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CKJ Review

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CKJ REVIEW

ANCA-associated vasculitis flare might be provoked by COVID-19 infection: a case report and a review of the literature

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ABSTRACT

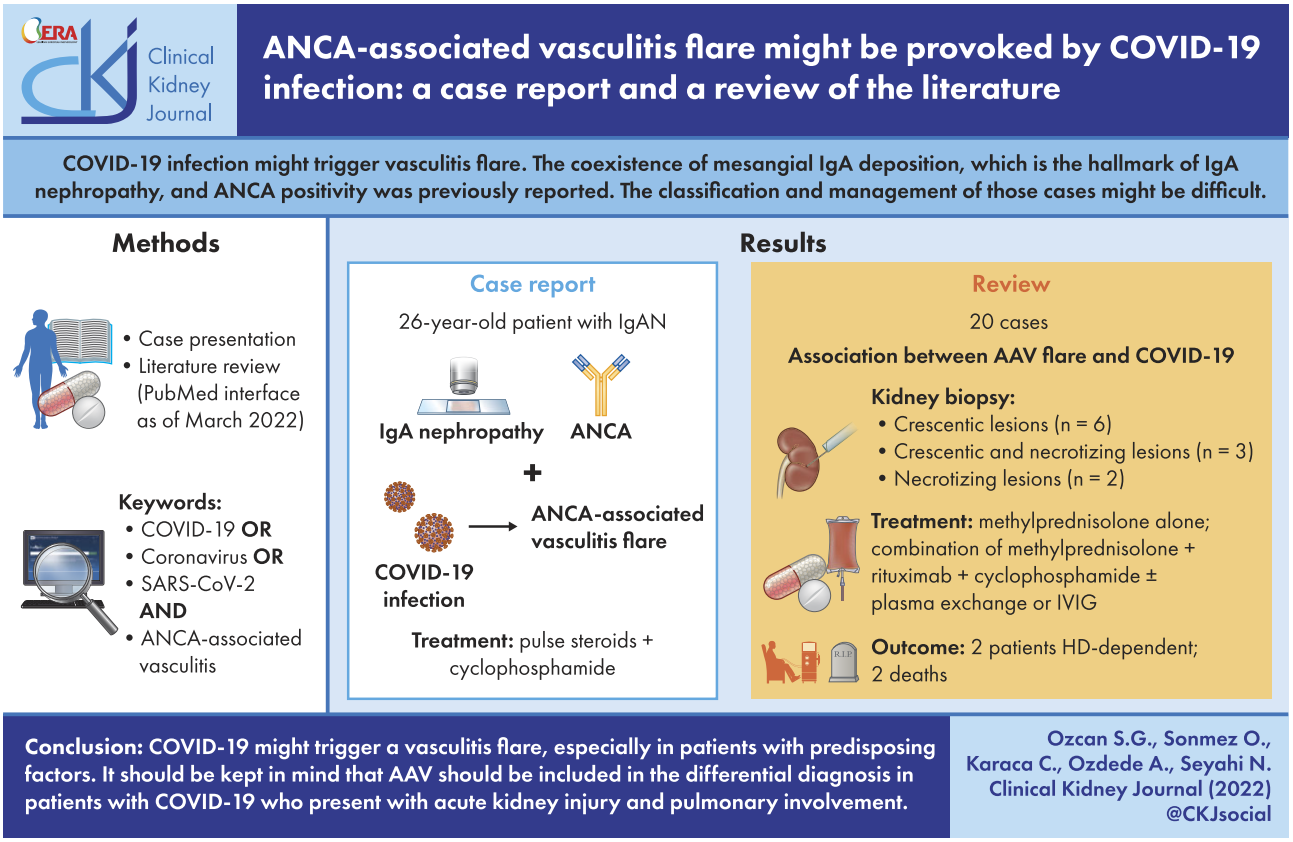
Mesangial immunoglobulin A (IgA) deposition is the hallmark of IgA nephropathy (IgAN). In some cases, crescentic involvement that might be associated with systemic leucocytoclastic vasculitis is documented. In such cases, the disease is called Henoch–Schönlein purpura (IgA vasculitis). Even more rarely, the coexistence of IgAN and anti-neutrophil cytoplasmic antibody (ANCA) seropositivity has been reported.

IgAN might be complicated by acute kidney injury (AKI) due to different causes. Herein we present a patient with mesangial IgA deposition and ANCA seropositivity who developed AKI, haematuria and haemoptysis during the course of coronavirus disease 2019 (COVID-19) disease and was diagnosed with ANCA-associated vasculitis based on clinical, laboratory and radiological findings. The patient was treated successfully with immunosuppressive therapy. We also made a systematic review of the literature to reveal and present the cases with COVID-19 and ANCA-associated vasculitis.

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GRAPHICAL ABSTRACT



Keywords: ANCA, ANCA-associated vasculitis, COVID-19, IgA nephropathy, vasculitis

INTRODUCTION

Coronavirus disease 2019 (COVID-19) may trigger the development or exacerbation of autoimmune diseases [1, 2]. Anti-neutrophil cytoplasmic antibody (ANCA)-associated vasculitis (AAV) affects small and medium-sized arteries and can progress to both kidney and lung involvement due to production of autoantibodies against the antigens myeloperoxidase (MPO) and

Table 1: Laboratory values obtained at the first admission (September 2020).

Parameters	Values	Reference range
Serum urea/creatinine (mg/dL)	37/0.99	17–49/0.5–0.9
24-hour urinalysis		
Creatinine clearance (ml/min)	218	
Total protein (mg/day)	3829	<140
Microalbumin (mg/day)	3330	<30
24-hour urine volume (ml)	1850	
Complete urinalysis	++	
Protein	+++	
Erythrocytes	5 white blood cells, abundant red blood cells, rare squamous epithelial cells	
Sediment	4.44	3.5–5.2
Serum albumin (g/dl)	13.4/39.6	12–16/36–48
Haemoglobin (g/dl)/haematocrit (%)	245.5	70–400
Serum IgA (mg/dl)	Negative	
ANA	12.5	<16
Anti-dsDNA (IU/ml)	1.47/0.3 (normal)	0.9–1.8/0.1–0.4
C3/C4 (g/l)	1.98	Negative: <12; borderline: 12–18; positive: >18
PR3-ANCA (IU/ml)	173	Negative: <12; borderline: 12–18; positive: >18
MPO-ANCA (IU/ml)		

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Table 2: Laboratory values at the time of diagnosis of COVID-19 (January 2022) and after treatment (February 2022).

Characteristics	On admission	After treatment	Reference range
Serum urea/creatinine (mg/dl)	57/2.48	51/0.99	17–49/0.5–0.9
24-hour urinalysis			
Creatinine clearance (ml/min)	NA	105	
Total protein (mg/day)		10 456	<140
Microalbumin (mg/day)		9687	<30
24-hour urine volume (ml)		2400	
Complete urinalysis	+++	+++	
Protein	+++	+++	
Erythrocytes	10 white blood cells, abundant red blood cells, 2 squamous epithelial cells	3 white blood cells, 27 red blood cells, 1 renal epithelial cell	
Serum albumin (g/dl)	3.48	3.15	3.5–5.2
CRP (mg/l)	67.2	0.84	<5
Ferritin (ng/ml)	391	97	30–400
D-dimer (mg/l)	0.94	0.83	0–0.5
Fibrinogen (mg/dl)	900	284,5	180–350
Haemoglobin (g/dl)/haematocrit (%)	11.1/32.4	13.3/37.9	12–16/36–48
Leucocytes (10^3/µl)	7300	14 300	4.3–10.3
Lymphocytes (10^3/µl)	800	2600	1.5–3.5
ANA	Negative	NA	
Anti-dsDNA (IU/ml)	8.38 (negative)	NA	<16
C3/C4 (g/l)	1.26/0.37 (normal)	NA	0.9–1.8/0.1–0.4
PR3-ANCA/MPO-ANCA (IU/ml)	0.56/235 (normal/increased)	NA	Negative: <12; borderline: 12–18; positive: >18

proteinase 3 (PR3) [3]. There are cases in the literature showing that COVID-19 triggers the development of AAV [4–12].

In this report we discuss a case with mesangial immunoglobulin A (IgA) deposition on kidney biopsy and ANCA seropositivity who developed acute kidney injury (AKI), haematuria and haemoptysis during COVID-19 infection and was diagnosed with AAV based on clinical, laboratory and radiological findings.

CASE PRESENTATION

A 26-year-old male patient presented to our nephrology clinic and was diagnosed with IgA nephropathy (IgAN) by renal biopsy on admission in September 2020, with 2 g/day proteinuria and haematuria. He had no history of chronic disease, smoking, alcohol, herbal substance or drug use. The kidney sizes, echogenicity and parenchymal thickness were normal on the urinary ultrasound. The laboratory values obtained at the first admission are shown in Table 1. His anti-nuclear antibody (ANA), anti-double-stranded DNA (dsDNA) and proteinase 3 (PR3)-ANCA were negative, while his myeloperoxidase (MPO)-ANCA was positive and complement 3 (C3) and C4 were normal. The total protein in 24-hour urine was 3829 mg/day. On the kidney biopsy there were 10 glomeruli, 2 of which were global sclerotic. Light microscopic examination revealed mesangial proliferation, with no crescents and no findings related to necrotizing vasculitis. According to immunofluorescence microscopy, there was (+++) IgA deposition in the mesangium and no staining with C1q. The Oxford classification for IgAN was M1E1S1T0. Despite the high MPO levels, the patient did not have any systemic vasculitis–related findings. Secondary causes of IgA were ruled out based on the clinical and laboratory data. The patient was started on ramipril 10 mg/day. On follow-up, prednisolone 40 mg/day was started due to the increase in proteinuria (total protein in 24-hour urine, 5357 mg/day; microalbumin, 4788 mg/day). Mycophenolate mofetil was added to the treatment in April 2021, due to persistent high levels of proteinuria (4075 mg/day). However, since persistent proteinuria continued, the patient's current immunosuppressive drug treatment was discontinued and he was accepted as unresponsive to current treatment in November 2021. The patient had two doses of Pfizer-BioNTech (BNT162b2) Comirnaty vaccine, with the last one given in June 2021.

In January 2022, the patient presented with complaints of severe progressive shortness of breath, haemoptysis, haematuria, dry cough, weakness and loss of appetite. His oxygen saturation in ambient air was 90% on admission. On auscultation, crepitant crackles were present in the mid and basal zones of the bilateral lungs. The laboratory results are presented in Table 2. The laboratory findings revealed AKI, normochromic normocytic anaemia, lymphopenia and increased C-reactive protein (CRP) and D-dimer levels. The urinalysis revealed an active sediment with haematuria and proteinuria. The patient was diagnosed with COVID-19 based on a reverse transcription polymerase chain reaction (RT-PCR) assay for deVere acute respiratory syndrome coronavirus 2 (SARS-CoV-2) on a nasopharyngeal swab. On chest computed tomography (CT), diffuse and predominantly peribronchovascular areas with ground glass opacity and consolidations were observed in both lung parenchymas and the findings were compatible with alveolar haemorrhage (Fig. 1). Subsequently the patient was hospitalized at the clinic for COVID-19 patients. During the follow-up, his haematocrit level decreased from 32.4% to 27.4% and his haemoglobin level decreased from 11.1 g/dl to 9.1 g/dl. The remeasured MPO-ANCA value increased to 235 IU/ml; other rheumatological markers [ANA, anti-dsDNA, rheumatoid factor, anti-cyclic citrullinated peptide, antiphospholipid syndrome antibodies, anti-Sm, anti Scl-70, anti-glomerular basement membrane

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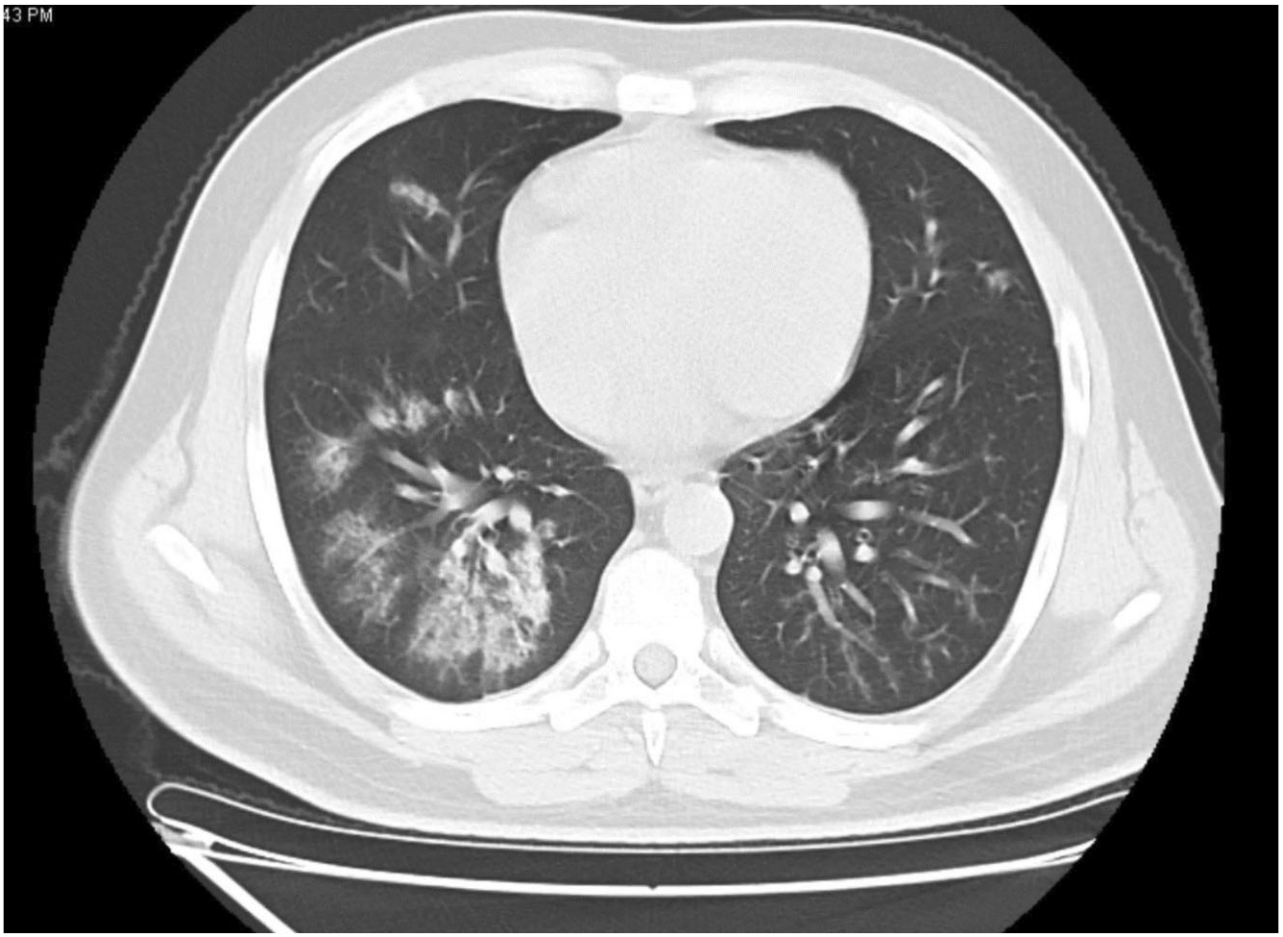


Figure 1: Ground glass opacities in the peribronchovascular areas and consolidations.

(GBM)] were negative. Increased MPO-ANCA level, AKI, alveolar haemorrhage and chest CT findings led to the diagnosis of AAV. Therefore 1000 mg/day of intravenous methylprednisolone was administered for 3 days, which led to remarkable symptomatic improvement in oxygen saturation levels. After pulse steroid treatment, the decrease in the haematocrit level stopped and complaints of haemoptysis and haematuria were significantly reduced. Prednisolone 1 mg/kg/day was initiated. In addition, cyclophosphamide administration 1000 mg/month was planned. Post-treatment laboratory values are shown in Table 2.

LITERATURE REVIEW

The presence of COVID-19 and AAV in the same patient might be a coincidence, however, the presence of similar previous cases might favour an association. Therefore we made a review of the literature.

We used the PubMed interface (pubmed.gov) to query using the combination of the following two keyword groups. The first group included the keywords 'COVID-19', 'coronavirus' and 'SARS-CoV-2' and the second group included the keyword 'ANCA-associated vasculitis'. Each keyword in the same group was combined using the logical operator OR, while the two groups were combined using the logical operator AND. We excluded reviews, editorials and case reports that were about COVID vaccine–related AAV.

We ran the query in March 2022 and found 102 articles in total that we manually examined. We excluded the reviews and editorials after reading the titles and abstracts. Then we read the full texts of all the articles and excluded the ones that were about COVID-19 vaccine–related AAV or those that included cases where the ANCA status was not reported or tested (Fig. 2).

The manual examination found 17 case reports describing 19 cases of AAV related to COVID-19 disease (Table 3).

DISCUSSION

We discussed a case that developed alveolar haemorrhage, haematuria, AKI and MPO-ANCA positivity in the course of COVID-19 and was diagnosed with AAV. There are few publications showing the development of vasculitis in patients with COVID-19 infection [4–23]. Our patient also received two doses of the messenger RNA (mRNA) COVID-19 vaccine. There are previous reports suggesting AAV flare following COVID-19 vaccine [24–27]. In most of the cases, the vaccine used was an mRNA vaccine [28]. However, in our case, the last vaccine dose was 6 months before the AAV flare. Therefore, in our patient, the temporal association favours a causal association between AAV flare and COVID-19 infection rather than an association between AAV flare and COVID-19 vaccine.

The coincidence of IgAN and AAV is a rare but previously reported condition [29–31]. It was reported that all ANCAs

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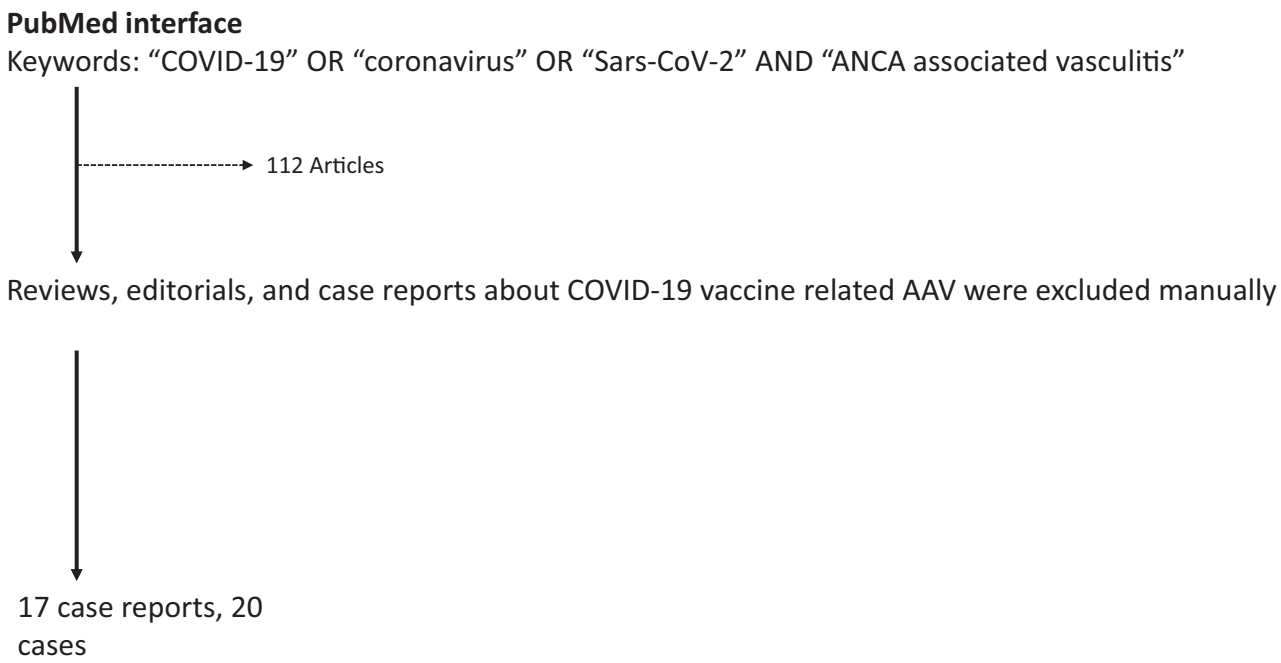


Figure 2: Steps of the literature review.

might not be associated with the pathology of IgAN and the classification of those overlap cases might be cumbersome. At the initial presentation, our patient did not have any systemic symptoms related to vasculitis and there was no crescentic involvement or necrotizing vasculitis on the kidney biopsy. Therefore IgA glomerulonephritis was considered in the patient with significant mesangial IgA deposition and the treatment was done accordingly. However, during the follow-up, the clinical evolution of the case suggested that the patient might be classified as renal-limited MPO-ANCA-associated vasculitis at the initial presentation.

According to our literature review, a total of 20 cases, including our case, showed an association between AAV flare and COVID-19. Obviously it is hard to infer causality based on these reports, however, a close temporal association might be considered a factor that favours causality. In 12 of the cases, COVID-19 and AVV flare were simultaneous. In the remaining eight cases, durations of 25 days to 6 months were reported (Table 3). Ten cases were male, with a median age of 46 years (range 17–86 years). Kidney biopsy was performed in 14 cases and crescentic lesions, crescentic and necrotizing lesions and necrotizing lesions were observed in 6, 3 and 2 of the cases, respectively. Methylprednisolone alone or the combination of methylprednisolone with rituximab, cyclophosphamide, plasma exchange and intravenous immunoglobulin (IVIG) were the preferred treatments. Eighteen cases were alive, of which two were haemodialysis dependent, and two cases died (Table 3).

The pathogenesis of COVID-19-induced vasculitis is unclear. However, the formation of neutrophil extracellular traps (NETs) that might be triggered in COVID-19 disease was proposed as a mechanism for the development of AAV [32]. There are high levels of NETs in the circulation in patients with AAV, as shown in the kidney biopsies of these patients [33]. NETs contain pro-inflammatory proteins and are directly associated with endothelial cell damage and complement system activation. It has been suggested that PR3 and MPO contribute indirectly to the development of vasculitis through the production of ANCA. We want to point out that our patient had a high level of MPO before COVID-19 infection, which might be a predisposing factor for AAV development [34].

It is difficult to distinguish between the kidney damage that develops during the course of COVID-19 disease and the kidney damage that develops due to AAV. Many different mechanisms have been proposed that lead to the development of kidney damage in COVID-19 patients. Of them, haemodynamic factors and endothelial dysfunction lead to viral tropism in the kidney tissue, fibrinoid necrosis and microthrombosis. In addition to the direct cytopathic effects of SARS-CoV-2 on the glomeruli and renal tubules, indirect effects such as cellular immunity and cytokine storm play a role in kidney damage [11]. Unfortunately, we did not perform a second kidney biopsy during the flare. Therefore we cannot reveal the exact cause of kidney damage in our patient. There are some other limitations in this current report, e.g. the patient was referred from a different centre. Therefore we do not have the results of CRP, ESR, cryoglobulin anti-GBM at the first presentation and electron microscopic examination was not performed in the kidney biopsy. Moreover, MPO titre after treatment is not available.

Considering the severity of the patient’s pulmonary AAV, cyclophosphamide was administered together with glucocorticoid therapy as the standard treatment, and the patient responded dramatically. Despite the presence of COVID-19 infection, as observed in similar cases in the literature, our patient also had a good response to immunosuppressive therapy.

In conclusion, COVID-19 might trigger a vasculitis flare, especially in patients with predisposing factors. It should be kept in mind that AAV should be included in the differential diagnosis in patients with COVID-19 who present with AKI and pulmonary involvement.

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Table 3: Review of the COVID-19-related AAV cases (sorted by the release date from oldest to newest).

Reference	Country	Age (years)	Gender	Time between COVID-19 and AAV	Serology	Lung involvement	Renal biopsy	Treatment	Outcome
Moeinzadeh *et al.* [11]	Iran	25	Male	Simultaneous	c-ANCA	Alveolar haemorrhage	Crescentic GN	Methylprednisolone + plasma exchange, cyclophosphamide	Living
Uppal *et al.* [5]	USA (African American)	64	Male	Simultaneous	MPO (p-ANCA)	Bilateral patchy infiltrates	Crescentic GN	Methylprednisolone + rituximab	Living
Uppal *et al.* [5]	USA (South Asian)	46	Male	Simultaneous	PR3 (c-ANCA)	Resolving peripheral ground glass opacities	Focal necrotizing DN	Methylprednisolone + rituximab	Living
Hussein *et al.* [6]	Saudi Arabia	37	Female	Simultaneous	PR3 (c-ANCA)	Alveolar haemorrhage	No, AKI	Methylprednisolone + plasma exchange, IVIG	Died
Jalalzadeh *et al.* [13]	USA (Hispanic)	46	Female	6 months	MPO (p-ANCA)	Bilateral pleural effusions and pulmonary infiltrates	Crescentic GN, pauci-immune	Methylprednisolone	Living
Selvaraj *et al.* [14]	USA	60	Female	4 weeks	PR3 (c-ANCA)	Alveolar haemorrhage	Crescentic and necrotizing GN, TIN	Methylprednisolone + plasma exchange, rituximab	Living
Izci Duran *et al.* [7]	Turkey	26	Male	Simultaneous	MPO (p-ANCA)	Subpleural and parenchymal dispersed consolidative ground glass opacities	Crescentic GN	Methylprednisolone + cyclophosphamide, plasma exchange	Living, HD dependent
Izci Duran *et al.* [7]	Turkey	36	Female	Simultaneous	PR3 (c-ANCA)	Bilateral cavitary lesions	Necrotizing crescentic GN	Methylprednisolone + cyclophosphamide	Living
Lind *et al.* [15]	USA	40	Male	5 weeks	PR3 (c-ANCA)	Alveolar haemorrhage	Focal crescentic GN	Methylprednisolone + rituximab	Living
Allena *et al.* [9]	USA	60	Female	Simultaneous	MPO (p-ANCA)	Alveolar haemorrhage	Focal segmental necrotizing, crescentic and sclerosing GN, pauci-immune type	Methylprednisolone + plasma exchange, rituximab	Living

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Table 3: Continued

Reference	Country	Age (years)	Gender	Time between COVID-19 and AAV	Serology	Lung involvement	Renal biopsy	Treatment	Outcome
Reiff *et al.* [16]	USA	17	Male	Simultaneous	PR3 (c-ANCA)	Pulmonary nodules	No, normal kidney function	Methylprednisolone + rituximab	Living
Maritati *et al.* [10]	Italy	64	Female	Simultaneous	PR3 (c-ANCA)	Bilateral interstitial pneumonia with ground glass opacities	Pauci-immune GN	Methylprednisolone + plasma exchange, cyclophosphamide	Living
Fireizen *et al.* [17]	USA	17	Male	2 months	MPO (p-ANCA)	Alveolar haemorrhage	Necrotizing GN with limited immune complex deposition	Methylprednisolone + cyclophosphamide	Living
Cobilinschi *et al.* [18]	Romania	67	Female	Simultaneous	p-ANCA	No	No, AKI	Methylprednisolone + cyclophosphamide	Living
Morris *et al.* [19]	USA (Hispanic)	53	Male	1 month	PR3 (c-ANCA), MPO ANCA (mildly +)	Multifocal pneumonia, alveolar haemorrhage	No, AKI	Methylprednisolone	Died
Kawashima *et al.* [20]	Japan	61	Female	3 months	MPO (p-ANCA)	No	Highly active nephritis associated with AAV	Methylprednisolone + cyclophosphamide	Living
Felzer *et al.* [21]	USA	86	Female	Simultaneous	MPO (p-ANCA)	Alveolar haemorrhage	No, AKI	Methylprednisolone + rituximab	Living
Wali *et al.* [22]	USA	26	Female	25 days	p-ANCA	Alveolar haemorrhage	GN associated with AAV	Methylprednisolone + rituximab	Living
Madanchi *et al.* [23]	USA	53	Male	4 months	MPO (p-ANCA)	Alveolar haemorrhage	Pauci-immune fibrocellular crescentic GN on top of glomerular sclerosis	Methylprednisolone + cyclophosphamide	Living, HD dependent
Current case	Turkey	26	Male	Simultaneous	MPO (p-ANCA)	Alveolar haemorrhage	No, AKI	Methylprednisolone + cyclophosphamide	Living

c-ANCA: cytoplasmic ANCA; GN: glomerulonephritis; p-ANCA: perinuclear ANCA; TIN: tubulointerstitial nephritis.

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The present work was conducted in accordance with the Declaration of Helsinki.

Informed consent was obtained from the patient for publication of this case report and accompanying images.

DATA AVAILABILITY STATEMENT

All data generated or analysed during this study are included in this article.

CONFLICT OF INTEREST STATEMENT

The authors have no conflicts of interest to declare.

REFERENCES

1. Caso F, Costa L, Ruscitti P *et al*. Could Sars-coronavirus-2 trigger autoimmune and/or autoinflammatory mechanisms in genetically predisposed subjects? *Autoimmun Rev* 2020;**19**:102524. https://doi.org/10.1016/j.autrev.2020.102524
2. Gracia-Ramos AE, Saavedra-Salinas MÁ. Can the SARS-CoV-2 infection trigger systemic lupus erythematosus? A case-based review. *Rheumatol Int* 2021;**41**:799–809. https://doi.org/10.1007/s00296-021-04794-7
3. Jennette JC, Falk R, Bacon P *et al*. 2012 revised International Chapel Hill Consensus Conference Nomenclature of Vasculitides. *Arthritis Rheum* 2013;**65**:1–11.
4. Tahir A, Sohail Z, Nasim B *et al*. Widespread cutaneous small vessel vasculitis secondary to COVID-19 infection. *Int J Dermatol* 2020;**59**:1278–9. https://doi.org/10.1111/ijd.15106
5. Uppal NN, Kello N, Shah HH *et al*. De novo ANCA-associated vasculitis with glomerulonephritis in COVID-19. *Kidney Int Rep* 2020;**5**:2079–83. https://doi.org/10.1016/j.ekir.2020.08.012
6. 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**:e9643.
7. Izci Duran T, Turkmen E, Dilek M *et al*. ANCA-associated vasculitis after COVID-19. *Rheumatol Int* 2021;**41**:1523–9. https://doi.org/10.1007/s00296-021-04914-3
8. Powell WT, Campbell JA, Ross F *et al*. Acute ANCA vasculitis and asymptomatic COVID-19. *Pediatrics* 2021;**147**:e2020033092. https://doi.org/10.1542/peds.2020-033092
9. Allena N, Patel J, Nader G *et al*. A rare case of SARS-CoV-2-induced microscopic polyangiitis. *Cureus* 2021;**13**:e15259.
10. 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**:214–20. https://doi.org/10.1159/000517513
11. Moeinzadeh F, Dezfouli M, Naimi A *et al*. Newly diagnosed glomerulonephritis during COVID-19 infection undergoing immunosuppression therapy, a case report. *Iran J Kidney Dis* 2020;**14**:239–42.
12. Nasr SH, Kopp JB. COVID-19–associated collapsing glomerulopathy: an emerging entity. *Kidney Int Rep* 2020;**5**:759–61. https://doi.org/10.1016/j.ekir.2020.04.030
13. Jalalzadeh M, Valencia-Manrique JC, Boma N *et al*. Antineutrophil cytoplasmic antibody-associated glomerulonephritis in a case of scleroderma after recent diagnosis with COVID-19. *Cureus* 2021;**13**:e12485.
14. Selvaraj V, Moustafa A, Dapaah-Afriyie K *et al*. COVID-19-induced granulomatosis with polyangiitis. *BMJ Case Rep* 2021;**14**:e242142. https://doi.org/10.1136/bcr-2021-242142
15. Lind E, Jameson A, Kurban E. Fulminant granulomatosis with polyangiitis presenting with diffuse alveolar haemorrhage following COVID-19. *BMJ Case Rep* 2021;**14**:e242628. https://doi.org/10.1136/bcr-2021-242628
16. Reiff DD, Meyer CG, Marlin B *et al*. New onset ANCA-associated vasculitis in an adolescent during an acute COVID-19 infection: a case report. *BMC Pediatr* 2021;**21**:333. https://doi.org/10.1186/s12887-021-02812-y
17. Fireizen Y, Shahriary C, Imperial ME *et al*. Pediatric P-ANCA vasculitis following COVID-19. *Pediatr Pulmonol* 2021;**56**:3422–4. https://doi.org/10.1002/ppul.25612
18. Cobilinschi C, Cobilinschi C, Constantinescu A *et al*. New-onset ANCA-associated vasculitis in a patient with SARS-COV2. *Balkan Med J* 2021;**38**:318–20. https://doi.org/10.5152/balkanmedj.2021.21129
19. Morris D, Patel K, Rahimi O *et al*. ANCA vasculitis: a manifestation of post-Covid-19 syndrome. *Respir Med Case Rep* 2021;**34**:101549. https://doi.org/10.1016/j.rmcr.2021.101549
20. Kawashima S, Kishimoto M, Hibino T *et al*. MPO-ANCA-positive microscopic polyangiitis following COVID-19 infection. *Intern Med* 2022;**61**:567–70.
21. 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**:e0894. https://doi.org/10.1002/rcr2.894
22. Wali H, Tabb D, Baloch SA. Diffuse alveolar hemorrhage (DAH) associated with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2): a case report. *Cureus* 2021;**13**:e20171.
23. 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**:e20331.
24. Shakoor MT, Birkenbach MP, Lynch M. ANCA-associated vasculitis following Pfizer-BioNTech COVID-19 vaccine. *Am J Kidney Dis* 2021;**78**:611–3. https://doi.org/10.1053/j.ajkd.2021.06.016
25. Villa M, Díaz-Crespo F, de José AP *et al*. A case of ANCA-associated vasculitis after AZD1222 (Oxford–AstraZeneca) SARS-CoV-2 vaccination: casualty or causality? *Kidney Int* 2021;**100**:937. https://doi.org/10.1016/j.kint.2021.07.026
26. Hakroush S, Tampe B. Case report: ANCA-associated vasculitis presenting with rhabdomyolysis and pauci-immune crescentic glomerulonephritis after Pfizer-BioNTech COVID-19 mRNA vaccination. *Front Immunol* 2021;**12**:762006.
27. Feghali EJ, Zafar M, Abid S *et al*. De-novo antineutrophil cytoplasmic antibody-associated vasculitis following the mRNA-1273 (Moderna) vaccine for COVID-19. *Cureus* 2021;**13**:e19616.
28. Baier E, Olgemöller U, Biggemann L *et al*. Dual-positive MPO-and PR3-ANCA-associated vasculitis following SARS-CoV-2 mRNA booster vaccination: a case report and systematic review. *Vaccines* 2022;**10**:653. https://doi.org/10.3390/vaccines10050653
29. Dias CB, Jorge LB, Woronik V *et al*. IgA nephropathy in patients with serum anti-neutrophil cytoplasmic autoantibody (ANCA) positivity: case series. *J Braz Nephrol* 2021;**44**:42–7. https://doi.org/10.1590/2175-8239-jbn-2021-0018
30. Huang X, Wang Y, Xie L *et al*. IgA nephropathy with anti-neutrophil cytoplasmic antibody seropositivity. *Clin Nephrol* 2015;**84**:156–64. https://doi.org/10.5414/CN108571

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31. Ştefan G, Terinte-Balcan G, Stancu S *et al*. IgA nephropathy with serum ANCA positivity: case series and literature review. *Rheumatol Int* 2021;**41**:1347–55. https://doi.org/10.1007/s00296-021-04888-2
32. Söderberg D, Segelmark M. Neutrophil extracellular traps in ANCA-associated vasculitis. *Front Immunol* 2016;**7**:256. https://doi.org/10.3389/fimmu.2016.00256
33. Sharma P, Uppal NN, Wanchoo R *et al*. COVID-19-associated kidney injury: a case series of kidney biopsy findings. *J Am Soc Nephrol* 2020;**31**:1948–58. https://doi.org/10.1681/ASN.2020050699
34. Yasukawa M, Kitagawa S, Togashi R *et al*. A patient with MPO-ANCA-positive IgA nephropathy diagnosed with the clinical onset of macrohematuria. *Intern Med* 2019;**58**:2051–56.

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