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Takotsubo syndrome after mitral valve replacement and defibrillation

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ABSTRACT

The article describes the case of Takotsubo syndrome, which arose in a 71-year-old female patient after a mitral heart valve replacement , performed due to its severe (3rd degree) insufficiency of non-rheumatic genesis. This pathology is quite rare. Questions of its etiology and pathogenesis remain controversial. A special feature of this case is the development of reversible left ventricular dysfunction in the early postoperative period after mitral valve replacement. The reduction of the left ventricular ejection fraction to 25% with hyperkinesia of its basal parts with subsequent recovery of the ejection fraction to 56% 3 weeks after the onset of the disease was observed. There were also electrocardiographic changes simulating acute circular myocardial infarction with the absence of hemodynamically significant lesions of the coronary arteries. The patient had risk factors for this syndrome (age, female gender, stress situation, surgical intervention, administration of dobutamine and fluoroquinolones). For the reasons given above, the presence of Takotsubo syndrome was suspected. This case demonstrates the possibility of developing takotsubo syndrome after cardiosurgical interventions and defibrillation.

Key words: takotsubo cardiomyopathy; valvular heart disease; ventricular fibrillation; mitral valve replacement; cardiac surgery; defibrillation.

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Синдром такоцубо после протезирования митрального клапана (клинический случай)

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РЕЗЮМЕ

В статье представлен случай синдрома такоцубо, резвившегося у 71-летней пациентки после протезирования митрального клапана сердца, выполненного в связи с его выраженной (3-я степень) недостаточностью неревматического генеза. Данная нозология является редко встречающейся. В литературе имеются единичные разрозненные сообщения о синдроме такоцубо после кардиохирургических вмешательств и электроимпульсной терапии. Вопросы этиологии и патогенеза остаются дискутабельными. Особенностью данного случая является развитие обратимой дисфункции левого желудочка в раннем послеоперационном периоде после протезирования митрального клапана – снижение фракции выброса левого желудочка до 25% с гиперкинезией его базальных отделов с ее последующим восстановлением до 56% через 3 нед после дебюта заболевания. Также имели место электрокардиографические изменения, имитирующие острый циркулярный инфаркт миокарда, при отсутствии гемодинамически значимых поражений коронарных артерий. У пациентки были выявлены факторы риска развития данного синдрома (возраст, женский пол, стрессовая ситуация, хирургическое вмешательство, дефибрилляция, применение добутамина и фторхинолонов). На основании перечисленного было заподозрено наличие синдрома такоцубо. Данный случай демонстрирует возможность развития синдрома такоцубо после кардиохирургических вмешательств и дефибрилляции.

Ключевые слова: кардиомиопатия такоцубо, приобретенные пороки сердца, фибрилляция желудочков, протезирование клапана сердца, кардиохирургия, дефибрилляция.

Конфликт интересов. Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с публикацией настоящей статьи.

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INTRODUCTION

Takotsubo syndrome (TTS), also known as Takotsubo cardiomyopathy, stress cardiomyopathy or broken heart syndrome, is a benign, reversible abnormality, characterized with a fugitive systolic ventricular dysfunction. Its clinical evidence is myocardial infarction with the absence of coronary arteries stenosis [1, 2]. The etiology of ST has not been fully studied, but scientists acknowledge its connection with excess catecholamines from physical or emotional stress. There exist several theories concerning TTS etiology: coronary artery spasm, left ventricular outflow tract obstruction,

increase in catecholamine levels, disorder in calcium regulatory system, oxidative stress and increase in production of transforming growth factor beta [3].

Despite numerous descriptions of TTS in scientific literature, there exist only isolated cases of this disorder in patients after cardiosurgical interventions. Such cases are only represented in foreign sources [4–8].

We present a case of TTS diagnosed in a patient after mitral valve replacement and defibrillation.

CASE REPORT

Female patient aged 71 was admitted to the cardiac surgery department for elective surgery. Patient had a diagnosis of non-rheumatic multiple valve disease: mitral valve prolapse, mitral valve insufficiency, aortic insufficiency, tricuspid insufficiency, permanent atrial fibrillation with normal sinus rhythm, heart failure with preserved ejection fraction, IIA, class IV.

Echocardiography was performed during the preoperative stage with the following results: left atrium – 6.2 cm; left ventricle end-diastolic size – 4.9 cm; left ventricle end-systolic size – 2.9 cm; left ventricle posterior wall – 1.1 cm; ventricular septum – 1,1 cm; ejection fraction (EJ) – 71%; right ventricle – 2.3 cm. The test also showed mitral valve's leaflets thickened into the left atrium (anterior mitral leaflet – >1.0 cm, posterior mitral leaflet – >0.9 cm) with significant mitral

valve regurgitation (peak mitral flow velocity – 470 cm per second, vena contracta diameter – 0.8 cm). An angiography of coronary arteries did not reveal any significant lesions. Preoperative electrocardiogram (ECG) shows atrial fibrillation with high systolic blood pressure (Fig. 1)

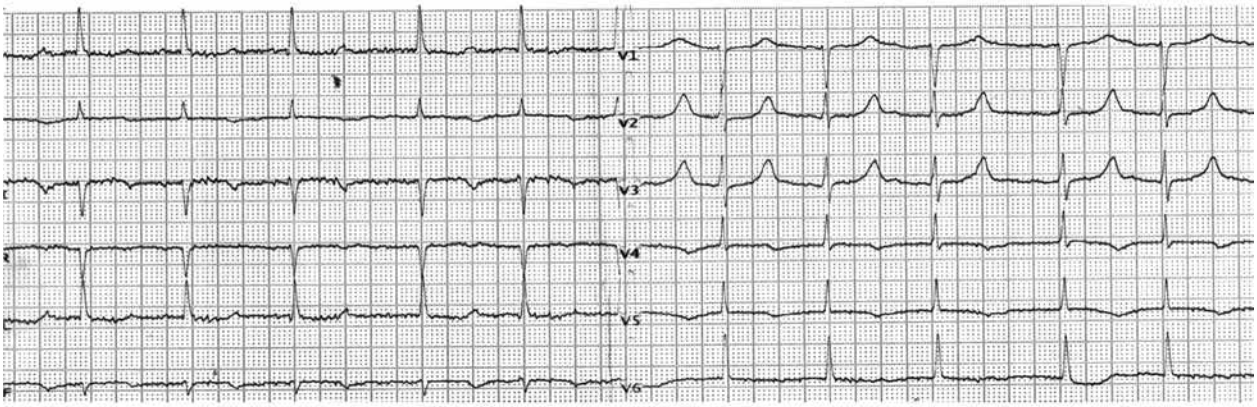

Fig. 1. Patient's preoperative ECG: scanning speed 25 mm/sec; voltage 1 мВ = 10 mm; heart rate 60–75 bpm; QTc 520 ms

The mitral valve replacement through median sternotomy with "Medtronic" HancockIIT510 trileaflet bioprosthesis, CinchSZ – 31 mm; reduction annuloplasty of tricuspid valve and left atrial appendage closure were performed in conditions of normothermic cardiopulmonary bypass and pharmaco-cold cardioplegia. Atrial fibrillation resumed by the end of the operation with a heart rate of 50–60 bpm.

Early postoperative period proceeded with predisposition to atrial fibrillation with heart rate <60 bpm (Fig. 2)

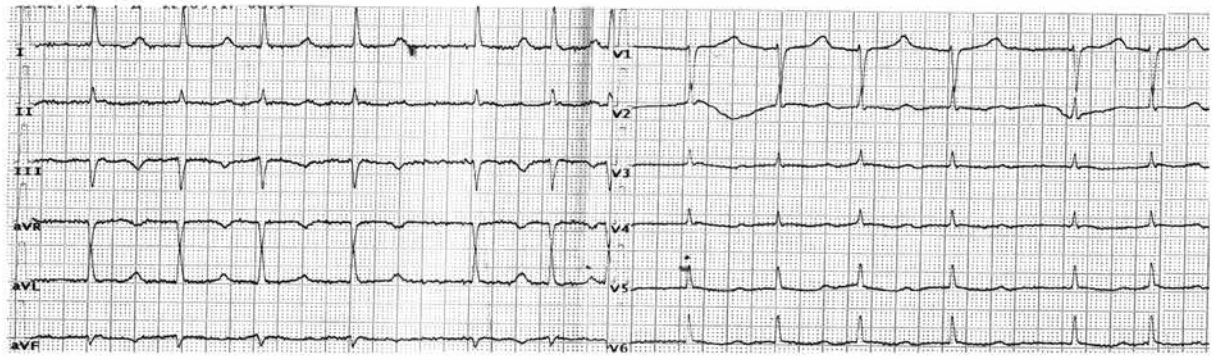

Fig. 2. Patient's ECG, 1st postoperative day: scanning speed 25 mm/sec; voltage 1 мВ = 10 mm; heart rate 58–100 bpm; QTc 557 ms (here and in pictures 2–4)

In the peri-operative period, the patient was receiving ciprofloxacin as a preventive measure for the prevention of infectious complications. Ventricular fibrillation developed in the patient on the third day after operation in the setting of relative well-being (normal general, electrolyte and biochemical blood values and central hemodynamics parameters). Biphasic defibrillation with 150 J was performed immediately. Atrial fibrillation was restored with the heart rate 55–65 bpm, arterial pressure 130/170 mmHg, central venous pressure 40 mm. H_2O. Electrocardiography revealed QT interval prolongation up to 754 ms and T-wave inversion in leads I, II, III, AVF, V_{3-6} (Fig. 3).

Pulmonary edema developed 6 hours after defibrillation. Echo-cardiography showed aneurysmal dilatation of left ventricular apex with ejection fraction reduction to 25% and hyperkinesis in the basal area of the left ventricle.

After patient's transfer to cardiac surgery department on the 9th day after ventricle fibrillation, the ECG was following (Fig. 4).

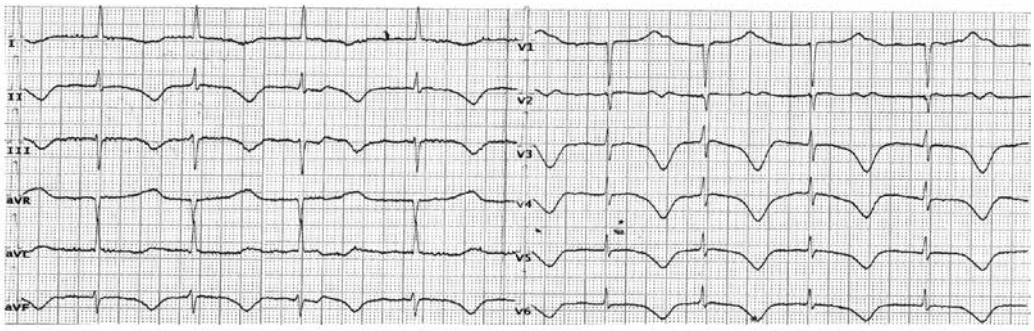

Fig. 3. Patient's ECG, 1st day after defibrillation

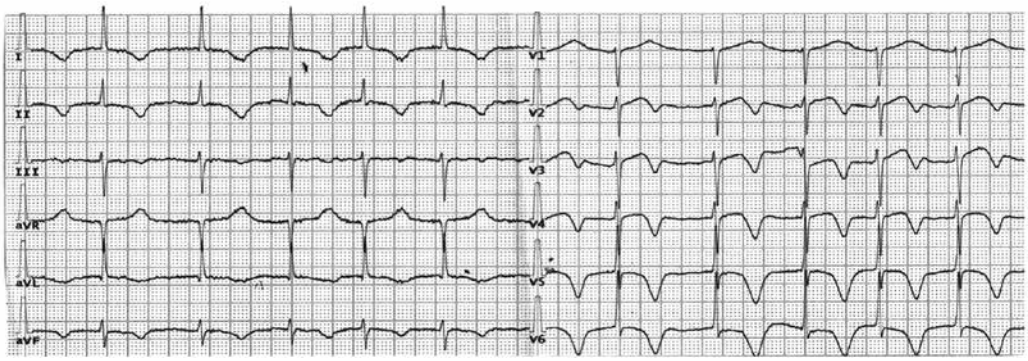

Fig. 4. Patient's ECG, 9th day after defibrillation

With the course of time, the patient stabilized and was discharged from the department in satisfactory condition on the 21st day. Discharge echo-cardiography: left atrium – 5.0 cm; left ventricle end-diastolic size – 5.3 cm; left ventricle end-systolic size – 3.8 cm; left ventricle posterior wall – 1.4–1.6 cm; ventricular septum – 1.5–1.8 cm; ejection fraction (EJ) – 56%; right ventricle – 3.2 cm; prosthetic mitral valve with pressure differential 15 mmHg; significant left ventricular myocardial hypertrophy; fluid traces in the upper part of pericardial cavity; aneurysm is not visible.

DISCUSSION

Clinical manifestations of Takotsubo syndrome are long retrosternal pains, dyspnea, sudden development of acute heart failure, severe ventricular arrhythmia which may lead to heart rapture [9].

Abnormal changes in ECG in acute phase (first 12 hours) include ST segment elevation or depression from the isoelectric period, newly developed left bundle-branch block and sometimes Q-wave formation. T-wave inversion and prolongation of QT interval is also possible in many derivations within 24–48 hours after the development of disease's clinical manifestation or triggering stress factor. QT interval prolongation is often significant (more than 500 ms) and, thus, predisposes to the development of torsades de pointes (TdP) and ventricle fibrillation [1].

Echocardiography reveals an area of regional myocardial contractility lesion that is larger than arterial blood supply area and various complications (left ventricular outflow tract obstruction, mitral valve regurgitation, thrombus formation and heart rapture) [1]. Angiography of coronary arteries shows the absence of hemodynamically relevant stenosis that could have been a reason for the myocardial contractility lesions mentioned above [1].

It may be noted that in the presented clinical case many TTS risk factors are present: age, female sex, stress situation, surgical intervention, dobutamine and ciprofloxacin injections. Since the influence of transforming growth factor beta on the TTS genesis and mitral valve prolapse development is proved, it can be assumed to influence the abnormality genesis in the presented case [10].

Diagnostic criteria, suggested by the specialists of Mayo clinic and further developed by Heart Failure Association (2016), exist nowadays [9]. Five of these criteria can be found in our patient: temporary regional myocardial contrac-

tility lesion after surgical intervention; regional myocardial contractility lesion takes place in the parts where blood is supplied by more than one coronary artery; absence of hemodynamically significant lesions of the coronary arteries; abnormal changes in the electrocardiogram (T-wave inversion and prolongation of QT interval up to 754 ms with its gradual shortening during the acute phase of the disease; restoration of the ejection fraction to 565.

Based on the mentioned above, the diagnosis of TTS was suggested in the patient.

CONCLUSION

The clinical case presented in this article demonstrates relevancy and significance of Takotsubo syndrome as a potential complication after surgical interventions on mitral valve or electrical cardioversion.

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