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## **DILATED CARDIOMYOPATHY IN A YOUNG PATIENT**

First identified by decompensation of chronic heart failure (CHF), requires an immediate diagnostic search for the cause of this clinical condition. Dilated cardiomyopathy (DCM) is the 3rd most common cause of CHF in the Russian Federation, as well as one of the main causes leading to heart transplantation. According to statistics, DCM is most often detected in males, more often of working age. Not only a large number of etiological factors can lead to the development of DCM, but also their combinations, which in turn poses a big problem for doctors of practical healthcare due to the difficulty of highly specific diagnosis and establishment of these causes. A special feature of the clinical case presented below is the young age of a male patient who has suffered an acute respiratory viral infection, with the formation of a further clinical picture of rapidly progressing heart failure. Our clinical case focuses the attention of the medical community not only on the complexity of timely diagnosis of possible causes of DCM, but also on the formation of further patient management tactics, with a parallel assessment of further likely consequences, and most importantly, the development of «alertness» among doctors in similar clinical situations...

Keywords: dilated cardiomyopathy, heart failure.

According to the data of the Federal State Statistics Service of the Russian Federation (RF) for 2022, diseases of the circulatory system, established for the first time in life, amounted to 4928.7, which is 33.6 per 1000 people of the population and exceeds the figures for 2021 (4455.7 and 30.3, respectively) [5]. Standardized mortality rates and their shares in the mortality structure are divided into groups: A (chronic coronary heart disease) (CHD) - 17.8±5.8%, B (cognitive impairment) - 13.9±5.9%, C (acute forms of coronary heart disease) - 4.6±2.8%, D (malformations, cardiopathies, endoand myocarditis - 4.8±2.7%) and group E (acute cerebral circulatory disorders)

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- 6,1±1,7% [8]. Dilated cardiomyopathy (DCM) is the most common cardiomyopathy. The incidence of DCM is estimated by various authors to be 5 - 7.5 cases per 100,000 population. According to statistics, men are most often ill with DCM (about 2-5 times more often than women), regardless of age. It is known that DCM is the 3rd most common cause of chronic heart failure (CHF) (RF: DCM is the cause of CHF in 0.8% of cases, and CHF of functional class (FC) III-IV - in 5.0%-5.4%), ranks 1st among all causes leading to heart transplantation [3]. Clinical experience shows that determining the etiological factor leading to the development of DCM is a rather difficult question. What could this be related to? The answer lies on the surface. If you look at the etiological classification of DCM, you can understand that it has a huge number of etiological factors from genetic causes, exposure to toxic substances, the infectious process, to autoimmune, auto-inflammatory and endocrine and metabolic disorders-related diseases [4].

In our opinion, the clinical case of a young patient with a clinical manifestation of CHF decompensation deserves special attention in conducting differential diagnosis in real clinical practice and timely treatment.

A clinical case. The patient, a 40-year-old man, was admitted to the cardiology department in August 2023 with complaints of shortness of breath that occurs with minimal physical exertion, swelling of the lower extremities, abdominal enlargement and palpitations.

Anamnesis of the disease. The patient associates the onset of the disease with the development of acute respiratory viral infection, accompanied by pronounced catarrhal phenomena.

For the first time, complaints of shortness of breath, frequent and severe cough with sparsely separated sputum, periodic chest pains on inhalation and an increase in temperature to 38-39°C appeared in early November 2017. By the end of the month, against the background of ineffective self-treatment, the patient sought medical help. The chest X-ray revealed signs of right-sided lower lobe pneumonia. With the persistence of complaints of shortness of breath, periodic discomfort in the chest when walking, fatigue, rapid heartbeat – the patient is referred for inpatient treatment. Against the background of therapy, he noted an improvement in his general condition. In early December 2017, the patient again sought medical help with complaints of chest discomfort when walking, shortness of breath and general weakness. An examination was conducted and, based on the data of anamnesis, clinic, instrumental and laboratory diagnostics, the following diagnosis was made: Basic: Coronary heart disease. Postinfarction cardiosclerosis (without date of establishment). Complication: stage I CHF, FC 2 according to NYHA. Concomitant: Community-acquired right-sided lower lobe pneumonia in the convalescence stage. In January 2018, he was hospitalized in the cardiology department, where coronary angiography was performed - no hemodynamically significant coronary artery stenoses were detected. Differential diagnosis was performed and the following diagnosis was made: DCM, treatment was prescribed. On a regular basis, the patient took medications: valsartan/sacubitril, eplerenone, torasemide, clopidogrel, amiodarone, meldonium dihydrate. Over the next 5 years, the patient was repeatedly hospitalized in various medical institutions due to the progression of heart failure (HF). He was consulted by a cardiac surgeon, and the patient was offered a heart transplant, which he refused. Due to the deterioration of his general condition due to decompensation of HF, he was hospitalized in the cardiology department.

Anamnesis of life. No heredity. Denies bad habits. Is a disabled person of the 3rd group due to a general disease. Suffers from chronic gouty polyarthritis, intermittent course, activity 1-2 st. Life history. No heredity. Denies bad habits. Is a disabled person of the 3rd group due to a general disease. Suffers from chronic gouty polyarthritis, intermittent course, activity 1-2 st..

Objective data. The patient's condition is serious. Body temperature is 36.7°C . Body weight 90.0 kg, height 175 cm, body mass index 29.39 kg/m<sup>2</sup>. The level of consciousness on the Glasgow scale is 15 points. The skin and visible mucous membranes are pale in color, dry, warm, the hairline is without features, there are no rashes/ hemorrhages, the face is puffy. Peripheral edema of the feet and shins. The lymph nodes available for palpation are not enlarged, painless, and not soldered to the surrounding tissue and skin. Musculoskeletal system without pathological changes. During percussion, there is a dulling of the percussion sound in the lower parts. In the lungs: vesicular breathing is weakened, widespread wet wheezing from the basal parts to the middle of the shoulder blades, respiratory rate (BPD) 23 per minute, SpO2 93%. Percutorially, the boundaries of relative cardiac dullness are expanded to the left by 1.5 cm, heart tones: arrhythmic, muted, heart rate (HR) = 110 per 1 minute, blood pressure (BP) 90/40 mmHg.. The tongue is physiologically colored, the papillae are well expressed, soft and elastic on palpation, mobility is preserved in full. Palpation of the abdominal organs is painless. The abdomen is enlarged in size, the liver protrudes from under the edge of the costal arch by 2 cm.

**Laboratory diagnostics:** General blood test: Hemoglobin – 148 g/l, erythrocytes – 5.07 x 10<sup>12</sup>/L, platelets – 151 x 10<sup>9</sup>

/L, leukocytes - 11.9 x 109/L, erythrocyte sedimentation rate - 5 mm/h. Biochemical blood analysis: glucose - 5.3 mmol/L, alanine aminotransferase - 14.1 U/L, aspartate aminotransferase - 27 U/L. creatine phosphokinase - 21.7 U/L, creatinine - 166 mg/L, urea - 22.89 mmol/L, total protein - 72.2 g/L, bilirubin - 51.7 mmol/L, K+ - 5.3 mmol/l, Na+ - 133 mmol/l, Ca - 0.98 mmol/l, pH - 7.320, LDH - 73.0, albumin - 36.4 g/l. General urine analysis: pH - 5.5, protein - 1.87 g/l, specific gravity - 1013 g/l. The dynamics show thrombocytopenia, moderate erythropenia, a progressive increase in the level of liver enzymes, creatinine and urea, as well as increasing proteinuria, uric acid - 799 mmol /l. Coagulogram: RT (sec) - 24.9, PT PT - 42.6, ARTT - 47.5, INR - 1.92. Lipidogram: total cholesterol - 1.95 mmol/L, low-density lipoprotein cholesterol - 0.8 mmol/L, Triglycerides - 0.99 mmol/L.

Instrumental diagnostics. According to the archive of electrocardiograms (ECG) since 2017, there are signs of focal changes in the anterior wall of the left ventricle (LV). In 2019, the following changes were registered for the first time: hypertrophy of the LV and left atrium (LA) with overload and impaired intraventricular conduction. In 2020, incomplete blockade of the right leg of the Gis bundle, atrioventricular blockade of the 1st degree. In May 2023, 3:1 atrial flutter was first recorded with an atrial contraction rate (AR) of 210 beats/min and a ventricular contraction rate (HR) of 70 beats/ min. An ECG in August 2023, at the time of hospitalization, recorded atrial flutter 2:1. heart rate 180 beats/min. heart rate 90 beats/min, overload of the right heart, low voltage (Figure No. 1).

Chest X-ray: On the chest X-ray in direct projection, the patient is rotated. Visible pulmonary fields – diffuse areas of heterogeneous infiltration of low intensity are determined in the right pulmonary field in the lower sections. There were no focal infiltrative changes in the left pulmonary field. The pulmonary pattern in the right lung is unevenly reinforced, low-structured, low intensity, deformed. In the left lung, it is not reinforced, low

intensity, not deformed. The shadow of the heart is expanded. The shadow of the roots of the lungs: the right root is slightly structured, not expanded, of medium intensity. The left root is not visualized. Sinuses: signs of free fluid in the right pleural cavity up to the level of the middle segment of the 4th rib, the left sinus is not visualized. The contours of the diaphragm on the left are clear and even, on the right they are not visualized. Signs of a paracostal closed hydrothorax on the right in the projection of a small interlobular gap. No bone - traumatic pathology was revealed. Conclusion: infiltrative changes in the right lung are probably of inflammatory etiology. X-ray signs of a right-sided hydrothorax with a liquid volume of 800 ml. X-ray signs of a paracostal obfuscated hydrothorax on the right. Signs of cardiomegaly (Figure No. 2).

Ultrasound examination of abdominal organs: ultrasound signs of moderate changes in the liver, pancreas, kidneys. Hepatosplenomegaly. Ascites. Fluid in the pleural cavity. Instrumental diagnostics

According to the echocardiography archive (ECHOCG) (2017). Enlargement of the LA. Zones of a-/hypokinesis of the anteroseptal region of the LV. Regurgitation on the mitral valve (MV) 0-1 st. Ejection fraction (EF) of the LV 50%. In mid-2019 - Hypo-, akinesis of the LV in the area of the interatrial septum (IVS), anterolateral wall (ALW) of the LV, anterior wall of the LV, tricuspid valve insufficiency (TVI), mitral valve insufficiency (MVI), dilation of the chambers. LV EF 38%. A year later - dilation of all cavities of the heart. Global contractility of the LV is reduced. Left ventricular diastolic dysfunction (LVD) st. 1 st. LV EF 35%. In 2022 - dilation of all cavities of the heart. Global LV contractility is reduced. LV diastolic dysfunction, diffuse hypo-, akinesis of the anterior septal wall and apex. LVD stage 3. EF 33%. By May 2023 - dilation of all heart cavities. Global LV contractility is reduced. Left ventricular diastolic dysfunction, diffuse hypo-, akinesis of the anterior septal wall and apex. LVD stage 3. EF 33%.

At the time of hospitalization in the



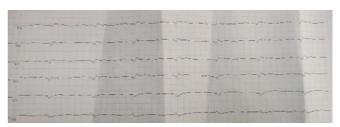


Fig. 1. ECG at the time of hospitalization (August 2023)

cardiology department in August 2023 - LP - 45 mm, end-diastolic size (CDR) LV – 60 mm, end-diastolic volume (CDR) LV - 180 ml, EF (according to Teichgolz) - 30%, interventricular septum (LV) - 11 mm, posterior the wall of the left ventricle (LV) is 10 mm, the right ventricle (RV) is 38 mm, the pulmonary artery (LA) is 22 mm. Dilation of all cavities of the heart. The LV myocardium is not thickened. Diffuse hypokinesis, hypo-, akinesis of the anterior septum wall and apex. Global LV systolic function is drastically reduced. The thoracic aorta is not dilated in all sections. The aortic half-moons are compacted, the opening is sufficient. The wings of the MV are not elongated, not compacted, their opening is sufficient IMV 1.5 st. ITV 3 st. Tricuspid regurgitation. Regurgitation on the pulmonary artery valve. The pressure in the LA is increased (pulmonary hypertension of 1 ct). The inferior vena cava is not dilated. it collapses more than 50% when breathing. At the time of the study, a moderate amount of fluid in the pericardium (up to 500 ml) is determined. Diastolic divergence of pericardial leaflets: behind the posterior wall of the LV up to 16 mm, behind the lateral wall of the LV up to 15 mm, behind the free wall of the pancreas up to 8 mm (Figures No. 3 and No. 4).

The conclusion of the nephrologist. Prerenal acute renal failure (ARF), against the background of chronic renal failure (CRF) stage C3b. The glomerular filtration rate according to CKD-EPI is 37.16 ml/min/1.73 m<sup>2</sup>.

Based on complaints, anamnesis data, laboratory and instrumental diagnostic methods, a clinical diagnosis was made: The main one: Dilated cardiomyopathy. Rhythm disturbance according to the type of constant form of atrial flutter 2:1. Complications: stage II B CHF, with reduced systolic function (EF 30%). FC 4 by NYHA. Ascites. Hepatosplenomegaly. MVI 1-2 st. TVI 3 st. Regurgitation on the pulmonary artery valve. Pulmonary hypertension 1 art. Right-hand hydrothorax. Hydropericardium. Prerenal acute renal failure on the background of CRF, CKD C3B, glomerular filtration rate according to CKD-EPI 37.16 ml/min/1.73 m2. Concomitant: Gout.

Treatment was prescribed: diet No. 10, designed for diseases of the cardiovascular system in order to create favorable conditions for blood circulation, reduce the load on the heart, blood vessels and kidneys, normalize water-salt, ion metabolism, simplify the elimination of toxic products of metabolic processes. Medications: infusion therapy, metabolic therapy, valsartan/sacubitril, dapaglifloz-



Fig. 2. Chest X-ray

in, eplerenone, metoprolol, torasemide, clopidogrel.

The scales (CHA2 DS2 -VASc) were evaluated - 1 point (LVEF <40%) and (HAS-BLED) - 3 points (creatinine over 200 mg/l – 1 point. Impaired liver function (bilirubin increased by more than 2 times) - 1 point. Labile INR - 1 point) - the patient has indications for anticoagulant therapy, but due to the high hemorrhagic risk, disaggregant therapy is prescribed.

Against the background of the treatment, stabilization of the patient's condition is noted, however, a progressive decrease in PV and the severity of the patient's condition requires urgent consultation with a cardiac surgeon in order to determine indications for heart transplantation. The patient is recommended to: diet No. 10, dynamic monitoring by a cardiologist, nephrologist at the place of residence. At the stage of patient observation, cardiac MRI and Holter ECG monitoring data were not provided. It is recommended to perform echocardiography, daily monitoring of ECG and blood pressure in dynamics, ultrasound Dopplerography of the brachiocephalic arteries on an outpatient basis as planned. Recommended for permanent use: valsartan/sacubitril (under the control of blood pressure, blood electrolytes), dapagliflozin, eplerenone, metoprolol, clopidogrel, torasemide.

Discussion. Taking into account the complaints and anamnesis of the disease of a young man, at the diagnostic stage, the issue of differential diagnosis of diagnoses was solved: postinfarction cardiosclerosis according to ECG data, myocarditis with decompensation of CHF. The revealed scarring of the anterior LV wall on the ECG and the absence of hemodynamic significant stenoses in the coronary arteries according to coronary angiography led to the need for further examination of the patient and differential





Fig. 3. Echocardiography at the time of admission (August 2023). Dilation of all cavities of the heart, overload of the right parts, regurgitation on the tricuspid valve. Fig. 3. echocardiography at the time of admission (August 2023). Dilation of all cavities of the heart, overload of the right parts, regurgitation on the tricuspid



Fig. 4. echocardiography at the time of admission (August 2023). Regurgitation on the pulmonary artery valve

diagnosis.

The problem of diagnosis and treatment of myocarditis in clinical practice stands in a separate place due to the complexity and polyethologicity of the pathological process, the practically absent specific clinical picture limiting pretest diagnosis, the difficulty of conducting a lifetime endomyocardial biopsy (EMB) [1, 6], which is the "gold standard" for the diagnosis of this disease, the basis

for the formation of further tactics patient management [6]. The incidence of myocarditis is 20-30% of all non-coronary heart diseases [1]. With a subacute clinical form not recognized in time, accompanied by latent symptoms at the onset of the disease, with a prolonged course, subsequently leads to the development of structural and functional changes in cardiomyocytes, which causes a further decrease in LV EF and LV dilation [2, 9]

The outcome of all of the above is the transformation into DCM, which, according to clinical observations and autopsies, was detected in a fairly large percentage. There is also evidence of a high percentage of spontaneous recovery (57% on average). According to statistics, morphological signs of myocarditis were not confirmed in patients with DCM who underwent EMB in up to 16% of cases in adults and up to 46% of cases in children. The clinical course of myocarditis described above is important to take into account when making a diagnosis, however, the main and "gold standard" diagnosis that allows you to confirm the diagnosis is the implementation of EMB, performed after an MRI with paramagnetic contrast, to identify the sites of biopsy sampling (at least 3 samples), the data of which are evaluated using the Lake Louise criteria. It should be borne in mind that the sensitivity of this method depends on the number of samples taken (4-5-50%), 17 - 79%) [6, 7].

Criteria for the diagnosis of DCM: according to the results of echocardiography, LV dilation or both ventricles of the heart (index of final diastolic volume) LV > 74 ml/ m2 in men and > 61 ml/ m2 in women; significant (EF < 40%) or moderate (EF- 41-49%) decrease in LV EF and the presence of: symptoms and/or signs of CHF; diffuse LV hypokinesia; relative LV wall thickness less than 0.3 (except for diseases with an initially hypertrophic morphofunctional phenotype of the heart); the verified cause of dilation characteristic of DCM (genetic defect, toxic, endocrine, metabolic, alimentary, immune, autoimmune, postinfectious factor, pregnancy). The above criteria are used in the Russian Federation, but there is another classification developed by experts of the World Heart Federation - "MOGE(S)" (Morpho-functional, Organ/ system involvement, Genetic, Etiological annotation, Stage, 2013) (Table No. 1), which is based on a combination of letters and numbers that allow you to create each patient has an individual disease code that transmits maximum information about the patient [3].

The modern approach to the treatment

## Classification of cardiomyopathies according to "MOGE(S)"

| The key parameter and its letter code   |  |
|---|--|
| M – phenotype   | O – organs and/or systems involved           |
| D - dilated cardiomyopathy  | H – heart                                    |
| H – hypertrophic cardiomyopathy   | M – skeletal muscles                         |
| R – restrictive cardiomyopathy  | N – nervous system                           |
| A – Arrhythmogenic dysplasia of the right ventricle                                 | C – skin                                     |
| NC – non–compact myocardium   | E – eye                                      |
| NS – non–specific phenotype   | A – hearing                                  |
| NA – information is not available   | K – kidneys                                  |
| E – previously identified conditions with details (E(D), (E(H), (E(R), (E(A), etc.) | G – gastrointestinal tract                   |
|   | S – skeleton                                 |
|   | Lu – lungs                                   |
|   | Li – liver                                   |
|   | (O) – absence of defeat                      |
| <b>G</b> – type of inheritance  | E – etiology                                 |
| N – absence of family heredity  | G - genetic                                  |
| U - unknown   | <b>G-OC</b> – is the carrier of the mutation |
| AD – autosomal dominant   | <b>G-DN</b> – is a genetic new               |
| AR - autosomal recessive  | <b>G-C</b> – more than one mutation          |
| XLR – X-chromosome-related recessive  | <b>G-Neg</b> – no mutations                  |
| XLD – X-chromosome-related dominant   | <b>G-NA</b> – genetic study not available    |
| XL – X-chromosome-related   | G-A – genetic amyloidosis                    |
| M – on the mother's side  | A –amyloidosis                               |
| <b>DN</b> – new mutation  | <b>A-k</b> – amyloidosis, type K             |
| (O) - absence   | M – myocarditis                              |
|   | V – viral infections                         |
|   | Al – autoimmune                              |
|   | I – infections other than viral              |
|   | T - toxic                                    |
|   | Eo – eosinophilia                            |
|   | (O) – there is no genetic test               |
| S – stage of heart failure (ACC/AAC:A; B; C; D NYHA class: I; II; III; IV)          |  |

of patients with DCM has proven its effectiveness in many clinical studies. The tactics of drug management of patients with is based on the principles of therapy of patients with HF with low EF. Quadri therapy is recommended for patients with HF (FC II-IV) and PV less than 40%. This therapy is necessary to reduce the risk of disease progression and includes the use of: angiotensin converting enzyme (ACE) inhibitors or angiotensin II receptor antagonists (ARA II) or group drugs (ARNI) (valsartan/sacubitril), beta-blockers (BB), ivabradine (in case of intolerance to BB), mineralocorticoid receptor antagonists (AMCR), type 2 sodium-glucose cotransporter inhibitors (iNGLT2, SGLT2 inhibitors), with rapid progression of CHF with the development of edematous syndrome - diuretics. Stable dynamics was achieved against the background of drug support, but the prognosis is more likely

unfavorable due to the progression of HF and dilation of all parts of the heart.

A young patient with terminal HF needs not only drug therapy, but also timely resolution of the issue of donor heart transplantation, which is not always available in clinical practice, due to the relatively small percentage of transplants performed. According to statistics from the National Medical Research Center for Transplantation and Artificial Organs named after Academician V.I. Shumakova performed 148 heart transplantations in 2024 [10], but we must not forget that the need for heart transplantation is many times higher than the current statistical indicators of successful operations.

**Conclusion**. The presented clinical case of a patient with the consequences of an acute respiratory viral infection in a young patient draws the attention of the medical community not only to the need

for timely diagnosis and the choice of the right management tactics, but also to the prediction of possible complications. Assessing the growing proportion of patients with CHF in the population and the socio-economic damage, it is necessary to consider the modernization of the approach to diagnosis and treatment, and most importantly, the prevention of these clinical situations.

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## CLINICAL CASE OF SEVERE **CORONAVIRUS INFECTION COMPLICATED** BY BILATERAL HOSPITAL-ACQUIRED PNEUMONIA IN A PREGNANT WOMAN

This article presents a clinical case of a severe course of COVID-19 in a 27-year-old pregnant woman. The infectious process was complicated by bilateral out-of-hospital pneumonia. A woman was admitted to the obstetric infectious disease unit at 39 weeks gestation, complaining of an attack-like cough with scanty sputum, a feeling of tightness in the chest, runny nose, weakness, increased body temperature up to 37.9 C. SARS-Cov-2 virus RNA was diagnosed by PCR. The condition progressively worsened overnight, the pregnant woman was transferred to non-invasive ventilation in the intensive care unit, with lung parenchyma lesions up to 50%.

Against the background of progressing respiratory failure, acute respiratory distress syndrome woman delivered by emergency cesarean section at 39 weeks' gestation. A live, premature baby girl was born, without asphyxia. The patient's condition remained extremely serious. On the 2nd day of the postoperative period sepsis was diagnosed, the lesion of lung parenchyma amounted to 75-100%

Multicomponent therapy, including recombinant monoclonal antibodies to the interleukin-6 receptor, resulted in improvement.

Keywords: COVID-19, pregnancy, respiratory failure, community-acquired pneumonia, multicomponent therapy

**Introduction.** The global pandemic of a novel COVID-19 coronavirus infection caused by SARS-CoV-2 virus has had a strong impact on the whole world [3, 4, 8]. Since then, a tremendous breakthrough in science has been

made and new diagnostic techniques, prevention, treatment and organizational approaches have been developed in the management of individuals with COVID-19 [5,9].

COVID-19 infection is characterized