CASE STUDY

SARS-COV-2 INFECTION AS A POSSIBLE TRIGGER FOR MICROSCOPIC POLYANGIITIS: CASE REPORT AND MINI-REVIEW

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ABSTRACT

The paper presents a clinical case of MPA in a 67-year-old woman following COVID-19, characterized by significant difficulties when working with the early etiological verification of diagnosis. The patient presented with polyarthritis affecting the upper and lower limbs, fever, and comorbid urological pathology in the form of urolithiasis and recurrent cystitis. This clinical presentation, hyperuricemia, azotaemia and anemia were mistakenly interpreted as chronic kidney disease: gouty nephropathy, gouty arthritis, which masked the underlying disease for a long time delaying the timely MPA diagnosis and treatment. Given that MPA is a multisystemic disease, it is essential to enhance awareness and knowledge of healthcare professionals of various specialties regarding AAVs and MPA in particular, as evidenced by the online survey data during COVID-19 pandemic among doctors in 21 countries.

KEY WORDS: ANCA-associated vasculitis, microscopic polyangiitis, clinical presentation, COVID-19, novel SARS-CoV-2 infection

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INTRODUCTION

Microscopic polyangiitis (MPA) is a necrotising vasculitis of small vessels, one of the clinical phenotypes of a group of rare systemic diseases known as vasculitis associated with antineutrophil cytoplasmic antibodies (ANCA). ANCA-associated vasculitis (AAVs) encompass a heterogeneous group of disorders characterized by severe destructive systemic vasculitis of small vessels and the development of autoantibodies against neutrophilic proteins, especially myeloperoxidase (MPO-ANCA) or proteinase 3 (PR3-ANCA). Currently available tests for MPO-ANCA and PR3-ANCA are highly sensitive and specific serological markers for the diagnosis of MPA and granulomatosis with polyangiitis (GPA) [1].

The main clinical and pathological variants of AAVs are MPA, GPA (formerly known as Wegener's granulomatosis), eosinophilic granulomatosis with polyangiitis (formerly Churg-Strauss syndrome) and single-organ AAV (such as renal-limited AAV) [2].

MPA was officially recognised at the first International Consensus Conference Chapel Hill Consensus Conference in 1994, which introduced the term MPA, referring to pauci-immune (i.e. minimal or absent immune deposits) necrotising vasculitis affecting small vessels with or without involvement of medium-sized arteries [3].

Despite ANCA-associated vasculitis remaining a rare autoimmune disease, the incidence and prevalence of AAV overall, including MPA have increased over the past 30 years [4, 5]. According to updated epidemiological data, the average standardized incidence rate for MPA per million was 5.04 in the 1990s, increased to 9.2 in the 2000s [5] and continues to rise. Our own clinical experience confirms this increase both before and after the COVID-19 pandemic [6**].**

The global COVID-19 pandemic caused by the novel coronavirus infection SARS-CoV-2 has been a major concern for the global medical community in recent years. It is known that apart from respiratory syndrome, the SARS-CoV-2 virus causes damage to the cardiovascular system, kidneys, joints, nervous system, etc., significantly affecting the clinical course of the disease and mortality. Furthermore, studies by Varga et al. indicate the presence of direct viral infection of endothelial cells of numerous organs and diffuse endotheliitis in COVID-19 patients leading to widespread endothelial dysfunction associated with apoptosis [7]. Cytokine-induced endothelial inflammation and vascular pathology in COVID-19 have been well described in post-mortem biopsies and several clinical cases reporting micro/macro thrombotic events in small, medium, large vessels and vasculitis in multiple organs [8-10].

Furthermore, there is growing evidence that SARS-CoV-2 is another virus which can trigger the emergence of new or exacerbate existing autoimmune diseases in children and adults [10-14].

Recently, the scientific literature has been increasingly reporting on a potential link between SARS-CoV-2 infection and AAVs: firstly, because lung lesions in COVID-19 can mimic the changes observed in patients with AAVs [14-17]; secondly, two diseases can occur concurrently; and thirdly, COVID-19 can trigger AAV [14-17]. The development of AAV following COVID-19 has been reported in many clinical case reports [10, 14, 16-19]. It is quite obvious, that diagnosing new cases of AAVs in a patient with COVID-19 is a challenging task for physicians, since some symptoms and clinical manifestations are common to both conditions [14].

Most published reports have focused on clinical case reports regarding the occurrence of two AAVs serological types associated with MPO-ANCA or PR3-ANCA without specifying the clinical phenotype of ANCA-associated vasculitis in patients who have experienced a novel coronavirus disease. Moreover, it should be emphasised that only a few publications specifically address new cases of MPA development following SARS-CoV-2 infection [8, 20-22].

Thus, the rarity of AAVs in general and MPA in particular, poorly known to a wide range of practitioners, the increasing incidence in recent years, diverse clinical manifestations of the disease and lack of specific symptoms, similarity to other syndrome-like diseases, the complexity of early diagnosis and management of such patients contribute to the practical relevance of this problem in everyday medical practice. The problem of AAVs and MPAs is especially relevant in the period of the novel SARS-CoV-2 coronavirus infection, given the common anatomical areas of COVID-19 and systemic inflammation [23].

CASE REPORT

A 67-year-old patient A. was admitted to the Nephrology and Dialysis Centre of Poltava Regional Clinical Hospital (PRCH) on April 22, 2022 with complaints of general weakness, intermittent heaviness in the lumbar region, pain in the knee, hip, elbow, shoulder and wrist joints, as well as feet. She felt ill for about two months, when after COVID-19 (verified by PCR) in February, she developed pain in the lower back and above-mentioned joints, their swelling, pain in the chest and along the spine, an increase in body temperature to 37.4-37.8 °C, and general weakness. The patient self-administered NSAIDs which provided temporary relief.

At the end of February her family doctor prescribed other NSAIDs which did not improve her condition. In April, the joint pain intensified, and she was treated by rheumatologist, who suspected rheumatoid arthritis. After additional examination (X-ray of the hands and feet, biochemical blood tests for inflammatory markers, rheumatoid factor, anti-cyclic citrullinated peptide antibodies), the diagnosis of rheumatoid arthritis was excluded. The diagnosis was secondary arthropathy involving the shoulder, elbow, hip, knee, ankle joints and II functional disability level. When examining, additional findings detected azotaemia (creatinine 300 µmol/l), anemia, hyperuricemia (HUA), which led to the referral of the patient to a nephrologist at PRCH and hospitalization to the Nephrology and Dialysis Centre.

Medical history data: patient experiences recurrent cystitis, urolithiasis (UL) for over 3 years. She received urological outpatient treatment, but did not undergo biochemical blood test. She has been diagnosed with autoimmune thyroiditis and hypothyroidism for the past 8 years and takes replacement therapy. Physical examination revealed tenderness on percussion over the sternum and ribs as well as when palpating the shoulder, elbow, hip, knee joints, and at the areas of the rib attachments to the sternum; ankle joints were painful on palpation, swelling of the ankle and foot joints; the skin over them was unchanged, without local temperature increase. Passive and active joint movements were limited. Bp – 160/90 mmHg, pulse rate – 80 bpm., rhythmical and satisfactory.

The patient was tested for chronic hepatitis markers (HBV, HCV), HIV infection – the results were negative. PCR testing for SARS-CoV-2, taken from the nasopharynx and oropharynx was negative. Changes in laboratory parameters during hospitalization detected: complete blood count (CBC) – anemia (Hb – 89-86 g/l, erythrocytes – 2.95-2.98·10 12 /l) lymphopenia (12%), a sharp increase in ESR (61-62 mm/h); BAC – signs of azotaemia (creatinine 360.2-293.8 µmol/l, urea 21.8-26.4 mmol/l); HUA (uric acid 557.2-395.5 µmol/l), hypercholesterolaemia (total cholesterol (TC) 5.26-6.3 mmol/l), hypocalcaemia (Ca 1.23-1.24 mmol/l), hyperfibrinogenaemia (5.2 g/l), elevated ferritin levels (208.4 ng/ml with a normal range of 12-150 ng/ml for women), decreased serum transferrin level – 1.51 g/l (normal range: 1.7-4.7 mg/l).

General urinalysis (GUA) detected the signs of urinary syndrome: proteinuria (0.885-1.88 g/l), erythrocytes – unchanged from ⅔to 40 per ppm, leukocytes – 8-14 per ppm. The Zimnitskyi test showed a specific gravity 1007-1014, nocturia (daytime diuresis 620.0 ml, and night-time diuresis 1170.0 ml). Daily proteinuria: 1.39 g/day. ECG revealed sinus rhythm and leftward deviation of EWV, signs of left ventricular hypertrophy.

Ultrasonography of the abdominal cavity and pancreas detected ultrasound signs: GI, chronic cholecystitis, pancreatitis, nephritis, urolithiasis of both kidneys, cysts of both kidneys. X-ray of hands and

feet indicated Rö signs of stage II-III polyosteoarthritic changes, particularly affecting the metatarsophalangeal joint of the first toe on the left foot). FGDS revealed duodenal-gastric reflux. The patient was examined by related specialists, including rheumatologist, haematologist, urologist and their conclusions were included in the clinical diagnosis. The patient refused from sternal puncture. The clinical diagnosis determined: CKD IV (GFR by SKD-EPI – 15 ml/min/1.73m^2): secondary gouty nephropathy in combination with pyelonephritis, latent course; UTI: small calculi in both kidneys; arterial hypertension, grade 3, very high risk; moderate severity anemia of chronic patient; chronic gouty arthritis; duodenal gastric reflux; chronic calculous cholecystitis; chronic pancreatitis, latent course. The patient was prescribed detoxification, antihypertensive, antihypertensive, membrane-stabilising, anticoagulant, antioxidant, antineoplastic therapy, as well as anti-inflammatory and anti-anemic drugs. In early May, the patient was discharged with slight improvement: the lumber pain subsided and the joint pain decreased.

In the following period, the patient was regularly monitored by rheumatologist, nephrologist, urologist, gastroenterologist and sought medical care from other related specialists. In particular, in May, she visited neurologist regarding myofascial pain syndrome and dorsalgia. In May-June, she received treatment from dermatologist for eczema-like rash on her right hand and foot. The diagnosis was pustular psoriasis. At the end of June, the patient was consulted by a cardiologist for shortness of breath and chest pain. The diagnosis was made based on the examination data, namely, metabolic cardiomyopathy, stage II arterial hypertension, grade 3, high risk; atrial fibrillation. After treatment, the patient's condition improved somewhat, but she continued to experience joint and rib pain and pain along the spine. Taking into account her complaints, chest X-ray was performed, which revealed radiological signs of bilateral (more left-sided) hydrothorax, left-sided upper lobe pneumonia. She was hospitalized and treated in the therapeutic department.

In September, the patient visited neurologist again regarding the chest pain and appearance of shooting pain in the spine. She was diagnosed with chronic thoracalgia, disseminated encephalopathy with bilateral reflex insufficiency. MRI of the spine was recommended, which indicated signs of left-sided C-shaped scoliosis of the thoracic spine, grade1; osteochondrosis of the thoracic spine; B Th4 haemangioma; intracorporeal Schmorl's nodes; deforming spondylosis, spondylarthrosis of the thoracic spine; bilateral hydrothorax.

In October 2022, the patient noticed a deterioration in her health, namely, increased pain in the joints and their swelling, frequent night-time urination (up to 3 times), which led her to visit a nephrologist. Blood tests showed creatinine of 350 μmol/l, urea – 15.4 mmol/l, which made it possible to diagnose gouty nephropathy. The patient was hospitalized at the regional nephrology centre for further examination and treatment.

At the time of admission on November 8, 2022, the patient complained of pain in the shoulder, elbow, hip, knee, ankle joints, swelling in the feet and lower legs, acute pain along the spine, general stiffness of movements due to pain, inability to take deep breaths, shortness of breath with minimal physical exertion (such as dressing and getting up), periodic squeezing pain in the heart area, increase in the body temperature up to 37.4 °C, weight loss of 12 kg over 4 months, general weakness, occasional dizziness. Objectively: shoulder, elbow, hip, knee, ankle joints were painful on palpation, swelling of the ankle and foot joints was observed. The skin over the joints showed no changes, without local temperature elevation. Passive and active joint movements were moderately limited. Patient experienced pain in the bones of the hands, legs and ribs on palpation.

The data of inpatient examination revealed anemia (Hb – 102-98 g/l), increased ESR (54-47 mm/h), azotaemia (creatinine 328.7-303.7 μmol/l, urea 20.7-26.9 mmol/l), HUA (uric acid 524.5 μmol/l, despite taking the drug febuxostat), hypercholesterolaemia (FPG 6.4 mmol/l), hypocalcaemia (Ca 1.21 mmol/l). A sternal puncture was performed without deviation from the normal range.

The urinalysis detected moderate proteinuria (0.488-0.657 g/l), microhaematuria (unchanged erythrocytes from 9-11 to 20-25 per day), mild leukocyturia (1-3 to 10-12 per day). Daily proteinuria was 1.09 g/day. Urine culture for microflora showed no growth of aerobic bacteria.

ECG: sinus rhythm, heart rate 69 beats/min. EWV deviated to the left, incomplete blockade of the anterior left branch of the His bundle, signs of LV strain, paired supraventricular extrasystole. Echocardiography detected adequate contractile function of LV. The heart cavities were not enlarged. Initial hypertrophy of the LV myocardium with type I diastolic dysfunction, changes in the aorta and aortic valve with regurgitation of I degree, regurgitation of the MC, TC of the I degree were noted. LVEF was 62 %. CT scan of the thoracic cavity organs showed signs of left-sided hydrothorax, hydropericardium, left-sided upper lobe pneumonia. FGDS revealed erythematous gastroduodenopathy, duodenal-gastric reflux.

Immunological blood tests dated November 16, 2022 detected myeloperoxidase (MPO), Ig G antibodies

(ELISA) – 192.36 U/ml (normal < 20 U/ml); antinuclear antibodies (ANA, IFT method) – 1:1000 (normal < 1:100); double-stranded DNA (ds DNA), Ig G antibodies – 1.5 IU/ml (normal < 10.0 IU/ml, negative result).

Thus, the test data can be interpreted as verified systemic ANCA (MPA+) vasculitis. The patient was examined by related specialists: rheumatologist, haematologist, gastroenterologist, pulmonologist, endocrinologist, urologist, neurologist, psychiatrist, angiosurgeon. It is worth mentioning that after discharge from the hospital in early May, the patient visited various related specialists a total of 20 times, and was consulted by a psychiatrist who ruled out psychiatric pathology. The psychiatrist's conclusion: astheno-neurotic syndrome.

It should be noted that the diagnosis of MPA was substantiated in accordance with the new criteria MPA classification ACR/EULAR 2022 [24], which are now confirmed for the use in clinical trials. These criteria assign the highest score to MPO-ANCA (or perinuclear ANCA, pANCA).

DISCUSSION

Systemic vasculitis, ANCA associated: microscopic polyangiitis (MPO+), grade III activity, chronic course, with kidney damage (CKD V (eGFR SKD-EPI=12 ml/min/1.73m^2): rapidly progressive glomerulonephritis, urinary syndrome; arterial hypertension, grade III, very high risk); pulmonary involvement (hydrothorax, pneumonia, pulmonary fibrosis, grade II); cardiac involvement (pericarditis, myocarditis, CHF IIA, FC III); central nervous system involvement (dyscirculatory encephalopathy with bilateral reflex pyramidal failure); peripheral nervous system (polyneuropathy); joints (polyarthralgia). Associated pathologies corresponded to the previous clinical diagnosis. Prescribed pathogenetic treatment included pulse therapy: "Solu-Medrol" (methylprednisolone) 250 mg IV drip No. 3, "Endoxan" (cyclophosphamide) 400 mg IV drip 1 time in 2 weeks (in the morning) in 200 ml of 0.9% sodium chloride solution, and then 1 time in 3 weeks 3-4 months (depending on the patient's condition), mesna solution (for prevention of toxic effects of cyclophosphamide on the urinary tract): during endoxan infusion, 4 hours and 8 hours after the infusion; after "Solu-Medrol", switch to "Medrol" 32 mg tablets per day with further dose reduction according to the regimen. Additionally, the patient received detoxification, antihypertensive, antianemic therapy, as well as hepatoprotectors, sorbents.

She was discharged from the hospital with significant improvement (in the patient's words, "born again") to continue outpatient treatment under the supervision of a multidisciplinary team of specialists (family doctor, nephrologist, rheumatologist, etc.). Currently, she continues to receive pathogenetic therapy and feels satisfactory.

It is widely acknowledged that the novel coronavirus disease 2019 (COVID-19) has become a global problem since its outbreak in December 2019, as it is associated with a great number of pathologies, affecting numerous organs. Moreover, SARS-CoV-2 is associated with the development of rheumatic diseases, especially small vessel vasculitis and arthritis. Typically, the onset occurs in several days or weeks after antigenic infection and in patients with mild COVID-19 [10, 11]. Based on the data of the analysed literary sources in the PubMed database, we determined that in 40% of cases AAV was diagnosed 1-6 months after COVID-19, while in 50-60% of cases, the diseases were concurrent [17,19].

The presence of a link between COVID-19 and autoimmune diseases is evidenced by the international team of scientists [13], who report that COVID-19 is similar to autoimmune diseases in terms of clinical manifestations, immune responses and pathogenic mechanisms. Persistent immune responses are involved in the pathogenesis of both diseases [13]. Similar to systemic autoimmune diseases, COVID-19 can manifest itself with heterogeneous and systemic clinical manifestations [13], which make it difficult to diagnose new cases of MPA in patients experienced COVID-19. Moreover, Thu Aung Z and others [17], in turn, emphasise that COVID-19 and AAVs are multisystem diseases. It is believed that there is a causal link between both conditions, which is supported by reports of numerous clinical cases [8, 14, 16-22]. Therefore, physicians should be aware of the potential spectrum of systemic and autoimmune diseases that can be triggered by SARS-CoV-2 infection. This will provide a timely diagnosis and treatment initiation [10], which can save not only lives, but also protect other organs from damage. Moreover, COVID-19 pandemic has changed the approach of various clinicians to ANCA-related vasculitis [25]. For this reason, it is essential to consider the importance of a high suspicion index for AAV in people experienced COVID-19.

A team of experts from the UK, India, Kazakhstan and Ukraine, eight of the world's leading research centres developed online questionnaire with 28 questions based on relevant global practice guidelines and AAVs recommendations for the evaluation of the level of knowledge and perceptions of physicians regarding ANCA-associated vasculitis, as well as conducted online survey during COVID-19 pandemic [25]. The report on this issue was published on November 21, 2022, where 113 respondents from 21 countries indicated the need for enhanced medical education, which could increase

practitioners' awareness and knowledge regarding ANCA-associated vasculitis.

It is commonly known that timely recognition, rapid diagnosis and early initiation of active specific treatment for MPA remain one of the most challenging tasks in real clinical practice, as MPA is characterised by heterogeneous initial manifestations and variable clinical presentation. Given that MPA is a systemic vasculitis, the involvement of many organs can lead to a wide range of signs and symptoms [26-28]. The kidneys and lungs are the most typical organs involved in MPA. Initially nonspecific and then heterogeneous symptoms can mislead the diagnostic search and cause a significant delay in adequate treatment for AAV [27].

In 2020, a group of French researchers [29] published the data of a retrospective analysis of clinical presentation in 378 patients with MPA from the French Vasculitis Study Group Registry. At the time of diagnosis, the main clinical manifestations of MPA included: renal dysfunction (74%), arthralgia (45%), skin (41%) and lungs (40%) involvement, mononeuritis (32%), and less frequent alveolar haemorrhage (16%), cardiomyopathy (5%) and severe gastrointestinal signs (4%); mean serum creatinine was 217 µmol/l [29].

Thus, MPA is a multisystem disease characterised by polymorphism nonspecific clinical signs and diverse clinical manifestations depending on the affected organs. It is worth noting that systemic necrotising vasculitis are great masqueraders, and sometimes their manifestations can often differ significantly from the typical recognised disease patterns, and therefore, received the descriptive name "the Great Masquerades" [30]. It is important to note that AAVs can also present with unusual manifestations of the disease [30], which can vary. In addition, the variability in clinical symptoms may also be related to the existing comorbidity. From clinical perspective, comorbid pathology complicates the course of underlying disease and leads to changes in the usual clinical presentation. Moreover, existing comorbid conditions can mislead the doctor and direct the diagnostic search in the wrong direction, as the clinical case presented clearly demonstrates. Therefore, COVID-19 may trigger a new-onset MPA, especially in patients with predisposing factors. Thus, AAV should definitely be included in the differential diagnosis in patients with COVID-19 who present with polymorphic clinical manifestations.

CONCLUSIONS

During the COVID-19 pandemic, the early diagnosis of MPA for healthcare professionals of different specialties remains as challenging as it was before, since these two diseases are multisystem disorders sharing common pathogenetic mechanisms of development and clinical manifestations, as well as casual relationship between them.

Polymorphism of nonspecific clinical signs and multiorgan lesions are characteristic features of MPA which can vary from patient to patient, mislead physicians and leading to a misdirected diagnostic search. Rapid and early diagnosis of MPA is important for initiating therapy, which can save not only lives, but also protect other organs from damage.

Since MPA is a multidisciplinary problem, there is a need to increase the awareness and knowledge of practitioners of all specialities regarding AAVs and MPA in particular, as evidenced by the data of online survey conducted during the COVID-19 pandemic among doctors from 21 countries worldwide.

REFERENCES

1. Bossuyt X, Cohen Tervaert JW, Arimura Y et al. Position paper: Revised 2017 international consensus on testing of ANCAs in granulomatosis with polyangiitis and microscopic polyangiitis. Nat Rev Rheumatol. 2017;13(11):683-692. doi: 10.1038/nrrheum.2017.140.
2. Jennette JC, Falk RJ, Bacon PA et al. 2012 revised International Chapel Hill Consensus Conference Nomenclature of Vasculitis. Arthritis Rheum 2013; 65(1):1–11. doi:10.1002/art.37715.
3. Jennette JC, Falk RJ, Andrassy K et al. Nomenclature of systemic vasculitides. Proposal of an international consensus conference. Arthritis Rheum. 1994;37(2):187-92. doi: 10.1002/art.1780370206.
4. Hashmi MF, Jain V, Tiwari V. Microscopic Polyangiitis. In: StatPearls. Treasure Island (FL): StatPearls Publishing. 2023. https://www.ncbi.nlm.nih.gov/books/NBK531484/#_NBK531484_pubdet [date access 09.02.2023]
5. Mohammad AJ. An update on the epidemiology of ANCA-associated vasculitis. Rheumatology. 2020;59(3): iii42–iii50. doi:10.1093/rheumatology/keaa089.
6. Katerenchuk IP, Tkachenko LA, Yarmola TI et al. Microscopic polyangiitis - a view of the problem through the lens of a nephrologist. Wiad Lek. 2021;74(4):1024-1031.
7. Varga Z, Flammer AJ, Steiger P et al. Endothelial cell infection and endotheliitis in COVID-19. Lancet. 2020;395(10234):1417-1418. doi: 10.1016/S0140-6736(20)30937-5.
8. Allena N, Patel J, Nader G et al. A Rare Case of SARS-CoV-2-Induced Microscopic Polyangiitis. Cureus. 2021;13(5):e15259. doi: 10.7759/cureus.15259.

9. Sovgirya SN, Filenko BN, Roiko NV et al. Morfo-patofiziologicheskie aspektyi izmeneniya pochek pri COVID-19 [Morphological and pathophysiological aspects of kidney changes in COVID-19]. Azerbaijan Medical Journal. 2022; 1:125–130. doi:10.34921/amj.2022.1.020. (in Russian)
10. Mendes JL, Venade G, Manuel P et al. Virus and Autoimmunity: Can SARS-CoV-2 Trigger Large Vessel Vasculitis? Eur J Case Rep Intern Med. 2022;9(8):003486. doi: 10.12890/2022_003486.
11. Gracia-Ramos AE, Martin-Nares E, Hernández-Molina G. New Onset of Autoimmune Diseases Following COVID-19 Diagnosis. Cells. 2021;10(12):3592. doi: 10.3390/cells10123592.
12. Sacchi MC, Tamiazzo S, Stobbione P et al. SARS-CoV-2 infection as a trigger of autoimmune response. Clin Transl Sci. 2021;14(3):898-907. doi: 10.1111/cts.12953.
13. Liu Y, Sawalha AH, Lu Q. COVID-19 and autoimmune diseases. Curr Opin Rheumatol. 2021;33(2):155-162. doi: 10.1097/BOR.0000000000000776.
14. Izci Duran T, Turkmen E, Dilek M et al. ANCA-associated vasculitis after COVID-19. Rheumatol Int. 2021;41(8):1523-1529. doi: 10.1007/s00296-021-04914-3.
15. Gelzo M, Cacciapuoti S, Pinchera B et al. A Transient Increase in the Serum ANCAs in Patients with SARS-CoV-2 Infection: A Signal of Subclinical Vasculitis or an Epiphenomenon with No Clinical Manifestations? A Pilot Study. Viruses. 2021;13(9):1718. doi: 10.3390/v13091718.
16. 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. doi: 10.7759/cureus.20331.
17. Thu Aung Z, Oluyombo R, Karim M et al. SARS-CoV-2 Infection: A Forerunner or Precursor in Anti-neutrophil Cytoplasmic Antibody-Associated Vasculitis With Kidney Injury. Cureus. 2022;14(9):e28705. doi: 10.7759/cureus.28705.
18. Morris D, Patel K, Rahimi O et. al. ANCA vasculitis: A manifestation of Post-Covid-19 Syndrome. Respir Med Case Rep. 2021;34:101549. doi: 10.1016/j.rmcr.2021.101549.
19. Ozcan S, Sonmez O, Karaca C et al. ANCA-associated vasculitis flare might be provoked by COVID-19 infection: a case report and a review of the literature. Clinical Kidney Journal. 2022; 15(11): 1987–1995. doi:10.1093/ckj/sfac186.
20. Fares E, Pathak K, Damiano Ch et al. Diffuse alveolar hemorrhage as a consequence of microscopic polyangiitis due to COVID-19. Critical care. 2020; 158(4): A775. doi: 10.1016/j.chest.2020.08.722.
21. Kawashima S, Kishimoto M, Hibino T et al. MPO-ANCA-positive Microscopic Polyangiitis Following COVID-19 Infection. Intern Med. 2022;61(4):567-570. doi: 10.2169/internalmedicine.8615-21.
22. Patel R, Amrutiya V, Baghal M et al. Life-Threatening Diffuse Alveolar Hemorrhage as an Initial Presentation of Microscopic Polyangiitis: COVID-19 as a Likely Culprit. Cureus. 2021;13(4):e14403. doi: 10.7759/cureus.14403.
23. Giryes S, Bragazzi NL, Bridgewood C et al. COVID-19 Vasculitis and vasculopathy-Distinct immunopathology emerging from the close juxtaposition of Type II Pneumocytes and Pulmonary Endothelial Cells. Semin Immunopathol. 2022;44(3):375-390. doi: 10.1007/s00281-022-00928-6.
24. Suppiah R, Robson JC, Grayson PC et al. DCVAS Study Group. 2022 American College of Rheumatology/European Alliance of Associations for Rheumatology Classification Criteria for Microscopic Polyangiitis. Arthritis Rheumatol. 2022;74(3):400-406. doi: 10.1002/art.41983.
25. Auanassova A, Yessirkepov M, Zimba O et al. Physicians' perceptions about antineutrophil cytoplasmic antibody-associated vasculitis: an online survey report in the time of the COVID-19 pandemic. Clin Rheumatol. 2023;42(3):831-837. doi: 10.1007/s10067-022-06452-0.
26. Kallenberg CG. The diagnosis and classification of microscopic polyangiitis. J Autoimmun. 2014;48-49:90-3. doi: 10.1016/j.jaut.2014.01.023.
27. Molnár A, Studinger P, Ledó N. Diagnostic and Therapeutic Approach in ANCA-Associated Glomerulonephritis: A Review on Management Strategies. Front Med (Lausanne). 2022;9:884188. doi: 10.3389/fmed.2022.884188.
28. Dousdampanis P, Assimakopoulos SF, Trigka K. Microscopic Polyangiitis: from Pathogenesis to Treatment. Urol Nephrol Open Access J. 2017;5(2): 00167 doi: 10.15406/unoaj.2017.05.00167.
29. Nguyen Y, Pagnoux C, Karras A et al. Microscopic polyangiitis: Clinical characteristics and long-term outcomes of 378 patients from the French Vasculitis Study Group Registry. J Autoimmun. 2020;112:102467. doi: 10.1016/j.jaut.2020.102467.
30. Sharma A, Gopalakrishan D, Nada R et al. Uncommon presentations of primary systemic necrotizing vasculitis: the Great Masquerades. Int J Rheum Dis. 2014;17(5):562-72. doi: 10.1111/1756-185X.12223.

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