13. Rus M, Ardelean AI, Andronie-Cioara FL, Filimon GC. Acute Myocardial Infarction during the COVID-19 Pandemic: Long-Term Outcomes and Prognosis -A Systematic Review. Life 2024; 14 (2):202. doi: 0.3390/life14020202.
14. Zhang L, Richards A, Barrasa MI, Hughes SH, Young RA, Jaenisch R. Reverse-transcribed SARS-CoV-2 RNA can integrate into the genome of cultured human cells and can be expressed in patient-derived tissues. Proceedings of the National Academy of Science of the United States of America. 2021; 118: e2105968118. doi: 10.1073/pnas.2105968118.
15. Zuin M, Rigatelli G, Battisti V, Costola G, Roncon L, Bilato C. Increased risk of acute myocardial infarction after COVID-19 recovery: A systematic review and meta-analysis. International Journal of Cardiology. 2022 Dec 16; 372:138–143. doi: 10.1016/j.ijcard.2022.12.032.

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COVID-INDUCED OVERLAP SYNDROME OF MICROSCOPIC POLYANGIITIS, SYSTEMIC LUPUS ERYTHEMATOSUS, AND AUTOIMMUNE THYROIDITIS THROUGH THE PRISM OF A CLINICAL CASE

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The problem of the overlap syndromes development is gaining more and more relevance, especially in the post-COVID period. This is due to the debut of these diseases after a coronavirus infection, the severity of their course, and the difficulty of timely diagnosis and treatment. The aim of the study was to analyze the clinical case as unique with regard to the features of the debut and clinical course of the COVID-induced overlap syndrome of microscopic polyangiitis, systemic lupus erythematosus and autoimmune thyroiditis. The article analyzes data from the medical records of a 46-year-old inpatient who recently had a coronavirus infection. In the post-COVID period, the disease debuted as a pulmonary-renal syndrome against a background of fever. The presence of immunological markers of microscopic polyangiitis, systemic lupus erythematosus, and autoimmune thyroiditis gave rise to the identification of an overlap syndrome of these diseases. The results of a nephrobiopsy confirmed the presence of morphological changes in the kidney tissue typical for microscopic polyangiitis. The relevance of this clinical case is due to the extreme rarity of descriptions in the professional medical literature development an overlap syndrome of microscopic vasculitis and systemic lupus erythematosus in combination with autoimmune thyroiditis.

Key words: overlap syndrome, microscopic polyangiitis, systemic lupus erythematosus, autoimmune thyroiditis, coronavirus infection, antineutrophil myeloperoxidase antibodies, antibodies to double-stranded deoxyribonucleic acid.

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КОВІД-ІНДУКОВАНИЙ ОВЕРЛАП-СИНДРОМ МІКРОСКОПІЧНОГО ПОЛІАНГІЇТУ, СИСТЕМНОГО ЧЕРВОНОГО ВОВЧАКА ТА АУТОІМУННОГО ТИРЕОЇДИТУ ЧЕРЕЗ ПРИЗМУ КЛІНІЧНОГО ВИПАДКУ

Проблема розвитку оверлап-синдромів набуває все більшої актуальності, особливо – в постковідний період. Це пов’язано, як із дебютом цих захворювань після перенесеної коронавірусної інфекції, тяжкістю їхнього перебігу, так із складністю своєчасної його діагностики та лікування. Мета роботи: проаналізувати клінічний випадок, як унікальний щодо особливостей дебюту й клінічного перебігу ковід-індукованого овелап-синдрому мікроскопічного поліангіїту, системного червоного вовчака та аутоімунного тиреоїдиту. У статті проаналізовано дані медичної документації стаціонарної хворого 46-ти річного віку, який нещодавно перехворів на коронавірусну інфекцію. У постковідному періоді захворювання дебютувало легенево-нирковим синдромом на тлі гарячки. Наявність у хворого імунологічних маркерів мікроскопічного поліангіїту, системного червоного вовчака та аутоімунного тиреоїдиту дало підставу виявити оверлап-синдром цих захворювань. Результати нефробіопсії підтвердили наявність типових для мікроскопічного поліангіїту морфологічних змін ниркової тканини. Надзвичайна рідкісність опису в фаховій медичній літературі клінічних випадків розвитку оверлап-синдрому мікроскопічного васкуліту та системного червоного вовчака в поєднанні з аутоімунним тиреоїдитом обумовлює актуальність висвітлення даного клінічного випадку.

Ключові слова: оверлап синдром, мікроскопічний поліангіїт, системний червоний вовчак, аутоімунний тиреоїдит, коронавірусна інфекція, антинейтрофільні антитіла до мієлопероксидази, антитіла до дволанцюгової дезоксирибонуклеїнової кислоти.

The study is a fragment of the research project “Substantiation of the use of traditional and non-traditional methods of prevention and rehabilitation therapy in patients with cardiovascular pathology with high cardiovascular risk”, state registration No. 0124U000095.

The COVID-19 pandemic, the peculiarities of the immune system response of different people to infection, the presence of a chronological sequence of the response to viral infection and the debut of some diseases have caused another wave of curiosity about the role of coronavirus in the activation of

autoimmune processes [2, 7]. It is reported that coronavirus infection can be a predictor of the development both: a single autoimmune disease and their combination – so-called overlap syndrome (OS) [6, 12].

Overlap syndrome is a crossover syndrome defined as the combination of two or more clearly characterized autoimmune diseases that meet their clinical and serological classification criteria. It most often occurs in the intersection of systemic connective tissue diseases (SCTD) such as: systemic lupus erythematosus (SLE), systemic sclerosis (SS), polymyositis (PM) or – dermatomyositis (DM), Sjögren's syndrome and/or – inflammatory arthritis (juvenile idiopathic arthritis (JIA), rheumatoid arthritis). It is much less common in the specialized medical literature to find reports of the SCTD combination with systemic vasculitis (SV) or other autoimmune diseases [12]. Noteworthy that OS develops not only with the combination of SCTD or SV. They can occur in combination with autoimmune diseases of the thyroid gland (autoimmune thyroiditis (AIT), liver (autoimmune hepatitis, primary biliary cirrhosis and sclerosing cholangitis), lungs (bronchial asthma, chronic obstructive pulmonary disease), both among themselves and with other SCTD or with SV [6].

At the same time, in the professional medical literature, the cases of the overlap syndrome of SV associated with antineutrophil antibodies (AAB) and SLE are described too rarely. According to data known in the literature, 49 such cases have been reported worldwide. In 45 of them, the SLE/microscopic polyangiitis (MPA) overlap syndrome developed, of which 1 had a documented onset after a previous coronavirus disease, 3 had SLE/granulomatosis with polyangiitis (GPA), and 1 case of SLE/MPA/thrombotic thrombocytopenic purpura overlap syndrome. In all described cases, the patients were female, whose age was not particularly important [6]. We did not find reports on the development of OS MPA, SLE and AIT, which determines the relevance of the case described.

Epidemiological studies on the incidence and prevalence of all OS have not been carried out, as they are rare pathological conditions. The estimated prevalence of the SLE/AAB overlap syndrome is only 2 % [10].

The etiology and pathogenesis of SLE, MPA and AIT are not fully understood. Provoking factors may include: epigenetic, genetic, environmental and infectious (including slow retroviruses and RNA-containing viruses, such as SARS-CoV-2) factors, intolerance to drugs, vaccines, serums, photosensitization and others [5].

The MPA/SLE/AIT overlap syndrome is an autoimmune process characterized by a combination of symptoms of these autoimmune diseases. However, the non-simultaneity of their clinical manifestations often contributes to the delay in diagnosis. According to the literature, OS MPA/SLE debuts with glomerulonephritis (GN) and / or interstitial lung disease (ILD), often against the background of nonspecific symptoms: arthralgia, myalgia, weight loss, dyspeptic, hyperthermic or intoxication syndromes, AIT – with hypothyroidism [6].

The lack of criteria for assessing disease activity and clear recommendations for the treatment of OS leads to a worsening prognosis and increased mortality in these patients. Therefore, we consider it necessary to highlight this clinical case to accumulate clinical experience in the management of such patients.

The purpose of the study was to analyze the clinical case as unique with regard to the features of the debut and clinical course of the COVID-induced overlap syndrome of microscopic polyangiitis, systemic lupus erythematosus and autoimmune thyroiditis.

The study analyzes the medical records of a 46-year-old patient G., who suffered a coronavirus infection in early June 2022. The diagnosis of COVID-19 was verified using a polymerase chain reaction (PCR) test [7]. During inpatient treatment at the nephrology center, in addition to the clinical and biochemical methods of blood and urine examination generally accepted in nephrology, electrocardiogram (ECG), echocardioscopy (ECHO), ultrasound examination (US) of the abdominal cavity, esophagogastroduodenoscopy (EGD), radiography (Ro-graphy) and computed tomography (CT) of the chest organs were performed, the presence of antibodies (AB) to human immunodeficiency virus (HIV), syphilis (Wasserman reaction (RW)), viral hepatitis: A (antiHAV), B (antiHBV), C (antiHCV), the level of ferritin, transferrin, C-reactive protein (CRP), seromucoid was determined. To verify the diagnosis, the presence of immunological markers was checked: antinuclear antibodies (ANA) to double-stranded deoxyribonucleic acid (dsDNA), the level of antineutrophil cytoplasmic antibodies (ANCA) to myeloperoxidase (MPO) and PR-3, antibodies (AB) to glomerular basement membrane (GBM) [6]. Chronic kidney disease (CKD) stage was determined by clinical classification based on the Kidney Disease Outcomes Quality Initiative (KDOQI, 2012), by calculating glomerular filtration rate (eGFR) using the CKD-EPI online calculator [9, 11].

The diagnosis of MPA and SLE was established based on the classification criteria of the American College of Rheumatology and the European League Against Rheumatism (EULAR/ACR) 2019 [13] and 2022 [2, 3] and/or morphological changes in renal tissue, which is based on the morphological classification of the International Society of Nephrology/Society of Renal Pathology (ISN/RPS) 2003 [1, 3, 13]. The diagnosis of hypothyroidism and AIT was based on the results of the patient's hormonal status and the detection of antibodies (AB) to thyroglobulin (TG) and/or to thyroperoxidase (TPO) [1].

Written informed consent was obtained from the patient for the publication of his clinical history.

Patient G, 46 years old, a shoe factory employee, has been receiving treatment at the Nephrology and Dialysis Center of the municipal enterprise "Poltava Regional Clinical Hospital of the Poltava Regional Council" since December 1, 2022. The main complaints during hospitalization were: shortness of breath with minor physical exertion, headache, uncontrolled increase in blood pressure (up to 200/100 mm Hg), edema of the lower extremities, hand tremor, increased body temperature to 38.0 °C, and severe general weakness.

From the anamnesis, it is known that the patient began his medical history on June 1, 2022, when, during a coronavirus disease, he noticed hemoptysis and red urine against the background of an increase in body temperature to 39.5 °C. After 2 weeks, shortness of breath appeared and began to progress, general weakness developed. The condition progressively worsened. A week later, the body temperature rose again to 38 °C. In addition to the above complaints, he began to notice periodic dizziness. On June 21, 2022, oliguria developed. He consulted his family doctor and was examined. A blood test first revealed an increase in creatinine and urea levels to 270 μmol/l and 9.25 mmol/l, respectively, a decrease in hemoglobin (Hb) to 118 g/l, protein in the urine (1 g/l), signs of bilateral lower-lobar pneumonia on Ro-gram, and ultrasound signs of nephritis. The diagnosis was: Systemic inflammatory response syndrome (post-COVID), bilateral polysegmental pneumonia. Hemoptysis. LF I. Mild anemia. Acute kidney injury (AKI), prerenal form, oliguric stage. Since then, he has been treated as an outpatient and inpatient at his place of residence. The course of the disease was persistent with severe exacerbations and progressive deterioration of his general condition. The kidney test performed (July 25, 2022) revealed a further increase in creatinine levels to 334 μmol/l, and urea levels to 41 mmol/l, proteinuria within the urinary syndrome and mild anemia persisted. In this regard, he was treated as an inpatient in the therapeutic department of the Kremenchuk city hospital. Autoimmune thyroiditis with decreased thyroid function was first detected. According to the Ro-graphy, bilateral interstitial pneumonitis was found. Consulted by a pulmonologist, hematologist, nephrologist, endocrinologist. Antibacterial, antianemic, detoxification, hormone replacement and symptomatic therapy was ineffective. The patient began to notice a constant increase in blood pressure (BP) (up to 150/100 mm Hg), and the dynamics of biochemical blood analysis (BBA) showed increasing azotemia (creatinine – 524 μmol/l, urea – 20.7 mmol/l), increased levels of CRP (18.1 mg/l (normal up to 10.0 mg/l), seromucoid (6.9 U (normal 0.12–0.2 U)) and D-dimer (1.23 μg/ml (normal up to 0.5 μg/ml)). Despite treatment, the patient's condition did not improve, the creatinine level remained within the range: 503–376 μmol/l, a hemorrhagic rash appeared on the skin of the trunk. Due to the ineffectiveness of the prescribed therapy, on October 31, 2022, he was hospitalized to the Nephrology and Dialysis Center of the municipal enterprise "Poltava Regional Clinical Hospital of the Poltava Regional Council".

During examination: general condition is severe. Skin and visible mucous membranes are pale. Dyspigmentation of the skin of the trunk is noted, due to a preceding hemorrhagic rash. Swelling of the upper eyelids. Respiratory rate (RR) – 22/min. Auscultation: in the lungs – hard breathing, especially in the lower parts, no wheezing. Pulse – 88 beats/min. rhythmic, satisfactory properties. Blood pressure 150/100 mm Hg. The left border of relative cardiac dullness is shifted outward by 0.5 cm from the midclavicular line in the VI intercostal space. Cardiac activity is regular, sounds are muffled, systolic murmur above the apex and at Botkin's point, accent of the second sound over the aorta. The abdomen is soft and painless on palpation. The percussion symptom is negative on both sides. Daily diuresis – 2 liters.

In the dynamics of changes in laboratory-instrumental parameters: general blood analysis (GBA) – anemia (Hb – 116–128 g/l), transient leukocytosis (7.8–16.4x10^9/l), thrombocytopenia (169x10^9/l), accelerated ESR (34–27 mm/h); BBA: hypoproteinemia (total protein – 62.0 g/l), hypoalbuminemia (37.0 g/l), hyperazotemia (creatinine (430.3–486.1 μmol/l), urea – 23.0–31.9 mmol/l), hyperuricemia (uric acid 484.0–390.1 μmol/l), hypocalcemia (1.16 mmol/l), hyperferritinemia (177.2 (N – 15–150) ng/ml for men), reduced transferrin level – 1.64 (N – 1.7–4.7) mg/l; in the general urine analysis (GUA) protein – 0.48 g/l, erythrocytes – 1–3 in the field of vision (f/v), leukocytes – 2–6 in the f/v, cylinders – 1–2 in the f/v, slight bacteriuria. GFR according to the SKD-EPI formula was 11.4 ml/min/1.73m^2. In the urine analysis according to Nechiporenko: leukocytes – 5750, erythrocytes – 11250, cylinders – 90 in 1 ml. Daily protein excretion – 3.52 g/day. Antibody to HIV, RW, antiHAV, antiHBV, antiHCV are negative. IgM to MPO –

160.29 U/ml (>20.0 positive result), AT index to PR-3 – 0.06 (>1.0 positive), AT to BMC – 0.3 U/ml (<7.00 – negative), ANA to ds DNA – 38.6 IU/ml (>15 positive). ECG from 01.11.2022: sinus rhythm, heart rate 92 beats/min. The electrical axis of the heart is deviated to the left. Incomplete blockade of the anterior branch of the left bundle branch. Signs of hypertrophy of the left heart. According to: FGDS (01.11.2022): – gastroduodenopathy, Ultrasound of the abdominal cavity (07.11.2022) – Ultrasound signs of chronic nephritis, cyst of the right kidney, CT scan of the chest organs (08.11.2022) – CT signs of "ground glass" areas in both lungs, Echocardiography (11/10/2022) – left ventricular (LV) contractility was sufficient (LV ejection fraction – 56 %), signs of LV myocardial hypertrophy with type I diastolic dysfunction, aortic stenosis, and grade I mitral (MV) and tricuspid (TV) valve insufficiency. He was consulted by a cardiologist, gastroenterologist, urologist, rheumatologist, pulmonologist, and ENT. A nephrobiopsy was performed. Histological changes in the renal tissue indicated the development of necrotizing glomerulonephritis with crescents, vacuolar dystrophy, and necrosis of the nephron tubular epithelium.

Based on complaints, anamnesis data, results of laboratory and instrumental studies, conclusions of related specialists and elevated levels of antibodies to MPO, ANA to dsDNA, and nephrobiopsy data, the diagnosis was revised. Immunological markers of MPA, SLE and AIT allowed us to identify an overlap syndrome of these diseases with kidney damage (CKD V (CKD-EPI – 11.4 ml/min): glomerulonephritis, nephrotic syndrome complicated by AKI with arterial hypertension, anemia), lungs (bilateral interstitial pneumonitis, LF 0, history of hemoptysis), heart (postmyocarditic cardiosclerosis, MV and TV insufficiency of the I degree, heart failure I, functional class II against the background of secondary (uremic, systolic) cardiomyopathy).

Symptomatic therapy and 3 sessions of pulse therapy with Solu-medrol 500 mg and 1 session of Endoxan (500 mg) were performed. On November 24, 2022, he was discharged with a slight improvement in clinical and laboratory parameters and recommendations to continue pathogenetic therapy at the place of residence.

Overlap syndrome is a pathological condition when one patient simultaneously or sequentially shows signs of several autoimmune diseases. In our case, it is a combination of MPA, SLE, and AIT. The problem of its development is one of the main ones for modern rheumatology, nephrology, endocrinology, family medicine and doctors of other specialties, taking into account the overlap of their manifestations and the complexity of timely diagnosis, which is associated, first of all, with multi-organ damage, the diversity of the clinical picture and variants of the course of the disease, with the blurring, atypicality and non-simultaneity of the appearance of clinical manifestations [4, 8]. The problem of its development is one of the main ones for modern rheumatology, nephrology, endocrinology, family medicine and doctors of other specialties, taking into account the overlap of their manifestations and the complexity of timely diagnosis, which is associated, first of all, with multi-organ damage, the diversity of the clinical picture and variants of the course of the disease, with the blurring, atypicality and non-simultaneity of the appearance of clinical manifestations [4, 8]. This is confirmed by the clinical case presented here.

Microscopic polyangiitis is a rare, poor-prognosis vasculitis characterized by necrotizing immune-mediated inflammation of small and medium-sized blood vessels. The kidneys (glomerulonephritis), lungs (pneumonitis with alveolar hemorrhage), peripheral nervous system (mononeuropathy), and skin (petechial rash or purpura) are most commonly affected [6].

Systemic lupus erythematosus is a chronic relapsing-remitting, organ-nonspecific SCTD with heterogeneous clinical manifestations, primarily with symptoms of kidney damage (lupus nephritis), joints (arthralgia, arthritis), central nervous system (CNS), serositis (pericarditis, pleurisy), skin (butterfly, discoid lupus, etc.) and blood (anemia, thrombocythemia) [8, 10].

Autoimmune thyroiditis is an autoimmune disease of the thyroid gland that occurs as a result of a violation of immune regulation and leads to aseptic inflammation and the development of hypothyroidism as a result of damage to the blood-thyroid barrier [4]. The development of COVID-induced AIT can be traced in the medical history of a given patient.

The diagnosis of MPA is verified based on the EULAR/ACR 2019 and 2022 classification criteria and/or morphological changes in the renal tissue in the form of nephritis, focal-segment necrotizing or diffuse GN with crescents, or signs of lupus nephritis detected during nephrobiopsy [3, 13]. Elevated levels of antibodies to MPA and morphological signs of necrotizing GN with crescents, vacuolar dystrophy, and necrosis of the nephron tubular epithelium confirmed its presence in the patient and excluded secondary COVID-induced vasculitis. At the same time, an increase in the level of SLE-specific ANA indicated the

presence of SLE in the patient, the typical symptoms of which may develop much later, as noted in the literature [2, 5].

The most common cause of pulmonary-renal syndrome is vasculitis, primarily ANCA-associated vasculitis, namely: MPA and GPA (formerly Wegener's syndrome), anti-GBM disease (formerly Goodpasture's syndrome). In our patient, the presence of GPA and anti-GBM disease were excluded, and the first manifestations of MPA were fever, gross hematuria, and hemoptysis. However, the presence of ANA to ds DNA did not exclude the superimposition of lupus nephritis and pneumonitis symptoms, which, according to the literature, is detected in 40 % of patients with SLE [2, 10, 14]. Generalization of the pathological process with the development of hyperazotemia, anemia, and hypercholesterolemia occurred less than three weeks after the COVID-19 infection, which corresponded to the acute course of the disease and is consistent with the literature [2] indicates that it was the coronavirus infection that became a predictor of his disease development.

The pathogenetic mechanisms of the various autoimmune diseases association are not completely understood. It is assumed that the key role in the formation of polyautoimmunity is played by defects in the immune system associated with polymorphisms of various genes, in combination with exogenous and endogenous factors, especially viral infections and hormonal disorders [1, 4]. The disease in this patient, who worked in a shoe factory for a long time and had contact with glue, paints, and other toxic substances, arose against the background of coronavirus infection, consistent with the data in the literature.

Recent publications suggest a potential link between COVID-19 infection, SLE, and MPA, as, first, lung lesions during COVID-19 may mimic changes seen in patients with SLE and MPA; second, these two diseases may occur simultaneously; and third, COVID-19 may be a predictor of MPA, SLE, and AIT [2, 6]. The SARS-CoV-2 virus is an RNA-containing virus belonging to the β-coronavirus family. Recognition of the viral RNA by immunocompetent human cells results in the activation of chemokines, migration and activation of neutrophils to the lesion site, which together quickly leads to disruption of the alveolar-capillary wall [7, 5, 14].

The reason for the increase in ANA titer in viral infections may be a combination of the immune system features (hyperactivity of B lymphocytes, their ability to polyclonal activation; predominance of helper activity of T lymphocytes over suppressor activity, impaired clearance of immune complexes) and the presence of antigenic mimicry (similarity of antigenic determinants of the infectious agent and the body's own) and the release of so-called neutrophil extracellular traps (NETs) by activated neutrophils. Under this condition, the permeability of the alveolar-capillary wall is impaired, thrombovasculitis, apoptosis, and lysis of affected cells develop as a result of a “cytokine storm,” circulatory hypoxia, and hyperactivation of platelets, endothelial cells, and cells of the reticuloendothelial system [5, 6, 14]. Thrombosis is caused not only by direct injury of the vessels by the virus, but also by infiltration of the vessels by immune cells, which is accompanied by activation of pathological fibrinolysis (a marker of which is considered to be an increase in D-dimer) with numerous hemorrhages of various localizations [3]. This was evidenced by the patient's hemoptysis and increased D-dimer at the beginning of the disease. The presence of a “cytokine storm” as a third mechanism of infection generalization caused by SARS-CoV-2 was suggested in the patient based on clinical signs (hyperthermia), high levels of acute phase indicators (leukocytosis, accelerated ESR, increased levels of CRP, seromucoid and ferritin) [7].

For the treatment of these patients, pathogenetic therapy is used: glucocorticoids (GCS), cytostatics or biological agents (rituximab) or mycophenolic acid preparations. In the case of the development of severe pulmonary-renal syndromes – plasmapheresis. GCS and cytostatics were used in the treatment of the patient with a slight positive effect.

Conclusions

1. Coronavirus infection caused the development of an overlap syndrome of microscopic polyangiitis, systemic lupus erythematosus, and autoimmune thyroiditis in a patient.

2. Polymorphism of nonspecific clinical signs and multiorgan involvement can mislead physicians and misdirect diagnostic search.

3. Verification of the microscopic polyangiitis and systemic lupus erythematosus diagnosis is carried out on the basis of an increase in the level of their immunological markers included in the EULAR/ACR 2019 and 2022 classification criteria and/or on nephrobiopsy data.

4. Patients with overlap syndrome of rheumatic diseases are subject to constant monitoring by an interdisciplinary team of doctors in order to prevent the development of complications and ensure long-term remission, which will extend life expectancy and improve its quality.

Prospects for further research lie in developing an algorithm for diagnosis, differential diagnosis, and treatment of the overlap syndrome of SLE, AAV, and AIT.

References

1. Viunitska LV, Gavrilenko TI, Pidgaina OA, Ryzhkova NO, Protsenko GO. Osoblyvosti laboratornoi diahnostyky kolahenoziv. Ukr J Rheumatol. 2022;2(88):25-33. doi: 10.32471/rheumatology.2707-6970.88.17072. [in Ukrainian].
2. Yefremenkova LN, Kovalevska LA, Vrublevska SV, Hrytsyk VP, Kachurova IM, Syvyi SM, et al. Debiut systemnoho chervonoho vovchaka pislia perenesenoho COVID-19. Visn. morskoi med. 2023;1(98):86-91. doi: http://dx.doi.org/10.5281/zenodo.7806274. [in Ukrainian].
3. Kovalenko VM, Rekalov DH, Yatsyshchyn RI, Holovach IYu, Stanislavchuk MA, Ter-Vartanian SKh, et al. Systemnyi chervonyi vovchak. Klinichni rekomendatsii. Kyiv; 2020. 76 p. [in Ukrainian].
4. Oshlianska OA, Nadtochii TH, Yats KA. Perekhresni syndromy v praktytsi dytiachoho revmatoloha. Ukr. revmatolohichnyi zhurn. 2024;2(96):18-25. DOI: 10.32471/rheumatology.2707-6970.96.18766. [in Ukrainian].
5. Oshlianska OA, Okhotnikova OM, Zakharchuk OV, Rusak S.O. Multysystemnyi zapalnyi syndrom, asotsiiovanyi z infektsiieiu sars-cov-2, u ditei: nova khvoroba chy syndrom na prykladi klinichnoho vypadku. Ukr. revmatolohichnyi zhurn. 2021;2(84):1-10. DOI: 10.32471/rheumatology.2707-6970.84.16258. [in Ukrainian].
6. Talash VV. Overlap-syndrom systemnoho chervonoho vovchaka z vaskulitamy, asotsiiovanymy z antyneitrofilnymy tsytoplazmatychnymy antytilamy (ohliad literatury). Aktualni problemy suchasnoi med. 2025;25(1):260-266. doi: 10.31718/2077-1096.25.1.260. [in Ukrainian].
7. Kaidashev I, Shlykova O, Izmailova O, Torubara O, Yushchenko Y, Tyshkovska T, et al. Host gene variability and SARS-CoV-2 infection: A review article. Heliyon. 2021;7(8):e07863. doi: 10.1016/j.heliyon.2021.e07863.
8. Kato K, Kawamura T, Terashima R, Tsuchiya Y, Takahashi Y, Kasai K, et al. A Case of Systemic Lupus Erythematosus/Antineutrophil Cytoplasmic Antibody-Associated Vasculitis Overlap Syndrome with Dissociated Pathological and Immunological Findings. Case Rep Nephrol. 2020;2020:5698708. doi: 10.1155/2020/5698708.
9. KDIGO 2012 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. Kidney Int Suppl [Internet]. 2012 [cited 2024 Sept 11];3(1):1-150. Available from: https://kdigo.org/wp-content/uploads/2017/02/KDIGO_2012_CKD_GL.pdf.
10. Mashinchi B, Aryannejad A, Namazi M, Moradi S, Masoumi Z, Parsaei A, et al. A case of C-ANCA-positive systematic lupus erythematous and ANCA-associated vasculitis overlap syndrome superimposed by COVID-19: a fatal trio. Mod Rheumatol Case Rep. 2023;7(1):117-121. doi: 10.1093/mrcr/rxab007.
11. National Kidney Foundation. KDOQI Clinical Practice Guideline for Diabetes and CKD: 2012 Update. Am J Kidney Dis. 2012;60(5):850-886. doi: 10.1053/j.ajkd.2012.07.005.
12. Overlap syndromes. BMJ Best Practice. [Internet]. 2023. [cited 2025 Apr 24]. Available from: https://bestpractice.bmj.com/topics/en-gb/1018.
13. Pyo JY, Lee LE, Park YB, Lee SW. Comparison of the 2022 ACR/EULAR Classification Criteria for Antineutrophil Cytoplasmic Antibody-Associated Vasculitis with Previous Criteria. Yonsei Med J. 2023;64(1):11-17. doi: 10.3349/ymj.2022.0435.
14. Senda A, Sasai R, Kato K, Nishibata Y, Masuda S, Ishizu A, et al. Involvement of neutrophil extracellular traps in the pathogenesis of glomerulonephritis in a case of systemic lupus erythematosus and antineutrophil cytoplasmic antibody-associated vasculitis overlap syndrome. CEN Case Rep. 2022;11(3):339-346. doi: 10.1007/s13730-021-00682-y.

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