LIFE-THREATENING ARRHYTHMIAS

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Abstract. *The difficulty of making a diagnosis for chest pain in SCST is due to the similarity of those in MI, aortic dissection, stable and unstable angina. The importance of timely diagnosis of SCST is associated with the risk of sudden cardiac death, acute MI, syncope, severe, often life-threatening disturbances of rhythm and conduction (ventricular tachycardia, ventricular fibrillation, high -grade atrioventricular blockade).*
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When examining a patient with SCST, it should be taken into account the possibility of the clinic's debut with epileptiform seizures, esophagospasm, the presence of thyrotoxicosis (up to 20% of such patients suffer from BCC) [43-45]. At the same time, it is advised to determine the level of thyroid hormones in patients with SBST, especially with its resistance to drug therapy [45]. It is necessary to carefully study non-specific complaints that can be regarded as a mental disorder (especially in women) [46]. It is considered important to differentiate the SCT, especially the refractory course, with the Kounis syndrome, which is not so rare, but it is rarely diagnosed, resembles ACS according to the clinic, but is associated with the activation of mast cells and platelets in the development of hypersensitivity and allergic reactions [47, 48]. Life-threatening arrhythmias are a serious manifestation of ischemic attack due to CFS and are associated with syncope or sudden death [1, 49]. It should be taken into account that the inducibility of polymorphic ventricular tachycardia or ventricular fibrillation, increased QT dyspsia, T wave alteration and early repolarization during the asymptomatic period are considered risk markers development of ventricular arrhythmias during CA spasm [49]. Studies have shown that ventricular arrhythmias were observed in 90% of cases during a BCC attack, 2/3 of them were short–term episodes of ventricular tachycardia [50]. The frequency and significance of ventricular arrhythmias is associated with the duration of the vasospastic episode ($p<0.005$), the degree of elevation of the ST segment ($p<0.006$), the presence of ST–T wave alteration ($p<0.005$), an increase in the height of the R wave by more than 25% ($p<0.025$). During an attack of Prinzmetal's angina pectoris , other rhythm disturbances were often noted and cardiac conduction, which play an important role in the "arrhythmogenic mood of the heart": a) supraventricular tachycardia, grade II AV block and complete AV block during right SC spasm, AV dissociation, sinus node dysfunction [51].

Results of the multicenter National register Japanese Association for the Study of Coronary Spasm [from 1429 patients with BCC [35 (2.8%) survived community- acquired cardiac arrest] showed that patients with BCC who suffered cardiac arrest are a high-risk group, and they should timely consider the possibility of implanting a cardiofibrillator for secondary prevention of cardiac arrest [19, 20, 52]. In addition, in patients with BCC with suspected arrhythmia due to vasospasm, moni should be performed- electrocardiogram toring (ECG; with the possible use of an implantable recorder), electrophysiological examination and coronary angiography (including acetylcholine provocation) [49].

A BCC attack can simulate ACS with damage to three coronary vessels [53]. It should also be taken into account that ventricular tachycardia associated only with syncope can be a variant of BCT without heart pain, which requires a provocative intracoronary test with ergonovine [54]. The ECG reveals characteristic changes in the form of ST segment elevation (class I, C1), but in practice, ECG recording during an attack is not always possible [55]. Outpatient ECG monitoring during 24–48h (IIa, C) or more in patients with SCST, in whom this condition is suspected, allows us to determine episodes of atypical chest pain and assess the extent of ischemia load, the severity of ischemia during the patient's daily activity [7, 9, 56]. It is also recommended to use implantable recorders (loop-recoder) for a long time, which makes it possible to more clearly identify rarely occurring, life-threatening arrhythmias [7]. Coronary angiography with provocative testing (IIa, C) with ergonovine and acetylcholine is the gold standard for the diagnosis of SCST [42, 57].

At the same time, the response to provocative stimuli:

a) reproduction of normal chest pain;

b) occurrence of ischemic changes on ECG;

d) vasoconstriction >90% (according to angiography) [42].

In the absence of at least one of the components, the test is considered questionable. It should be noted that spasm can occur both in atherosclerosis-affected CA and in intact vessels [58, 59]. So, according to the German research CASPAR (Coroary Artery Spasmi Patients with Acute Coroary SydRome), every fourth patient with ACS does not have hemodynamically significant CA lesions. Among these patients, the KA spasm documented with intracoronary administration of acetylcholine is noted in half of the cases [60]. At the same time, it is shown that even normal according to the data. An ECG of a spacecraft under closer examination [according to intravascular ultrasound examination (ultrasound)] may have minimal signs of atherosclerosis [61].

It should be emphasized that a negative angiographic provocative test does not allow (!) definitively exclude coronary vasospasm as the cause of the VSST, confirming the golden rule of domestic clinicians: "a positive symptom confirms the diagnosis, a negative one does not reject it" [62].

Research Group for the Study of Coronary Vasomotor Disorders (Coroary Vasomotor Disorders Iteratioal Study Group [COVADIS]) has developed international standards for diagnostic criteria for coronary vasomotor disorders.

"Unconditional BCC" is diagnosed if nitrateresponsive angina is evident during immediate episodes, and also transient ischemic changes in the cardiogram during immediate episodes or criteria for spasm are present. A "suspected BCC" is diagnosed if nitrate-responsive angina is evident during immediate episodes, but transient ischemic changes in the cardiogram are doubtful or unavailable and the criteria for spasm are questionable. And finally, the relationship with the risks and complications of provocative testing in the case of transient changes ECG during a nitrate-responsive angina episode, as defined by COVADIS, which can be refrained from [9, 42].

The possibility of using other techniques is being studied for the diagnosis of BCC (the class of recommendations is indicated in parentheses, according to the Japanese Association of Blood Circulation): physical activity test (class IIb);

hyperventilation in patients with low (class IIa) and high (class IIb) frequency of angina attacks; endothelial dysfunction detection test (class IIb);

multi-detector computer tomography (Class IIB); cold test or mental load with a stable condition of the patient and suspicion of SCST (Class IIb);

coronary blood flow examination (IIb);

measurement of coronary sinus lactate levels during induced coronary spasm (Class IIB);

coronary angioscopy; intravascular ultrasound [10, 22].

The possibility of using cohort optical tomography and single-photon computed emission tomography with 123I β-methyl-branched fatty acid for differential diagnosis of BCST and cardiomyopathy is being studied [63, 64].

Treatment of patients with SCST should be comprehensive with a mandatory combination of non-drug, drug-based methods of exposure and timely determination of indications for surgical treatment.

1. Correction of risk factors includes (all classes I):

a) quitting smoking;

b) blood pressure control;

c) maintaining an ideal weight;

d) correction of impairedglucose tolerance;

e) correction of lipid metabolism disorders;

f) avoiding excessive fatigue and mental stress;

g) refusal from drinking alcohol;

h) exclusion of trigger factors [10].

2. Drug therapy is carried out with the following drugs:

1. Nitrates – sublingual administration, intravenous administration during an attack (class I), prolonged for the prevention of coronary spasm (IIa) [10]. Therefore, the emergence of tolerance to nitrates may limit their use as first-line therapy [6, 22].

2. Nicorandil (Class IIa) is a CATF activator, has a nitrate–like effect and also suppresses attacks BCST [65].

3. Calcium channel blockers (BCCs) are considered first–choice drugs for the treatment of VSST. They control the condition of patients well and provide a generally favorable long- term prognosis (class I) [10, 66]. It is important that long–acting BCCs are taken before bedtime in such a way that the time of their maximum activity falls on the period of a possible attack (midnight-early morning) [22]. They begin with the use of high doses of long-acting BCC (nifedipine 80 mg / day, amlodipine 20 mg / day, diltiazem 360 mg / day or verapamil 480 mg / day). In case of refractory FAC , the appointment of a combination is recommended BCC consists of two different classes (dihydro- and non-dihydropyridine) in combination with the intake of nitrates [22].

Concomitant combined use of beta-blockers is possible only with SCT with significant CA stenosis (IIa). Monotherapy with β-blockers can be harmful (class III) due to the potential increase in the frequency of seizures and their duration due to vasospasm caused by the predominance of stimulation of α-adrenergic receptors during blockade of β-adrenergic receptors [10, 67]. Moreover, beta- blockers of the third generation (for example, nebivolol), despite their vasodilating properties, can also induce CFS with the development of SCST [68].

In patients with multivessel spasm, BCC should not it can be canceled even with a decrease in the number of seizures and their absence during treatment, since mute ischemia, which often occurs in such patients, is a trigger for the development of lethal arrhythmias and sudden cardiac death [22]. It is possible to use other medications that are considered effective in suppressing KVS: vitamin E (IIb), estrogens in postmenopausal women with BCST (IIb), corticosteroids (IIb), statins (IIb), inhibitors of the renin-angiotensin-aldosterone system (IIb), direct inhibitor of Rho-kinase

in refractive- VSST [10, 69]. It has also been shown that intravenous therapy allows to quickly stop the attack, leading to vasodilation of all CA [70].

Nevertheless, even despite the correctly selected treatment, about 20% of patients suffering from BCT do not respond to therapy with two BCCs in combination with long-acting nitrates [6].

Therefore, in the treatment of refractory vasospastic angina, the following strategies may be used, which may be effective:

a) high doses of BCC (verapamil or diltiazem 960mg/day and/or nifedipine 100 mg/day);

b) antiadrenergic drugs, including guanetedin or clonidine;

c) corticosteroids;

d) blockade of the left stellate ganglion;

e) selective percutaneous accommodation [71-76].

Unfortunately, even optimal treatment is not able to eliminate the entire risk of recurrence of SCT due to the development of life-threatening ventricular arrhythmias [19]. Interventional interventions. Data on the use of interventional interventions in patients with SCD are contradictory [77]. The main problems of stenting CA in SCST is a recurrence of spasm proximal or distal to the installed stent, as well as its restenosis [10, 78]. A number of studies have shown that drug-coated stents in some cases can cause endothelial dysfunction, contributing to the development of diffuse coronary spasm [79]. According to the recommendations for SCST, percutaneous intervention can be performed in patients with adequate therapy in combination of vasospastic angina with severe organic stenosis (class IIa) [10, 66]. Currently, the search for new methods continues treatment and diagnosis of VSST. Thus, the possibilities of renal denervation to reduce the tone of the sympathetic nervous system are being actively studied in people with arterial hypertension, life-threatening cardiac arrhythmias [80]. The use of a Holter smartphone, loop recoders, smartphones with the provision of a study of the function of the cardiovascular system and the possibility of remote analysis, which will determine the further tactics of patient management, are considered promising [56, 81, 82]. Finally, the question of the use of intravascular brachytherapy is being studied in case of restenosis of a drug-coated stent [83]. In patients with BCC and pain syndrome without lethal arrhythmias with adequate drug therapy, the prognosis is favorable (98% survival and 92% survival after significant cardiovascular events [cardiac death, non-fatal MI, hospitalization for unstable angina and heart failure]) during 5-year follow-up [57]. At the same time, the survival rate of patients after cardiac arrest at the prehospital stage compared to patients who survived in hospital conditions is significantly lower (72% vs. 92% after 5 years, $p<0.001$) [20]. Moreover, the potential risk of recurrent seizures SCST (from 3.9 to 18.6% of cases) exists for a long time after diagnosis and is often underestimated [6, 84, 85]. So, Korean scientists E.M. Lee et al.

The state of chest pain syndrome was assessed for 5 years in 4644 patients with CV (with angiographic stenosis of CA <50% who passed the test with acetylcholine) and showed that long-term use of BCG, nitrates and statins is an important predictor of recurrence of chest pain [86].

Currently, the search for prognostic markers continues. For example, Japanese scientists T. ihei et al. suggest using the determination of Rho-kinase activity in circulating leukocytes to stratify the prognosis in patients with BCST [87].

Thus, despite progress in the study of pathogenesis and in the development of diagnostic methods, a relatively favorable prognosis against the background of ongoing therapy, angina symptoms persist in 1/3 of patients with BCC, which worsens the quality of life of patients and

requires the development of new approaches to treatment and careful clinical and dynamic monitoring.

Prinzmetal angina is one of the malignant functional disorders with a high risk of developing cardiovascular complications, sudden coronary death, including from severe life-threatening ventricular arrhythmia. The available data indicate that SCD is a multifactorial disease.

In addition, since vascular smooth muscle cells are nonspecific, potential triggers of coronary vasospasm can cause angina attacks under various conditions. Therefore, the identification of the FAC is important for determination of the strategy for the treatment of KA lesions. Therefore, the need for further studies of Prinzmetal angina is important both for understanding the pathophysiology of this disorder and for developing new effective methods of treatment and prevention in order to improve the prognosis, quality and life expectancy of patients.

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