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ABSTRACT

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THE LINK BETWEEN POST-TRAUMATIC STRESS DISORDER AND CARDIOVASCULAR EVENTS DEVELOPMENT (LITERATURE REVIEW)

Post-traumatic stress disorder (PTSD) is a complex illness that occurs as a result of exposure to extremely traumatic events such as war, violence, and the threat of death. The presence of symptoms such as intrusive memories of a traumatic event, anxiety, mood swings, irritability, outbursts of anger, depression, sleep disturbances, and nightmares for a month or more are diagnostic criteria that confirm this diagnosis.

The aim of the study is to find out the pathophysiological mechanisms of PTSD development and its impact on the cardiovascular system, as well as the pathogenetic links that combine the development of PTSD with the development of cardiovascular diseases.

Materials and methods. The authors have processed the results of scientific publications in the world literature on the problems of diagnosis, symptoms, and pathophysiological mechanisms of PTSD development, as well as its connection with the occurrence of the most common cardiovascular diseases, which will deepen the understanding of these problems and improve the approach to diagnostic and therapeutic methods.

Results. It has been established that PTSD is closely associated with impaired autonomic regulation, decreased activity of the parasympathetic nervous system, increased tone of the sympathetic nervous system, and activation of inflammatory processes. PTSD-related disorders cause an increased risk of developing hypertension, coronary heart disease, as well as conditions such as type 2 diabetes, metabolic syndrome, autoimmune and neurodegenerative diseases. Morphological changes in brain structures, such as the hippocampus, amygdala, and prefrontal cortex, confirm the profound neurobiological

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impairment in PTSD. These findings highlight the necessity of early diagnosis, a multidisciplinary approach to treatment, and the development of prevention strategies among populations vulnerable to PTSD.

Conclusions. Post-traumatic stress disorder (PTSD) significantly affects the functioning of the nervous and cardiovascular systems, contributing to the development of various chronic diseases. The identified neurobiological disorders, including changes in brain structures, confirm the deep pathological processes associated with PTSD. The findings underscore the importance of a comprehensive approach to early diagnosis, treatment and prevention, which will reduce the risk of developing serious comorbidities.

Keywords: Post-traumatic stress disorder, cardiovascular diseases, autonomic dysfunction, hypothalamic-pituitary-adrenal system, cardiovascular risk.

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ЗВ'ЯЗОК МІЖ ПОСТТРАВМАТИЧНИМ СТРЕСОВИМ РОЗЛАДОМ ТА РОЗВИТКОМ СЕРЦЕВО-СУДИННИХ ПОДІЙ (ОГЛЯД ЛІТЕРАТУРИ)

Посттравматичний стресовий розлад (ПТСР) є складним захворюванням, що виникає внаслідок впливу надзвичайно травматичних подій, таких як війна, насильство та загроза смерті. Наявність таких симптомів як нав'язливі спогади про травмуючу подію, тривожність, різкі зміни настрою, дратівливість, спалахи гніву, депресія, порушення сну, поява кошмарів протягом місяця і більше є діагностичними критеріями, що підтверджують даний діагноз.

Мета дослідження – з'ясувати патофізіологічні механізми розвитку ПТСР та його вплив на серцево-судинну систему, а також патогенетичні ланки, що поєднують розвиток ПТСР із розвитком серцево-судинних захворювань.

Матеріали та методи. Авторами опрацьовано результати наукових праць зі світової літератури, що стосуються проблем діагностики, симптоматики, та патофізіологічних механізмів розвитку ПТСР а також його зв'язок з виникненням найбільш поширених серцево-судинних хвороб, що дозволить поглибити розуміння даних проблем та покращити підхід до діагностично-терапевтичних підходів.

Результати. Встановлено, що ПТСР тісно пов'язаний з порушенням вегетативної регуляції, зниженням активності парасимпатичної нервової системи, підвищенням тонусу симпатичної нервової системи та активізацією запальних процесів. Розлади, пов'язані з ПТСР, спричиняють підвищений ризик розвитку артеріальної гіпертензії, ішемічної хвороби серця, а також таких станів, як діабет 2-го типу, метаболічний синдром, аутоімунні та нейродегенеративні захворювання. Морфологічні зміни в структурах мозку, таких як гіпокамп, мигдалина і префронтальна кора, додатково підтверджують глибокі нейробіологічні порушення при ПТСР. Отримані результати

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підкреслюють необхідність ранньої діагностики, мультидисциплінарного підходу до лікування та розробки стратегій профілактики уразливих до ПТСР груп населення.

Висновки. Посттравматичний стресовий розлад (ПТСР) суттєво впливає на функціонування нервової та серцево-судинної систем, сприяючи розвитку різних хронічних захворювань. Встановлені нейробіологічні порушення, зокрема зміни у структурах мозку, підтверджують глибокі патологічні процеси, пов'язані з ПТСР. Отримані дані підкреслюють важливість необхідність комплексного підходу до ранньої діагностики, лікування та профілактики, що дозволить знизити ризики розвитку серйозних супутніх захворювань.

Ключові слова: Посттравматичний стресовий розлад, серцево-судинні захворювання, вегетативна дисфункція, гіпоталамо-гіпофізарно-надниркова система, серцево-судинний ризик.

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INTRODUCTION

Post-traumatic stress disorder (PTSD) is a serious and debilitating illness that develops as a result of exposure to life-threatening or traumatic events, such as participation in hostilities, the threat of death, violence or being under the threat of violence. According to the recommendations of the American Psychiatric Association, which were set out in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), the symptoms accompanying the development of PTSD were grouped into appropriate categories. Symptoms include frequent memories of the traumatic event, intrusive thoughts, sleep disturbances, nightmares, anxiety, symptoms of increased nervous excitement, mood lability, irritability, outbursts of anger, depression, avoidance of activities associated with memories of the traumatic event [1, 2, 3]. The diagnostic criterion is the presence of these symptoms for 1 month or more. To assess the severity of PTSD symptoms, the clinically administered 5th edition scale is used (CAPS 5) [4].

Post-traumatic stress disorder is a widespread illness and occurs among both civilians and the military. It has been found that up to 20% of veterans and almost 8% of civilians suffer from lifelong PTSD symptoms [5]. A study of the prevalence of PTSD among US residents revealed the following data. Among civilians, the incidence rate ranged from 2.3 to 9.1% per year, and from 3.4% to almost 27% over the course of a lifetime. In the case of military officers, 6.7-50.2% were diagnosed with PTSD during the year and 7.7-17.0% during their lifetime [6]. There is evidence that being female is associated with a higher risk of PTSD developing. According to a study conducted in the United States, the lifetime incidence of PTSD in men was up to 6%, while among the female population, this figure reached 13% [3, 7]. The prevalence of post-traumatic stress disorder among residents of the United Kingdom was almost 8%, South Korea – almost 27%, Lebanon – 33.3%, and the Republic of China – more than 53% [8].

The analysis revealed a trend towards a progressive increase in the incidence of PTSD in recent years. Given the existing and potentially new military conflicts and wars, natural tragedies and the number of precedents of violence among people, scientists predict a further increase in the number of patients with PTSD [5]. Ukraine has also faced such challenges, as the number of diagnosed cases of PTSD has increased significantly due to the war, both among civilians and military members. According to the National Health Service of Ukraine (NHSU), the incidence of PTSD in the first two months of 2024 was almost the same as in the whole of 2021, and in 2023 it was four times higher than in 2021. However, it is not yet possible to establish the exact number of people who will need medical care, and the real extent of the consequences of the hostilities can only be assessed after the war is over.

AIM

The aim of this study was to investigate the mechanisms of posttraumatic stress disorder development, as well as the pathophysiological pathways that combine the development of PTSD with cardiovascular disease and mutually aggravate symptoms and clinical course.

MATERIALS AND METHODS

The authors have analysed the literature on pathophysiological mechanisms that play an important role in the development of post-traumatic stress disorder and may be involved in the progression and consequences of cardiovascular disorders.

RESULTS AND DISCUSSION

Post-traumatic stress disorder leads to serious health consequences and affects a large number of organs and systems. There are studies that confirm the impact of PTSD on the increased risk of arterial hypertension and coronary heart disease. Not all the mechanisms that lead to an increase in blood pressure and the subsequent risk of cardiovascular events are known. However, the performed research confirms the role of inflammatory processes and disorders of vegetative balance. During a state of rest, a decrease in the level of activity of the PNS (parasympathetic nervous system) is registered, and in contrast, an increase in the activity of the SNS (sympathetic nervous system) in times of stress [5, 9]. Epidemiological studies have shown an increased risk of cardiovascular disorders among people with diagnosed PTSD. Cardiovascular diseases are multifactorial and the mechanisms of their development may include dysfunction of the autonomic nervous system and an exaggerated response to stress factors [16].

Individuals with PTSD experience changes in brain functioning, namely the appearance of morphological changes in areas that play an important role in the response to stress. These include the prefrontal cortex, amygdala, hippocampus, anterior cingulate gyrus, and insula [14]. Studies have shown that the volume of the hippocampus, which is known to be involved in the formation of memories, is significantly reduced in patients with PTSD [15]. The role of the amygdala is to regulate memory consolidation and generate affective feelings. It has been established that the amygdala plays an important role in the pathophysiological mechanisms of PTSD development, and the extent of its size reduction is associated with the severity of symptoms. A more pronounced response of the amygdala in response to repeated stress factors was found in women compared to men. Perhaps this mechanism is one of the reasons for the more frequent formation of post-traumatic stress disorder in women, despite the same type of traumatic factor [5].

Recent studies have confirmed the link between PTSD and the development of changes and disorders in various organs and systems. These include autoimmune, inflammatory diseases such as rheumatoid arthritis; metabolic and neuroendocrine disorders (diabetes mellitus, metabolic syndrome); disorders related to the central nervous system, such as sleep disorders and dementia, as well as cardiovascular diseases, including arterial hypertension and the consequences of atherosclerotic vascular disease, such as coronary heart disease and cerebrovascular disease [11, 12, 13].

It is believed that one of the driving mechanisms that lead to changes in the reactivity of the autonomic system is impaired of the arterial baroreflex sensitivity (BRS), which has been confirmed among military veterans diagnosed with PTSD [10]. The study of the correlations between the severity of symptoms characteristic of PTSD and the risk of cardiovascular events, the state of the autonomic nervous system, and the development of inflammatory changes led to the following conclusions. It has been established that an increase in resting heart rate, increased dysfunction of arterial baroreflex sensitivity, a decrease in the level of activity of the parasympathetic autonomic system under stress, and an increase in indicators indicating the development of inflammatory changes are associated with a more severe course of PTSD symptoms [9].

Post-traumatic stress disorder is a significant risk factor for cardiovascular disease, as evidenced by impaired functioning of the hypothalamic-pituitary-adrenal and autonomic systems [17, 18]. Regulatory dysfunction of these systems can lead to the development of atherosclerotic vascular changes, arterial hypertension, thromboembolic venous conditions and acute cardiac disorders, which can develop in patients with PTSD [19]. The results of a meta-study confirmed the role of PTSD in the occurrence of cardiovascular diseases. PTSD has been recognized as an independent predictor of cardio-vascular disease, and controlling symptoms of depression and such risk factors for vascular events as blood pressure and lipid profile changes does not reduce the role of PTSD. A 49% increase in the risk of myocardial infarction and hospitalization related to heart disease in people with PTSD has been found [20]. It is known that the disruption of autonomic balance observed in PTSD leads to an increase in the level of basal activity and, as a result, an increasing load on the cardiovascular system. During the development of PTSD, the activity of the sympathetic nervous system increases, as evidenced by an increase in catecholamine production and an increase in heart rate, compared to individuals without PTSD. As a result of a short-term acceleration of the heart rate, hemodynamic changes occur, the effect of shear stress on the endothelium of the vascular wall increases, which can lead to its damage, rupture of the atherosclerotic plaque and, as a result, to cardiovascular or cerebrovascular consequences. Endothelial cell dysfunction is a recognized early sign and threat of further cardiovascular diseases, as it is a key link in the development of the atherosclerotic process,

inflammatory changes and prothrombotic mechanisms [21]. Scientists led by Grenon et. al. studied the association of PTSD with endothelial dysfunction. They found a significant decrease in the level of dilatation processes of the brachial artery in patients with diagnosed post-traumatic stress disorder [22].

The activation of the sympathetic nervous system produces catecholamines, the increased concentration of which has been confirmed in the blood plasma, cerebrospinal fluid, and urine of people with PTSD. After exposure to stress factors, an increase in the level of noradrenaline in the blood plasma was detected. Comparison of the results of urine tests of patients with PTSD and control group without signs of PTSD revealed a significantly higher concentration of noradrenaline in the urine of patients in the main group with PTSD [23]. Increased levels of noradrenaline due to stress can lead to an increase in the production of cytokines, which play an important role in the formation of an inflammatory response [5]. Long-term changes in inflammatory reactions may be the reason for the association of post-traumatic stress disorder with the risk of cardiovascular complications [24]. It is recognized that several proinflammatory cytokines are involved in the mechanisms of development and progression of cardiovascular events [25]. According to the results of the meta-analysis, the blood plasma of patients with confirmed PTSD showed an increase in inflammatory markers such as C-reactive protein, tumor necrosis factor-α, interleukin-1β, interleukin-6, compared to the control group of healthy people without PTSD. It is also reported that the gut microbiome has an important impact on the regulation of the immune response, the development of inflammatory reactions and metabolic changes in individuals with PTSD. A significant association was found between increased plasma concentrations of proinflammatory cytokines in patients with PTSD and exaggerated activation of the sympathetic nervous system and excessive cardiovascular responses to stress, which indicates the role of inflammation, autonomic responses and vascular changes during the development of PTSD [11].

According to studies conducted among the population of different countries, high cholesterol, hyperglycemia, abdominal obesity, and type II diabetes mellitus have been linked to the development of PTSD. In addition, the risk of developing metabolic syndrome was twice as high in patients with PTSD compared to the control group [26]. Dysfunction of glucose regulation, diabetes mellitus and metabolic syndrome are confirmed factors that increase the likelihood of cardiovascular disease [11]. The relationship between PTSD and metabolic syndrome was analyzed. The results revealed a significant association with obesity and confirmed the role of genetic factors. Long-term studies have led to the conclusion that PTSD can cause an increase in BMI, especially among women. Therefore, this may explain the high prevalence of obesity among patients with PTSD (OR = 1.55, 95% CI: 1.32–1.82) [27, 28]. The presence of dysregulation of the limbic system and prefrontal cortex in people with PTSD, as well as high concentrations of stress hormones, leads to an increased desire to consume large amounts of carbohydrates and ultra-processed foods. As a result, an eating disorder is formed with overeating and a subsequent increase in the likelihood of neurodegenerative and inflammatory disorders in people with PTSD. In addition, PTSD and the development of obesity are associated with an increased risk of food and nicotine addictions, attention and sleep disorders, migraines, arterial hypertension, and carbohydrate metabolism disorders with subsequent development of diabetes. Disorders of metabolic processes and cardiovascular reactions in people with PTSD can cause fatal consequences, including irreversible pathological conditions such as dementia and early mortality [29].

There is evidence linking post-traumatic stress disorder to the development and progression of atherosclerotic vascular changes, angina pectoris, ischemic heart disease, and cerebrovascular disorders [30, 31]. According to the results of studies, 25% of people who suffered cerebral stroke developed PTSD, which was a poor prognostic sign for further cardiovascular disorders. Despite the fact that PTSD was first studied mainly among men, PTSD was subsequently found to be associated with ischemic heart disease in both women and men [32]. The study of the level of indicators showing calcification of the coronary arteries in healthy individuals of the control group compared to military veterans of combat operations confirmed a significantly higher level of calcification among veterans, even in the subgroup without signs of coronary heart disease. It is believed that endothelial dysfunction, which is a predictor and early pathophysiological sign of atherosclerotic vascular changes, accompanies the development and course of PTSD [33]. In addition, among patients with PTSD, the development of myocardial ischemic processes associated with stress has been identified, which further increases the risk of developing cardiovascular diseases [30].

CONCLUSIONS

Post-traumatic stress disorder (PTSD) is a complex psychopathological condition that has been confirmed to be a significant predictor of cardiovascular disease by numerous clinical and epidemiological studies. The obtained data indicate a close link between PTSD and an increased risk of developing cardiovascular diseases, such as hypertension and coronary heart disease. The

identified mechanisms include dysfunction of the autonomic nervous system, increased inflammation, disruption of the baroreflex system and morphological and functional changes in brain structures. PTSD is accompanied by a decrease in the activity of the parasympathetic nervous system at rest and excessive activation of the sympathetic nervous system during stressful events, which contributes to an imbalance in autonomic regulation. Disorders of the hypothalamic-pituitary-adrenal and autonomic systems in patients with PTSD lead to excessive stress on the cardiovascular system, causing the development of hypertension, atherosclerosis, thromboembolism and acute cardiac events. In addition, morphological changes are observed in the brain regions responsible for emotional regulation and memory, in particular in the hippocampus, amygdala and prefrontal cortex.

It has been established that the severity of PTSD symptoms correlates with the level of inflammatory burden, so PTSD not only has a psycho-emotional dimension, but is also a serious risk factor for a wide range of somatic diseases, including autoimmune, metabolic, neuroendocrine and cardiovascular disorders.

PTSD has been recognised as an independent risk factor for cardiovascular disease, which does not decrease even when controlling symptoms of depression, blood pressure and lipid profile. Patients with PTSD have increased sympathetic nervous system activity and catecholamine levels, which disrupts vascular endothelial function and contributes to the formation of atherosclerotic plaques. These individuals also have elevated levels of proinflammatory cytokines, which support the chronic inflammatory process associated with cardiometabolic disorders.

PTSD is often accompanied by the development of metabolic syndrome, obesity, diabetes mellitus and eating disorders, which further increases the risk of cardiovascular events. There is also an increased susceptibility to addictions and sleep disorders, which have an indirect impact on cardiovascular health. The endothelial dysfunction observed in patients with PTSD is an important pathophysiological link between the mental state and somatic consequences. PTSD is associated with an increased risk of coronary heart disease, stenocardia and cerebral stroke. These findings highlight the necessity of early diagnosis, a multidisciplinary approach to treatment, and the development of prevention strategies among populations vulnerable to PTSD.

PROSPECTS FOR FUTURE RESEARCH

The pathogenesis of PTSD has been studied to a large extent, but further research should focus on a deeper analysis of the processes that accompany its course, as well as the pathophysiological mechanisms that contribute to cardiovascular disorders. The undoubted influence of genetic factors on the occurrence of disorders associated with stress and traumatic events requires detailed research. The study of epigenetic changes in the development of PTSD will open up new opportunities for understanding pathogenetic aspects and will allow us to identify specific markers of this disorder.

AUTHOR CONTRIBUTIONS

All authors substantively contributed to the drafting of the initial and revised versions of this paper. They take full responsibility for the integrity of all aspects of the work.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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