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FEATURES OF THE MANAGEMENT OF A COMORBID PATIENT WITH METABOLIC SYNDROME AND CHRONIC HEART FAILURE WITH REDUCED EJECTION FRACTION

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Angiotensin-converting enzyme inhibitors or the combination of sacubitril and valsartan, beta-blockers, mineralocorticoid receptor antagonists, and sodium-dependent inhibitors of glucose-like cotransporter type 2 (SGLT2) are the standard of care for chronic heart failure with reduced ejection fraction (HFrEF). The implementation of an SGLT2 (Empagliflozin) in heart failure phenotype with reduced ejection fraction with/without type 2 diabetes and eGFR ≥ 20 mL/min/1.73 m^2 is the result of the randomized, double-blind, placebo-controlled clinical trial EMPEROR-Reduced. Empagliflozin reduces the combined risk of death and hospitalization by 25 % and 30 % respectively, and slows the development of chronic renal failure in individuals with heart failure with reduced ejection fraction with/without type 2 diabetes. The presented clinical case corresponds to the results of the EMPEROR- Reduced clinical trial and shows that empagliflozin 10 mg/day in comorbid patients with the heart failure phenotype with reduced ejection fraction contributes not only to the improvement of left ventricular systolic function and quality of life, increased exercise tolerance and reduced frequency of hospitalizations, but also to the reduction of glycemia, normalization of blood lipid spectrum and indicators of nitrogen excretory renal function.

Key words: heart failure, coronary heart disease, myocardial infarction, cardiomyopathy, sodium-dependent inhibitors of glucose-like cotransporter type 2, metabolic syndrome.

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ОСОБЛИВОСТІ ВЕДЕННЯ КОМОРБІДНОГО ПАЦІЄНТА З МЕТАБОЛІЧНИМ СИНДРОМОМ ТА ХРОНІЧНОЮ СЕРЦЕВОЮ НЕДОСТАТНІСТЮ ЗІ ЗНИЖЕНОЮ ФРАКЦІЄЮ ВИКИДУ

Інгібітори ангіотензинперетворювального ферменту /сакубітрил+валсартан, бета-блокатори, антагоністи мінералокортикоїдних рецепторів та інгібітори натрійзалежного котранспортера глюкози 2-го типу є стандартом лікування хронічної серцевої недостатності зі зниженою фракцією викиду. Показання до застосування інгібітору натрійзалежного котранспортера глюкози 2 типу емпагліфлозина при фенотипі серцевої недостатності зі зниженою фракцією викиду з/без цукровим діабетом 2 типу та рШКФ ≥ 20 мл/хв/1,73 $м^2$ є результатом рандомізованого подвійного сліпого плацебо-контрольованого клінічного дослідження EMPEROR-Reduced. Емпагліфлозин знижує комбінований ризик смерті та госпіталізації на 25 % та 30 % відповідно і уповільнює розвиток хронічної ниркової недостатності у осіб з серцевою недостатністю зі зниженою фракцією викиду з/без цукровим діабетом 2 типу. Представлений клінічний випадок відповідає результатам клінічного дослідження EMPEROR-Reduced та показує, що емпагліфлозин 10 мг/добу у коморбідних пацієнтів із фенотипом серцевої недостатності зі зниженою фракцією викиду сприяє не лише покращенню систолічної функції лівого шлуночка та якості життя, підвищенню толерантності до фізичного навантаження та зменшенню частоти госпіталізацій, але й зниженню глікемії, нормалізації ліпідного спектру крові та показників азотовидільної функції нирок.

Ключові слова: серцева недостатність, ішемічна хвороба серця, інфаркт міокарда, кардіоміопатія, інгібітори натрійзалежного котранспортера глюкози 2 типу, метаболічний синдром, гіпертонічна хвороба, цукровий діабет 2 типу.

The work is a fragment of the research project "Cardiovascular remodeling, structural and functional state of the liver and kidneys, and their relationship with cardiometabolic risk factors in patients with cardiac pathology and comorbidities. Possibilities of treatment optimization", state registration No. 0124U002036.

In addition to a poor prognosis, congestive heart failure (CHF) significantly reduces the quality of life of patients who have difficulty with daily activities, mobility problems, and cannot take good care of themselves. The universal definition of HF includes phenotypes, depending on the left ventricular (LV) ejection fraction (EF). In patients with HF with reduced EF (HFrEF), treatment should begin with the use of concomitant renin-angiotensin system (RAAS) inhibitors (angiotensin-converting enzyme inhibitors (I,A) or sacubitril-valsartan combination (I,B)), beta-blockers (I,A), mineralocorticoid receptor antagonists (I,A), sodium-dependent glucose-like cotransporter type 2 inhibitors (SGLT2) (I,A) in low doses, or sequentially [1]. Indications for the use of the selective competitive SGLT2 empagliflozin in HFrEF phenotype with/without type 2 diabetes mellitus (DM) and eGFR ≥ 20 ml/min/1.73 m2are the result of a randomized, double-blind, placebo-controlled clinical trial EMPEROR-Reduced, which was conducted with the participation of 3730 patients with chronic HF (New York Heart Association (NYHA) functional

class (FC) II-IV and LVEF≤40 %) to evaluate the efficacy and safety of empagliflozin 10 mg/day as an adjunct to standard HF therapy [4, 12]. In this study, empagliflozin was associated with a lower combined cardiovascular risk of death or hospitalization for HF than placebo, as well as with a slower progression of renal deterioration in patients with the HFrEF phenotype, regardless of the presence/absence of type 2 diabetes. It is believed that the beneficial effect of SGLT2 in HF is due to a decrease in sympathetic tone, severity of oxidative stress, and glycemic fluctuations, increased natriuresis, improved renal function and cardiorenal interaction, reduced pre- and post-load and stress on the LV walls, and reduced interstitial edema.

The purpose of the study was to evaluate the effectiveness of treatment of HFrEF in a comorbid patient when SGLT2 (empagliflozin 10 mg/day) was added to conventional therapy in real clinical practice.

Clinical observation of a comorbid male patient S., 45 years old with HFrEF phenotype, with the addition of Sodium-Glucose Transport Protein 2 (SGLT2) Inhibitors (empagliflozin (Jardines®, Germany) 10 mg/day) to conventional therapy in real clinical practice.

As part of this clinical observation, the patient underwent a clinical, laboratory, and instrumental examination. At the first visit (06.02.2024), the man was concerned about a dry cough and shortness of breath, which appeared 3 months before the visit during severe COVID-19 in November 2023. Symptoms worsened 3 weeks before the first visit. Consciousness is clear. Blood pressure 140/100 mm Hg. Pulse 78 beats/minute, arrhythmic. Respiratory rate 24/minute. Breathing is significantly weakened in the lower parts of the lungs, especially on the left; there is no wheezing. Heart sounds are reduced and arrhythmic. Systolic murmurs over the mitral valve (MV) and tricuspid valve (TV). Accent of the 2nd tone over the pulmonary artery. The left border of the heart is displaced outward by 3.5 cm. The abdomen is painless, swollen. Leg edema. Height 178 cm. Body weight 120 kg. Body mass index 37.9 kg/m ^{2}. Cardiovascular heritage is not burdened.

ECG (02/06/2024): Sinus irregular rhythm, 78 beats /minute, ventricular extrasystole. The electrical axis of the heart is deviated to the left. Fibrosis of the posterior wall of the left ventricle. Atrioventricular (AV) block I.

Transthoracic echocardiography (TTE) (01/22/2024) revealed reduced LV EF (28 %) due to diffuse hypokinesia of its walls, and moderate mitral and tricuspid regurgitation. LV EDD 68.0 mm. LV ESD 59.0 mm, RWT 0.31. LV EDV 245 ml. LV ESV 175 ml. LV SV 69.0 ml, EF -28 %. Tachycardia and slight left-sided hydrothorax were observed.

From 02/14/2024 to 02/26/2024, the patient was hospitalized at the Vinnytsia Regional Clinical Hospital named after M.I. Pirogov with a diagnosis of DCMP, HF stage IIB, III FC (NYHA) with reduced EF (EF 28 %). Left-sided hydrothorax. AV block Ist. Ventricular Extrasystole. Obesity stage 2, mixed genesis, stable course.

Laboratory examination data (02/15/2024). Laboratory examination data (02/15/2024). Complete blood count: RBC – 6.14*1012/l, Hb 173 g/l, Plt. 341*109/l, ESR 20 mm/h, WBC. – 9.8*10^{12}/l. Urine analysis (02/15/2024): transparency is complete, reaction is acidic, specific gravity 1027, protein is absent, squamous epithelium – 0–2 in ppm, leuk. – 2–4 in ppm, mucus – +. Plasma glucose 6.8 mmol/l, HbA1c 5.9 %, fibrinogen 3.6 g/l, potassium 4.5 mmol/l, sodium 141 mmol/l, prothrombin index 81 %, total bilirubin 8.6 μmol /l, direct bilirubin 0 μmol /l, ALT 51.4 units/l, AST 25.3 units/l, urea 7.2 mmol/l, creatinine 105.8 μmol /l, GFR (CKD – EPI) 76.34 ml/ min /1.73m^2, cholesterol 6.7 mmol/l, triglycerides 1.9 mmol/l, uric acid 425 μmol /l, NTproBNP 9755 pg/mL.

Repeated TTE (26.02.2024) did not reveal any significant dynamics of the heart geometry. Additionally, global longitudinal strain (GLS), showed a decrease in kinetics (-10.5 %), which is below the normal limit for Cannon Ultrasound (-18.2 %).

Daily ECG monitoring (02/21/2024) in a hospital setting showed a trend of average daily heart rate towards normosystole (77 per minute). The average value of the PQ interval is 219 msec. with an extension at night to 250 msec. The average value of the QT interval is 403 msec. (within normal limits). Single monomorphic ventricular extrasystolic complexes were registered in a small number (135 per day). Signs of early ventricular repolarization syndrome were noticed. Diagnostically significant ST-segment dynamics are absent.

The patient was prescribed treatment: bisoprolol 2.5 mg/day, sacubitril/valsartan combination 50 mg 2 times/day, eplerenone 50 mg/day, and torasemide 5 mg/day.

It should be emphasized that the patient has components of metabolic syndrome (arterial hypertension, impaired glucose tolerance, hyperlipidemia, obesity, hyperuricemia), which are significant cardiometabolic risk factors.

Due to the severity of the condition, the patient was referred for further examination to the Amosov National Institute of Cardiovascular Surgery, where a magnetic resonance imaging (MRI) of the heart was

performed ("Toshiba Vantage Orian" 1.5 T) with gadolinium contrast (04.03.2024). The heart is typically located. Aortic root 39 mm. Ascending aorta 41 mm. Pulmonary trunk 30 mm. Mediastinal lymph nodes are not enlarged. LA longitudinal size of 7.6 cm; transverse size of LA 4.6 cm. RV: Longitudinal size of RV 5.6 cm; transverse size of RV 5.1 cm. LV: EF 24 %; KDO 320 ml; EDI 136, ml/m²; ESV 244 ml; ESI 104 ml/m²; SV 76 ml 76; CO: 6.53 l/min; CI 1.77 l/min/m²; myocardial mass 260 g; LVMI 111 g/m². EDD 73 mm. IVS thickness 16 mm, lateral wall – 11 mm. On the background of global hypokinesia, areas of deep hypokinesia and akinesia of the lower and lower-lateral segments at the basal and middle levels are noted, as well as areas of deep hypokinesia of the anterior, anterior-septal segments at the middle and apical levels. Minimal regurgitation on TC and MK. RV diameter 45 mm. Myocardial thickness 5 mm. T1 mapping /T2 mapping: T2 relaxation time of the LV myocardium was within normal limits (T2 relaxation time up to 42 ms) – no signs of myocardial edema were detected.

Global and regional T1 relaxation times from the LV myocardium were within normal limits (T1 relaxation time up to 1003 ms).

Gadolinium contrast was performed to study coronary blood flow and myocardial accumulation. As a result, the signs of post-ischemic cardiofibrotic changes were observed in the LAD basin with a lesion depth of up to 50–75 % and in the RCA basin with a lesion depth of up to 75–100 %. Dilatation of the left ventricle, decreased LV systolic function (LV EF=24 %), and minimal cardiac fibrosis of non-ischemic genesis – a phenotype of ischemic DCMP. Minimal insufficiency of the MV and TV. Hypertrophy of the LV myocardium. Dilatation of the ascending aorta. Small hydropericardium. Left-sided small hydrothorax.

The patient was recommended to take empagliflozin 10 mg/day, rosuvastatin 20 mg/day, and aspirin 100 mg/day in addition to initial prescriptions (bisoprolol, sacubitril/valsartan, eplerenone, torasemide).

On 04/09/2024, the patient was hospitalized at the Amosov National Institute of Cardiovascular Surgery, where he underwent treatment until 04/11/2024.

Laboratory examination: Blood group O (1), Rh +; HbS-Ag negative; HCV negative; HIV and syphilis antibodies negative (03/22/2024). Cito test for coronavirus antigen negative. Complete blood count: Hb 144 g/l, RBC 5.18 10^{12}/l, ESR 20 mm/h, WBC – 10.6 x 10^{9}/l, Plt. 272.6 x 10^{9} /l (04/09/2024).

On ECG (09.04.2024): normalization of PQ 0.18 msec was detected. Posterior block of the left bundle branch.

Echocardiogram (09.04.2024) – without significant dynamics, except for the increase in EF to 32 %.

CAG (10.04.2024): 30 % stenosis of the proximal third, 100 % chronic occlusion of the middle third of the RCA (scar depth 75–100 % according to MRI): 70 % stenosis of the RCA, collateral in the anterior interventricular branch of the LCA; 70 % stenosis of the middle third of the circumflex LCA and 50–70 % stenosis of the distal third of the anterior interventricular branch of the LCA.

Based on the data obtained, the following diagnosis was established: Ischemic heart disease (IHD). Ischemic cardiomyopathy. Post-infarction cardiosclerosis in the anterior and anterior-septal segments of the LV at the middle and apical levels (depth of lesion up to 50–75 %, LCA circumflex basin): in the lower and lower-lateral segments at the basal and middle levels (depth of lesion up to 75–100 %, left anterior descending artery basin). Myocardial infarction (MI) of unknown duration (MRI from 04.03.2024). Stenotic atherosclerosis of the coronary arteries (CA): 30 % stenosis of the proximal third of the RCA, 100 % occlusion of the middle third of the RCA, 70 % stenosis of the sinus node branch of the RCA, collateral to the anterior interventricular branch of the LCA; 70 % stenosis of the middle third of the circumflex branch of the LCA; 50–70 % stenosis of the distal third of the anterior interventricular branch of the LCA (CAG dated 10.04.2024). Angina pectoris of the I FC. Mild mitral a tricuspid regurgitation. Dilatation of the ascending aorta (4.1 cm). Intermittent blockade of the posterior inferior branch of the left bundle branch. HF IIA stage with HFrEF (EF 32 %), I FC. Essential Hypertension stage 3, CV risk very high. After the revision of the treatment plan, it was recommended to continue the previously prescribed drug therapy. The patient was placed on the waiting list for heart transplantation.

On 07/31/2024, the patient was reconsulted at the Amosov National Institute of Cardiovascular Surgery and underwent TTE. Cardiac ultrasound (07/31/2024): Aortic valve is tricuspid. Aortic diameter at the level of the annulus is 2.3 cm; bulbar section 3.9 cm; ascending 4.2 cm; pressure gradient 5 mm Hg; no reverse leakage. TV is normal; small reverse leakage. Pulmonary artery valve: normal. The outflow tract is enlarged. EDD 70 mm, ESD 55 mm, PWT 12.0 mm, IVS 10.0 mm. Type 1 diastolic dysfunction. Hypokinesia. EDV 255 ml; ejection fraction 36 %; CI 106.3 ml/m^2. LVM 363.1 g, LVMI 151.3 g/m^2. The diameter of the LA 45 mm. RV diameter was normal. Conclusion: The phenotype of DCM with HFrEF (ejection fraction 36 %) is more due to hypo-akinesia of the posterior wall of the LV. Minor mitral

insufficiency. Minor tricuspid insufficiency. Initial dilation of the ascending aorta (4.2 cm) without aortic valve dysfunction. No evidence of significant pulmonary hypertension. It should be noticed that the heart parameters did not change significantly, but the EF increased to 36 %. The diagnosis didn't change.

Follow-up of the patient at the "Vinnytsia Regional Clinical Hospital named after M.I. Pirogov" (05/20/2025). showed positive changes of nitrogen excretion of kidneys (urea 7.0 mmol/l, creatinine 103.1 μmol/l, GFR (CKD – EPI 78.75 ml/min /1.73m^2), blood lipid spectrum (cholesterol 4.8 mmol/l, triglycerides 1.3 mmol/l), uric acid (330 μmol/l), reduction of glycemia level (5.5 mmol/l, HbA1c 5.6 %,) and laboratory stabilization of HF (NTproBNP 199 pg/mL) when adding empagliflozin 10 mg/day to the classical treatment of HFrEF phenotype in a comorbid patient. In the background of the treatment with sacubitril/valsartan, a beta blocker, a mineralocorticoid receptor antagonist, and SGLT2, an improvement in quality of life, a decrease in complaints, and an increase in physical tolerance were noted.

Thus, during MRI, the patient was found to have cardiomegaly of ischemic genesis (comorbidity of CHD); no inflammatory nature of the changes was established at the time of the study. CAG also confirmed the diagnosis of CHD and post-infarction cardiosclerosis. Nevertheless, we do not exclude the connection of verified ischemic events with the severe course of COVID-19, taking into account the rapid destabilization of the patient's condition during infection.

It is known that the metabolic syndrome present in our patient is a significant cardiovascular risk factor. At the same time, it has been proven that the presence of metabolic disorders is associated with a more severe course of COVID-19 and the development of various complications. The results of a meta-analysis that included more than 20 million observations of the course of COVID-19 showed that acute MI occurred in approximately 0.5 % of patients with COVID-19. The incidence and risk of MI in patients who had COVID-19 were approximately 93 % higher compared with the control group during a median follow-up period of 8.5 months [15].

Direct viral effects on cardiomyocytes, which cause transcriptional changes in the cell code and cell death, endothelial dysfunction, complement activation, complement-mediated coagulopathy, microangiopathy, RAAS dysregulation, and increased pro-inflammatory cytokines, may be the cause of the onset or progression of cardiovascular diseases in COVID-19, in particular, coronary heart disease and myocardial infarction [6].

The current hypothesis states that integration of the SARS-CoV-2 genome into human DNA may occur as chimeric transcripts combining viral nucleotide sequences with cellular ones [14]. The binding of SARS-CoV-2 to angiotensin-converting enzyme 2 (ACE2) receptors results in downregulation of these receptors and increased activity of angiotensin II, triggering mechanisms of systemic vasoconstriction, apoptosis, inflammation, endothelial proliferation, cardiomyocyte damage, and ischemia. Another identified mechanism of MI development in patients with COVID-19 is coronary embolism, which is detected in 3 % of patients with acute MI [13].

Coronary artery thrombosis is more common in patients with COVID-19 due to destabilization of existing atherosclerotic plaque in the CA with a pronounced inflammatory process. Hypertrophied cardiomyocytes express higher numbers of ACE2 receptors, which may explain the clinical deterioration of patients with cardiovascular disease infected with COVID-19 [11]. However, other receptors, such as CD147, neuropilin-1, dipeptidyl peptidase 4, alanylaminopeptidase, and glutamylaminopeptidase, also interact with the S protein of SARS-CoV-2 [2]. The S protein of SARS-CoV-2 is detected in the peripheral blood of COVID-19 patients even after recovery and can itself cause damage to the endothelium of the coronary arteries. SARS-CoV-2 uses the endosomal cysteine proteases cathepsin B/L (CTSL and/or CTSB) to enter cells, particularly cardiomyocytes. The S protein promotes increased migration of cardiac pericytes, decreased endothelial cell network formation, stimulation of cytokine secretion, and increased levels of proapoptotic factors, leading to endothelial cell death [7].

Although the mechanisms of myocardial damage associated with COVID-19 are still not fully understood, this process is often linked to downregulation of ACE2 receptors, endothelial dysfunction, procoagulant status, and increased levels of proinflammatory cytokines and autoimmune aggression.

Analyzing our clinical case from a pathophysiological point of view, it can be assumed that the inflammatory process in COVID-19 could have caused a persistent state of hypercoagulability [2, 15] and autoimmune aggression [10], which, against the background of accelerated atherosclerotic process, could contribute to thrombus formation in the coronary artery, progression of coronary artery disease and the occurrence of acute MI. At the same time, other concomitant factors, such as the formation of neutrophil extracellular traps with intraplaque and circulating neutrophils, increased platelet activity, impaired fibrinolysis and a general decrease in the anticoagulant function of the endothelium, could also contribute to coronary thrombosis [3].

The literature indicates that patients with COVID-19 have an increased risk of thrombotic events and inflammatory heart disease (mainly myocarditis) [8]. However, the fact that cardiovascular risk is increased even 12 months after the acute phase of COVID-19 suggests the need for careful screening for cardiovascular disease and follow-up of patients after COVID-19.

However, although we do not have evidence to support our hypothesis of ischemic cardiomyopathy in this patient, it is safe to say that the addition of empagliflozin to standard therapy was associated with regression of heart failure and improvement in metabolic status and renal function. Our clinical observation is consistent with the results of the EMPEROR-Reduced trial, in which empagliflozin reduced the combined risk of death and hospitalization by 25 % and 30.5 %, respectively, in individuals with HFrEF with/without type 2 diabetes [4, 9, 12]. Empagliflozin also improves patients' quality of life and has a beneficial effect on HF progression according to NYHA criteria across the entire spectrum of LVEF.

In particular, when added to standard HFrEF treatment, Empagliflozin 10 mg/day resulted in normalization of NT-proBNP and glycosylated hemoglobin levels and a clinically significant increase in EF (from 28 % to 36 %).

There is a hypothesis that SGLT2 can inhibit the progression of atherosclerosis by affecting the levels of pro-inflammatory interleukins in the blood serum, inhibiting endothelial dysfunction, proliferation, and migration of vascular adhesion molecules, macrophages, foam cell formation, platelet activation, and free radical formation. This potentially beneficial effect of using SGLT2 for more than 6 months on the blood lipid spectrum from a pathophysiological point of view can be explained by a decrease in systemic glucose toxicity [5] and is consistent with the results of the EMPA-REG OUTCOME study, which showed a favorable effect of the drug on the prognosis of patients with coronary artery disease.

The treatment of the HFrEF phenotype is important regardless of the etiologic factors of HF. The clinical case proves that for patients with the HFrEF phenotype, quality of life, reduction of symptoms, and hospitalizations are as important as LVEF.

Conclusion

The experience accumulated to date indicates that severe SARS-CoV-2 infection is associated with an increased risk of ischemic events. This clinical case may be confirmation of this.

Long-term follow-up of a patient with HFrEF of ischemic origin revealed a positive effect of adding Empagliflozin to standard therapy, both in terms of improving cardiovascular function, renal function, and glucose metabolism. Objective evidence of this is a decrease in NT-proBNP, an increase in EF, a decrease in clinical manifestations of HF, an increase in GFR, and normalization of blood glucose and HbA1c levels.

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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.

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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