ОГЛЯД ЛІТЕРАТУРИ

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Modern Problems of Stress and Mental Disorders – Anxiety and Depression

Abstract. The review highlights researches that concern the problems of excessive stress and mental disorders, anxiety and depression in cardiac patients. A close relationship between these conditions and cardiovascular pathologies is shown, including the impact on the balance of nutrients, platelet activity, inflammation, alterations in immunological system, the hypothalamic – pituitary – thyreoid – adrenal axis and increased activity of the nervous system, which act as large triggers of cardiac events.

Key words: excessive stress, anxiety, atrial fibrillation, depression, adaptation.

In times of scientific-technical progress and rapid development of scientific disciplines [1], the problem of psychosocial stress and associated anxiety and depression acquires great clinical significance. After all, they are recognized as independent risk factors of circulatory system diseases [2]. These conditions increase the risk of adverse cardiovascular events – sudden circulatory arrest, myocardial infarction, arrhythmias, hemorrhagic and ischemic stroke, and also complicate the course of already available diseases. The most frequent manifestations of mental violations of the mental sphere, which occur in the practice of the doctor-intern, are anxiety and depression [3, 4]. The depression states are clinically met at an average of 20% of patients, and in the use of formalized diagnostic scales and questionnaires this figure increases from 45 to 56% [4, 43]. The prevalence of depressive spectrum disorders in Ukraine in 2008 to 2012 increased from 65.37 to 73.6 persons, and the incidence from 8.74 to 9.06 per 100 thousand of population. Their frequency in general medical practice reaches 22-33% [44]. It is proven that depression is common among patients with cardiovascular disease (CVD), coronary heart disease (CHD), and contributes to deterioration in functional and cardiovascular outcomes [4, 6, 43]. It worsens the ability of patients to the treatment, reduces the quality of life and social adaptation affects the course of the post-infarction period and leads to earlier mortality [5, 6, 7]. Depression is considered as an independent risk factor for progression of coronary heart disease, angina pectoris, heart failure (HF), myocardial infarction and cardiovascular mortality [7, 8]. Distribution of depression is 17-27% of hospitalized patients with CHD. The majority of studies have also demonstrated an adverse prognostic effect in patients with comorbide depression [6]. Many studies conducted over the past few years have confirmed that depression and anxiety are related with adverse cardiovascular consequence independently of traditional risk factors [9, 10, 11]. Among them, multinational clinical study EUROASPIRE III Study included patients with CHD from 22 countries. They are assessed using hospital scale of anxiety and depression (HADS). The prevalence of depression (HADS - depression "8") varied from 8.2% to 35.7% in men and from 10.3% to 62.5% in women, and the prevalence of anxiety (HADS-dial alarm "8") varied from 12.0% to 41.8% in men and from 21.5% to 63.1% in women. Depression and anxiety were more often observed in women than in men. Their frequency increased with age and decreased with higher levels of education. There was no connection between the scale indices of depression and anxiety and blood pressure (BP), but identified a tendency to more frequent smoking, low physical activity, dyslipidemia and diabetes mellitus [12, 13, 14]. Anxiety has been associated with sudden death, risk of myocardial infarction and a low quality of life [15, 16, 17, 18, 19]. In addition, it is clarified that anxiety is correlated with sympathetic nervous system activity and endothelial dysfunction and increases the risk of atherosclerosis and CVD in general [13, 20]. Formation of depression and anxiety has a number of pathogenetic factors. So, Greden and Stephenson found that the need of methylated xanthines (caffeine) can cause symptoms of diffuse anxiety, as well as insomnia. Their action is based on the ability to stimulate a psycho-physiological stress reaction (SR), first of all through the sympathetic activation [21, 22]. There are few behavioral and physiological mechanisms that strengthen intercommunication between depression and diseases of heart. Among them change of activity of thrombocytes, that testifies the strengthening of their reaction on physiological stress and can cause the unfavorable ischemic events of coronal arteries [6, 8, 13, 23, 28]. For patients with CHD plasma concentrations of thrombocyte factor of IV (PF - 4), b- thromboglobulin (b - TG), markers of intermediate steps in the cascade of rolling up of thrombocytes is higher at presence of depression, than for patients without it [6, 13, 25]. It is proved that inflammatory cytokines, in particular C-reactive protein (C-RP), interleukin-1 (IL-1), interleukin-6 (IL-6) and tumor necrosis factor alpha (TNF-&), which are the risk factors for cardiovascular mortality, are released in depressed patients in damage of endothelial dysfunction [6, 24, 25]. For such patients changes take place in the immunological system and increase of activity of the sympathetic nervous system that is related to development of metabolic syndrome and arrhythmias [26]. There are proofs that patients with depression in a greater measure are apt to the well known risk of cardiovascular pathologic factors, less apt to the changes of unfavorable way of life, and less amenable to cardioprotective medications [27, 28]. Aim-analysis, conducted by Walsin and Singal showed that even after control over traditional risk factors (hypodynamia, smoking, arterial hypertersion) the presence of the depressed symptoms is a meaningful independent risk in the origin of CHD. The American cardiologic association recently published recommendations for exposure and treatment of depression for patients from CVD, and proves that early recognition and treatment of depression for patients from CVD provide exact diagnostics and effective treatment [6, 28, 29, 30, 37]. Examination of cardiologic patients with the use of questionnaires of health of patient PHQ - 2 and PHQ - 9 allows reveal depression in this category of patients [31, 32, 33]. The depressed reactions arise up also, as one of psychological displays of excessive stress [22]. Yes, stress is examined, as an adaptive reaction of organism that develops in reply to a threat and results in violation of homoeostasis [1, 2, 42]. A stress reaction is activated by the systems accountable for mobilization of energy and resources necessary to overcome this homoeostatic violation. Herein an important role is played by a hypothalamic – pituitary – tirion – adrenal and thyroid axis and sympathetic adrenomedular system [22, 34, 36]. In realization of stress reaction a heterospecific stimulus influences on the neurosecretory mews of middle hump of hypothalamus, excreting corticotropin-releasing-hormone (CRH) [1, 38], that then causes the discharge of adrenocorticotropic hormone (ACTH) from a front fate to the hypophysis that stimulates the bark of adrenals [1, 34], distinguishing glucocorticosteroids (GCS) [1, 3, 22, 40, 41]. They, in their turn, stimulate neoglicogenesis, that provides an organism with the supplies of ready-to-use energy necessary

for adaptation in a stress situation [1, 39, 40]. It is important to remember, that activating of every axis can mutually recover the effects of other axes. These mechanisms and axes can not jointly lead to an action every time, when man runs into stress. Most obvious is circumstance that no stressor causes a sympathetic or parasympathetic effect. All possible axes of stress reactions can not be activated at once for the same individual. However, question about a method that chooses the organism of man the special character on what organ and through what axis a stress reaction influences it, remains open for reflections [22]. It is known that due to hormones (ACTH, CRH, GCS) a negative feed-back is provided, in an order to overcome a reaction on stress and turn an organism to the homoeostasis [34, 40, 42]. From the other side, if the action of stressor will proceed, then in a result energy of adaptation, that are adaptive mechanisms that participate in support of the stage of resistance will exhaust itself, and it will result in exhaustion [22, 39]. And greater part of organs will yield to atrophic and degenerative changes [1]. The cardiovascular system is considered by many researchers and clinicians as the basic eventual organ of stress reaction. Essential high blood pressure and arrhythmias also belong to cardiovascular disorders that are most often associated with excessive stress [22]. Blood pressure rises due to the vessel-narrowing action of epinephrine that is distinguished by the cerebral layer of adrenals. In addition, the final result, that may stimulate the increase of blood pressure, can be through the changes of tendency of carotid sine and baroreceptors of aorta or irritation of completions of the depressed nerve that is in the wall of arc of aorta [22, 41]. Psychological stress changes potential of action of cardiac tissue in the organism of a man that results in electric instability that can be the result of origin of arrhythmias [34, 35]. It is important to remember for a clinician, that in a patient's life a greater part of excessive stress is initiated and produced by himself. Nevertheless a patient chooses the method of stressful stimulation (meal, drink, smoking) [22, 30]. Levi proves that there are sympathomimetic substances that can cause SR simply by means of their inclusion in the exchange processes of organism are not involving the higher interpretation mechanisms of brain here, but only more lower sensory mechanisms and mechanisms of digestion [22]. It is assumed that at a stress reaction the supplies of vitamin C in an organism are exhausted, and necessities of organism, increase respectively (Seley, 1976). Results of Hodges, Glerer, Cottsealh (1970) testify that outflow of ascorbic acid with urine in a person increases at the action of stressors. Finally, there are data that SR can cause exhaustion of supplies of group B vitamin (Moanteastle) and the vitamin B_6 and Zn (Preiffen). Thus, a stress reaction is nevertheless related to responsibility of individual for a choice from the consequences of which he suffers from [22, 42].

Conclusions

1. Modern methods of diagnosis of depression and anxiety allow us timely diagnose them and provide appropriate medical care.

2. An answer of the organism for a stress reaction in the conditions of depression and stress determines the course of cardio-vascular pathology that stipulates the necessity of development of diagnostic and curative program of such patients' management.

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Received: 15.05.2017
Revised: 15.06.2017
Accepted: 15.06.2017