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TAKOTSUBO SYNDROME WITH CARDIOGENIC SHOCK IN A CAUCASIAN WOMAN

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ABSTRACT

Takotsubo syndrome (TS) is a transient, reversible dysfunction of the left ventricle (LV) without damage to the coronary arteries, which clinically manifests as acute coronary syndrome (ACS) and/or heart failure (HF). We are reporting the clinical case of a 58-year-old Caucasian woman who was admitted to our hospital with a clinical presentation of ACS and HF, but finally was diagnosed with TS. As it is known to us, this is the first case report of TS from Georgia. Accordingly, our goal is to promote scientific interest in this pathology because, till now, there are no modern guidelines or recommendations on the treatment and management of this disease.

INTRODUCTION

TS is a transient, reversible dysfunction of the LV without damage to the coronary arteries, which clinically manifests as ACS and/or HF. [1] The word "Takotsubo" in Japanese culture is the name of the clay vessel used to hold the octopus, which is very similar in shape to the radiological image obtained in the ventriculogram of the apical ballooning of the LV. [2] This phenomenon was first described in 1990, in Japan patients with myocardial infarction, who showed specific changes in the systolic configuration of the LV. [3]

TS is now well known in Europe, the USA, the UK and many other countries. The prevalence of patients presenting with ACS is approximately 2%, and most cases are in postmenopausal women. [2,3]

The exact mechanism of TS development is not well known. Over-activation of the sympathetic system, microvascular pathology, metabolic disorders of the myocardium, and coronary vasospasm are considered as the major causes. Often, the catecholamine release mechanism is a strong, negative psycho-emotional stress, such as family conflicts, fear, psychological trauma, death of a close person. However, in a small number of cases, positive stress was also described, for example, birthday surprise, winning the jackpot, etc. [1] In addition to all mentioned, extreme physical exertion is considered to be another trigger. For example, by Chams S. et al, in 2018, a clinical case of a 38-year-old woman was described, which was associated with the so-called Zumba fitness. [4]

CASE REPORT

A history of Present Illness: A 58-year-old Caucasian woman presented to the ER with retrosternal, squeezing pain and shortness of breath. The above-mentioned symptoms, but with less intensity, started one day before admission, which was preceded by strong emotional stress.

Physical Exam and vitals: On admission, physical examination revealed wheezing in the lungs, sinus tachycardia, and increased jugular vein pressure. No other clinically significant pathological changes were observed. The heart rate was 103 beats per minute (bpm), the SpO2 level was 92%, the respiratory rate was 24 bpm, and the blood pressure was 102/75 mmHg.

Past Medical History and medications: The patient doesn't have a history of any other disease except hypothyroidism, which was managed with Levothyroxine. She reported that she had been in menopause for the past few years. In the ER, based on clinical presentation, the condition was evaluated as ACS complicated by AHF (NYHA functional class III).

Laboratory: Based on provisional diagnosis, troponin I, a specific biomarker of myocardial necrosis, was measured. It was 0.77 ng/ml. The obtained result was approximately 33 times the upper limit of normal (ULN = 0,023 ng/ml). The hemoglobin level was 133 g/L (VN: 110–160 g/L), the white blood cell count was 9,8×109/L (VN: 4-10×109/L), and the platelet count was 267×109/L (VN: 100-300×109/L) Human. Plasma creatinine, urea, and hepatic panel were normal.

Instrumental diagnostics: Electrocardiography (ECG) detected an inversion of T waves in I and aVL leads and QT interval prolongation up to 551 msec. The patient was diagnosed with acute myocardial infarction and AHF and was admitted to the coronary intensive care unit (CICU). An echocardiogram showed apical hypo-akinesia and severe systolic dysfunction. The LV ejection fraction (LVEF) was 24%.

Managment: A clinical picture of cardiogenic shock appeared within a few hours of hospitalization. Accordingly, an IV infusion of Norepinephrine was started at a rate of 0.02 mcg/kg/min, which was increased to 0.03 mcg/kg/min. Dobutamine I.V was added approximately 2 hours later at a rate of 1 mcg/kg/min since oliguria was noted.

Taking into account the main diagnosis, coronary artery angiography was performed. Hemodynamically significant stenosis wasn't detected. On the same day of hospitalization, follow-up ECG revealed an inversion of T waves in the precordial leads. The dynamics of T waves and QT interval changes are given in Table - 1.

		Day 1 (ER)	Day 1 (CICU)	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8
Qtc/msec		551	576	609	498	525	470	492	479	511
	I	-0.5	-0.5	-0.5	≈0	-1	0	+	-	-0.5
	aVL	-1	-1	-0.5	≈0	+	0	0	-	-0.5

	V1	0	-1	-0.5	0	+	-0,5	+	+	+
	V2	+	-1	-1	+	+	+	+	+	+
	V3	+	-1.5	-1	+	+	+	+	-2	+
	V4	+	-1.5	-1.5	-0,5	+	-0,5	-1	-2	-4
	V5	+	-1	-2	-1	+	-1	-1.5	-2.5	-3
	V6	+	-1	-2	-0,5	+	-1	-1	-2.5	-2

Table - 1. Dynamic changes of T waves and QT interval according to days of hospitalization. QTc was calculated by Bazett's formula. The table shows the ECG leads in which specific changes were observed. A negative number indicates an inversion of the T wave, 0 - an isoline, and + a positive deflection from the isoline.

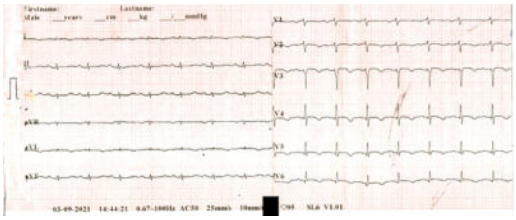

Image – 1. ECG on the day of admission (CICU) showing inversion of T waves in I, aVL, V1-V6 leads.

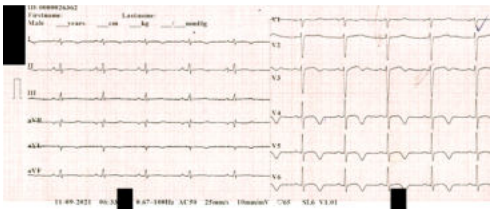

Image – 2. ECG on the day of discharge, which shows an increase in the degree of inversion of T waves in V4-V6 leads, and a decrease in V1-V3.

Diagnosis: The patient was diagnosed with TS according to the Mayo Clinic criteria.

Treatment: The patient was kept on double inotropic support and positive dynamics were observed. An adequate amount of urine was released and blood pressure stabilized. Decreasing doses of Dobutamine were stopped on the 3rd day of initiation, and Norepinephrine was stopped on the 5th day. In addition, the patient received 50 mg of Spironolactone and 75 mg of Aspirin per day. ACEi/ARB could not be administered due to hemodynamic instability.

Outcome: The treatment was successfully completed on the 8th day of admission. The symptoms of AHF were completely resolved. On the day of discharge, control echocardiography showed improvement in systolic function and regional contractility. On the same day, LVEF was 35%, and 2 weeks after discharge, 45%. Thyroid-stimulating hormone (TSH) at that time was within the normal range – 1.53 μIU/ml (VN: 0.35-5.5 μIU/ml).

DISCUSSION

TS, as in our case, is clinically manifested with anginal complaints and AHF, by ECG changes (ST elevation, QT prolongation, inversion of T waves, etc.) and an increase in biomarkers of myocardial necrosis. That makes TS even more similar to ACS, but in contrast to it, the presence of hemodynamically significant lesions is not recorded on coronary angiography.

To facilitate the differential diagnosis, the Mayo Clinic provides the following criteria: (1) Transient hypokinesia, dyskinesia, or akinesia of LV segments with or without apical involvement; (2) Absence of significant coronary artery damage; (3) New-onset ECG changes (ST-segment elevation and/or T-wave inversion) or significant elevation of troponins; (4) absence of pheochromocytoma and myocarditis; [5]

The clinical outcome of TS is benign in most cases, as evidenced by the synonym of this disease - "reversible dysfunction of the LV". According to a large epidemiological study, TS is characterized by a 4,2% in-hospital mortality rate [2], although it can be complicated by severe, life-threatening conditions such as cardiogenic shock. Cardiogenic shock raises the risks of both in-hospital and 60-day mortality (23,35% versus 2,3%, P 0,001). [6]

The described case is a clear example of TS mimicking ACS with its clinical course and laboratory-diagnostic tests. As it is known to us, this is the first case report of TS from Georgia.

CONCLUSION

TS is a newly discovered acute cardiological pathology, the exact cause of which has not yet been established. Therefore, there are no recommendations for its treatment and management. That is why it is of great interest both in the scientific community and among doctors with clinical practice. Because it mimics ACS, it is very difficult to differentiate it at the initial stage of the disease. The presented clinical case is a classic example of this disease, which rarely occurs in our region. Our goal as scientists is to increase interest in this pathology in the national scientific community.

COMPETING INTERESTS

The author(s) declares that they have no competing interests.

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REFERENCES

[1] Lyon, A. R., Citro, R., Schneider, B., Morel, O., Ghadri, J. R., Templin, C., & Omerovic, E. (2021). Pathophysiology of Takotsubo Syndrome: JACC State-of-the-Art Review. Journal of the American College of Cardiology, 77(7), 902–921. https://doi.org/10.1016/j.jacc.2020.10.060

[2] Gupta, S., & Gupta, M. M. (2018). Takotsubo syndrome. Indian heart journal, 70(1), 165–174. https://doi.org/10.1016/j.ihj.2017.09.005

[3] Y-Hassan, S., & Tornvall, P. (2018). Epidemiology, pathogenesis, and management of takotsubo syndrome. Clinical autonomic research : official journal of the Clinical Autonomic Research Society, 28(1), 53–65. https://doi.org/10.1007/s10286-017-0465-z

[4] Chams, S., El Sayegh, S., Hamdon, M., Kumar, S., & Kulairi, Z. (2018). Zumba-induced Takotsubo cardiomyopathy: a case report. Journal of medical case reports, 12(1), 160. https://doi.org/10.1186/s13256-018-1696-x

[5] Amin, H. Z., Amin, L. Z., & Pradipta, A. (2020). Takotsubo Cardiomyopathy: A Brief Review. Journal of medicine and life, 13(1), 3–7. https://doi.org/10.25122/jml-2018-0067

[6] Di Vece, D., Citro, R., Cammann, V. L., Kato, K., Gili, S., Szawan, K. A., Micek, J., Jurisic, S., Ding, K. J., Bacchi, B., Schwyzer, M., Candreva, A., Bossone, E., D'Ascenzo, F., Sarcon, A., Franke, J., Napp, L. C., Jaguszewski, M., Noutsias, M., Münzel, T., … Templin, C. (2019). Outcomes Associated With Cardiogenic Shock in Takotsubo Syndrome. Circulation, 139(3), 413–415. https://doi.org/10.1161/CIRCULATIONAHA.118.036164