Tako-tsubo cardiomyopathy

Tako-tsubo cardiomyopathy (sometimes called apical ballooning cardiomyopathy, broken heart syndrome, stress cardiomyopathy) is a **rare disease of unclear etiology**. Today, the theory of myocardial stunning by high concentrations of circulating **catecholamines** is the most accepted . Catecholamines are directly toxic to the myocardium and secondarily cause transient microvascular dysfunction.[1][2] The disease **mimics acute coronary syndrome** and is often manifested by acute heart failure.[2] Although the disability is usually reversible, serious and life-threatening complications can also occur.

The word Tako-tsubo comes from Japan and refers to a special basket of Japanese fishermen, designed to catch squid, whose shape is similar to a classic light bulb.

Etiopathogenesis

The pathophysiology of tako-tsubo cardiomyopathy **is still unclear**. One hypothesis works with myocardial stunning by circulating **catecholamines**. Some papers question this theory and call elevated catecholamine levels a compensatory mechanism.[2][3] The theory **of noradrenaline and neuropeptide Y**, which are neurotransmitters of sympathetic presynaptic neurons, is investigated.[3] These substances can be toxic to the myocardium and lead to epicardial and microvascular dysfunction. It is not clear why some people develop the disease, some develop it after exposure to factors and others do not. **Genetic predisposition** probably plays a roleside effects of catecholamines and other substances on the myocardium.[3][4]

The triggering factor is not detected in up to one third of the patients.[3] **Triggers are in most cases physical or emotional factors, but it is not always the case. Physical evoking factors predominate.**[4] Among the emotional factors (approximately 5 days before the manifestation of the difficulties) are strong stressful situations such as loss of a partner, violence, natural disaster, great financial loss. Among the physical ones are, for example, acute serious injuries and diseases, surgery, meningitis, stroke, pulmonary embolization, chemotherapy, administration of catecholamines, sepsis, exacerbation of COPD, etc.[2][3][4]

Epidemiology

Tako-tsubo cardiomyopathy very often mimics acute coronary syndrome. Of the total number of coronary angiographies performed for suspected acute coronary syndrome, tako-tsubo cardiomyopathy is finally diagnosed in approximately 1-2% of patients (data from the USA).[2][3] Up to 90% of them are women, the majority being **postmenopausal patients over 65 years of age**.[3] This is due, among other things, to the combination of increased sympathetic tone, endothelial dysfunction and increased neuropeptide Y secretion with lower coronary reserve.[3]

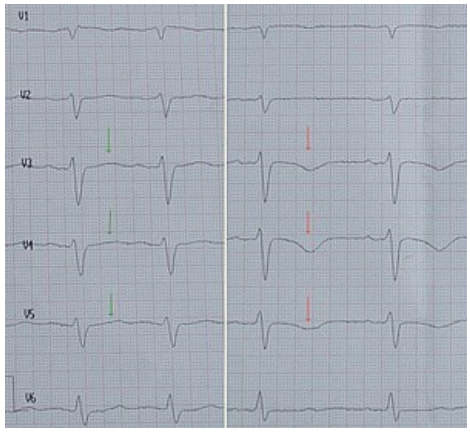
Clinical picture

Postmenopausal women are most often affected (90%). The predominant symptom is **acute chest pain** of angina pectoris.[1][3] Other common symptoms are **shortness of breath, dizziness and syncope**. The last two symptoms are related to hypotension and hypoperfusion and may indicate developing cardiogenic shock or ventricular tachyarrhythmia.[3]

Diagnosis

The diagnostics follow the recommendations of professional companies.[3][4][5] The clinical picture, typical changes in imaging methods and markers of myocardial damage apply. An important diagnostic criterion is also that **left ventricular systolic dysfunction is reversible** within a few weeks to months of the onset of the problem.[3] Acute coronary syndrome, myocarditis and pheochromocytoma based cardiomyopathy must be ruled out in differential diagnosis.[1][3]

A typical picture in imaging methods (including ventriculography) is the reduced contractility of the apical segments of the left ventricle, which is compensated by the increased contractility of the basal segments. This, together with the forward movement of the anterior tip of the mitral valve, can lead to obstruction of the left ventricular outflow tract and the formation of apical thrombi. However, the basal or middle segments of the left ventricle may also be hypokinetic. The biventricular type may also occur. In these forms, hemodynamic instability is often greater. Myocardial kinetics disorder extends beyond the distribution of a single coronary artery.[1][2][3][4]



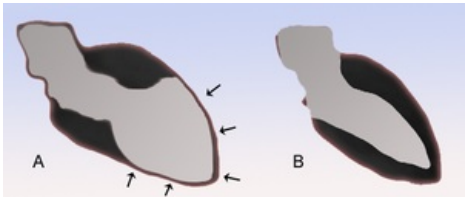
ECG finding in Tako-tsubo cardiomyopathies. T-wave inversions in precordial leads are visible.

The ECG is abnormal in most cases. **ST elevations and T wave inversions** are usually present. **QT prolongation** is also common, associated with an increased risk of ventricular tachycardia and ventricular fibrillation.[3] Patients must therefore be monitored telemetrically. **ECG does not clearly distinguish between tako-tsubo cardiomyopathy and acute coronary syndrome!**[1]}}

Diagnostic coronary angiography is performed on patients to **rule** out acute coronary syndrome . Examination should not reveal significant atherosclerotic changes in the coronary arteries in most cases.[3]

In addition to typical contractility disorders, **echocardiography** also shows possible obstruction of the left ventricular outflow tract and related mitral regurgitation.[2][3][5] It is basic and due to the dynamics of the disease a very important imaging method in monitoring the patient's condition and **stratifying the risk of complications**.

Biomarkers of myocardial damage tend to have different dynamics than acute coronary syndrome. **Elevation of troponin and CK-MB is usually mild**. In contrast, most patients have a **marked elevation of natriuretic peptides** (BNP or pro-BNP), which is usually higher than coronary syndrome.[2][3][4][5]



Left ventriculography schematically - Tako-tsubo cardiomyopathy - apical form (A), normal finding (B)

Magnetic resonance imaging of the heart is useful for the **differential diagnosis** and differentiation of tako-tsubo cardiomyopathy from myocarditis and ischemic myocardial infarction. Myocardial edema is usually present. The advantage of using magnetic resonance imaging is that it has better resolution of myocardial segments, including the right lateral compartments, and possible thrombi in the tip of the left ventricle.[1][3]

Complication

Tako-tsubo cardiomyopathy is a transient disease, but **the patient's condition** must be **closely monitored**. Systolic **heart failure** is present in up to half of the patients. [3][5] Current mitral regurgitation, left ventricular outflow tract obstruction, and right-sided involvement can significantly worsen heart failure.[3] At the same time, they pose a higher risk of developing cardiogenic shock. **Arrhythmias** occur in about a quarter of patients.[3][5] They are a character of atrial fibrillation or due to QT prolongation torsade to pointes. Other complications include **thrombi** in the left ventricular cavity and rarely rupture of the left ventricular wall.[1][3][5]

Therapy

In the acute phase, **the patient's ECG and echocardiographic parameters must be monitored repeatedly** . Even patients without prolonged QT interval should be hospitalized in the coronary unit for the first time due to possible complications.[1][3] Pharmacological treatment for this disease is highly individual and, in addition to the severity of heart failure, is largely governed by the presence and severity of left ventricular outflow tract obstruction and associated hypotension. Therefore, **caution should be exercised when using ionotropics**.[1][3][5] ACE inhibitors,diuretics and beta-blockers are used in the treatment of heart failure which also have a certain antiarrhythmic effect and can improve the obstruction.[1][3][5] In some cases, venodilators are used. [3] Anticoagulation is used when thrombi are detected. In cardiogenic shock patients, mechanical cardiac support, ECMO, or intra-aortic balloon pump may be indicated.[3]

There is no study of chronic medication that confirms the positive effect of long-term use of ACE-inhibitors, beta-blockers or other drugs. The use of beta-blockers is supported by experts, especially in patients with increased sympathetic tone.[3][5]

Prognosis

Hospital mortality is 2-5%. Due to the reversibility of left ventricular dysfunction, the long-term prognosis is mostly good, however, the recurrence of cardiomyopathy within 10 years is about 20%.[3] Metabolic and myocardial structure changes are also described in some patients.[3]

Links

related articles

- Cardiomyopathy

References

- CZECH, Richard, et al. Internal. 1st edition. Prague: Triton, 2010. 855 pp. ISBN 978-80-7387-423-0
- PALEČEK, Tomáš. Non-sarcomeric hypertrophic cardiomyopathies, Takotsubo cardiomyopathies: II. Department of Internal Medicine, Cardiology and Angiology, 1st Faculty of Medicine, Charles University, General University Hospital, Prague; ICRC-FNUSA, Brno [online]. [feeling. 2014-03-27]. < http://www.kardio-

cz.cz/resources/upload/data/474_palecek-nesarkomerickehypertrofickekmp.pdf/ >.
- MANN, Douglas L, et al. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine. 10th Edition edition. 2015. ISBN 978-0-323-29429-4.
- MEDINA DE CHAZAL, Horacio, Marco Giuseppe DEL BUONO and Lori KEYSER-MARCUS. Stress Cardiomyopathy Diagnosis and Treatment. Journal of the American College of Cardiology. 2018, year. 16, vol 72, pp. 1955-1971, ISSN 0735-1097. DOI: 10.1016 / j.jacc.2018.07.072.
- GHADRI, Jelena-Rima, Ilan Shor WITTSTEIN and Abhiram PRASAD. International Expert Consensus Document on Takotsubo Syndrome (Part I): Clinical Characteristics, Diagnostic Criteria, and Pathophysiology. European Heart Journal. 2018, year. 22, pp. 2032-2046, ISSN 0195-668X. DOI: 10.1093/eurheartj/ehy076 (https://academic.oup.com/eurheartj/article/39/22/2032/5025412).

External links

- Takotsubo kardiomyopatia (TECHmED) (https://www.techmed.sk/takotsubo-kardiomyopatia/)

Reference

1. PALEČEK, Tomáš. *Non-sarcomeric hypertrophic cardiomyopathies, Takotsubo cardiomyopathies: II. Department of Internal Medicine, Cardiology and Angiology, 1st Faculty of Medicine, Charles University, General University Hospital, Prague; ICRC-FNUSA, Brno* [online]. [feeling. 2014-03-27]. < http://www.kardio-cz.cz/resources/upload/data/474_palecek-nesarkomerickehypertrofickekmp.pdf/ >.
2. MANN, Douglas L, et al. *Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine.* 10th Edition edition. 2015. ISBN 978-0-323-29429-4 .
3. MEDINA DE CHAZAL, Horacio, Marco Giuseppe DEL BUONO and Lori KEYSER-MARCUS. Stress Cardiomyopathy Diagnosis and Treatment. *Journal of the American College of Cardiology.* 2018, year. 16, vol 72, pp. 1955-1971, ISSN 0735-1097. DOI: 10.1016 / j.jacc.2018.07.072 .
4. GHADRI, Jelena-Rima, Ilan Shor WITTSTEIN and Abhiram PRASAD. International Expert Consensus Document on Takotsubo Syndrome (Part I): Clinical Characteristics, Diagnostic Criteria, and Pathophysiology. *European Heart Journal.* 2018, year. 22, pp. 2032-2046, ISSN 0195-668X. DOI: 10.1093 / eurheartj / ehy076 .
5. GHADRI, Jelena-Rima, Ilan Shor WITTSTEIN and Abhiram PRASAD. International Expert Consensus Document on Takotsubo Syndrome (Part II): Diagnostic Workup, Outcome, and Management. *European Heart Journal.* 2018, year. 22, vol. 39, pp. 2047-2062, ISSN 0195-668X. DOI: 10.1093 / eurheartj / ehy077 .