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ŞƏKƏRLİ DİABET VƏ ARTERİAL HİPERTENZİYA FONUNDA MİOKARD İNFARKTININ QULAQCIQ FİBRİLYASİYASI SAYƏSİNDƏ GİZLİ KEÇMƏSİ TƏSADÜFÜ

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Məqalədə sol mədəciyin arxa divarında II tip şəkərli diabet və arterial hipertenziya fonunda törənmiş kəskin subendokardial miokard infarktı təsadüfü haqqında məlumat verilmişdir. Müəlliflərin öz müşahidələrinə əsasən haqqında məlumat verilən xəstədə ürəyin kəskin zədələnməsinin yeganə əlaməti qulaqcıqların uzun sürən taxisistolik formalı fibrillyasiyası olmuşdur. Aparılan kompleks terapevtik müalicə kompleksinə baxmayaraq, xəstə ölmüşdür. Xəstədə kəskin subendokardial miokard infarktı olduğu isə yalnız ölümdən sonra patanatomik təsrih zamanı müəyyənləşdirilmişdir.

Məqalədə təsvir edilən klinik hadisə miokard infarktının qulaqcıqların gizli keçən fibrilyasiyası fonunda atipik şəkildə keçə bildiyini nümayiş etdirir. Bu klinik təsadüf qulaqcıq fibrilyasiyası olan pasiyentlərin kompleks şəkildə müayinə edilməli olduğunu və ilk növbədə miokardda baş verə bilən geriyədinməz zədələnmələrin olub-olmadığını araşdırmağın vacibliyini göstərir.

Açar sözlər: *səyrici aritmiya, miokard infarktı, ürəyin işemik xəstəliyi*

Ключевые слова: *мерцательная аритмия, инфаркт миокарда, ишемическая болезнь сердца*

Key words*: atrial fibrillation, heart attack, coronary heart disease*

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A CASE OF HIDDEN MYOCARDIAL INFARCTION BY ATRIAL FIBRILLATION AGAINST THE BACKGROUND OF DIABETES MELLITUS AND ARTERIAL HYPERTENSION

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This article presents information about acute subendocardial myocardial infarction of the posterior wall of the left ventricle, which developed against the background of type 2 diabetes mellitus and arterial hypertension. The only clinical symptom of critical heart disease was a tachysystolic form of permanent atrial fibrillation. Despite a complex of therapeutic manipulations, the patient died. The final diagnosis of acute subendocardial myocardial infarction of the posterior wall of the left ventricle was established only after autopsy.

The presented clinical case demonstrates an atypical course of myocardial infarction manifested by latent atrial fibrillation. This case focuses on the need for a comprehensive examination of patients with atrial fibrillation and the exclusion, first of all, of irreversible myocardial damage.

Introduction. In most cases, atrial arrhythmias are the cause of early mortality in acute myocardial infarction [1, 2]. Atrial fibrillation (AF) is a very common heart rhythm disturbance and belongs to actual problems of modern cardiology and arrhythmology. Among adults, it reaches 0.5–2.0%, while in the elderly people, it occurs in

10-15% [3]. According to VALIANT randomized controlled trial (RCT), which was referred by the European Congress of Cardiology (2004) [4], 3 years after myocardial infarction AF is significantly associated with increased incidence of combined cardiac pathology (cardiovascular mortality, recurrent myocardial infarction, heart failure (HF), resuscitation, sudden cardiac death).

Diabetes mellitus (DM) plays an important role in the progression of coronary artery disease and atrial arrhythmia. This is one of the independent risk factors increasing the frequency of atrial arrhythmia by about 2-fold, and in the presence of arterial hypertension – by 3-fold. It can be assumed that the occurrence of AF in diabetes contributes to the development of so-called diabetic cardiomyopathy and diabetes-associated cardiac autonomic neuropathy and disturbance of microcirculatory bed [5] with systemic endocrine disturbance in menopause espessialy [6]. The prevalence of diabetes mellitus among the population is growing rapidly and is projected to increase from 2.8% in 2000 to 4.4% in 2030, so its combination with AF dramatically affects the prognosis for these patients. This combination is going to become more and more common [7, 8].

A heart attack is a very dangerous condition. About 30% of all cases treated at a hospital within the first hour of symptom onset are fatal. Among inpatients, 13% to 28% die during the first 28 days. In the first year following a heart attack, 4% to 10% of patients die, while among those over 65 years old – 35% of patients die [9, 10]

The combination of diseases such as coronary heart disease, atrial fibrillation, myocardial infarction, and diabetes causes difficulties for diagnosis and treatment, since sequential pathogenetic processes are disrupted generally leading to undesirable results. Our patient had clinical signs characteristic of such a difficult diagnostic case, and for this reason we decided to investigate the history of the disease in detail.

Case presentation. *A 69-year-old woman with overweight was admitted to the Otorhinolaryngology Unit of Sumy Regional Hospital on 11 Sep 2020 with a diagnosis of epistaxis. Objective findings: Nosebleed, weakness, shortness of breath, tense rapid breathing, which made her seek medical help. From her anamnesis, it was revealed that she had secondary nephropathy, type II diabetes mellitus. She took a diuretic and cardioselective beta-blockers for arterial hypertension and metformin for diabetes every day.*

Complete blood count: HGB – 52 g/l (normal range: 110–160), RBC – $1.67*10^{12}$/l (normal range: 3.9–5.3), WBC – $18.6*10^{9}$/l (normal range : 4–9), ESR – 20 mm/h (normal range: 1–10), PLT – $150.3*10^{9}$/l (normal range: 150–390), Segm – 80% (normal range: 47–72), Mon – 6% (normal range: 3–10), Lym – 5% (normal range: 19–37). Biochemical blood test dated 11.09.2020: Total Protein – 74.2 g/l (normal range: 66–87), Urea – 16.6 mmol/l (normal range: 3–9.2), Creatinine – 274 μmol/l (normal range: 62–124), ALT – 0.36 mmol/l (normal range: 0.12–0.88), AST – 0.46 mmol/l (normal range: 0.1–0.45), Total Bilirubin – 9.9 μmol/l (normal range: 8.6–20.5), Glucose – 6.7 mmol/l (normal range: 4.56–6.38). Coagulogram dated 12.09.2020: Activated Partial Thromboplastin Clotting Time – 40 seconds (normal range: 30–40), Plasma tolerance to heparin – 14min (normal range: 7–15), Fibrinogen – 2.64 g/l (normal range: 2–4), PTI – 75% (normal range: 70–130). Urinalysis: yellow, transparent, acidic, specific gravity – 1018, protein – 0.035 g/l, leukocytes – 10–15 per FoV, mucus in large quantities.

Physical examination showed that the patient's condition was of moderate severity. Blood pressure – 130/80 mm Hg. Consciousness clear, skin pale. Legs and abdomen edematous. Palpitations with signs of arrhythmia, muffled tones, 115 beats/min. In the lungs: difficult, suppressed breathing in the lower part. NPV – 16/min. She was treated by an ENT doctor: anterior and posterior nasal packing was performed.

The patient's condition aggravated, and she was transferred to the department of anesthesiology and intensive care. She was evaluated by an endocrinologist, cardiologist, pulmonologist, surgeon, ophthalmologist, and hematologist. Spinal and sternal punc-

tures were performed; esophagogastroduodenoscopy (EGDS) could not be performed due to the condition severity. Electrocardiography (ECG) (Fig. 1), abdominal ultrasound, chest X-ray were performed. The patient had infusion therapy, blood transfusion.

Ophthalmologist consultation: angiosclerosis, retinal angiopathy of both eyes.

Cardiologist consultation: coronary heart disease, diffuse cardiosclerosis, tachysystole form of permanent atrial fibrillation, premature ventricular contraction, heart failure (class IIB–III). Liver cirrhosis, hydrothorax, hydropericardium. Ascites, type I diabetes in the stage of subcompensation. Erosive nasal bleeding, gastroduodenitis. Severe anemia. Recommendation: coagulants (Digoxin), diuretic (Furosemidin), S01EV – cardiac drugs (Thiotriazolini), potassium-preserving diuretic (Verospiron). ECG: there was no data on myocardial infarction, because an additional V7-9 lead, which is responsible for the posterior wall of the myocardium, was not performed (Figure).

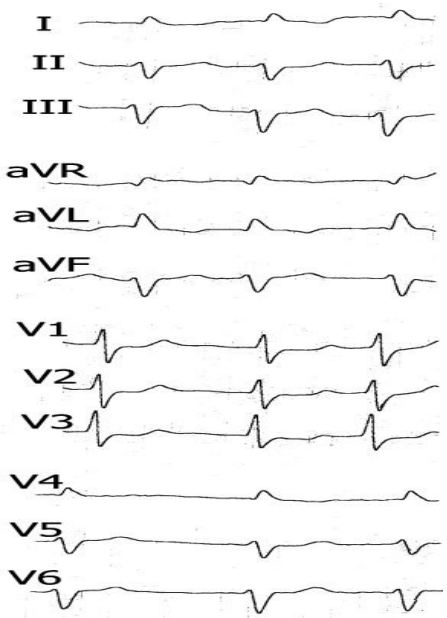

Figure. *Electrocardiogram of the patient. 10 mm / mV, 50 mm / s, F 50 Hz, F 35*

Ultrasound of the abdominal cavity dated 11 Sep 2017: hepatomegalia, liver hyperechogenicity; spleen, pancreas, kidneys are not visible (due to flatulence). Free fluid in the abdominal cavity. Chest X-ray dated 11 Sep 2020: congestion in the lungs against the background of cardiac pathology, aortoatherosclerosis. Pleurodiaphragmatic adhesions on the right side. Analysis of cerebrospinal fluid: liquid – transparent, colorless; protein – 0.33 g/L; cytosis – 4 lymphocytes; chlorides – 98 mmol/L; leukocytes 0-1-2 in the field of view.

Despite all the medical care provided, the patient's condition remained extremely severe and she died. A post-mortem study of the heart showed red focus partially extending to the endocardium and myocardium on the posterior wall of the left ventricle. Areas of growth of connective tissue are observed as small foci of gray fibrous layers in the left ventricular wall. Areas of the contours of individual muscle fibers without nuclei or in a state of karyolysis have been revealed, with free blood cells between them. The formation of a barrage of leukocytes indicates that the necrosis (infarction) developed about 72 hours ago.

Discussion. Simulation of medical process is important part of different field in modern medicine [12, 13]. Atrial fibrillation is associated with a variety of cardiovascular diseases that contribute to the development and progression of cardiac arrhythmias. These include: arterial hypertension; class II-IV heart failure according to NYHA; acquired defects (usually mitral) of the heart valve; congenital heart defects (atrial septal defect, ventricular wall abnormality, transposition of the great arteries, etc.); cardiomyopathy (especially dilated); coronary heart disease (occurs in 20% of patients with atrial fibrillation); inflammatory processes (pericarditis, myocarditis); tumors of the heart (myxoma, angiosarcoma, etc.). Organic heart disease can cause structural remodeling of the atria and ventricles. In the atria, this process depends on the proliferation and differentiation of fibroblasts into myofibroblasts, as well as on increased proliferation of connective tissue and fibrosis. All this leads to dissociation and heterogeneity of electrical activity of muscular fascicles, thereby contributing to the development of AF [14].

There are many hypotheses for the mechanisms of AF development, but the most common are the theory of focal mechanisms and the hypothesis of multiple small waves.

Moreover, these mechanisms can be combined with each other. Focal mechanisms include triggered activity and microtype reentry. According to this theory, AF occurs as a result of multiple impulses from autonomous foci, which are most often located in the orifices of the pulmonary veins or along the posterior wall of the left atrium near the junction with the pulmonary vein. The tissues in these zones have a shorter refractory period, thus, resembling the properties of the sinus node cells. As paroxysmal form progresses to constant form, the foci of increased activity are distributed throughout the atria. According to the hypothesis of multiple small waves, AP persists as a result of chaotic conduction of many independent minor excitations [15,16].

Changes in the atria can also occur after the onset of AF. In this case, the refractory period of the atria is shortened by reducing the calcium current through the L-type channels and increasing the potassium entry into the cells. Also, the contractile function of the atria decreases due to a decreased entry of calcium ions into cells, impaired release of calcium ions from intracellular depots, and impaired energy metabolism in myofibrils. The blood flow in the atria slows down due to a contractility disorder, which leads to blood clot formation, mainly in the left atrial appendage. As a result, complications may develop, for example, myocardial infarction [17].

It is well known that the prevalence of coronary artery disease is 2 to 4 times higher in the patients with type 2 diabetes mellitus, 3 times higher in the patients with essential hypertension, and 4 to 7 times higher in the patients with acute myocardial infarction. In the patients without prior myocardial infarction, IM risks were 3.5% (in nondiabetic subjects) and 20.2% (in diabetic subjects), while in diabetic patients with prior myocardial infarction the IM risks equaled 45% [18]. In conclusion, we can say that the risk of unfavorable outcome in patients with type 2 diabetes mellitus is as high as in patients with ischemic heart disease or postinfarction cardiosclerosis. In addition, that the progression of damage to the heart muscle and nerve fibers is exacerbated by impact of diseases of another organs. It should be noted that the clinical manifestations of coronary artery disease in diabetic patients have a number of features. First, the frequency of the disease is irrespective of gender, while among diabetic patients, the coronary heart disease is more common in men. Secondly, patients with diabetes mellitus often develop painless ("silent") forms of coronary heart disease. Silent myocardial infarction in the presence of autonomic neuropathy is accountable for late diagnosis and delayed treatment and, as a consequence, a higher frequency of complications [19]. Ischemic heart disease often leads to sudden death due to arrhythmia, and in diabetic subjects, the risk of such an outcome increases two-fold [20].

If ECG yields little information in a patient with suspected myocardial infarction, it is recommended to assay markers of necrosis (both early and late) after 2 to 4, 6 to 9, and 12 to 24 hours [21-23]. In this regard, the specialists of the US National Academy of Clinical Biochemistry recommend considering the moment of patient's admission, rather than the time of pain onset. Thus, blood tests should be carried out no earlier than 6 hours after pain episode onset. A significant increase in diagnostic markers level in two sequential analyzes is sufficient for MI diagnosis. A single measurement indicating increased cardiac troponin level is sufficient to diagnose myocardial infarction [23, 24].

In summary, we can note that combination of all the unfavorable factors associated with atrial fibrillation is a very difficult condition, especially in case of persistent atrial fibrillation, as well as untimely, insufficient pain relief; poor blood pressure correction; inadequate therapy, followed by the resulting imbalance in blood coagulation system and the blood clot formed in the cardiac coronary arteries led to a heart attack episode in the patient. We failed to detect myocardial infarction due to the lack of clinical signs, diagnostic criteria, and corresponding assays.

REFERENCE

1. Wyndham C. Atrial Fibrillation: The Most Common Arrhythmia // Tex Heart Inst J. 2000; 27(3): 257–267.
2. Maesen B., Nijs J., Maessen J., Allessie M. et al. Post - operative atrial fibrillation: a maze of mechanisms // Europace. 2012; 14 (2): 159-174.
3. Zoni-Berisso M., Lercali F., Carazza L., Domenicucci S. Epidemiology of atrial fibrillation: European perspective. Clin Epidemiol. 2014;6:213–220.
4. Denga O., Pyndus T., Gargin V., Schneider S. Influence of metabolic syndrome on condition of microcirculatory bed of oral cavity // Georgian Med News. 2017;(273):99-104.
5. Kober L., Swedberg K., McMurray J.J. et al. Previously known and newly diagnosed atrial fibrillation: a major risk indicator after a myocardial infarction complicated by heart failure or left ventricular dysfunction // Eur J Heart Fail. 2006;8(6):591–8.
6. Gargin V.V., Alekseeva V.V., Lupyr A.V., Urevich N.O., Nazaryan R.S., Cheverda V.M. Correlation between the bone density of the maxillary sinus and body mass index in women during the menopause // Problemi Endokrinnoi Patologii 2019(2):20-26. doi: 10.21856/j-PEP.2019.2.03
7. Herlitz J., Malmberg K., Karlson B.W. etal. Mortality and morbidity during a five-year follow-up of diabetics with myocardial infarction // Acta Med Scand. 1988;224(1):31-38.
8. Benjamin E.J., Wolf P.A., D'Agostino R.B. et al. Impact of atrial fibrillation on the risk of death: the Framingham Heart Study // Circulation. 1998;98(10):946-952. doi:10.1161/01.cir.98.10.946
9. Alpert J.S., Thygesen K., Antman E., Bassand J.P. Myocardial infarction redefined – a consensus document of The Joint European Society of Cardiology/American College of Cardiology Committee for the redefinition of myocardial infarction [published correction appears in J Am Coll Cardiol 2001 Mar 1;37(3):973] // J Am Coll Cardiol. 2000;36(3):959-969.
10. Wattigney W.A., Mensah G.A., Croft J.B. Increased atrial fibrillation mortality: United States, 1980-1998 // Am J Epidemiol. 2002;155(9):819-826. doi:10.1093/aje/155.9.819
11. Gargin V., Radutny R., Titova G. et al. Application of the computer vision system for evaluation of pathomorphological image. 2020 IEEE 40th International Conference on Electronics and Nanotechnology, ELNANO 2020 - Proceedings; 2020. 469-473, doi:10.1109/ELNANO50318.2020.9088898.
12. Bondarenko A.V., Pokhil S.I., Lytvynenko M.V., Bocharova T.V., Gargin V.V. Anaplasmosis: experimental immunodeficient state model // Wiad Lek. 2019;72(9 cz 2):1761-1764.
13. Fesenko D., Glazunov O., Nakonechna O., Nazaryan R., Gargin V. Consequences of microsequences of microcirculatory distrurbances of oral mucosa in modeling of rheumatoid arthritis // Georgian Med News. 2019;(295):137-140.
14. Chiang C.E., Naditch-Brûlé L., Murin J. et al. Distribution and risk profile of paroxysmal, persistent, and permanent atrial fibrillation in routine clinical practice: insight from the real-life global survey evaluating patients with atrial fibrillation international registry // Circ Arrhythm Electrophysiol. 2012;5(4):632-639.
15. January C.T., Wann L.S., Alpert J.S., et al. 2014 AHA/ACC/HRS guideline for the management of patients with atrial fibrillation: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on practice guidelines and the Heart Rhythm Society // Circulation. 2014;130(23):2071-2104.
16. Shmendi A., Pirie F., Naidoo D.P., Tlou B., Pilloy W., Motala A.A. Myocardial perfusion imaging for evaluation of suspected ischemia and its relationship with glycemic control in South African subjects with diabetes mellitus // Diabetes Metab Syndr Obes. 2014;7:545-552.
17. Ostgren C.J., Merlo J., Råstam L., Lindblad U. Atrial fibrillation and its association with type 2 diabetes and hypertension in a Swedish community // Diabetes Obes Metab. 2004;6(5):367-374.
18. Haffner S.M., Lehto S., Rönnemaa T., Pyörälä K., Laakso M. Mortality from coronary heart disease in subjects with type 2 diabetes and in nondiabetic subjects with and without prior myocardial infarction // N Engl J Med. 1998;339(4):229-234.
19. Morrow D.A., Cannon C.P., Jesse R.L. et al. National Academy of Clinical Biochemistry Laboratory Medicine Practice Guidelines: clinical characteristics and utilization of biochemical markers in acute coronary syndromes // Clin Chem. 2007;53(4):552-574.
20. Shemyakin A., Kövamees O., Rafnsson A. et al. Arginase inhibition improves endothelial function in patients with coronary artery disease and type 2 diabetes mellitus // Circulation. 2012;126(25):2943-2950.
21. Kavsak P.A., MacRae A.R., Newman A.M. et al. Effects of contemporary troponin assay sensitivity on the utility of the early markers myoglobin and CKMB isoforms in evaluating patients with possible acute myocardial infarction // Clin Chim Acta. 2007;380(1-2):213-216.
22. Bondarenko A.V., Torianyk I.I., Pokhil S.I. et al. Seroprevalence of babesiosis in immunocompetent and immunocompromised individuals // Pol Merkur Lekarski. 2021;49(291):193-197.
23. European Heart Rhythm Association; European Association for Cardio-Thoracic Surgery, Camm AJ, et al. Guidelines for the management of atrial fibrillation: the Task Force for the Management of Atrial Fibrillation of the European Society of Cardiology (ESC) // Eur Heart J. 2010;31(19):2369-2429. doi:10.1093/eurheartj/ehq278
24. Kirchhof P., Auricchio A., Bax J. et al. Outcome parameters for trials in atrial fibrillation: recommendations from a consensus conference organized by the German Atrial Fibrillation Competence NETwork and the European Heart

Rhythm Association // Europace. 2007;9(11):1006-1023.
25. Apple F.S., Parvin C.A., Buechler K.F., Christenson R.H., Wu A.H., Jaffe A.S. Validation of the 99th percentile cutoff independent of assay imprecision (CV) for cardiac troponin monitoring for ruling out myocardial infarction // Clin Chem. 2005;51(11):2198-2200.

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СЛУЧАЙ СКРЫТИЯ ИНФАРКТА МИОКАРДА ФИБРИЛЛЯЦИЕЙ ПРЕДСЕРДИЙ НА ФОНЕ САХАРНОГО ДИАБЕТА И АРТЕРИАЛЬНОЙ ГИПЕРТЕНЗИИ

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В статье представлено сведение об остром субэндокардиальном инфаркте миокарда задней стенки левого желудочка, который развился на фоне сахарного диабета второго типа и артериальной гипертензии. Единственным клиническим симптомом острого поражения сердца была тахисистолическая форма перманентной фибрилляции предсердий. Несмотря на комплекс проведенных терапевтических манипуляций, пациентка умерла. Окончательный диагноз острого субэндокардиального инфаркта миокарда задней стенки левого желудочка был установлен только после вскрытия.

Представленный клинический случай демонстрирует нетипическое протекание инфаркта миокарда, который проявлялся скрытой фибрилляцией предсердий. Данный случай акцентирует внимание на необходимость комплексного обследования пациентов при фибрилляции предсердий и исключения в первую очередь необратимых поражений миокарда.

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