DOI: 10.20514/2226-6704-2025-15-4-303-309
УДК 616.831-005.1-06:616.127-005
EDN: NZTTQX

М.А. Тяпкина, А.А. Ордякова, Е.Ю. Пономарева, Е.В. Яковлева

Федеральное государственное бюджетное образовательное учреждение высшего образования Саратовский государственный медицинский университет имени В.И. Разумовского Министерства здравоохранения Российской Федерации, кафедра госпитальной терапии, Саратов, Россия

ЦЕРЕБРАЛЬНАЯ МАСКА СИНДРОМА ТАКОЦУБО

M.A. Tyapkina, A.A. Ordyakova, E.Yu. Ponomareva, E.V. Yakovleva

Ministry of Health and Social Development of the Russian Federation State Educational Institution of Higher Professional Education Saratov State Medical University named after V.I. Razumovsky, hospital therapy department, Saratov, Russia

Cerebral Mask of Takotsubo Syndrome

Резюме

Синдром такоцубо (СТ) — это остро развивающаяся и обычно обратимая дисфункция миокарда преимущественно левого желудочка, клинически и электрокардиографически напоминающая острый коронарный синдром. Среди причин развития данной патологии указывают тяжелые психоэмоциональные потрясения и физические состояния, в числе которых тяжелые заболевания головного мозга. Самыми частыми симптомами заболевания в острый период являются боль за грудиной и одышка, реже — сердцебиение, синкопальные состояния. Тяжелое течение СТ может осложняться кардиогенным шоком, отеком легких, острым нарушением мозгового кровообращения, остановкой сердца. Представленное клиническое наблюдение демонстрирует необычное течение СТ, при котором тяжесть состояния пациента определялась поражением центральной нервной системы.

Мужчина 68 лет госпитализирован в тяжелом состоянии в реанимационное отделение: после тяжелого психоэмоционального напряжения обнаружен дома без сознания. При осмотре констатированы сопор, правосторонний парез взора, опущение правого угла рта, тяжелый неврологический дефицит по шкале NIHSS. При компьютерной томографии головного мозга неотчетливо определялся ишемический очаг в бассейне левой средней мозговой артерии. На электрокардиограмме зафиксирован двухфазный и отрицательный зубец Т в AVL, V3-V6. При эхокардиографии выявлено снижение фракции выброса левого желудочка до 32 %, выраженные нарушения локальной сократимости, в том числе циркулярные. Отмечено повышение маркеров некроза миокарда. Выставлен диагноз сочетанного ишемического повреждения головного мозга и сердца, начато лечение. На следующий день значимая положительная динамика — пациент в сознании, неврологический дефицит отсутствует. При магнитно-резонансной томографии головного мозга данных за ишемический инсульт не получено. На электрокардиограмме в динамике отсутствуют отрицательные и двухфазные зубцы Т, по данным эхокардиографии — нормализация фракции выброса левого желудочка, отсутствие зон гипокинезии.

Связь начала заболевания с тяжелым эмоциональным напряжением, несоответствие зон нарушения локальной сократимости бассейну одной коронарной артерии с циркулярным поражением левого желудочка, отсутствие морфологического субстрата, объясняющего столь выраженный неврологический дефицит в остром периоде, быстрое полное восстановление кардиологической и неврологической дисфункции позволили сделать вывод о первичной форме СТ. Катехоламиновый выброс вызвал острую дисфункцию левого желудочка, которая осложнилась гипоперфузией мозга с прогрессирующим его отеком.

Таким образом, СТ в остром периоде может имитировать не только типичную, ангинальную форму инфаркта миокарда, но и церебральную.

Ключевые слова: *Синдром такоцубо, ишемический инсульт, инфаркт миокарда*

Конфликт интересов

Авторы заявляют, что данная работа, её тема, предмет и содержание не затрагивают конкурирующих интересов

Источники финансирования

Авторы заявляют об отсутствии финансирования при проведении исследования

Соответствие принципам этики

Пациент дал согласие на опубликование данных лабораторных и инструментальных исследований в статье «Церебральная маска синдрома такоцубо» для журнала «Архивъ внутренней медицины», подписав информированное согласие

Статья получена 03.03.2025 г.
Одобрена рецензентом 18.04.2025 г.
Принята к публикации 30.04.2025 г.

Для цитирования: Тяпкина М.А., Ордякова А.А., Пономарева Е.Ю. и др. ЦЕРЕБРАЛЬНАЯ МАСКА СИНДРОМА ТАКОЦУБО. Архивъ внутренней медицины. 2025; 15(4): 303-309. DOI: 10.20514/2226-6704-2025-15-4-303-309. EDN: NZTTQX

Abstract

Takotsubo Syndrome (TS) is an acutely developing and typically reversible myocardial dysfunction, predominantly affecting the left ventricle, which clinically and electrocardiographically resembles acute coronary syndrome. Among the etiological factors of this pathology, severe emotional stress and physical conditions, including severe brain diseases, are noted. The most common symptoms in the acute phase include chest pain and dyspnea, while palpitations and syncope occur less frequently. Severe cases of TS may be complicated by cardiogenic shock, pulmonary edema, acute cerebrovascular accident, cardiac arrest. The presented clinical case demonstrates an atypical course of TS, in which the severity of the patient's condition was determined by central nervous system involvement

A 68-year-old man was admitted in critical condition to the intensive care unit: after experiencing severe emotional stress, he was found unconscious at home. Upon examination, the patient was in a state of sopor with right-sided gaze paresis, right-sided mouth corner drooping, and severe neurological deficit, as assessed by NIHSS.

Computed tomography of the brain revealed a poorly defined ischemic lesion in the vascular territory of the left middle cerebral artery. Electrocardiography showed biphasic and negative T waves in AVL, V3-V6. Echocardiography revealed a reduction in left ventricular ejection fraction to 32 % and the regional walls motion abnormality including circular hypokinesis. Elevated levels of myocardial necrosis markers were observed. A diagnosis of combined ischemic brain and myocardial injury was established, and treatment was initiated. The following day, significant positive dynamics were observed—the patient regained consciousness, had no neurological deficit. Magnetic resonance imaging of the brain did not reveal evidence of stroke. There are no negative and biphasic T waves on the electrocardiogram, echocardiography demonstrated normalization of left ventricular ejection fraction and the absence of hypokinetic zones.

The correlation between disease onset and severe emotional stress, the discrepancy between the regional walls motion abnormality and the vascular territory of a single coronary artery with circular involvement of the left ventricle, the absence of a morphological substrate explaining the pronounced neurological deficit in the acute phase, and the quick, complete recovery of cardiac and neurological function led to the conclusion of a primary form of TS. A catecholamine surge induced acute left ventricular dysfunction, which was further complicated by cerebral hypoperfusion with progressive brain edema.

So, in the acute phase TS may mimic not only the typical anginal form of myocardial infarction but also a cerebral event.

Key words: *Takotsubo Syndrome, acute ischemic stroke, myocardial infarction*

Conflict of interests

The authors declare no conflict of interests

Sources of funding

The authors declare no funding for this study

Conformity with the principles of ethics

The patient consented to the publication of laboratory and instrumental research data in the article «Cerebral mask of Takotsubo Syndrome» for the journal «The Russian Archives of Internal Medicine» by signing an informed consent

Article received on 03.03.2025
Reviewer approved 18.04.2025
Accepted for publication on 30.04.2025

For citation: Tyapkina M.A., Ordyakova A.A., Ponomareva E.Yu. et al. Cerebral Mask of Takotsubo Syndrome. The Russian Archives of Internal Medicine. 2025; 15(4): 303-309. DOI: 10.20514/2226-6704-2025-15-4-303-309. EDN: NZTTQX

TC — Takotsubo cardiomyopathy, ACS — acute coronary syndrome, ACVA — acute cerebrovascular accident, ECG — electrocardiography, echoCG — echocardiography, MRI — magnetic resonance imaging

Takotsubo cardiomyopathy (TC) is an acute and usually reversible myocardial dysfunction, mostly in the left ventricle, which is associated with circular reduction in myocardial contractility, seen clinically and on ECG as acute coronary syndrome (ACS) [1]. In Japanese, takotsubo means an octopus trap, a ceramic pot with a round basis and narrow neck. This is the form of the left ventricle at the end of the systole due to transient ball-shaped widening of apical segments and midsegments.

N. Sato et al. were the first to describe TC in 1990. Publications emphasised the correlation between this syndrome and negative psychoemotional factors. For this reason, TC is called the broken heart syndrome, stress-induced cardiomyopathy. However, results of later studies demonstrate that physical factors are more often the cause of TC than emotional [1]. Physical triggers include GI bleeding, surgery, severe brain conditions (acute cerebrovascular accidents, subarachnoid haemorrhage, cerebrocranial accidents), thyrotoxicosis, pheochromocytoma, severe pain (pneumothorax, renal or biliary colic), abstinence symptom (alcohol, opiates), β2-adrenoceptor agonist overdose, etc. Since the physical factors, which can trigger acute ballooning of myocardium sections, are numerous, it was suggested to identify all variants of TC, caused by physical factors, as secondary TC, whereas the classical disease progression under the influence of psychoemotional stress is called primary TC [2]. It is assumed that the main pathogenetic causes are direct cardiotoxic action of catecholamines, myocardial stupor (hybernation), coronary artery spasm, oxidative stress, vegetative imbalance, inflammatory and metabolic conditions of the myocardium [1, 2].

The accurate incidence of TC is currently unknown. According to the 2018 global consensus, the incidence of the syndrome is 1–3% of all patients hospitalised with suspected acute myocardial infarction with elevated ST,

and in women the rate of TC can reach 5–6%. Patients are mostly postmenopausal women; however, recently the disease is reported in men of 50–75 years of age [3].

Clinical presentation of acute TC is often similar to ACS, with or without elevated ST. The most common symptoms of this disease during the acute phase are retrosternal pain (up to 75% of cases) and shortness of breath (up to 52% of cases). Palpitations (up to 12%) and syncope (up to 9%) are less common. The main clinical manifestations of TC caused by physical triggers are the symptoms of the underlying physical illness. Severe TC can be complicated by cardiovascular shock, pulmonary oedema, acute cerebrovascular accident (ACVA), cardiac arrest [3].

The presented clinical case demonstrates atypical TC progression, where the main clinical manifestations and patient's condition severity depended on the rate of central nervous system involvement.

Male, 68 years old, was admitted to the A&E of Saratov Regional Clinical Hospital at 3.00 pm on April 15, 2024. Upon admission, he did not have any complaints due to his severe condition. Past medical history was presented by his relatives. The patient was the head of a farm business, where a large fire occurred recently, the roof collapsed and people died. He lived alone, called his relatives at around 8.00 am, and did not complain of anything. At 11.00 am he was found at home unconscious. The past history shows that he did not have any bad habits and known cardiovascular diseases; he had no family history of cardiovascular pathologies. The patient underwent regular medical examinations and led an active life.

Upon admission, his condition was serious. His consciousness was close to semi-consciousness; Glasgo Coma Score: 10 points. When painful stimulus and loud call were used, the patient opened his eyes for a short time; he did not follow instructions. The skin was of normal colour. Overweight (body mass index: 27.7 kg/m^2). Cardiac sounds were muffled, rhythmic. The pulse was of equal volume and exertion, 96 beats per minute. Blood pressure: 130/80 mm Hg. Vesicular respiration, without rale, 19/min. Right gaze palsy. The face is asymmetrical: the right angle of mouth is lower than the normal level. Sensorimotor aphasia. 24 points on the NIHSS, which corresponded to severe neurologic impairment.

Brain computer tomography: projection of the left parietal region with an unclear area of lower density, 13×11×12 mm, 26 HU, which was interpreted as a sign of possible ischaemic stroke in the left medial cerebral artery system (Figure 1). The quantification scale of early ischaemic changes in brain substance based on ASPECTS CT results: 9 points, i.e. a small ischaemic site. Duplex scanning of brachiocephalic arteries showed hemodynamically insignificant stenosis (10–15%) in the bifurcation of the left common carotid artery.

Electrocardiography (ECG) showed sinus tachycardia with heart rate of 100 beats per minute, signs of blocked anterior branch of the left bundle of atrioventricular bundle, two-phase and negative T wave in the septal-apical-side area of the left ventricle (Figure 2). Echocardiography (eco-CG) showed a drop in ejection fraction to 32% (Simpson). Insignificantly widened left atrium cavity (index left atrial volume: 36 mL/m^2), grade2 mitral, tricuspid regurgitation; marked hypokinesia of lower, lower lateral segments at the apical, middle, basal levels; lower septal, anterior septal segments at the middle and basal levels of the left ventricle; grade 1 pulmonary hypertension (systolic pressure in the pulmonary artery: 43 mm Hg).

Laboratory test results showed leucocytosis, hyperglycaemia, dyslipidemia, minor hyperazotemia, high myocardial necrosis markers, high natriuretic peptides. The most important laboratory parameters upon hospital admission and their changes are presented in Table 1.

A preliminary diagnosis was determined. Primary diseases: 1. Brain infarction (unknown origin) in the left medial cerebral artery system, dated April 15, 2024. Sensorimotor aphasia. Central right facial palsy. Right gaze palsy. 2. Ischaemic heart disease. Acute non-Q septal-apical-lateral myocardial infarction without elevated ST, dated April 15, 2024. Atherosclerosis of aorta and carotid arteries without hemodynamically significant stenosis. Relative mitral valve insufficiency, grade 2; tricuspid valve insufficient, grade 2. Grade 1 pulmonary hypertension. Overweight (body mass index: 27.7 kg/m^2). Dyslipidemia. Complications**:** Brain swelling. IIA circulation failure with reduced ejection fraction (32%).

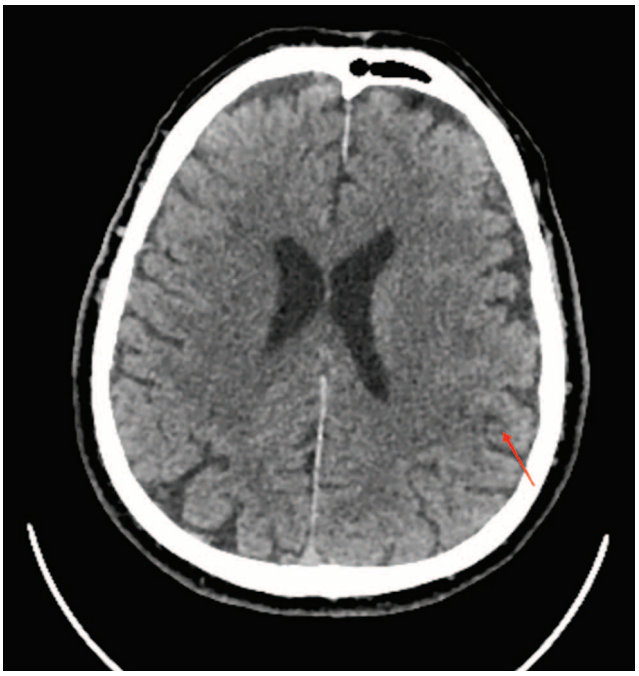

Figure 1. *Computer tomography of the brain, first day of illness*

Note. A zone of low-density measuring 13×11×12 mm with density characteristics of 26 HU is extremely indistinctly defined on the left parietal lobe

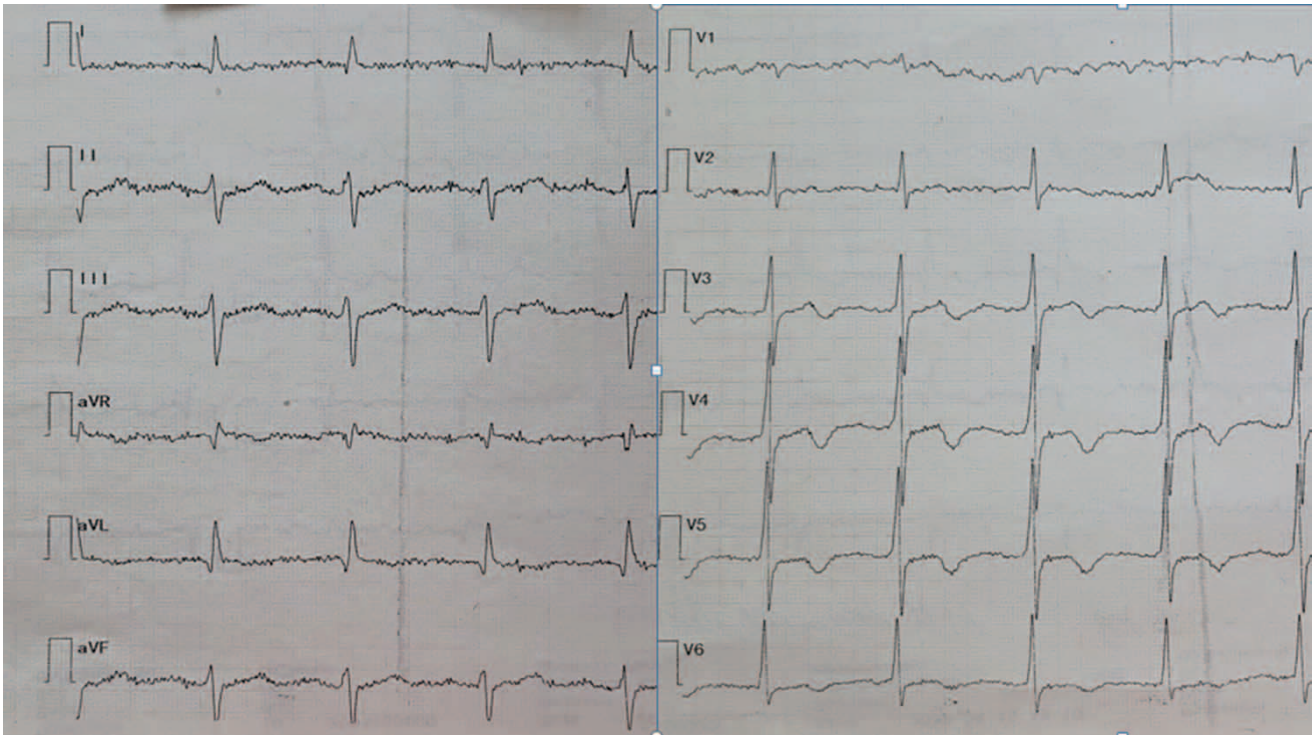

Figure 2. *ECG of the patient upon admission, first day of illness*
Note. Sinus tachycardia. Incomplete right bundle brunch block, biphasic and negative T wave in AVL, V3-V6

Table 1. *Laboratory tests of the patient upon admission to hospital and during dynamic observation*

Test, units of measurement	Upon admission to hospital	On the 6th-7th day of hospitalization	Reference values
Leukocytes, 10^9/l	11,2	4,6	4-9
Glucose, mmol/l	10,5	5,1	<6,1
Total cholesterol, mmol/l	5,6	4,3	-
LDLs, mmol/l	3,9	2,4	-
Creatinine, mmol/l	130	103	56-115
GFR (CKD-EPI) / ml/min/1,73 м^2,	48	64	>60
Troponin-I hs, ng/ml	1,7	0,4	<0,5
CPK tot, Ed/l	517	251	<200
CPK-MB, Ed/l	69,5	24	<25
NT-proBNP, пг/мл / pg/ml	824	561	<125

Note. LDLs — low-density lipoproteins, GFR (CKD-EPI) — glomerular filtration rate calculated by the CKD-EPI, Troponin-I hs — Troponin-I, high sensitivity, CPK tot — Creatine Phosphokinase total, CPK-MB — Creatine Phosphokinase MB, NT-proBNP — N-terminal prohormone of brain natriuretic peptide
Reference values for total cholesterol and LDLs are not given because target values for these parameters are calculated individually.

According to the studies, acute myocardial infarction increases the risk of ACVA and vice versa. In 2010, Omar et al. suggested a concept of acute cardiocerebral infarction. Despite its low incidence (1–6% of cases), this combination worsens prognosis for patients and limits the therapy of each individual condition [4]. Clinically, there are three forms of this pathology, depending on the timing of the damage [5]. Type one: simultaneous myocardial and cerebral infarction. This is possible in embolism from the left heart in atrial fibrillation; in crossed embolism from the central circulatory system because of the open foramen ovale; and in aortic dissection, if the affected area includes both coronary and carotid arteries. Type two: primary heart involvement. In acute myocardial infarction, strokes are usually cardioembolic due to the development of such complications as atrial fibrillation and ventricular cavity thrombosis. Also, acute reduction in myocardial contractility in some myocardial infarction patients causes brain hypoperfusion, progressive brain swelling, and neurological symptoms. Type three: primary brain involvement. Cardiac changes during acute craniocerebral accident are associated with vegetative nervous system dysfunction and increased catecholamine

production, which can cause TC and decompensation of existing ischaemic heart disease. Therefore, the examination and follow-up in this patient were performed to clarify the origin of the ischaemic myocardial and cerebral damage.

The patient was treated with cerebral protectants (ethylmethylhydroxypyridine succinate, Cerebrolysin), antiedemics (magnesium sulphate), double disaggregation (acetylsalicylic acid, clopidogrel), lipid-lowering (atorvastatin), cardiotropic drugs (metoprolol succinate, enalapril), and gastroprotectors (esomeprazole). Anticoagulants were prescribed for preventive purposes because of early severe stroke (enoxaparin). Given that the patient was in semiconscious state, he was put on artificial lung ventilation in order to ensure clear airways and prevent secondary hypoxic damage to the brain. No thrombolytic therapy for the stroke was performed because the patient was admitted to the hospital more than 4.5 hours after disease onset. It was decided not to perform coronary angiography and possible percutaenous coronarography intervention due to suspected large brain damage and serious condition of the patient.

The therapy resulted in significant improvement of the patient's condition and neurological status. The next day after admission, the patient is conscious, lucid, knows who he is, knows the place and time, is reasonable, answers questions asked, fully follows instructions, does not have any complaints. NIHSS score is 0 points, no neurological deficit. In order to assess the nature and extent of the brain damage, brain magnetic resonance imaging (MRI) was performed 24 hours after the previous imaging (T1, T2W and diffusion). There were no areas of high magnetic resonance signal in the brain and cerebellum matter, i.e. there were no signs of ACVA (Figure 3).

ECG results (April 17, 2024): no tachycardia; no signs of impaired repolarization in the form of negative and two-phase T waves (Figure 4).

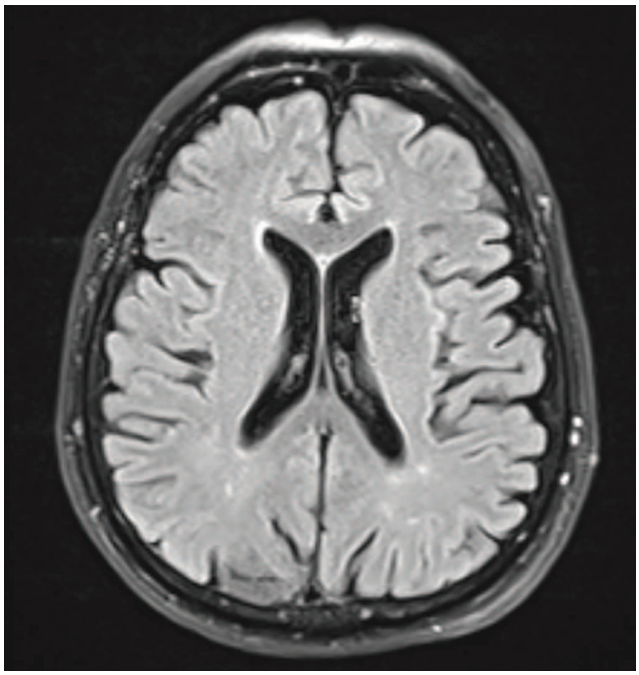

Figure 3. *Magnetic resonance imaging of the brain, second day of illness. Stroke not detected.*

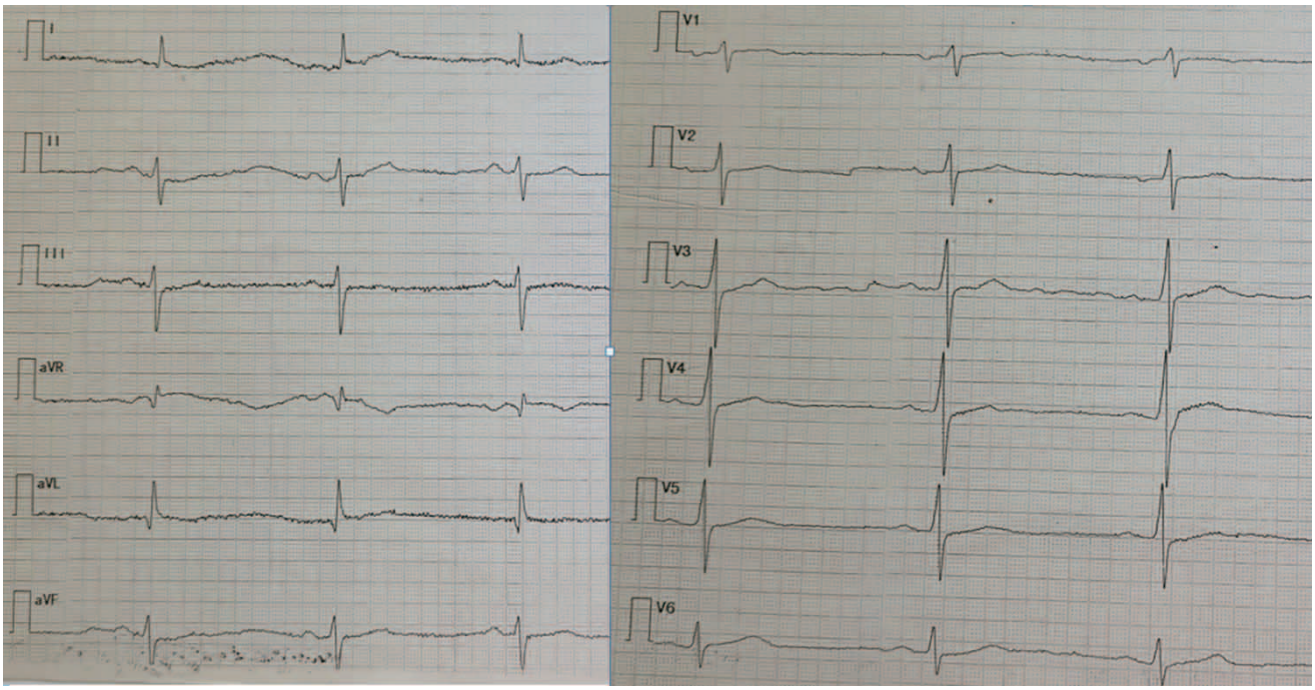

Figure 4. *ECG of the patient, third days of illness. Tachycardia has been stopped, there are no repolarization disorders*

ECG results (April 17, 2024) show significant favourable changes: ejection fraction normalised (61%, Simpson) and no signs of impaired local contractility. Laboratory test results showed normal WBC count, normal levels of creatinine, serum glucose, and markers of myocardial necrosis (Table 1). Glicated haemoglobin value was 5.2%, and it was concluded that hyperglycaemia observed upon admission was a stress reaction and not a sign of long-lasting carbohydrate metabolism impairment. Holter ECG monitoring results (April 17, 2024): sinus rhythm with an average rate of 60 beats per minute, individual auricular extrasystoles; no QT elongation, ST excursions, irregular repolarization processes.

Complete correction of neurological deficit within 24 hours without any MRI changes allowed to rule out stroke and diagnose transient ischaemic attack. Fast normalisation of ECG manifestations and echoCG parameters, absence of the typical clinical signs caused some doubts about the diagnosis of acute ischaemic myocardial damage. Given the correlation of disease onset with emotional stress, mismatch between areas of impaired local contractility and one coronary artery with circular damage to the left ventricle, significantly higher levels of N-terminal pro brain natriuretic peptide with insignificant increase in troponin concentrations, higher laboratory parameters, which indirectly point out to hypercatecholaminemia, TC was suggested.

The international diagnostic criteria for TC (InterTAK Diagnostic Criteria) were proposed in 2018 [3]. The patient had the following criteria of this pathology: transient signs of left ventricle dysfunction, regional abnormality of left ventricle wall movement beyond the blood flow in one coronary artery, prior emotional trigger, changes seen on ECG (including inverted T wave), moderately elevated cardiospecific markers, higher levels of natriuretic peptide. There was no solid clinical evidence of infectious myocarditis; heart MRI was not performed due to technical reasons. Once his condition stabilised, the patient was offered coronary angiography, but he refused. Of note, according to this document [3], even obstructive lesion of the coronary system did not rule out TC. The international diagnostic criteria emphasise that acute neurological disorders can trigger Takotsubo cardiomyopathy.

This clinical situation brought up a question: What occurred first? Did acute neurological accident trigger Takotsubo cardiomyopathy, or vice versa? Clear correlation of disease onset with psychoemotional stress, absence of morphologic substrate (extensive ischaemic stoke, head injury), which could explain marked neurological deficit during the acute phase, fast and complete restoration of the cardiological and neurological function allowed concluding the presence of the primary form of Takotsubo cardiomyopathy. Acute left ventricle dysfunction in this patient was likely to be complicated by cerebral hypoperfusion and progressive brain swelling, which caused these marked neurological symptoms. The same pathogenetic mechanism underlies cerebral clinical form of acute myocardial infarction. The main difference is that in ischaemic necrosis of the myocardium, the damage is persistent, while in this patient the left ventricle function recovered quickly, and neurological deficit completely disappeared.

The patient's therapy was adjusted: disaggregants, blood thinners, cerebral and GI protectants were cancelled. The recommended therapy in outpatient settings: lipid-lowering drugs (rosuvastatin 20 mg + ezetemibe 10 mg), low doses of angiotensin-converting enzyme inhibitors (perindopril 2.5 mg daily). Beta-blockers were not prescribed since the patient was susceptible to bradycardia. Two and six months after discharge from the hospital, the patient's state of health was satisfactory; he had good exercise tolerance; no complaints, kept active lifestyle without bad habits, and followed recommendations. With the therapy, blood pressure is normotensive (110–120/70–79 mm Hg); low-density lipoproteins two months later: 1.36 mmol/L, which is a target value for patients with a very high cardiovascular risk.

Therefore, during the acute phase, Takotsubo cardiomyopathy can look similar not only to the typical, anginal form of myocardial infarction, but also to the cerebral form. Fast recovery of the left ventricle function in this patient resulted in complete disappearance of neurological deficit and favourable outcome, despite severe clinical manifestations during the acute phase. Physicians should be made aware of this pathology; it will allow avoiding overdiagnosing acute ischaemic heart disease and excessive drug load.

Вклад авторов:

Все авторы внесли существенный вклад в подготовку работы, прочли и одобрили финальную версию статьи перед публикацией

Тяпкина М.А.: сбор, анализ, интерпретация данных, разработка общей концепции и дизайна статьи, написание рукописи, проверка критически важного интеллектуального содержания, принятие окончательного решения о готовности рукописи к публикации, согласие быть ответственным за все аспекты работы.

Ордякова А.А.: сбор, анализ, интерпретация данных, разработка общей концепции и дизайна статьи, написание рукописи, проверка критически важного интеллектуального содержания.

Пономарева Е.Ю.: разработка общей концепции и дизайна статьи, написание рукописи, проверка критически важного интеллектуального содержания.

Яковлева Е.В.: разработка общей концепции и дизайна статьи, написание рукописи, проверка критически важного интеллектуального содержания.

Author Contribution:

All the authors contributed significantly to the study and the article, read and approved the final version of the article before publication

Tyapkina M.A.: examination, evaluation, diagnosis, data's analysis, development of general concept and article design, writing of the manuscript, verification of critical important intellectual content, making a final

determination regarding the publication readiness of the manuscript, responsibility for all work aspects.

Ordyakova A.A.: examination, evaluation, diagnosis, data's analysis, development of general concept and article design, writing of the manuscript, verification of critical important intellectual content.

Ponomareva E.Yu.: development of general concept and article design, writing of the manuscript, verification of critical important intellectual content

Yakovleva E.V.: development of general concept and article design, writing of the manuscript, verification of critical important intellectual content

Список литературы/References:

1. Болдуева С.А., Евдокимов Д.С. Кардиомиопатия такоцубо. Обзор данных литературы: понятие, эпидемиология, патогенез. Часть I. Российский кардиологический журнал 2022;27(3S):4993. doi: 10.15829/1560- 4071-2022-4993. EDN BQQYSD.
   Boldueva S.A., Evdokimov D.S. Takotsubo cardiomyopathy. Literature review: concept, epidemiology, pathogenesis. Part I. Russian Journal of Cardiology 2022;27(3S):4993. doi: 10.15829/1560-4071-2022-4993. EDN BQQYSD [in Russian].
2. Шилова А.С., Шмоткина А.О., Яфарова А.А. и др. Синдром такоцубо: современные представления о патогенезе, распространенности и прогнозе. Рациональная Фармакотерапия в Кардиологии 2018; 14(4):598-604. doi: 10.20996/1819-6446-2018-14-4-598-604.
   Shilova A.S., Shmotkina A.O., Yafarova A.A. at al. Takotsubo Syndrome: Contemporary Views on the Pathogenesis, Prevalence and Prognosis. Rational Pharmacotherapy in Cardiology 2018; 14(4):598-604. doi: 10.20996/1819-6446-2018-14-4-598-604 [in Russian].
3. Ghadri JR, Wittstein IS, Prasad A., at al. International Expert Consensus Document on Takotsubo Syndrome (Part I): Clinical Characteristics, Diagnostic Criteria, and Pathophysiology. Eur Heart J. 2018 Jun 7; 39(22):2032-2046. doi: 10.1093/eurheartj/ehy076.
4. Ibekwe E, Kamdar HA, Strohm T. Cardio-cerebral infarction in left MCA strokes: a case series and literature review. Neurol Sci. 2022 Apr;43(4):2413-2422. doi: 10.1007/s10072-021-05628-x. Epub 2021 Sep 29.
5. Yeo LL, Andersson T, Yee KW, at al. Synchronous cardiocerebral infarction in the era of endovascular therapy: which to treat first? Thromb Thrombolysis. 2017 Jul;44(1):104-111. doi: 10.1007/s11239-017-1484-2.

Информация об авторах:

Тяпкина Мария Александровна — кандидат медицинских наук, доцент кафедры госпитальной терапии ФГБОУ ВО «Саратовский государственный медицинский университет имени В.И. Разумовского» Минздрава России, Саратов, e-mail: mariya-tyapkina@yandex.ru, ORCID ID: https://orcid.ord/0000-0002-1860-3171

Ордякова Анна Александровна — ассистент кафедры госпитальной терапии ФГБОУ ВО «Саратовский государственный медицинский университет имени В.И. Разумовского» Минздрава России, Саратов, e-mail: anna-ordyakova@mail.ru, ORCID ID: ttps://orcid.ord/0000-0002-6088-6259

Пономарева Елена Юрьевна — кандидат медицинских наук, доцент кафедры госпитальной терапии ФГБОУ ВО «Саратовский государственный медицинский университет имени В.И. Разумовского» Минздрава России, Саратов, e-mail: ponomareva_elena1@mail.ru, ORCID ID: https://orcid.ord/0000-0001-6187-7525

Яковлева Елена Викторовна — кандидат медицинских наук, доцент кафедры госпитальной терапии ФГБОУ ВО «Саратовский государственный медицинский университет имени В.И. Разумовского» Минздрава России, Саратов, e-mail: elenaviktorova@yandex.ru, ORCID ID: https://orcid.ord/0000-0001-6700-8531

Information about the authors

Maria A. Tyapkina — MD, PhD, Associate Professor, Department of Hospital Therapy, Federal State Budgetary Educational Institution of Higher Education "Saratov State Medical University named after V.I. Razumovsky" of the Ministry of Healthcare of the Russian Federation, Saratov, e-mail: mariya-tyapkina@yandex.ru, ORCID ID: https://orcid.ord/0000-0002-1860-3171

Anna A. Ordyakova — Assistant, Department of Hospital Therapy, Federal State Budgetary Educational Institution of Higher Education "Saratov State Medical University named after V.I. Razumovsky" of the Ministry of Healthcare of the Russian Federation, Saratov, e-mail: anna-ordyakova@mail.ru, ORCID ID: ttps://orcid.ord/0000-0002-6088-6259

Elena Yu. Ponomareva — MD, PhD, Associate Professor, Department of Hospital Therapy, Federal State Budgetary Educational Institution of Higher Education "Saratov State Medical University named after V.I. Razumovsky" of the Ministry of Health of the Russian Federation, Saratov, e-mail: ponomareva_elena1@mail.ru, ORCID ID: https://orcid.ord/0000-0001-6187-7525

Elena V. Yakovleva — MD, PhD, Associate Professor of the Department of Hospital Therapy, Federal State Budgetary Educational Institution of Higher Education "Saratov State Medical University named after V.I. Razumovsky" of the Ministry of Health of the Russian Federation, Saratov, e-mail: elenaviktorova@yandex.ru, ORCID ID: https://orcid.ord/0000-0001-6700-8531

Автор, ответственный за переписку / Corresponding author