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Stress-Induced Cardiomyopathy: Modern Concepts And Neurohumoral Issues Of Pathogenesis

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Abstract: *In recent years, special attention has been paid to the study of a new form of cardiomyopathy – stress-induced takotsubo cardiomyopathy (SCMP). SCMP takotsubo is a new nosological form of acquired CMP, characterized by transient left ventricular (LV) dysfunction in response to physical or mental stress, clinically and electrocardiographically resembling acute coronary syndrome (ACS), described mainly in postmenopausal women. period without signs of ischemic heart disease and characterized by a relatively favorable prognosis.*

Keywords: stress-induced cardiomyopathy, acute sympatical activity, acute left ventricular dysfunction.

Introduction

Stress-induced cardiomyopathy (SCM) takotsubo is a new nosological form of acquired CMP [1], characterized by transient left ventricular (LV) dysfunction in response to physical or mental stress, clinically and electrocardiographically resembling acute coronary syndrome (ACS), described mainly in postmenopausal women. period without signs of ischemic heart disease and characterized by a relatively favorable prognosis [1,3,5,9].

The term "takotsubo" in Japanese means "a ceramic pot with a round base and a narrow neck for catching octopuses in the sea" [45]. It is this form of hypokinesia of the apex with hyperkinesia of the basal LV sections that is observed in SCMP during echocardiographic (EchoCG) studies (Fig. 1.1) [6,7,8].

For the first time SCMP was described in 1990 by Japanese cardiologists H. Sato and N. Yagihara [44,45] as a transient spherical (balloon-like) expansion of the apex of the heart (apical ballooning) with simultaneous hyperkinesia of the basal segments of the LV, accompanied by apical ventricular dysfunction. Previously, similar changes associated with blood transfusion were described by S. Kawai et al. (2007) [39]. For the next decade, SCMP was described only by Japanese cardiologists [8,9,11,43]. The maximum number of observations of SCMP (88 patients) was presented by K. Tsushikashi et al. (2001) [48]. In the European population, the first description of 13 patients with SCMP was made by WJ Desmet et al. (2003) [20].

Materials and methods

In modern medical literature, SCMP has many synonyms: "transient catecholaminergic stunning", "transient spherical (balloon) expansion of the LV apex", "ampullar (amphora-like) cardiomyopathy", "broken heart syndrome" [1,2,10,16]. The term "transient left ventricular dysfunction syndrome" seems to be the most correct term for most researchers of the SCMP [12].

Emotional and physical stress, accompanied by an acute ejection of CA [4,10], increased sensitivity of adrenergic receptors (AR), impaired sympathetic innervation of the heart, and autonomic dysfunction due to the stress response of the body [18], are dominant among the possible factors of the onset of SCMP. The cause of the disease and the pathogenesis are not exactly known [20]. It is believed that SCMP is an abortive (interrupted) variant of acute myocardial infarction (AMI) [29,43] or is a rare reversible form of neurogenic CMP [30]. The hypothesis of myocarditis associated with toxic damage to the myocardium of CA [17] is based on the signs of focal myocytolysis, interstitial fibrosis with monocytic infiltration or connective tissue necrosis obtained in some patients, however, in general, refuted by the data of biopsy of the endocardium and myocardium, as well as serological studies [26,33]. There is an opinion that SCMP belongs to metabolic CMP [1,5], and disturbances in energy, carbohydrate, and fat metabolism may be a definite connection between acute stress and myocardial stunning [13,14]. Cases of fatty infiltration of the heart [17] and cardiac steatosis [31] have been described.

Japanese authors propose to consider SCMP as a primary (idiopathic) disease that differs in its mechanism of development from other acute conditions accompanied by the development of transient LV dysfunction, such as stannous myocardium, subarachnoid hemorrhage, pheochromocytoma crisis, acute myocarditis, tachycardic cardiopathy [19,36,37,38,42].

Currently, the following possible theories of the pathogenesis of SCMP are being considered: increased sympathoadrenal activity [12,41,49], catecholamine-induced multiple coronary spasm [37,45], coronary microvascular dysfunction [31], direct cardiotoxic action of CA and catecholamine stunning (stannization) of the myocardium [34,47]. Clinical analysis of the state of the autonomic nervous system in SCMP showed a transient increase in the tone of the sympathetic nervous system with a simultaneous decrease in the parasympathetic division [47].

The leading role is assigned to extreme or prolonged stress and participation in this CA [3,18]. YJAkashi et al. (2003) noted an increase in the level of adrenaline in the blood plasma (maximum in the acute stage) in all four patients with SCMP [9,10]. With

SCMP, significantly higher levels of CA (norepinephrine, adrenaline, and dopamine) in blood plasma are determined than in AMI [2,23]. In a study by ISWittstein et al. (2005) [50] the concentration of epinephrine in the blood of patients with SCMP compared with AMI was 1264 versus 376 pg / mL, and norepinephrine was 2284 versus 1100 pg / mL [50].

The rise in the levels of CA in the blood plasma is a natural evolutionary response of the organism to a sudden shock, fright or danger [1,18]. At physiological and elevated concentrations, norepinephrine released from sympathetic nerve endings acts on cardiomyocytes (CMC) mainly through β1-AR, showing positive inotropic and chronotropic effects [6,40]. Epinephrine also interacts with β1-AR, but it has a greater affinity for β2-AR. The ratio of β1-AR and β2-AR is 4:1 [16,46]. Under physiological conditions, adrenaline has a cardiotonic effect, but at high concentrations it has a negative inotropic effect [10,12]. After reducing the level of adrenaline circulating in the blood to physiological values, CMC restore their inotropic function.

Sympathetic stimulation of AR in the ventricular myocardium is achieved in two ways [6]: local synthesis of norepinephrine in sympathetic nerve endings with direct excitation of the myocardium and the effect of circulating CAs in the blood. It is known that the LV basal region in mammalian and human hearts has the highest density of sympathetic nerve endings [5,6], approximately 40% higher than in the apical region [5,6]. Under physiological conditions, most of the norepinephrine released from nerve endings and secreted by the adrenal cortex does not have a detrimental effect on the myocardium [1,6]. Under stress reaction conditions, the degree of influence of CA circulating in the blood plasma on the myocardium will depend on the local AR density in different areas of the myocardium [8,9].

The density of β-AR is more pronounced in the apical part of the heart, with a decrease in the concentration gradient towards the basal region of the myocardium [8,9], as a result of which the response to the action of CA from the apex will be more pronounced than that of the basal myocardium [8,9]. Such a difference in the ratio of nerve endings and AR in different parts of the heart can be explained by the fact that an increase in the density of β-AR in the apical myocardium is necessary to compensate for the sympathetic innervation, reduced due to the lower density of nerve endings, for an adequate response to stress, in which the main mediator is adrenaline [21,22].

In vivo experiments simulating cardiac arrest upon administration of isoprenaline, it has been shown that the apex of the heart is more sensitive to the action of CA than the basal regions, and is subject to a greater "constrictive" response to stress [15], and preliminary administration of α- and β-blockers normalizes LV dysfunction [24]. When CA levels return to normal, LV function and apical contractility are restored within days or weeks after the stressful event. Norepinephrine in a stress reaction causes a spasm of the coronary vessels. Thus, due to the different density of AR in different parts of the heart, "extra" CAs selectively affect, first of all, the apical region of the LV [15,24].

This leads to transient LV dysfunction, classified as "stanned myocardium with normal coronary blood flow" [23]. With a stunned (stunned) myocardium, the cardiomyocyte does not function due to ischemia or other factors, but remains alive and, under more favorable conditions of blood circulation, restores its functional activity [23,29]. "Stanned myocardium with normal coronary blood flow" [28,36] is a phenomenon common in cerebral pathology (especially in acute subarachnoid hemorrhage – SAH) [19,25,28] and in pheochromocytoma [36], in which, due to excessive sympathetic activation and sharp fluctuations in CA levels reveal acute phase ECG changes, increased levels of cardiac enzymes and acute, but reversible ventricular dysfunction in normal coronary arteries [25,28,36].

CA and their oxidation products also have a direct toxic effect on CMC. Myocardial biopsy of patients with pheochromocytoma and SCMP has similar pathological changes: reversible myocytolysis, mononuclear infiltrates and connective tissue fibrosis [36,37,38].

The role of cyclic adenosine monophosphate (cAMP) in the pathogenesis of SCMP is considered [1]. It is known that cAMP modulates enzymes involved in any oxidative process, and CAs are cAMP modulators. An increase in the level of cAMP causes a decrease in the functioning of the K + -channel, which leads to an increase in depolarization and lengthening of the QT interval, which is often concomitant with SCMP [6]. Receptors that increase the level of cAMP include P1-AR, P2-AR, P3-AR, and A2-adenosine receptors. β2-AR, A1-adenosine receptors, and muscarinic M2 and M3 receptors decrease cAMP levels. Phenotypic features in the distribution of these receptors in the myocardium are a dynamic process and make different areas of the myocardium more or less sensitive [5,6].

Main part

According to the hypothesis explaining the development of CT, mainly in postmenopausal women, the decrease in the level of estrogen after menopause can be considered as the root cause of SCMP [35]. It is known that the basal plasma levels of adrenaline are lower in women than in men [35], and that estrogens have cardioprotective effects that prevent acute myocardial injury [35]. Gender-related differences in the response of the adrenal cortex to sudden, high-intensity sympathoadrenal activation and gender differences in epinephrine pharmacokinetics could explain the increased representation of women in SCMP [35,37]. T. Ueyama et al. (2007) found that female rats exposed to stress and not receiving estrogens develop SCMP significantly more often than rats receiving estrogens.

In experimental models, stress activates premature gene expression both in the central nervous system and in the ventricular myocardium, while estrogens reduce these changes in gene expression and prevent apical ventricular dysfunction, partially preventing the stress-induced hypothalamic-sympathoadrenal response [45]. In in vivo experiments, stress-induced SCMP was prevented by blockade of α- and β-adrenergic receptors, and estrogen supplementation reduced changes in the heart and brain

structures with the representation of central sympathetic neurons and neurons with immunoreactive estrogen receptors in the hypothalamic nuclei. Estrogens also regulate the levels of cardioprotective substances such as heat shock protein and atrial sodium uretic peptide [14,22].

It is known that the density of β1-ARs after oophorectomy increases and that β2-ARs of smooth muscle cells are more sensitive than in men [37]. Apparently, estrogen deficiency can disrupt the ratio of β1-AR and β2-AR, then the cardiotoxic effect of CA is manifested in the area of the highest β-AR density. A case of SCMP associated with estrogen deficiency in Turner syndrome in the postpartum period has been described [29].

According to the hypothesis of multivessel spasm, several simultaneous spasms of the coronary arteries can lead to transient impairment of microcirculation [22,27]. However, intracoronary administration of acetylcholine is accompanied by coronary spasm in only 1 of 13 patients [36]. According to another version, microvascular vasospasm and microvascular dysfunction, which are not visible in coronary angiography, lead to insufficient oxygen supply to the myocardium [32]. Contributing factors are also considered hypovolemia, adrenergic stimulation and some anatomical features of the heart: S-shaped structure of the interventricular septum, small diameter of the LV outflow tract and a smaller LV volume [25,44,45,49].

The true prevalence of SCMP is unknown. It is believed that in 0.7-2.5% of cases, SCMP is the cause of the misdiagnosis of AMI [43]. In Japan, SCMP is diagnosed in 1.7-2.2% of patients with ACS [9,10]. In the United States, 2-2.2% of patients with ACS are subsequently diagnosed with SCMP [33,34]. M. Gianni et al. (2006) [29], using the search engines MEDLINE (from 1966 to August 2005) and EMBASE (from 1980 to August 2005), analyzed 286 patients with SCMP aged 10-89 years (only 2.7% were persons under 50 years). SKMP accounted for 2% of the ACS. Women predominated (88.8%) [29].

The analysis of the largest cohort of patients with SCMP in Europe is presented by Swiss authors who studied 13715 coronary angiographies and 2459 patients with ACS over 3 years, which made it possible to determine the incidence of SCMP among ACS in 1.7% and in 0.3% of performed coronary angiographs [30,32]. The average age was 65 years, women predominated (85%) [30]. A good forecast and a low probability of event repetition were noted [30]. According to Italian researchers [30,41], SCMP occurs in 1% of patients with ACS (a total of 2233 patients with ACS were studied from 2001 to 2006). All patients were women; at the age of 76 ± 7 years, 82% experienced stress in the previous days, half had chest pain and shortness of breath [30]. A favorable prognosis was noted [30]. Retrospective analysis of 26593 coronary angiographies performed in the period 2003-2006, allowed German authors to identify SCMP in 0.08% of cases [33]. The average age was 68.4 years, women predominated (90.5%) [32].

Mental and physical stress is the main triggering factor for SCMP. The development of SCMP after treadmill, hypothermia, lightning, after intercourse has been described [1,5,6]. Mental stress plays a decisive role in the development of SCMP [1,6,9,45]. Strong psychological experiences, such as the death of a loved one, bad financial news, legal problems, natural disasters, health problems, reliably precede the development of "broken heart syndrome" [9,45]. An increase in the frequency of the syndrome has been noted after earthquakes and shipwrecks [9,45].

Other stressful circumstances include acute neurological diseases [28,38], urgent states [2,12], surgical procedures [41], imbalance of CA [49] and estrogens [37,44]. Currently, SCMP, as a relatively new cardiological nosology, is undergoing a period of intensive description in a wide variety of conditions and diseases [16,18,48]. According to the data available in the literature, SCMP is observed in such urgent conditions as pneumothorax [27], pneumopericardium [27], sepsis [1], hemodialysis [6], thyrotoxic crisis [1], status asthma, artificial ventilation of the lungs, status epilepticus [15], hyperglycemic hyperosmolar coma [41], hypoglycemia [41], acute alcohol intoxication, cocaine and opiate poisoning [13], opiate withdrawal syndrome in the treatment of osteoarthritis [44].

SCMP is observed in neurological patients with craniocerebral trauma, meningitis and epidural abscess, encephalitis, cerebral infarction, with ischemic, hemorrhagic and cardioembolic stroke, with SAH [49]. SCMP has been described in patients with epilepsy [25]. There is an opinion that patients with epilepsy, as well as with major ischemic or hemorrhagic stroke, represent a risk group for developing SCMP [11]. Apparently, there is a friendly pathophysiological mechanism of dysregulation of cerebrocardial disorders [11]. The literature describes cases of SCMP with metastasis to the brain, amyotrophic lateral sclerosis, hereditary motor and sensory neuropathy, angiopathy, myasthenia gravis, leukodystrophy [11,19,25,28,38].

SCMP occurs in the mentally ill with disorders of consciousness, anorexia nervosa, neuroleptic malignant syndrome, during electroconvulsive therapy, as well as in patients after neurosurgical operations [11,15,19,25,28,44]. SCMP is observed during local and general anesthesia [6], during many surgical operations and in the postoperative period, including cardiac surgery and organ transplantation [29,38,41].

Cases of SCMP in patients with endocrinological pathology have been described: in Addison's disease (hypocorticism), adrenocorticotropic hormone deficiency, in excess of cortisol, in pheochromocytoma, thyrotoxicosis, hyperthyroidism, autoimmune polyendocrine syndrome, such as bronchial asthma, paraganglioma, esophageal cancer, Turner syndrome, myopathy, immunodeficiency syndrome [36,37,49].

In cardiology, SCMP is described in acute pericarditis, Dressler's syndrome, cardiac sarcoidosis, hypertrophic CMP, in ventricular septal defect, Kounis syndrome, systemic lupus erythematosus, long QT syndrome, atrioventricular block, radiofrequency ablation of the atrioventricular [22,24,25,27]. A case of the development of SCMP in a young woman with sporadic prolongation of the QT interval after syncope caused by torsades de pointes has been described [42].

Drug-induced SCMP is observed during treatment with adrenaline, dobutin, amphetamine, disopyramide, antidepressants,

some antibiotics (levofloxacin), as a result of chemotherapy [1,5,6,8,12]. A recent large systematic review has shown that patients with SCMP have lower levels of traditional cardiac risk factors such as hypertension, hyperlipidemia, diabetes, smoking, or a family history [23].

The disease occurs mainly in elderly and senile females, however, there have been cases of SCMP in young women [1,7,5], adolescents, including a boy and a two-year-old girl [1,5]. SCMP is found in obstetric practice: in early pregnancy, during ectopic pregnancy and in the postpartum period [4,5].

Reviewed by W.J. Desmet et al. (2003) [20], the majority of patients with SCMP were Asian (57.2%) or "Caucasian" (40%) nationality [20]. However, given the fact that SCMP is a relatively new concept in cardiology practice, an even national representation should be expected. There is a report about the SKMP in a Russian woman [35].

Apparently, heredity has a certain importance for the development of CT, since only a minority of postmenopausal women develop SCMP [1,7,45]. A case of SCMP was described in a 44-year-old woman whose mother at the age of 49 had an “atypical” AMI. Analysis of the medical history, coronary angiography and ventriculography of the patient's mother allowed the authors to retrospectively diagnose her SCMP [39].

Thus, SCMP is a rare idiopathic disease classified by modern cardiology in the framework of acquired CMP, which, due to hypercatecholaminemia, develops a state of “stunnedness” of a certain part of the myocardium, which gives an infarct-like clinical, electrocardiographic, echocardiographic and biochemical picture.

Conclusion

In foreign countries, publications on this issue are few and carried out mainly in the genre of reviews. The Russian medical literature describes clinical observations of SCMP in patients with arterial hypertension and in young people with heart failure. Unfortunately, such clinical cases of SCMP have not been described in Uzbekistan.

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