myalgia, and rhinorrhea and he was managed with conservative treatment. A few days later, he developed right distal upper and lower limb paresthesia and was admitted to another center with suspicion of Guillain-Barre syndrome (GBS) due to COVID-19. His electromyography (EMG) and nerve conduction velocity (NCV) results were consistent with chronic bilateral asymmetric sensorimotor axonal polyneuropathy. Therefore, GBS was ruled out from the diagnosis. Lumbar puncture (LP) was normal except for positive COVID-19 polymerase chain reaction (PCR). Intravenous immunoglobulin (IVIG), dexamethasone 8 mg daily, and remdesivir were started for the patient. After 2 days, the patient's paresthesia worsened and he experienced a right-sided foot drop. He was referred to our center for further assessment. Upon examination, vital signs were normal; however, the neurologic exam showed right wrist and foot drop, reduced right distal extremity forces (1/5), and reduced right distal extremity deep tendon reflexes (1+). The EMG-NCV was repeated and the result was subacute axonal sensorimotor polyneuropathy. Laboratory data showed a WBC count of 17,000 cells/mcL, a hemoglobin level of 10 g/dL, a platelet count of 509,000 platelets/mcL, an erythrocyte sedimentation rate (ESR) level of 95 mm/hour, and a C-reactive protein (CRP) level of 116 mg/dL. Other lab data including liver function test, creatinine level, urine analysis, and urine culture were normal. Perinuclear anti-neutrophil cytoplasmic antibody (MPO-ANCA or P-ANCA) was positive with a titer of 47.5 U/mL. Other potential causes of positive MPO-ANCA such as hepatitis, tuberculosis, human immunodeficiency virus (HIV), endocarditis, and use of certain medications (antithyroid drugs, hydralazine, and cocaine) were ruled out. According to the laboratory and paraclinical results, vasculitis was the main differential diagnosis. Therefore, other organ involvements were assessed. The spiral chest computed tomography (CT) scan showed sub-segmental atelectasis at the lower lobes and lingula, minimal right-sided pleural reaction, and minimal pericardial effusion. The spiral paranasal sinuses (PNS) CT scan was normal. The sural nerve biopsy was performed and the pathology result was obtained using immunohistochemistry (IHC) and hematoxylin & eosin (H&E) staining methods. It was consistent with small to medium-sized vessel vasculitis. There were endothelial swelling and lymphocytic infiltration including karyorrhectic nuclear debris within the endoneurial vessel wall in the pathology. Despite inflammation, there was no necrosis present in the pathology (Figure 1). Eventually, considering positive MPO-ANCA and nerve biopsy results, the patient was diagnosed with COVID-induced AAV based on the 2022 classification criteria of the American College of Rheumatology/European Alliance of Associations for Rheumatology.[8] He was prescribed three consecutive pulses of methylprednisolone 1 g and three consecutive pulses of cyclophosphamide 1 g every 2 weeks and was discharged with prednisolone 60 mg and cotrimoxazole 400/80 mg daily. One month later, he got infected with cytomegalovirus (CMV) and was treated with gancyclovir. As a result, the next pulse of cyclophosphamide was postponed to 1 month later and it was continued once every month for 6 months. Prednisolone was tapered down to 10 mg daily and azathioprine 2 mg/kg was started for him as maintenance therapy. He was followed up every 3 months. His foot drop did not fully recover, but his MPO-ANCA, ESR, and CRP levels gradually decreased.

3 | LITERATURE REVIEW

A literature review in PubMed and Scopus was conducted from December 1, 2019 to July 14, 2023 using the keywords "anti-neutrophil cytoplasmic antibody-associated vasculitis" and "COVID-19". Studies reporting new-onset AAV cases after COVID-19 infection were included. Studies that contained new-onset AAV patients after COVID-19 vaccination and COVID-19 infection in previously diagnosed AAV patients, reviews, and non-English articles were excluded. Eventually, 46 case reports (48 patients) were included for further assessment. The female/male ratio was 1.2 and the mean age was 45 years. Positive MPO-ANCA was seen in 26 cases (54%), while others were proteinase 3 anti-neutrophil cytoplasmic antibody (PR3-ANCA or C-ANCA) positive. MPO-ANCA is more commonly associated with renal involvement, such as glomerulonephritis

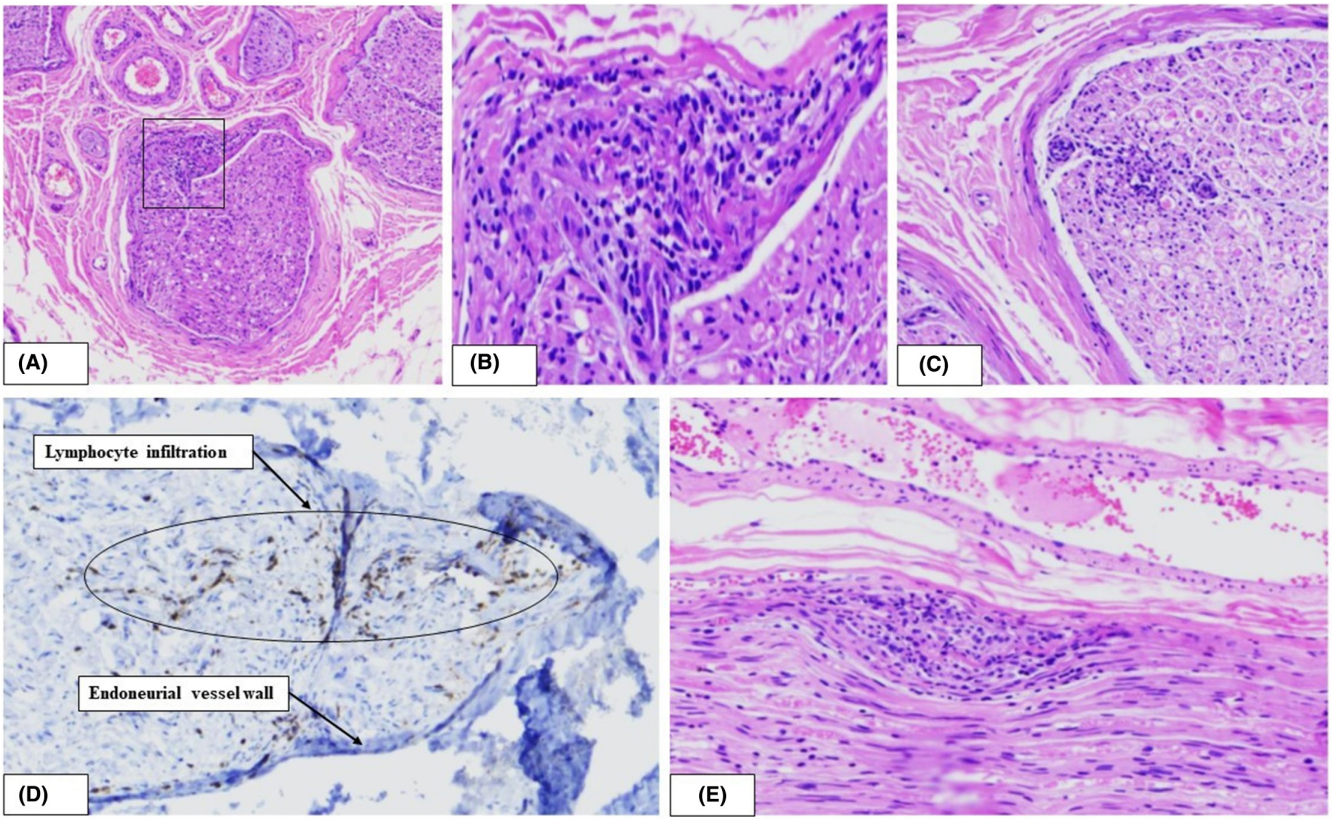

FIGURE 1 The pathology of the sural nerve biopsy. (A) Transection of nerve with lymphocytic infiltration within the endoneurial vessel wall (H&E). (B) High power field view of the selected area (H&E). (C) Transaction of nerve with endothelial swelling and lymphocytic infiltration within the endoneurial vessel wall (H&E). (D) Longitudinal section of nerve showing infiltration of CD3 positive lymphocytes within the endoneurial vessel wall (IHC). (E) Longitudinal section of nerve showing peri and within vessel wall lymphocytic infiltration including some karyorrhectic nuclear debris and endothelial swelling (H&E).

and renal failure while PR3-ANCA is often associated with respiratory tract involvement, including sinusitis, lung nodules, and lung hemorrhage.[9–11] However, in this literature review, it was challenging to differentiate them in uncommon presentations of COVID-induced AAV.

The most frequently affected organ was kidneys (28 cases) presented mainly with glomerulonephritis (GN) or acute kidney injury (AKI). The symptoms were primarily pallor, hypertension, edema, and hematuria. Diagnosis relied on laboratory findings such as anemia, high ESR and CRP, azotemia, and positive MPO-ANCA and confirmed through biopsy.

The second prevalent affected site was the pulmonary system (19 cases) presented with diffuse alveolar hemorrhage (DAH) which was marked by respiratory distress, hypoxemia, and hemoptysis and the diagnosis was confirmed with a CT scan as well as laboratory findings including high ESR and CRP and positive MPO-ANCA.

There have been only three other patients with neurological presentations following COVID-19 until now that were diagnosed through laboratory data and CT scan or magnetic resonance imaging (MRI). One of them was a 17-year-old girl with no previous medical history who had facial allodynia.[5] The second patient was a 13-year-old girl with a history of immune thrombocytopenia purpura (ITP) who experienced walking difficulties and numbness of the lower limbs 1 week after being exposed to COVID-19 and generalized tonic–clonic status epilepticus 2 weeks later. She was diagnosed with transverse myelitis, brain demyelination, and pachymeningitis.[3] The third one was a healthy 14-year-old girl who experienced sinusitis and migratory arthritis 3 weeks after COVID-19 infection. Later, she had severe abdominal pain and chest pain. She underwent treatment with the diagnosis of acute coronary syndrome (ACS). A few days later, she had tonic–clonic seizures and experienced hemiparesis 3 weeks later. Her brain MRI showed an intracranial hemorrhage (ICH).[4]

Other organs of the body were also affected with two cases of each cardiac involvement (myocarditis and ST-elevation myocardial infarction (STEMI)), gastrointestinal involvement and hearing loss. In summary, AAV can involve any part of the body and in post-COVID patients with similar presentations, it should be considered a primary differential diagnosis.

The characteristics of the included studies are shown in Table 1.

TABLE 1 Characteristics of the included studies.

Author	Gender	Age	Presentation	Antibody type	Treatment
Allena et al.[12]	F	60	GN	MPO-ANCA	Corticosteroids, RTX, and plasmapheresis
Babu et al.[13]	M	59	GN	MPO-ANCA	Corticosteroids, CTX, and MMF
Bołtuć et al.[14]	M	34	DAH and RPGN	MPO-ANCA	Corticosteroids and CTX
Bryant et al.[7]	F	16	Chronic serous otitis media and CHL	PR3-ANCA	Corticosteroids, and MMF
Cobilinschi et al.[15]	F	67	Necrotic ulcer-like lesions on both legs	MPO-ANCA	Corticosteroids and CTX
Felzer et al.[16]	F	86	DAH	MPO-ANCA	Corticosteroids and RTX, and convalescent plasma
Fireizen et al.[17]	M	17	DAH and AKI	MPO-ANCA	Corticosteroids, CTX, and plasmapheresis
Giles et al.[18]	M	28	GI vasculitis	PR3-ANCA	Corticosteroids and RTX
Izci Duran et al.[19]	M	26	GN	MPO-ANCA	Corticosteroids, CTX, HD, and plasmapheresis
Izci Duran et al.[19]	F	36	GN and total hearing loss	PR3-ANCA	Corticosteroids and CTX
Jalalzadeh et al.[20]	F	46	GN	MPO-ANCA	Corticosteroids and RTX
Kataria et al.[21]	F	79	GN	MPO-ANCA	Corticosteroids and RTX
Kawashima et al.[22]	F	61	RPGN	MPO-ANCA	Corticosteroids and CTX
Lind et al.[23]	M	40	DAH	PR3-ANCA	Corticosteroids and RTX
Madanchi et al.[24]	M	53	DAH, AKI, and GN	MPO-ANCA	Corticosteroids, CTX and AZA
Maritati et al.[25]	F	64	GN and APS	PR3-ANCA	Corticosteroids, RTX, CTX, and HD
Merveilleux et al.[26]	M	59	Hypereosinophilic bronchiolitis	MPO-ANCA	Corticosteroids and AZA
Morris et al.[27]	M	53	DAH and AKI	PR3-ANCA	Corticosteroids and HD
Nakamura et al.[5]	F	17	Facial allodynia	PR3-ANCA	Corticosteroids and RTX
Powell et al.[28]	F	12	DAH	MPO-ANCA	Corticosteroids, RTX, and CTX
Reiff et al.[29]	M	17	MIS-C	PR3-ANCA	Corticosteroids and RTX
Selvaraj et al.[30]	F	60	GN and TIN	PR3-ANCA	Corticosteroids and RTX
Singh et al.[31]	F	48	DAH and AKI	MPO-ANCA	Corticosteroids, RTX, and plasmapheresis
Ta et al.[32]	M	67	Oral, mucosal, and cutaneous involvements	PR3-ANCA	Corticosteroids
Thabet et al.[3]	F	13	Transverse myelitis, CNS demyelination, and pachymeningitis	PR3-ANCA	Corticosteroids and RTX
Thu Aung et al.[33]	F	64	AKI	PR3-ANCA	Corticosteroids, RTX, CTX, and plasmapheresis
Uppal et al.[34]	M	64	GN	MPO-ANCA	Corticosteroids, RTX, and HD
Uppal et al.[34]	M	46	AKI	PR3-ANCA	Corticosteroids and RTX
Wali et al.[35]	F	26	DAH and GN	MPO-ANCA	Corticosteroids and RTX
Wang et al.[36]	F	56	DAH	MPO-ANCA	Corticosteroids and CTX
Asma et al.[37]	M	72	GN	PR3-ANCA	Corticosteroids and CTX
Garcia Vega et al.[38]	M	60	RPGN	MPO-ANCA	Corticosteroids and RTX
Patel et al.[39]	M	51	DAH and GN	PR3-ANCA	HD
Zakrocka et al.[40]	M	59	RPGN	MPO-ANCA	Corticosteroids, CTX, HD, and plasmapheresis

TABLE 1 (Continued)

Author	Gender	Age	Presentation	Antibody type	Treatment
Wintler et al.[41]	F	13	Necrosis of the perineum and rectum	PR3-ANCA	Corticosteroids and RTX
Chargui et al.[42]	M	49	TMA	MPO-ANCA	Corticosteroids, CTX, and plasmapheresis
Fares et al.[43]	F	55	DAH	MPO-ANCA	Corticosteroids and plasmapheresis
Hussein et al.[44]	F	37	DAH	PR3-ANCA	Corticosteroids, IVIG, and plasmapheresis
Moeinzadeh et al.[45]	M	25	GN	PR3-ANCA	Corticosteroids, CTX, IVIG, and plasmapheresis
Raeeskarami et al.[4]	F	14	STEMI, ICH, and GI perforation	PR3-ANCA	Corticosteroids, CTX, IVIG, AZA, and MMF
Ozcan et al.[6]	M	26	DAH and AKI	MPO-ANCA	Corticosteroids and CTX
Kostelníková et al.[46]	F	43	DAH	PR3-ANCA	Corticosteroids, CTX, and MMF
Caglayan et al.[47]	F	17	DAH and myocarditis	MPO-ANCA	Corticosteroids, IVIG, and CTX
Chandok et al.[48]	F	57	AKI and GN	MPO-ANCA	Corticosteroids and RTX
Kaynar et al.[49]	F	57	GN and AHF	MPO-ANCA	Corticosteroids, CTX, and AZA
Shah et al.[50]	M	32	TIN, TMA, acute scleritis, and pulmonary cavitary lesion	PR3-ANCA	Corticosteroids
Tahir et al.[51]	M	82	GN and PE	MPO-ANCA	Corticosteroids and RTX
Manivannan et al.[52]	F	41	DAH	PR3-ANCA	Corticosteroids, CTX, and plasmapheresis

Abbreviations: AHF, acute heart failure; AKI, acute kidney injury; APS, antiphospholipid syndrome; AZA, azathioprine; CHL, conductive hearing loss; CTX, cyclophosphamide; DAH, diffuse alveolar hemorrhage; F, female; GN, glomerulonephritis; GI, gastrointestinal; HD, hemodialysis; IVIG, intravenous immunoglobulin; ICH, intracranial hemorrhage; M, male; MIS-C, multisystem inflammatory syndrome in children; MMF, mycophenolate mofetil; MPO-ANCA, perinuclear anti-neutrophil cytoplasmic antibodies; PE, pericardial effusion; PR3-ANCA, Proteinase 3 anti-neutrophil cytoplasmic antibodies; RTX, rituximab; RPGN, Rapidly progressive glomerulonephritis; STEMI, ST-elevation myocardial infarction; TIN, tubulointerstitial nephritis; TMA, thrombotic microangiopathy.

4 | DISCUSSION

AAV can involve many organs in the body and appear in various presentations. glomerulonephritis (GN) and diffuse alveolar hemorrhage (DAH) are the most common ones. The typical renal appearance is GN with a decrease in renal function accompanied by hypertension, microscopic hematuria, and sub-nephrotic range proteinuria over days to a few months.[1,2,53,54] The dominant radiological pulmonary pattern in COVID-19 is peripherally and posteriorly distributed multifocal bilateral ground-glass opacities (GGO) and consequent overlapping of the consolidations and there may also be pleural effusion and cavitation.[1,53]

The current study presents a case of AAV after a COVID-19 infection with neuropathy and limb paresthesia that has been reported for the first time. Neurologic involvement in COVID-induced AAV has occurred in only three patients with manifestations ranging from facial allodynia and transverse myelitis to intracranial hemorrhage (ICH) previously.[3–5] Several studies have established a connection between SARS-CoV-2 infection and various neurological manifestations, such as headaches or specific conditions like Guillain–Barré syndrome or encephalitis.[55] Research indicates that over 35% of individuals with COVID-19 experience neurological symptoms, and in some cases, these symptoms may even be the initial presenting signs of the disease.[56] SARS-CoV-2 can enter the nervous system through various pathways. Therefore, it is important to consider COVID-19 as a potential cause of CNS infection, and testing for the virus in the CSF is warranted. However, the test is not always positive and the neurological symptoms can be due to other mechanisms which are mentioned below.[57]

Recent studies have suggested that COVID-19 infection may trigger or exacerbate autoimmune diseases such as AAV in susceptible individuals.[12,58] In a cohort study by Lim et al. including 354,527 individuals with a history of COVID-19 and 6,134,940 controls, it was proved that the risk of multiple autoimmune and autoinflammatory connective tissue disorders including AAV was

significantly higher in the post-COVID group compared to the controls.[58]

There are various potential mechanisms which induce autoimmunity after COVID-19 infection. One of them is molecular mimicry, leading to the immune system falsely attacking its self-tissues when a viral protein is analogous to a protein in the body.[13] Another mechanism is bystander activation, in which the immune system responds to the virus and inadvertently attacks nearby healthy tissue.[14] A third mechanism is epitope spreading, where the immune system initially targets the virus but then begins attacking other proteins in the body that are not directly related to the virus.[7] SARS-CoV-2 can also induce an immune response by elevating interleukin-6 (IL-6). It activates the inflammatory cascade and causes tissue destruction and organ failure. These reactions are responsible for most of the symptoms of an induced autoimmune disease after COVID-19 infection.[15,16]

There can be other mechanisms besides vasculitis that can justify the neurological presentations. COVID-19 has been associated with vascular issues, including blood clot formation. Vascular complications such as thrombosis or embolism could lead to reduced blood flow to the brain, causing neurological symptoms. Furthermore, it has been suggested that the SARS-CoV-2 virus might have neurotropic properties, meaning it can directly invade the nervous system. This invasion could result in various neurological manifestations. Moreover, patients with severe COVID-19 may experience critical illness, and the physiological stress of critical illness itself can lead to neurological complications such as encephalopathy or other functional disturbances. Finally, Severe respiratory distress and complications from COVID-19 can lead to hypoxia which may contribute to neuronal damage and neurological symptoms.[59–61]

There have been other types of vasculitis leading to neuropathy reported following COVID-19 infection. Giant cell arteritis (GCA) may be triggered by COVID-19 and can present with neurological manifestations.[62] Another type of vasculitis in which CNS can be involved after COVID-19 infection is lymphocytic vasculitis which has been reported before a few times.[63] Cryoglobulinemic vasculitis can also affect the peripheral nerves, causing neuropathy.[64]

5 | CONCLUSION

In this study, we presented a unique case of AAV induced by COVID-19 infection confirmed by nerve biopsy and clinical presentation. COVID-19 infection might trigger AAV with different manifestations in both adults and pediatrics. One of the rare presentations is neurologic involvement. Further research is needed to completely understand the relationship between COVID-19 and AAV.

AUTHOR CONTRIBUTIONS

Aida Mohamadi: Data curation; writing – original draft. **Somayeh Soroureddin:** Data curation. **Sepehr Nayebirad:** Conceptualization; writing – review and editing. **Zahra Tamartash:** Writing – review and editing. **Maryam Mohebbi:** Resources; supervision. **Hoda Kavosi:** Project administration; writing – review and editing.

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CONFLICT OF INTEREST STATEMENT

The authors have no relevant financial or non-financial interests to disclose.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

ETHICS STATEMENT

This study was approved by the Administration Committee of The Rheumatology Research Center, Tehran University of Medical Sciences, Tehran, Iran (Approval number: IR.TUMS.SHARIATI.REC.1402.005).

CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

ORCID

Aida Mohamadi https://orcid.org/0000-0002-6921-0108

REFERENCES

1. Wang D, Hu B, Hu C, et al. Clinical characteristics of 138 hospitalized patients with 2019 novel coronavirus-infected pneumonia in Wuhan. *China Jama*. 2020;323(11):1061-1069.
2. Knight JS, Caricchio R, Casanova JL, et al. The intersection of COVID-19 and autoimmunity. *J Clin Invest*. 2021;131(24):1-9.
3. Thabet F, Yahyaoui A, Besbes H, et al. c-ANCA-associated vasculitis with predominant CNS demyelination after COVID-19. *Archives de Pédiatrie*. 2023;30(1):74-76.
4. Raeeskarami SR, Mousavi SH, Ashouri M, Akbariasbagh P, Assari R, Naderi S. Could COVID-19 trigger presentation or

exacerbation of Vasculitides?(a 14-year-old girl with newly diagnosed GPA after COVID-19 infection). *Iran J Pedia*. 2021;31(3):1-7.
5. Nakamura Y, Okumura F, Nihei M, et al. Granulomatosis with Polyangiitis following COVID-19 in an adolescent. *Int Med*. 2023;62(4):589-593.
6. Ozcan S, Sonmez O, Karaca C, Ozdede A, Seyahi N. ANCA-associated vasculitis flare might be provoked by COVID-19 infection: a case report and a review of the literature. *Clin Kid J*. 2022;15(11):1987-1995.
7. Bryant MC, Spencer LT, Yalcindag A. A case of ANCA-associated vasculitis in a 16-year-old female following SARS-COV-2 infection and a systematic review of the literature. *Pedia Rheumatol Onl J*. 2022;20(1):65.
8. Yazici H, Tascilar K, Yazici Y. American College of Rheumatology/European Alliance of associations for rheumatology classification criteria sets for three types of antineutrophilic cytoplasmic antibody-associated vasculitis. *Current Opin Rheumatol*. 2023;2022:35.
9. Robson JC, Grayson PC, Ponte C, et al. American College of Rheumatology/European Alliance of associations for rheumatology classification criteria for eosinophilic granulomatosis with Polyangiitis. *Annal Rheum Dis*. 2022;81:309-314.
10. Robson JC, Grayson PC, Ponte C, et al. American College of Rheumatology/European Alliance of associations for rheumatology classification criteria for granulomatosis with polyangiitis. *Annal Rheumat Dis*. 2022;81:315-320.
11. Robson JC, Grayson PC, Ponte C, et al. American College of Rheumatology/European Alliance of associations for rheumatology classification criteria for microscopic polyangiitis. *Annal Rheum Dis*. 2022;81:321-326.
12. Allena N, Patel J, Nader G, Patel M, Medvedovsky B. A rare case of SARS-CoV-2-induced microscopic Polyangiitis. *Cureus*. 2021;13(5):e15259.
13. Babu SS, Senthilnathan G, Shah SN, Annigeri RA. Double anti-neutrophil cytoplasmic antibody and anti-glomerular basement membrane antibody-positive Crescentic glomerulonephritis, following SARS-CoV-2 infection. *Indian J Nephrol*. 2022;32(5):491-494.
14. Bołtuć K, Bielejewska A, Coloma-Millar A, et al. Case report: cyclophosphamide in COVID—when an absolute contraindication is an absolute necessity. *F1000Res*. 2021;10:829.
15. Cobilinschi C, Cobilinschi C, Constantinescu A, Draniceanu I, Ionescu R. New-onset ANCA-associated Vasculitis in a patient with SARS-COV2. *Balkan Med J*. 2021;38(5):318-320.
16. Felzer JR, Fogwe DT, Samrah S, et al. Association of COVID-19 antigenicity with the development of antineutrophilic cytoplasmic antibody vasculitis. *Respirol Case Rep*. 2022;10(1):e0894.
17. Fireizen Y, Shahriary C, Imperial ME, Randhawa I, Nianiaris N, Ovunc B. Pediatric P-ANCA vasculitis following COVID-19. *Pediatric Pulmonol*. 2021;56(10):3422-3424.
18. Giles T, Roy SP, Chandrasoma D, Oakley S, Lynnhtun K, Draganic B. Life-threatening gastrointestinal haemorrhage requiring surgical resection caused by SARS-CoV-2 induced ANCA associated vasculitis: a case report. *Int J Surg Case Rep*. 2022;98:107491.
19. Izci Duran T, Turkmen E, Dilek M, Sayarlioglu H, Arik N. ANCA-associated vasculitis after COVID-19. *Rheumatol Int*. 2021;41(8):1523-1529.
20. Jalalzadeh M, Valencia-Manrique JC, Boma N, Chaudhari A, Chaudhari S. Antineutrophil cytoplasmic antibody-associated glomerulonephritis in a case of scleroderma after recent diagnosis with COVID-19. *Cureus*. 2021;13(1):e12485.
21. Kataria S, Rogers S, Sadia H, et al. Antineutrophil cytoplasmic antibody (ANCA)-associated renal Vasculitis after COVID-19 infection: a case report. *Cureus*. 2022;14(6):e26111.
22. Kawashima S, Kishimoto M, Hibino T, et al. MPO-ANCA-positive microscopic Polyangiitis following COVID-19 infection. *Int Med*. 2022;61(4):567-570.
23. Lind E, Jameson A, Kurban E. Fulminant granulomatosis with polyangiitis presenting with diffuse alveolar haemorrhage following COVID-19. *BML Case Rep*. 2021;14(6):1-3.
24. Madanchi N, Stingo FE, Patrick KC, et al. Possible association between COVID-19 infection and De novo Antineutrophil cytoplasmic antibody-associated Vasculitis. *Cureus*. 2021;13(12):e20331.
25. Maritati F, Moretti MI, Nastasi V, et al. ANCA-associated glomerulonephritis and anti-phospholipid syndrome in a patient with SARS-CoV-2 infection: just a coincidence? *Case Rep Nephrol Dial*. 2021;11(2):214-220.
26. Merveilleux du Vignaux C, Ahmad K, Tantot J, Rouach B, Traclet J, Cottin V. Evolution from hypereosinophilic bronchiolitis to eosinophilic granulomatosis with polyangiitis following COVID-19: a case report. *Clin and Exp Rheumatol*. 2021;39 Suppl 128(1):11-12.
27. Morris D, Patel K, Rahimi O, Sanyurah O, Iardino A, Khan N. ANCA vasculitis: a manifestation of post-Covid-19 syndrome. *Resp Med Case Rep*. 2021;34:101549.
28. Powell WT, Campbell JA, Ross F, Peña Jiménez P, Rudzinski ER, Dickerson JA. Acute ANCA Vasculitis and asymptomatic COVID-19. *Pediatrics*. 2021;147(4):1-3.
29. Reiff DD, Meyer CG, Marlin B, Mannion ML. New onset ANCA-associated vasculitis in an adolescent during an acute COVID-19 infection: a case report. *BMC Pediatrics*. 2021;21(1):333.
30. Selvaraj V, Moustafa A, Dapaah-Afriyie K, Birkenbach MP. COVID-19-induced granulomatosis with polyangiitis. *BML Case Rep*. 2021;14(3):1-3.
31. Singh S, Vaghaiwalla Z, Thway M, Kaeley GS. Does withdrawal of immunosuppression in rheumatoid arthritis after SARS-CoV-2 infection increase the risk of vasculitis? *BML Case Rep*. 2021;14(4):1-4.
32. Ta H, Awada H, Kang P, et al. Antineutrophil cytoplasmic autoantibody (ANCA)-associated Vasculitis with mucosal involvement following COVID-19 pneumonia. *Cureus*. 2022;14(11):e31441.
33. Thu Aung Z, Oluyombo R, Karim M, Wong Sun Wai J, Ugni S. SARS-CoV-2 infection: a forerunner or precursor in antineutrophil cytoplasmic antibody-associated Vasculitis with kidney injury. *Cureus*. 2022;14(9):e28705.
34. Uppal NN, Kello N, Shah HH, et al. De novo ANCA-associated Vasculitis with glomerulonephritis in COVID-19. *Kidney Int Rep*. 2020;5(11):2079-2083.
35. Wali HA, Tabb D, Baloch SA. Diffuse alveolar hemorrhage associated with severe acute respiratory syndrome coronavirus 2. *Cureus*. 2021;13(12):e20171.
36. Wang E, Lee T, Sharma P, Epstein E, Bouland D, Gupta V. The perfect storm: diffuse alveolar hemorrhage after SARS-COV-2 infection. *Chest*. 2021;160(4):A1212-A1213.

37. Asma H, Mariem BS, Nouha BM, et al. POS-930 ANCA associated vasculitis with CRESCENTIC glomerulonephritis complicating COVID 19 in a 72-year-old man. *Kidney Int Rep*. 2022;7(2):S406.
38. Garcia Vega A, Santoyo-Fexas L, Maya-Quintá R. POS-851 MPO-ANCA associated vasculitis after COVID-19. *Kidney Int Rep*. 2022;7(2):S367-S368.
39. Patel K, Morris D, Iardino A. Post-COVID-19 complications: hemoptysis in a middle-aged man. *TP31 TP031 Interesting Cases Associated with SARS-COV-2 Infection*. American Thoracic Society; 2021:A2012.
40. Zakrocka I, Korolczuk A, Załuska W. Antineutrophil cytoplasmic autoantibody-associated vasculitis with rapid progressive glomerulonephritis following SARS-CoV-2 infection: a cause or coincidence. *Pol Arch Intern Med*. 2022;132:e242-e245.
41. Wintler T, Zherebtsov M, Carmack S, Muntean R, Hill SJ. Acute PR3-ANCA vasculitis in an asymptomatic COVID-19 teenager. *J Pedia Surg Case Rep*. 2021;75:102103.
42. Chargui S, Mbarek M, Harzallah A, Aouidia R, Ouinissi M, Abdallah TB. POS-172 renal thrombotic microangiopathy, ANCA vasculitis and COVID 19 infection: an intriguing association. *Kidney Int Rep*. 2021;6(4):S70.
43. Fares E, Pathak K, Damiano C, Kuntz C. Diffuse alveolar hemorrhage as a consequence of microscopic polyangiitis due to COVID-19. *Chest*. 2020;158(4):A775.
44. Hussein A, Khalil KA, Bawazir YM. Anti-neutrophilic cytoplasmic antibody (ANCA) vasculitis presented as pulmonary hemorrhage in a positive COVID-19 patient: a case report. *Cureus*. 2020;12(8):1-5.
45. Moeinzadeh F, Dezfouli M, Naimi A, Shahidi S, Moradi H. Newly diagnosed glomerulonephritis during COVID-19 infection undergoing immunosuppression therapy, a case report. *Iranian Journal of Kidney Diseases*. 2020;14(3):239-242.
46. Kostelníková P, Skácelová M, Špíšek M, Šimíček M, Horák P. Diffuse alveolar hemorrhage as a life threatening manifestation of newly diagnosed granulomatosis with polyangiitis following COVID-19 infection—a case report. *Vnitřní Lékařství*. 2022;68(5):290-294.
47. Caglayan S, Yener GO, Ulu K, et al. A pediatric case of Takayasu's arteritis with anti-neutrophil cytoplasmic antibody-associated vasculitis triggered by COVID-19 infection. *North Clin Istanb*. 2023;10(3):393-397.
48. Chandok T, Nasr R, Uday KA. A case of Antineutrophil cytoplasmic antibody Vasculitis-associated acute kidney injury in a patient with asymptomatic COVID-19 infection. *Cureus*. 2023;15(2):e35006.
49. Kaynar K, Güvercin B, Demir S, Mungan S, Çifçi ET. A case report of antineutrophil cytoplasmic antibody-associated vasculitis and glomerular immune depositions. *Hippokratia*. 2022;26(2):86-88.
50. Shah V, Patel H, Oza J, et al. Atypical immunologic manifestations of COVID-19: a case report and narrative review. *SN Comprehensive Clinical Medicine*. 2023;5(1):108.
51. Tahir A, Walia J, Daly T, Gradzka A, Banai R. Rapidly progressive glomerulonephritis: a COVID-19 case report. *Cureus*. 2023;15(4):e37767.
52. Manivannan S, Jain K. ANCA vasculitis presenting as hemoptysis post COVID-19 infection. *J Am Soc Nephrol*. 2021;32:92.
53. Geetha D, Jefferson JA. ANCA-associated Vasculitis: Core curriculum 2020. *Am J Kid Dis*. 2020;75(1):124-137.
54. Nayebirad S, Ramandi A, Nili F, et al. Glomerulonephritis associated with systemic sclerosis: a case report. *J Med Case Rep*. 2023;17(1):1-5.
55. Whittaker A, Anson M, Harky A. Neurological manifestations of COVID-19: a systematic review and current update. *Acta Neurologica Scandinavica*. 2020;142(1):14-22.
56. Jiang F, Deng L, Zhang L, Cai Y, Cheung CW, Xia Z. Review of the clinical characteristics of coronavirus disease 2019 (COVID-19). *J Gen Int Med*. 2020;35(5):1545-1549.
57. Elmakaty I, Ferih K, Karen O, et al. Clinical implications of COVID-19 presence in CSF. *Syst Rev Case Rep Cel*. 2022;11(20):1-5.
58. Lim SH, Ju HJ, Han JH, et al. Autoimmune and autoinflammatory connective tissue disorders following COVID-19. *JAMA Network Open*. 2023;6(10):e2336120.
59. Zirpe KG, Dixit S, Kulkarni AP, et al. Pathophysiological mechanisms and neurological manifestations in COVID-19. *Indian J Crit Care Med*. 2020;24(10):975-980.
60. Jha NK, Ojha S, Jha SK, et al. Evidence of coronavirus (CoV) pathogenesis and emerging pathogen SARS-CoV-2 in the nervous system: a review on neurological impairments and manifestations. *Journal of Molecular Neuroscience*. 2021;71(11):2192-2209.
61. Wenting A, Gruters A, van Os Y, et al. COVID-19 neurological manifestations and underlying mechanisms: a scoping review. *Frontiers in Psychiatry*. 2020;11:860.
62. Stojanovic M, Barac A, Petkovic A, et al. Large-vessel Giant cell arteritis following COVID-19-what can HLA Typing Reveal ? *Diagnostics (Basel)*. 2023;13(3):1-4.
63. Timmons GM, Rempe T, Bevins EA, et al. CNS lymphocytic Vasculitis in a young woman with COVID-19 infection. *Neurol Neuroimmunol Neuroinflamm*. 2021;8(5):1-3.
64. Martín MJ. Cryoglobulinemic purpura and COVID-19 infection. *Med Clin (Engl Ed)*. 2022;159(10):e68-e69.

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