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Case report

Takatsubo syndrome as a differential diagnostic dillema versus acute coronary syndrome - case report

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Abstract

Introduction. Takatsubo syndrome is not an infrequent clinical entity in which there is transient left ventricular systolic dysfunction, often induced by emotional stress, organic disease or their combination. Clinically, it is often hard to distinguish it from acute coronary syndrome and it poses a differential-diagnostic dilemma in everyday practice. The aim of our case presentation was to report a patient with Takotsubo syndrome, initially treated as an acute coronary syndrome, with a review and comment on the available literature.

Case report. A 66-year-old female patient, with a past medical history of treated arterial hypertension, presented to the health service because of chest tightness that she had been experiencing for the past 48 hours. She cites the intense emotional stress that preceded the beginning of her complaints - the loss of her dog and the fear that it could hurt someone. Because of the appreciated ST segment elevation in the precordial leads, she was referred for emergency coronary angiography to the PCI center. There were no angiographically significant lesions in the epicardial coronary arteries. Echocardiography was performed, which showed a severely impaired global left ventricular systolic function with characteristic pattern of akinesia of the apical and medial and hyperkinesia of the basal segments of all the walls of the left ventricle, which, along with the anamnestic and demographic data, supported the diagnosis of the apical subtype of Takotsubo syndrome. Natriuretic peptides and especially troponin were elevated. Heart failure therapy was started with the initial hemodynamic stability of the patient. The next day, pulmonary edema refractory to treatment developed and resulted in death.

Conclusion. Suspicion of Takotsubo syndrome in patients with appropriate demographic (postmenopausal women) or clinical profile (intense emotional stress or intercurrent organic disease) with echocardiographic or ventriculographic confirmation and mandatory exclusion of obstructive coronary disease by coronary angiography and risk stratification using clinical variables such as natriuretic peptides, troponin, left ventricular ejection fraction, the presence of thrombus in the left ventricle or obstruction of the left ventricular outflow tract determines the further direction and intensity of the patient's treatment.

Key words Takotsubo syndrome, heart failure, acute coronary syndrome, STEMI, echocardiography, natriuretic peptides, troponin, LVOT

Introduction

Takotsubo syndrome is a transient left ventricular systolic dysfunction, often induced by emotional or physical stressors in the absence of infectious myocarditis or pheochromocytoma[1]. It was first described in 1990 by Japanese authors[2] and because of the specific appearance of the left ventricle in the apical echocardiographic windows, it was termed after the Japanese octopus catching vessel. Since then, a large number of case reports was published around the world, and patient registries have been created for a better overview[3]. It is of paramount importance that this syndrome has a similar presentation to acute coronary syndrome, so differential diagnosis is important due to the different therapeutic approach in these clinical entities[1].

The aim of our case presentation was to report a patient initially treated as acute coronary syndrome referred for primary PCI procedure with a consequent diagnosis of Takotsubo syndrome with a discussion on the available literature.

Case presentation

A 66-year-old female patient presented to the emergency medical service of the Bor hospital because of ongoing chest tightness for the past two days. So far, she

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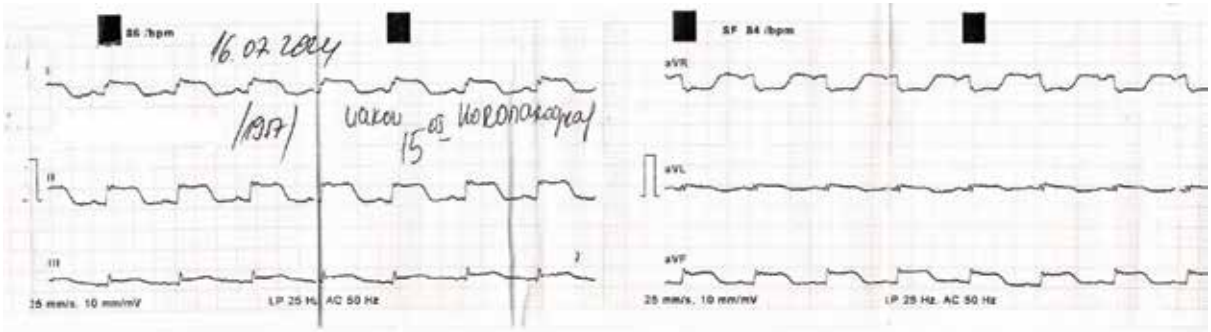

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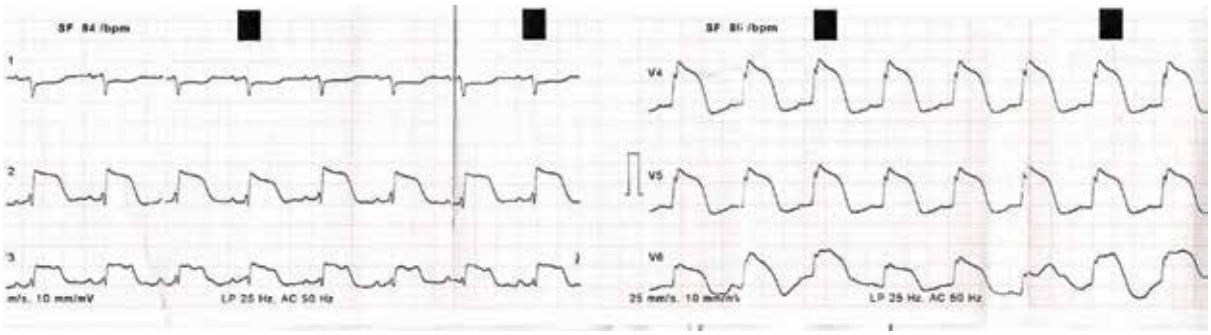

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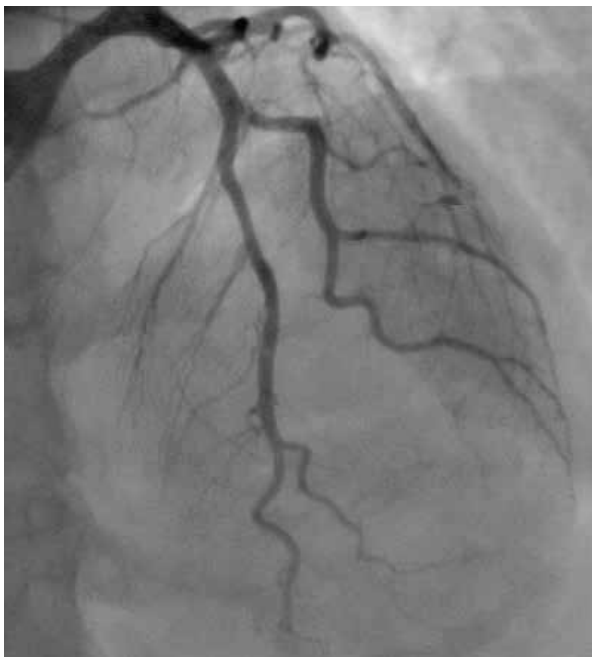

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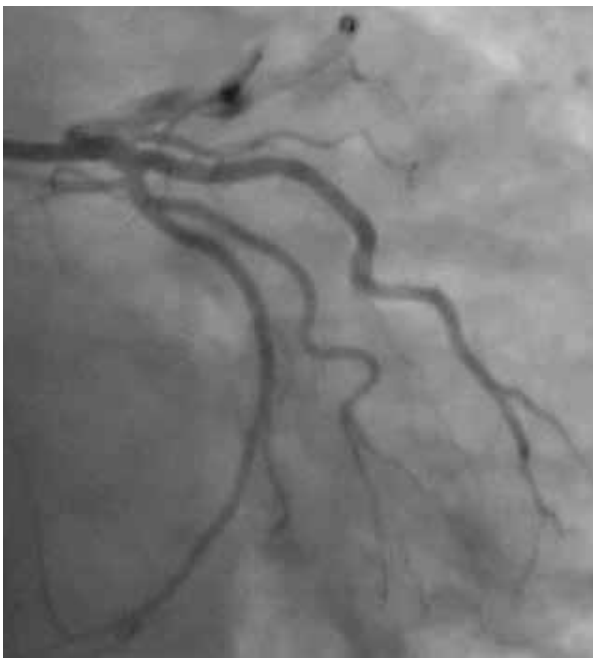
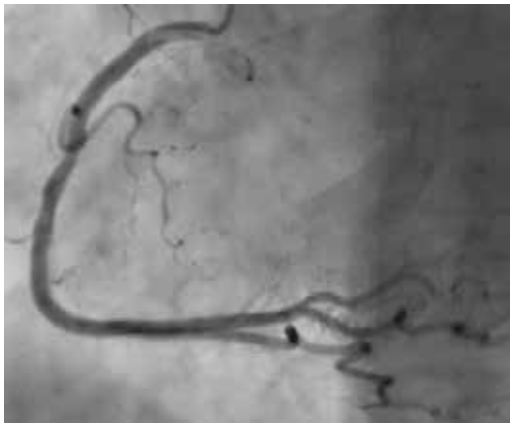


Figure 4.

has been treated for hypertension with a low dose of B-blockers. Electrocardiographically, ST segment elevation is registered in all leads except leads aVR and V1, in which ST segment depression is noted. ST elevation was most pronounced in leads V2-V6 over 10mm, without formed Q waves (Figures 1 and 2). She was referred for a primary PCI procedure at the Department of Invasive Cardiology under suspicion of anterior wall STEMI. During the exam, she reports chest pain during breathing and elements of hemodynamic instability are present - arterial blood pressure is 95/60 mmHg with signs of peripheral tissue hypoperfusion, heart rate of 85/min, normal SpO2 on room air and no pulmonary congestion. Abundant excoriations are visible all over the body, mostly on the leg area. The patient stated that 3 days ago she lost her dog, which she desperately tried to find on difficult terrain, with the aforementioned injuries and intense emotional distress. A selective coronary angiography was performed, which did not show any angiographically significant stenosis: a muscular bridge of the medial segment of the LAD was appreciated, which did not compromise the lumen of the artery in systole, and a 30% stenosis of the medial segment of the RCA (Figures 3, 4 and 5). Afterward, she was treated in the Coronary Care Unit and a transthoracic echocardiographic examination was performed. Akinesia of the apical and medial segments of both septums and all left ventricular walls was seen, along with hypercontractility of the basal segments and characteristic apical ballooning. Global systolic function was severely impaired



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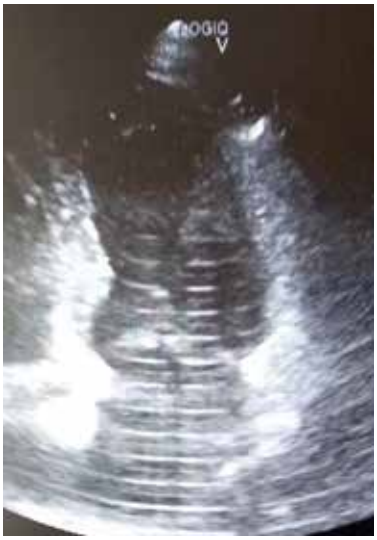

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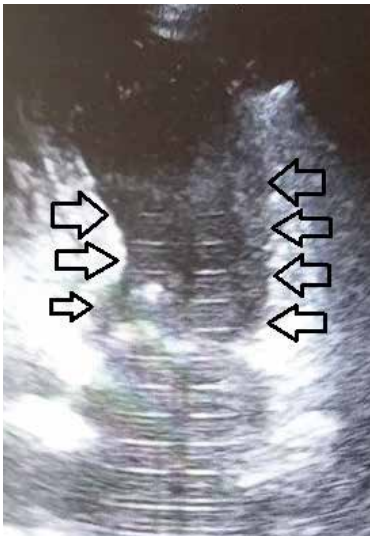

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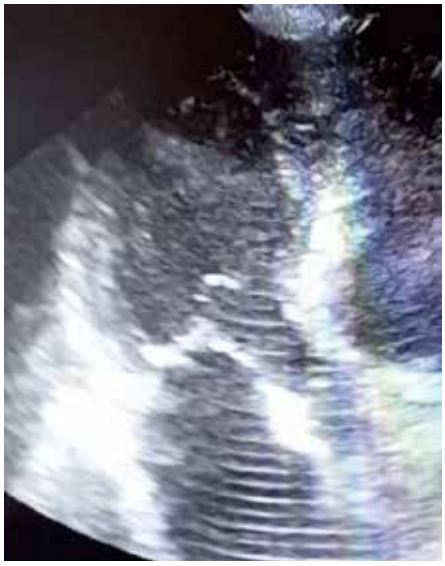

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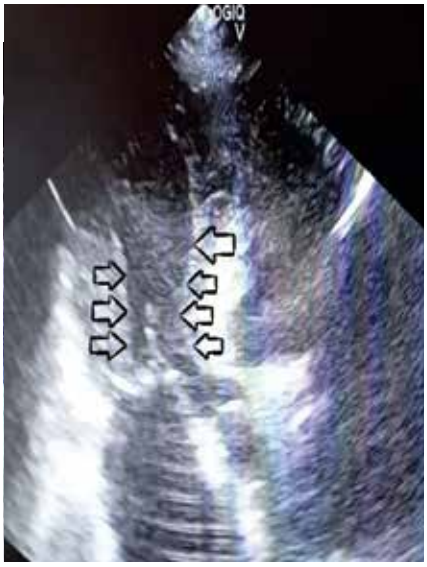

Figure 9.

with LVEF around 30%. The right heart chambers were of normal dimensions with a slightly enlarged left atrium (Figures 6, 7, 8 and 9). Considering the non-obstructive coronary artery disease, the characteristic echocardiographic findings, the patient's demographic data and the circumstances of intense emotional stress that preceded symptoms onset, a diagnosis of Takotsubo syndrome was made. Heart failure therapy was introduced. The high sensitivity (hs) troponin values were immeasurably high (over 25605 ng/L) and BNP on admission was 958 pg/ml. After the initial period of clinical stability, 24 hours after admission, pulmonary edema refractory to the applied therapy and the fatal outcome occured.

Discussion

Takotsubo syndrome is an acute left ventricular dysfunction with a characteristic pattern of wall motion abnormality, mainly induced by emotional or physical stress[3]. It is believed to occur in about 2% of patients referred to as acute coronary syndrome[4]. It is likely underreported because patients are misdiagnosed as acute coronary syndrome[3]. It occurs mostly in postmenopausal women. Most of the data was obtained from the results of the InterTAK registry, which published data on 1750 treated patients from 35 centers worldwide[3]. 90% of patients are female and in them Takotsubo syndrome occurs at an older age than in men (66.8 +/- 13 vs. 62.9 +/- 13.1 years). In 71.5% of patients there is a trigger for the onset of the syndrome: 27.7% emotional, 36% physical, 7.8% combined. It is important to note that 28.5% had no known trigger of the disease. Among the physical triggers, acute respiratory failure, postoperative and post-fracture conditions, and acute neurological diseases were most often present in descending order. Of the emotional triggers, among the most common are feelings of panic and anxiety (22.1% of all Takotsubo syndrome induced by emotional factors), which was also the case with our patient, because the onset of symptoms was preceded by intense stress and fear of losing an animal which, as the patient stated, could hurt someone. The pathophysiology of Takotsubo syndrome has not been fully understood. Some of the proposed mechanisms such as transient vasospasm have been rejected because the patterns of wall motion abnormalities do not correlate to the irrigation areas of individual coro-

nary arteries. The role of microvascular dysfunction, myocardial inflammation was considered, and catecholamine-mediated cardiotoxicity has the most support. It was observed that the level of circulating catecholamines is up to 3 times higher in patients with Takotsubo syndrome than in patients with acute myocardial infarction and post-infarction heart failure. It is also associated with hyperadrenergic conditions: in pheochromocytoma, during subarachnoid hemorrhage and after infusion of inotropes that act as sympathomimetics. It has been shown that in mammals, the sympathetic innervation of the basal parts of the myocardium is the most dense and the apical is scarce. Adrenergic receptors, on the other hand, are the most dense in the apical and the density decreases towards the basal parts of the myocardium. Catecholamines have a positive inotropic effect in low and medium concentrations, and in high, they seem to have a negative inotropic effect, which explains the greatest "stunning" and dysfunction of the apical segments of the left ventricular myocardium in most patients with Takotsubo syndrome[5].

Electrocardiographic changes are common in patients with Takotsubo syndrome, which leads to the differential diagnostic deliberation with acute coronary syndrome. ST segment elevation is described in 11-100% and negative T waves in 17-100% of patients. The differential diagnosis is mainly versus anterior wall STEMI, because the electrocardiographic changes in Takotsubo syndrome are most often present in the precordial leads. The ECG pattern depends to a great extent on the timing of the patient's presentation. It has been shown that there are 4 phases: in the acute phase, ST segment elevation is present, in the subacute phase (days 1-3), after the resolution of the ST elevation, -T waves appear initially; in the third, there is a reduced depth of -T waves, and in the fourth, a deepening of negative T waves is peresent, which can last for months, and when the echocardiographic and clinical recovery occurs. QT prolongation is usually pronounced, somewhat more than in anterior wall STEMI . Pathological Q waves are less common in Takotsubo syndrome than in myocardial infarction, and if they occur, they usually disappear later with the reappearance of R waves in the electrocardiogram, which indicates myocardial stunning rather than tissue necrosis[6]. In the study by *Frangieh et al*[7], 200 patients with Takotsubo syndrome from the InterTak registry were compared with 200 patients with acute coronary syndrome treated at the Zurich University Hospital (of which 106 patients with STEMI of different localizations).

It has been shown that ST segment elevation has excellent specificity for Takotsubo syndrome vs STEMI if with ST elevation in aVR: there is no elevation in V1 and pathological Q waves 97%; if there is ST elevation in the inferior leads 88%; or elevation in the anteroseptal leads 100%. We were particularly interested in ST segment depression in aVR because in our patient it was present in leads aVR and V1 with ST elevation in the other leads. In the aforementioned study, comparing the subgroups of patients with ST elevation Takotsubo syndrome and STEMI patients, it can be noted that ST depression in aVR was present in 43% of patients in the group with Takotsubo syndrome and 5% in the group of STEMI patients (p=0.001). ST depression in lead V1 has not been specifically reported. In a smaller series in which 43 patients with Takotsubo syndrome were compared with 407 patients with anterior wall STEMI, it was found that ST segment depression in AVR associated with the absence of ST elevation in the V1 lead has a diagnostic accuracy of 94% for distinguishing Takotsubo syndrome from patients with STEMI if is the occlusion is distal and 97% if the LAD occlusion is proximal to the first septal side branch[8]. This is in concordance with the electrocardiographic image seen in the patient we present in the paper.

Echocardiography plays a key role in identifying patients with Takotsubo syndrome. Echocardiography and ventriculography have described 4 subtypes of the disease: the typical apical subtype with hypo-, a- or dyskinesia of the apical and possibly medial with hyperkinesia of the basal segments of the left ventricle (about 80% of patients) and atypical forms of the syndrome (midventricular, basal, focal) that make up to 20% of all cases. In the atypical form of the disease the younger age, intercurrent neurological disease, electrocardiographic ST segment depression, less impaired systolic function and lower values of natriuretic peptides are more common. Patients with typical and atypical forms of the disease have comparable hospital outcomes and complication rates. Left ventricular ejection fraction below 45%, atrial fibrillation and the presence of a neurological disease were shown to be independent predictors of a poor prognosis (death in one year follow up)[3]. Our patient had an echocardiographic pattern of typical Takotsubo syndrome with akinesia of apical and medial segments of all walls, inferior and anterior septum with hyperkinesia of basal segments. The systolic function was greatly impaired (estimated ejection fractio of 30%), so it met one of the three previously mentioned poor prognostic criteria.

Since there is no single biomarker that distinguishes Takotsubo from acute coronary syndrome, troponin and natriuretic peptides (BNP/NT pro BNP) are often used in clinical assessment. In meta-analyses, troponin values have been shown to be significantly higher in acute coronary and natriuretic peptides in Takotsubo syndrome[9,10]. Due to divergent pattern of biomarker rise in these two clinical syndromes, a BNP/troponin I ratio has been proposed. It has been shown to be significantly elevated in patients with Takotsubo syndrome of 642 pg/mg compared to patients with NSTEMI - 184.5 and STEMI - 7.5[9]. This is consistent with the often severe left ventricular impairment seen in Takotsubo syndrome and yet preserved tissue viability, as demonstrated by the use of cardiac magnetic resonance imaging; in contrast to tissue fibrosis reflected in the presence of late gadolinium enhancement (LGE) seen in patients with acute coronary syndrome and MINOCA, Takosubo syndrome is characterized by edema and tissue inflammation, without fibrosis[11]. Both markers are considered to have prognostic value in patients with Takotsubo syndrome. In the study by *Stahli et al*[12] involving 2938 patients from the InterTak registry, the one-year mortality was 7.6%. An increase in troponin more than 28.8-fold the upper reference limit has been shown to represent clinically significant myo-

cardial damage. One-year mortality is significantly higher in these patients (p 0.001) and also five-year mortality (HR 1.58, p 0.002). The Takotsubo Syndrome Task force of the European Society of Cardiology has included natriuretic peptides as a risk stratifying factor. A BNP over 600 or NT pro BNP over 2000 pg/ml is among the minor factors in the risk assessment. In particular, NT pro BNP is considered an important indicator of clinical deterioration or recovery, and low admiession values predict a favourable prognosis[13].

Contrary to expectations in patients with Takotsubo syndrome, biomarkers of necrosis were more significantly elevated in our patient than heart failure markers, i.e. natriuretic pepetides. The BNP/troponin ratio was 38.4 pg/mg, which is less common in Takotsubo syndrome. It can be speculated whether this is due to the late presentation, as the patient seeked medical attention more than 48 hours after the symptom onset.

Although Takotsubo syndrome was previously considered to have a relatively benign clinical course, today it is known that this is not the case. Hospital mortality is comparable to that of STEMI patients[14,15]. After the acute event, patients with Takotsubo syndrome have a one-year mortality rate of 5.6% patient-years and a MACCE rate of 9.9 patient-years. As many as 1 in 8 patients will experience a recurrence of Takotsubo syndrome, often precipitated by a recurrent (often different from the initial episode) stress factor. To date, no clinical or physiological factors can predict the likelihood of recurrence[16].

Treatment of Takotsubo syndrome depends on the clinical manifestation. In the absence of severe left ventricular systolic dysfunction and other complications, clinical and echocardiographic follow up is usually sufficient. If overt heart failure is present, the same principles are applied as in the treatment of heart failure: diuretics as needed, ACE inhibitors, B blockers, nitrates[16]. Based on the proposed mechanism of catecholamine mediated cardiotoxicity, it would be expected that B blockers are the most effective therapeutic approach in Takotsubo syndrome. Clinical studies have shown, however, that B blockers do not reduce mortality[17] or recurrence rate[18]. The only exceptions are patients who develop left ventricular outflow tract (LVOT) obstruction and who are generally treated similarly to those with hypertrophic obstructive cardiomyopathy. About 20% of patients with Takotsubo syndrome develop LVOT obstruction and may also develop mitral regurgitation as a consequence of systolic anterior motion (SAM) of the anterior mitral leaflet. A mid ventricular gradient over 25 mmHg is considered significant, and over 40 mmHg is considered a high risk feature. Usually, LVOT obstruction resolves spontaneously after a few days[13]. In a small case-control study of *Santoro et al*.[19], IV administration of esmolol within the first 24 hours of admission followed by 1.5 mg of bisoprolol/day was shown to be beneficial by reducing the gradient in patients with LVOT obstruction. The use of ACE inhibitors has been shown to be beneficial in patients with Takotsubo syndrome because it reduces mortality in the one-year follow-up period[14] and recurrence rate[18]. Since Takotsubo syndrome is a self-limiting condition, treatment of heart failure (beta blockers, ACE inhibitors and, if needed, diuretics) should be given for a period of at least 3 months or until the systolic function recovery[20].

If cardiogenic shock develops without significant LVOT obstruction, it is recommended to cautiously administer inotropes such as dopamine and dobutamine. If there is a severe LVOT obstruction, the use of sympathomimetic inotropes is contraindicated. Intravenous fluids should be cautiously used in addition to levosimendan, if available, as it has been shown to be effective and safe in these circumstances[21]. In the most severe cases that are refractory to medication therapy, mechanical circulatory support (LVAD, ECMO) may be considered as a bridge to spontaneous recovery, while the use of an intra aortic balloon pump is avoided because it can worsen LVOT obstruction[13].

If rhythm disturbances occur, they are treated with antiarrhythmic drugs, including intravenous administration of magnesium in the treatment of torsade de pointes. The use of QT prolonging drugs (amiodarone, sotalol) is avoided because this interval is often very prolonged in the setting of Takotsubo syndrome.

Systemic thromboembolism is a significant and often overlooked complication of Takotsubo syndrome. In acute hospitalization, the rate of ischemic strokes is 1-2%, which is significantly higher than in acute coronary syndrome. The most likely origin of the embolus is a mural thrombus due to blood stasis in the akinetic segments of the myocardium. Spontaneous recovery of left ventricular systolic function can lead to embolization and subsequent stroke[16]. The risk of cerebral embolization is significant and according to the GEIST registry, it affects 17% of patients with confirmed left ventricular thrombus[22]. Patients with confirmed thrombus in the left ventricle should be treated with anticoagulant therapy for 3 months or until resolution[16].

Our patient was initially hypotensive, with signs of low stroke volume, without signs of pulmonary and systemic congestion, without LVOT obstruction, so she was treated with diuretics, mineralocorticoid receptor antagonists, SGLT2 blocker, B blocker, without ACE inhibitor due to hypotension. There were no heart rhythm disturbances. The next day there was a sudden clinical deterioration with the development of cardiogenic pulmonary edema refractory to diuretic therapy and non-invasive positive pressure ventilation. The episode proved fatal.

Conclusion

Takotsubo syndrome is an often unrecognized and not infrequent clinical entity that should be considered especially in patients with suggestive demographic (postmenopausal women) and clinical profile (intercurrent neuropsychiatric illness, respiratory insufficiency or intense emotional stress preceding the onset of symptoms) with chest pain and signs of heart failure. Early echocardiography and diagnostic coronary angiography, possibly also ventriculography are the cornerstone of a diagnosis. Biomarkers such as troponin and natriuretic peptides and, if available, cardiomagnetic resonance are important methods in the diagnostic algorithm of patients with Takotsubo syndrome.

References

1. Ghardi JR, et al. International Expert Consensus Document on Takotsubo Syndrome (Part I): Clinical Characteristics, Diagnostic Criteria, and Pathophysiology. Eur Heart J 2018;39(22): 2032–2046.
2. Sato H, et al. Takotsubo-like left ventricular dysfunction due to multivessel coronary spasm. Clinical Aspect of Myocardial Injury: From Ischemia to Heart Failure. Tokyo: Kagakuhyoronsha Publishing Co; 1990; 56–64.
3. Ghadri JR, Camman VL, Templin C. The International Takotsubo Registry: Rationale, Design, Objectives, and First Results. Heart Fail Clin 2016;12(4):597-603.
4. Scantelburi TC, Prasad A. Diagnosis of Takotsubo Cardiomyopathy. Circulation J. 2014;78(9):2129-2139.
5. Lyon A, Citro R, Schneider B, et al. Pathophysiology of Takotsubo Syndrome: JACC State-of-the-Art Review. JACC 2021;77(7):902–921.
6. Kosuge M, Kimura K. Electrocardiographic findings of takotsubo cardiomyopathy as compared with those of anterior acute myocardial infarction. J Electrocardiol 2014;47(5):684–689.
7. Fragieh AH, et al. ECG Criteria to Differentiate Between Takotsubo (Stress) Cardiomyopathy and Myocardial Infarction.J Amer Heart Assoc 2016;5(6).
8. Kosuge M et al. ST segment depresion in lead aVR combined with no ST segment elevation i lead V1 defferettiates Takatsubo cardiomyopathy from anterior acute myocardial infarction. JACC 2012;59(13):
9. Khan H, Gamble D, Meyincescu A, et al. A systematic review of biomarkers in Takotsubo syndrome: A focus on better understanding the pathophysiology. IJC Heart & vasculature 2021;34:100795.
10. Couch LS, Garrard J, Henry J, et al. Comparison of troponin and natriuretic peptides in Takotsubo syndrome and acute coronary syndrome: a meta-analysis. Open Heart 2024;11(1).
11. Citro R, Okura H, Ghadri JR, et al. Multimodality imaging in takotsubo syndrome: a joint consensus document of the European Association of Cardiovascular Imaging (EACVI) and the Japanese Society of Echocardiography (JSE). Eur Heart J Cardiovasc Imaging 2020;21(11):1184-1207.
12. Stahli EJ, Shindler V, Camman K, et al. Cardiac troponin elevation and mortality in takotsubo syndrome: new insights from the International Takotsubo (InterTAK) registry. Eur Heart J 2022;43(2):544-1466.
13. Lyon A, Bossone E, Shneider B, et al. Current state of knowledge on Takotsubo syndrome: A Position Statement from the Taskforce on Takotsubo Syndrome of the Heart Failure Association of the European Society of Cardiology. Eur J Heart Fail 2016;18(1): 8-27.
14. Templin C, Ghadri JR, Diekmann J, et al. Clinical features and outcomes of takotsubo (stress) cardiomyopathy. N Engl J Med 2015;373:929–938.
15. Uribarri A, Núñez-Gil IJ, Conty DA, et al. Short- and long-term prognosis of patients with takotsubo syndrome based on different triggers: importance of the physical nature. J Am Heart Assoc 2019;8:e013701.
16. Singh T, Khan H, Gamble DT, et al. Takotsubo Syndrome: Pathophysiology, Emerging Concepts, and Clinical Implications. Circulation 2022;145(13):
17. Isogai T, Matsui H, Tanaka H, et al. Early β-blocker use and in-hospital mortality in patients with Takotsubo cardiomyopathy. Heart 2016;102:1029–1035.
18. Singh K, Carson K, Usmani Z, et al. Systematic review and meta-analysis of incidence and correlates of recurrence of Takotsubo cardiomyopathy. Int J Cardiol 2014;174:696–701.
19. Santoro F, Ieva R, Ferraretti A, et al. Hemodynamic effects, safety, and feasibility of intravenous esmolol infusion during Takotsubo cardiomyopathy with left ventricular outflow tract obstruction: results from a multicenter registry. Cardiovasc Ther 2016;34:161–166.
20. Assad J, Femia G, Pender P, Badie T, Rajaratnam R. Takotsubo Syndrome: A Review of Presentation, Diagnosis and Management. Clinical Medicine Insights: Cardiology 2022;16.
21. Jaguszewski MJ, Gasecka A, Hering D, et al. Levosimendan improves the acute course of takotsubo syndrome: a pooled analysis. ESC Heart Fail. 2021;8(5):4360-4363.
22. Santoro F, Stiermaier T, Tarantino N, et al. Left ventricular thrombi in takotsubo syndrome: Incidence, predictors, and management: Results from the geist (german italian stress cardiomyopathy) registry. J Am Heart Assoc 2017; 6 e006990.

Sažetak

Takatsubo sindrom kao diferencijalno dijagnostička dilema u odnosu na akutni koronarni sindrom – prikaz slučaja

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***Uvod**. Takatsubo sindrom je ne tako redak klinički entitet u kome postoji tranzitorna sistolna disfunkcija leve komore, načešće indukovana emiocionalnim stresom, organskim oboljenjem ili njihovom kombinacijom. Klinički ga često nije lako razlikovati od akutnog koronarnog sindroma i nameće se kao diferencijalno-dijagnostička dilema u svakonevnom radu. Cilj rada je prikaz bolesnice sa Takatsubo sindromom inicijalno shvaćene kao akutni koronarni sindrom uz pregled i komentar dostupne literature.*

***Prikaz slučaja**. Bolesnica starosti 66 godina koja se do tada lečila od arterijske hipertenzije javlja se zdravstvenoj službi zbog opresija u grudima koje ima 48h unazad. Navodi intenzivan emocionalni stres koji je prethodio početku tegoba - gubitak psa i strah da bi isti nekoga mogao povrediti. Zbog viđene slike ST segment elevacije u prekordijalnim odvodima upućena je na urgentnu koronarografija u nadležni PCI centar. Nisu viđene angiografski značajne stenoze na epikardnim koronarnim arterijama. Urađen je ehokardiografski pregled kojim je viđena teško narušena globalna sistolna funkcija leve komore sa karakterističnom akinezijom apikalnih i medijalnih uz hiperkineziju bazalnih segmenata svih zidova leve komore što je uz anamnestičke i demografske podatke govorilo u prilog apikalnom podtipu Takatsubo sindroma. Natriuretski peptidi bili su povišeni a naročito je bio povišen hs troponin. Započeta je terapija srčane slabosti uz inicijalnu hemodinamsku stabilnost bolesnice. Narednog dana dolazi do razvoja edema pluća refraktarnog na primenjenu terapiju i smrtnog ishoda.*

***Zaključak.** Postavljanje sumnje na Takatsubo sindrom kod pacijenata odgovarajućeg demografskog (postemenopauzalne žene) ili kliničkog pofila (intenzivan emocionalan stres ili interkurentno organsko oboljenje) uz ehokardiografsku ili ventrikulografsku potvrdu a obavezno isključenje opstruktivne koronarne bolesti koronarnom angiografijom i stratifikacija rizika koristeći kliničke varijable kao što su natriurestki peptidi, troponin, ejekciona frakcija leve komore, prisustvo tromba u levoj komori ili opstrukcije izlaznog trakta leve komore određuje dalji pravac i intenzitet lečenja bolesnika.*

***Ključne reči:** Takatsubo sindrom; srčano popuštanje, akutni koronarni sindrom, STEMI, ehokardiografija, natriurestski pepditi, troponin, LVOT*