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ВЕСТНИК
МЕДИЦИНСКОГО КОЛЛЕДЖА
ИМ. МЕГРАБЯНА

BULLETIN
OF THE MEDICAL COLLEGE
AFTER MEHRABYAN

VOL. 15 ТОМ

ԵՐԵՎԱՆ 2023 YEREVAN

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12/19/2023

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ՄԵՀՐԱԲՅԱՆԻ ԱՆՎԱՆ ԲԺՇԿԱԿԱՆ ՔՈԼԵՋ

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РЕСПУБЛИКА АРМЕНИЯ

ВЕСТНИК

МЕДИЦИНСКОГО КОЛЛЕДЖА

ИМЕНИ МЕГРАБЯНА

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ОСОБЕННОСТИ ЭФФУЗИВНОГО ПЕРИКАРДИТА КАК ПОЗДНЕГО ОСЛОЖНЕНИЯ ПРИ COVID-19 И НЕКОТОРЫЕ ЕГО ФАРМАКОТЕРАПЕВТИЧЕСКИЕ АСПЕКТЫ
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Аннотация. Мы рассмотрели редкий случай острого перикардита с перикардиальным выпотом у 44-летнего мужчины с диагнозом COVID-19 за месяц до перикардита. У него были респираторные симптомы и положительный тест на COVID-19 за 4 недели до обращения в нашу больницу по поводу дискомфорта в груди и одышки, тахикардии, учащенного дыхания. Мы предполагаем, что у этого пациента был перикардит с выпотом в перикарде как отсроченное осложнение COVID-19, поскольку при поступлении у него был положительный результат полимеразной цепной реакции на COVID-19, и никакое другое случайное заболевание или инфекция не могли вызвать его симптомы. Хотя мы не фиксировали уровень провоспалительных цитокинов, неспецифические маркеры воспаления, такие как D-димер и С-реактивный белок, были повышены, что свидетельствует о воспалении. Ему была начата стандартная терапия перикардита – ибупрофен + колхицин, с улучшением симптомов. Во время пребывания в больнице его лейкоцитоз разрешился, уровень СРБ снизился, а боль в груди значительно уменьшилась. Этот случай подчеркивает важность продолжения расследования диагностики и лечения COVID-19 и связанных с ним симптомов. Целью данного описания случая является повышение осведомленности медицинского сообщества о возможности осложнений со стороны сердца на длительной фазе Covid-19.

Ключевые слова: *Симптомы Covid-19, перикардит, перикардиальный выпот, позднее осложнение – тахикардия.*

THE FEATURES OF EFFUSIVE PERICARDITIS AS A LATE COMPLICATION OF COVID-19 AND SOME PHARMACOTHERAPY MANAGEMENT ASPECTS (Case Report)

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Abstract. We report a rare case of acute pericarditis presenting with pericardial effusion in a 44-year-old man diagnosed with COVID-19 one month prior to pericarditis. He had respiratory symptoms and a positive test for COVID-19 4 weeks before presentation to our hospital for chest discomfort and shortness of breath, tachycardia, tachypnea. We propose that this patient had pericarditis with pericardial effusion as a delayed complication of COVID-19 because he was COVID-19 positive by polymerase chain reaction during admission and no other coincidental illness or infection could have caused his symptoms. Although we did not capture the level of proinflammatory cytokines, nonspecific markers of inflammation such as D-dimer and C-reactive protein were elevated, suggestive of inflammation. He was initiated with standard therapy for pericarditis – ibuprofen +colchicine, with improvement in his symptoms. During his hospital course, his leukocytosis resolved, CRP levels decreased, and chest pain significantly improved. This case emphasizes the importance of continued investigation regarding diagnosis and treatment of COVID-19 and its related symptoms. The aim of this case report is to raise awareness in the medical community on the possibility of complications targeting heart in the long-COVID-19 phase.

Keywords: *Covid-19 symptoms, pericarditis, pericardial effusion late complication, tachycardia.*

ԷՖՈՒԶԻՎ ՊԵՐԻԿԱՐԴԻՏԻ ԱՌԱՆՁՆԱՀԱՏԿՈՒԹՅՈՒՆՆԵՐԸ՝ ՈՐՊԵՍ COVID-19-Ի ՈՒՇ ԲԱՐԴՈՒԹՅՈՒՆ ԵՎ ՆՐԱ ՈՐՈՇ ՖԱՐՄԱԿՈԹԵՐԱՊԻԱՅԻ ԱՍՊԵԿՏՆԵՐԸ **(Կլինիկական դեպք)**

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Ամփոփագիր: Մենք ուսումնասիրեցինք սուր պերիկարդիտի հազվագյուտ դեպք՝ պերիկարդիալ էֆուզիա, 44-ամյա տղամարդու մոտ, ում մոտ ախտորոշվել է COVID-19 պերիկարդիտից մեկ ամիս առաջ: Նա ուներ շնչառական ախտանիշներ և դրական Covid-19 թեստ՝ կրծքավանդակի շրջանում անհանգստության և շնչառության, տախիկարդիայի և տախիպնեայի պատճառով: Մենք ենթադրում ենք, որ այս հիվանդը ունեցել է պերիկարդիտ՝ պերիկարդային էֆուզիայով, որպես COVID-19-ի հետաձգված

բարդություն, քանի որ ընդունվելիս նա դրական է գնահատել պոլիմերազային շղթայական ռեակցիան COVID-19-ի նկատմամբ, և որևէ այլ պատահական հիվանդություն կամ վարակ չի կարող առաջացնել նրա ախտանիշները: Չնայած մենք չենք արձանագրել բորբոքային ցիտոկինների մակարդակը, բայց ավելացել են բորբոքման ոչ սպեցիֆիկ մարկերները, ինչպիսիք են D-dimer-ը և C-ռեակտիվ սպիտակուցը, ինչը ցույց է տալիս բորբոքում: Նրան նշանակվեց պերիկարդիտի ստանդարտ թերապիա՝ Իբուպրոֆեն + կոլխիցին, ախտանիշները բարելավվեցին: Հիվանդանոցում գտնվելու ընթացքում նրա լեյկոցիտոզն անհետացել է, CRP-ի մակարդակը նվազել է և զգալիորեն նվազել է կրծքավանդակի ցավը: Այս դեպքն ընդգծում է շարունակական հետաքննության կարևորությունը COVID-19-ի և դրա հետ կապված ախտանիշների ախտորոշման և բուժման վերաբերյալ: Այս կլինիկական դեպքի նպատակն է բարձրացնել բժշկական հանրության իրազեկությունը COVID-19-ի երկարատև փուլում սրտի վրա ազդող բարդությունների հավանականության վերաբերյալ:

Հիմնաբառեր՝ *Կովիդ-19-ի ախտանիշներ, պերիկարդիտ, պերիկարդիալ էֆուզիա, ուշ բարդություն՝ տախիկարդիա:*

Introduction

Since its recognition in December 2019, Covid-19 has rapidly spread globally causing a pandemic. Covid-19 cases are reason of patient's hospital admission also today. Pre-existing comorbidities such as hypertension, diabetes, and cardiovascular disease are associated with a greater severity and higher fatality rate of Covid-19. Furthermore, Covid-19 contributes to cardiovascular complications, including acute myocardial injury as a result of acute coronary syndrome, myocarditis, stress-cardiomyopathy, arrhythmias, cardiogenic shock, and cardiac arrest. The cardiovascular interactions of Covid-19 have similarities to that of severe acute respiratory syndrome. patients typically present with respiratory symptoms; however, cardiac symptoms, such as pericarditis and pericardial effusion, have also been reported. We report a case of acute pericarditis and pericardial effusion as a delayed complication of Covid-19 [6, 11].

The pericardium is an avascular sac consisting of two layers. The outer layer (parietal pericardium) is composed of fibrous tissue, and the inner layer (visceral pericardium) is composed of serous mesothelial cells. The parietal pericardium is a thick, durable outer sac composed of collagenous connective tissue. It is attached to the diaphragm, sternum and costal cartilages. The parietal pericardium separates the heart from other mediastinal structures. The visceral pericardium is a thin layer adjacent to the epicardial surface of the heart and its epicardial fatty tissue [7, 19].

In recurrent acute pericarditis, autoinflammation has been implicated as a causative mechanism, limiting the uncertain diagnosis of «idiopathic» pericarditis. Cardiac magnetic resonance imaging, which detects persistent pericarditis, may be helpful in treating difficult-to-treat patients. Developing a risk score can help identify patients at high risk of developing complicated

pericarditis who require close monitoring and aggressive treatment. Treatment with IL-1 inhibitors has been shown to be effective in relapsing forms and has a good safety profile. Finally, acute pericarditis has recently attracted great interest as it has been reported as a side effect of Covid-19 vaccination and is suspected of being a possible accomplice of SARS-CoV-2 infection. Recent advances in the treatment of acute pericarditis have contributed to a better understanding of the disease and enable individualized treatment of the patient. However, there are still unresolved questions that require further research [5, 8, 9].

Acute pericarditis is generally a benign disease that self-limiting disease. However, in some unfortunate cases, complications may occur: short-term (eg, cardiac tamponade, which is a terrible and potentially fatal complication if not recognized and treated promptly), medium-term (recurrent pericarditis), or long-term pericarditis) [15]. The frequency of complications largely depends on the underlying etiology, which is higher in secondary forms, some characteristics of the patients: age and gender, as well as the choice of treatment, for example the use of glucocorticoids. Acute pericarditis can occur as an isolated disease or as part of a systemic disease. In the latter case, an interdisciplinary approach is necessary for a favorable result. Indeed, in so-called idiopathic forms, probably viral, patients are treated by cardiologists, and in secondary forms, collaboration between cardiologists and specialists from other medical specialties (for example, rheumatologists, oncologists) is necessary. Unlike initial acute pericarditis, recurrent pericarditis is a problematic disease that is often difficult to treat [15, 20].

The etiology of pericarditis includes infectious and non-infectious forms. Among the infectious forms, the most common is viral pericarditis, which accounts for approximately the majority of all cases of pericarditis. Recent clinically significant viral infection (upper respiratory tract infection or gastroenteritis) has been reported in many patients with acute pericardial inflammation. However, at the time of diagnosis of pericarditis, viral infection cannot be confirmed by a serological antibody test because IgM antibodies are often no longer detectable. Molecular techniques such as polymerase chain reaction on pericardial fluid or tissue from pericardiocentesis and pericardial biopsy, respectively, can help identify the infectious agent, but this is an invasive approach. and not recommended for otherwise benign illness [1, 3, 30].

Drug treatment for acute pericarditis includes NSAIDs, colchicine, and proton pump inhibitors to protect the stomach. The most commonly used NSAIDs in clinical practice are ibuprofen, aspirin, naproxen and indomethacin. Aspirin should be preferred in patients already receiving it for alternative indications (eg, coronary artery disease or peripheral artery disease). Although indomethacin has a consistent anti-inflammatory effect, concerns about side effects (mainly gastrointestinal) limit its use, especially in the elderly and patients with coronary artery disease. A gradual reduction in NSAID dosage is supported by most experts, although not supported by

high-quality evidence. Current recommendations are that the full dose should be taken over several days, and the dose of aspirin and ibuprofen should be gradually and individually reduced by 200 to 400 mg every 1 to 2 weeks over the following weeks. CRP levels should return to normal before dose reduction; otherwise, the patient is at risk of relapse. Recurrent pericarditis often requires longer-term use of NSAIDs. Interestingly, in a recent study, beta blockers in addition to standard anti-inflammatory therapy were associated with better symptom control by reducing heart rate and therefore friction between the layers of the pericardium [2, 28, 33].

In recent years, acute pericarditis has attracted attention due to new data on pathophysiological problems and new treatment methods, especially in refractory cases. The Covid-19 pandemic has further increased public and media attention to pericardial syndromes, as pericarditis is a potential complication occurring either as part of Covid-19 disease or as a complication following vaccination against it. Covid-19 and affects confidence and may affect vaccination [3, 18].

Despite great progress in the understanding and therefore treatment of acute pericarditis, there is still room for further research. In this context, the recent availability of animal models of pericarditis is of utmost importance to decipher recurrent pericarditis and possibly to identify patients prone to relapses, a major problem in the field of pericarditis. In recent years, drugs targeting the pathophysiology have been developed, such as colchicine, an NLRP3 inflammasome inhibitor, and anti-IL-1 drug. In this regard, additional medications that can cause pronounced and long-term remission after stopping treatment are extremely welcome. Although the latest ESC guidelines on pericardial syndromes shed more light on the murky picture of pericardial syndromes, they are still influenced by the highly evidence-based recommendations. Therefore, further research is needed to provide evidence-based evidence for optimal treatment of pericardial syndromes [10, 21, 29].

Goal

The main aim of the study was to analyze the features of effusive pericarditis as a late complication of Covid-19 and some pharmacotherapy intervention in case study.

Case report

A young man with no past medical history presented to the outpatient clinic with complaints: a five-day history of progressive orthopnea, conversational dyspnea, and chest pain radiating to the neck and shoulders. He reported Covid-19 infection 4 weeks prior to presentation, a mild non-productive cough and subjective fever were present, but had no other associated symptoms. He took no home medications. Recent onset progressive shortness of breath, increased heart rate at rest, which increases with slight physical activity, easy fatigue, decreased ability to

work developed progressively during last days. Activity: he has been a craftsman for years; He has been in contact with plasterboard and thermal isolation products. After Covid-19 infection he developed general weakness, decreased ability to work, easy fatigue, periodically episodes of temperature rise up to 37 degrees Celsius, he did not consult a doctor, did not conduct any diagnostic tests. The above-mentioned complaints started gradually with chest discomfort, progressed during the last 5-6 days, which became the reason for referral.

He denied a personal or family history of autoimmune conditions, a personal history of tuberculosis, also history of joint pain or swelling.

He was tachycardic and tachypneic with a blood pressure of 149/98 mmHg. Initial oxygen saturation was 96% on 5 liters nasal cannula and the patient was weaned successfully to 2 liters nasal cannula for symptomatic relief. Cardiopulmonary examination was significant for muffled heart sounds, friction rub, and bilateral rhonchi. A 12-lead electrocardiogram revealed sinus tachycardia with low voltage as well ST-elevation up to 0.5-1 mm and PR depression in I, AVL leads (See: *Figure 1*).

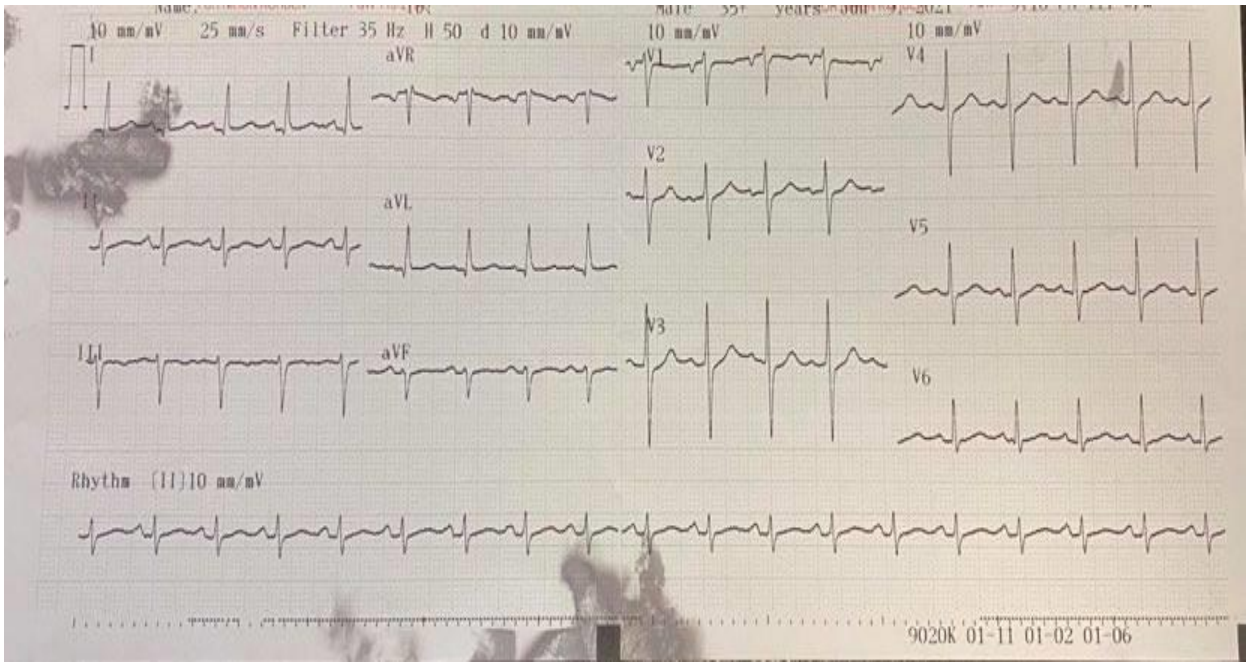

Figure 1. *A 12-lead electrocardiogram revealed sinus tachycardia with low voltage as well ST-elevation up to 0.5-1 mm and PR depression in I, AVL leads.*

A point of care echocardiographic study was performed, it revealed a pericardial effusion within 13 mm, without concern for tamponade physiology (See: *Figure 2*). Since the patient had progressive tachycardia, tachypnea during the last days, he was admitted to the ICU for close hemodynamic monitoring and special i-lation with negative airflow.

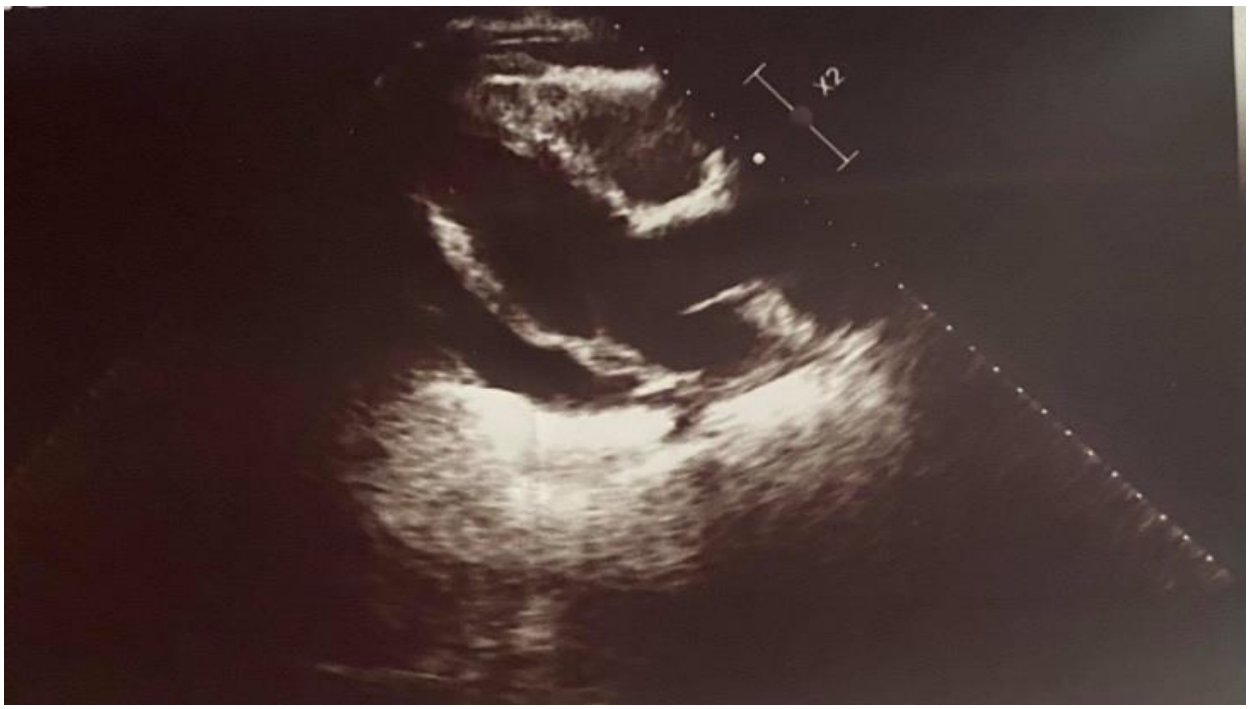

Figure 2. *Echocardiography revealed a pericardial effusion within 13 mm*

His clinical features were concerning for Covid-19, and testing via nasopharyngeal molecular polymerase chain reaction (PCR) for SARS-CoV-2 yielded a positive result. Chest X-ray was significant for cardiomegaly with the absence of pulmonary infiltrates.

Initial laboratory studies revealed leukocytosis white blood cells (WBC) 13.12 K/mcL (4.5–11 K/mcL) without lymphopenia, thrombocytosis 602 K/mcL (150–400 K/mcL), and elevated inflammatory markers including D-dimer 7.43 mcg/mL fibrinogen equivalent units (FEU) (<0.5 mcg/mL FEU), ferritin 803 ng/mL (30–400 ng/mL), lactate dehydrogenase (LDH) 423 U/L (100–250 U/L), C-reactive protein (CRP) 143 mg/L (0-10 mg/L), and rheumatoid factor 14.4IU/mL (0–14 IU/mL). Two high sensitivity cardiac troponin assays were drawn three hours apart and were both negative <9 ng/L (<14 ng/L). Procalcitonin, and lactic acid levels were within normal limits. Testing for human immunodeficiency virus (HIV) antibodies were negative. A comprehensive transthoracic echocardiogram confirmed a moderate circumferential pericardial effusion without respiratory variation to left ventricular inflow. Ibuprofen + colchicine has been started. He was initiated with this standard therapy for pericarditis with improvement in his symptoms. During his hospital course, his leukocytosis resolved, CRP levels decreased, and chest pain significantly improved. He was discharged home in stable condition with colchicine 0.5 mg two times daily for three months and ibuprofen 600 mg three times daily with plans for a gradual taper following complete resolution of symptoms and CRP normalization. Further increase of pericardial effusion and signs of tamponade were not observed, the condition stabilized, the patient subsequently continued medical therapy on an outpatient basis, one month fol-

lowing discharge, repeat echocardiogram showed complete resolution of his pericardial effusion, inflammation markers normalized.

Discussion

Pericarditis is a rare complication of the coronavirus 19 (Covid-19) infection, cases of the development of acute pericarditis have been described in the first days of the initial symptoms of the Covid-19 infection. In our report, we present a case of pericarditis, which gradually appeared over several weeks with little initial symptomatology in a young man, one months after the Covid infection.

Physicians typically assess myocardial damage (ie, necrosis) by testing serum cardiac troponins. However, myocarditis often occurs without necrosis, so the absence of elevated troponin levels does not exclude the presence of myocarditis, even severe myocarditis. Low-level SARS-CoV-2 infection can damage cardiomyocytes, leading to the release of cardiac myosin and activation of resident APCs such as mast cells and macrophages to promote inflammation in the heart. Autopsy studies conducted retrospectively to determine the number of cases of myocarditis due to Covid-19 often have a number of problems, including the need to detect inflammation and necrosis on histological examination, and the inability to report or analyze data by sex and age.

Myopericarditis is diagnosed in patients with a clinical picture of acute pericarditis with elevated troponin levels, but without disturbances in left ventricular ejection fraction or disturbances- in left ventricular wall motion [16, 31]. In contrast, patients with pleuritic chest pain, with or without pericardial effusion, but with evidence of left ventricular or biventricular dysfunction, are diagnosed with perimyocarditis and treated in the same way as patients with pure myocarditis. A subgroup of patients with myopericarditis is treated as «pure» acute pericarditis. However, ESC guidelines recommend prescribing lower effective doses of NSAIDs for shorter periods of time, as NSAIDs have been shown to increase inflammation and mortality in animal models. In addition, the currently available data are insufficient to recommend the use of colchicine in this situation.

Early comorbidities associated with Covid-19 included cardiovascular complications including arrhythmias, myocardial infarction, myocarditis, pericarditis, and thromboembolic events. Since then, numerous population-based studies have been conducted to examine the incidence or prevalence of myocarditis or pericarditis associated with SARS-CoV-2 or Covid-19 infection. Vaccines against SARS-CoV-2 have been rapidly developed, including a new mRNA vaccine platform that uses mRNA against the dominant virus antigen encapsulated in lipid nanoparticles, also known as extracellular vehicles (EVs). Soon after the initiation of vaccination pro-

grams, cases of myocarditis and pericarditis began to emerge from data from passive vaccination surveillance programs, hospital data, and countries with mandatory or integrated health care systems. Over time, many large population-based studies have examined the incidence or prevalence of vaccine-associated myocarditis [27, 32].

Pericarditis is a rare extrapulmonary complication of Covid-19 infection and has been described in isolated cases. Pericarditis should be considered as a differential diagnosis in patients with Covid-19 and unexplained persistent chest discomfort. An atypical latent infection may appear several months after the initial Covid-19 infection.

Although the exact pathophysiological mechanism of pericardial effusion in Covid-19 is unknown, it is believed to develop secondary to the cytotoxic and immune effects and systemic inflammatory response caused by SARS-COV-2. To date, there is no guideline for the treatment of pericarditis developed secondary to Covid-19, according to the single cases described in the literature, such patients were treated with standard therapy (ibuprofen + colchicine).

Rare cases of pericarditis in the initial stage of Covid-19 infection have been described, the presentation of pericarditis and myocarditis ranging from mild symptoms to lethal tamponade, however, it is difficult to connect pericarditis developed several months after the transfer of Covid infection to the transferred infection or reinfection without virus sequencing data.

The treatment of myocarditis caused by Covid-19 is essentially the same as the treatment of pre-Covid-19 myocarditis and is based on the recommendations of cardiologists and the guidelines of the European Society of Cardiology. There has been some controversy about the effect of immunosuppressive therapy such as glucocorticoids on myocarditis, although recent long-term data support their benefit. The success of anti-inflammatory approaches in patients with COVID-19 has been reported in the literature, likely due to the overwhelming pro-inflammatory and cytokine response observed shortly after SARS-CoV-2 infection.

Pericarditis can develop several weeks after the initial viral infection and is associated with coxsackievirus, herpesvirus, HIV infection or preceded by kidney pathology, myocardial infarction, trauma, tumor (breast, lung, leukemia, Hodgkin's and non-Hodgkin's lymphoma) medication – hydralazine, procainamide.

In our case, none of these pathologies preceded the manifestation of pericarditis, the symptoms began gradually and progressed with the accumulation of fluid in the pericardium, and the condition worsened.

This case is among the rare reports of pericardial effusion one month after mild Covid-19 infection. Several reports have described pulmonary involvement as the most common clinical presentation of severe infection with the severe acute respiratory syndrome coronavirus 2, extrapulmonary manifestations were increasingly identified, most often occurring concomitantly

with pulmonary disease. Pericardial disease in the absence of pulmonary findings, however, is rarely described [4]. We report acute pericarditis presenting with pericardial effusion in an adult man with prior Covid-19 as a late complication of the infection [24, 25, 34].

Covid-19 patients usually present with symptoms of respiratory infection; however, some patients complain of palpitations and chest tightness. Covid-19 patients with acute myocardial injury, acute pericarditis, and left ventricular dysfunction have been reported.

The degree of cardiac involvement does not necessarily correlate with the severity of respiratory distress, i.e. a patient may be clinically very ill with myopericarditis associated with Covid-19 but have relatively little lung involvement [4, 12, 17].

Pericardial fluid is usually exudative and does not contain viruses, so it probably represents an inflammatory rather than an infectious process. Furthermore, the degree of pericardial inflammatory reaction (and therefore the size/volume of the effusion) does not necessarily correlate with the degree of myocardial involvement, i.e., a large pericardial effusion Pericardial tamponade may be noted in the absence of acute/fulminant myocarditis [14, 23].

Patients with structural heart disease and/or underlying cardiac disease may be at increased risk of developing Covid-19 myopericarditis and cardiac tamponade [13, 26].

With pericardial injury, we can consider the possibility of 2 different scenarios: 1) a patient treated for pericarditis and subsequently infected with SARS-CoV-2 and 2) a patient with Covid-19 with pericarditis or pericardial effusion. In both cases, clinicians may have questions about the safety profile of the standard therapy drugs and the agents used in the treatment of refractory cases. (non-steroidal anti-inflammatory drugs (NSAIDs), corticosteroids, colchicine and biologic agents such as anti-IL1- anakinra [10, 20, 29].

To date, there are few data on pericarditis and pericardial effusion during and after Covid-19. Most published clinical cases are associated with myocardial involvement with troponin elevation. Although there are some cautions regarding the use of NSAIDs in Covid-19, which require additional studies, the use of corticosteroids, colchicine, and anakinra in the course of Covid-19 for the treatment of pericarditis is not contraindicated.

In this case, it is difficult to unambiguously connect pericarditis with previous Covid-19 infection, however, we should be careful in terms of extrapulmonary manifestations in patients with prolonged non-specific symptoms after transmission of Covid-19. Pericarditis should also be considered when differentiating, whether it is reinfection or latent Covid-19.

Conclusion

In conclusion, it can be said that the cardiovascular system is a potential target for Covid-19, although at this point, there are no studies that provide evidence of direct infection of heart

cells with SARS-CoV-2 and virus replication. Further studies and examination of autopsy materials will be very useful in clarifying the direct infection of the pericardium by SARS-CoV-2 and in determining the cause of pericarditis.

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