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Evaluation of levels of stress hormones in patients with atherosclerosis

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ABSTRACT Chronic exposure to stress hormones, including adrenocorticotropic hormone (ACTH) and cortisol, has complex effects on cardiovascular health. Cortisol has anti-inflammatory properties but contributes to AS by altering lipid profiles and vascular function. Chronic stress increases norepinephrine and catecholamine levels, which contribute to hypertension and vascular contraction through the sympathetic–adrenal–medullary system. The renin–angiotensin–aldosterone system also elevates angiotensin II levels, further influencing blood pressure and catecholamine activity. Imbalance of hormones brought about by chronic stress, mainly on the hypothalamic–pituitary–adrenal axis, intensifies the risk for cardiovascular diseases due to a disturbed vascular response and myocardial dysfunction. Socioeconomic development and individual physiological responses may mediate these effects. These mechanisms identify potential therapeutic targets, including stress hormone modulation, for preventing and managing CVD.

KEYWORDS atherosclerosis, adrenocorticotropic hormone, cardiovascular disease, cortisol

1. INTRODUCTION

Atherosclerosis is an inflammatory chronic disease that is responsible for the growth of plaque in the arterial wall, which represents a leading cause of cardiovascular diseases and a public health problem of great magnitude. While traditional risk factors such as high cholesterol, hypertension, and diabetes have long been established, the role of psychosocial stress and associated hormonal changes in the pathogenesis of atherosclerosis has, in recent years, received greater attention [1]. Chronic stress has been established to cause damaging effects on the vascular health with increased stress hormones like adrenaline and cortisol levels in the blood being associated with atherosclerosis initiation and progression process [2]. Such hormonal imbalances due to chronic stress are linked to enhanced levels of blood pressure, lipids, and inflammation, all contributing to plaque development and instability [2]. Stress has also been linked to a higher incidence of atherosclerosis in women, potentially due to its interactions with other risk factors such as diet and smoking [3].

Extensive research has demonstrated that psychosocial stress, a known cardiovascular risk factor, can modulate inflammatory processes involved in the pathology of atherosclerosis [3]. Furthermore, environmental stressors such as noise pollution have been shown to elevate stress levels, leading to increased atherosclerosis risk [2]. Profiling the levels of stress hormones in patients with atherosclerosis may provide valuable insights into the underlying mechanisms and contribute to the development of more targeted therapeutic approaches.

2. RISK FACTORS OF ATHEROSCLEROSIS

Certain risk factors and cardiovascular inflammation are connected throughout the pathophysiological processes leading to the development of atherosclerosis. Some classical risk factors are largely nonmodifiable. Being male, for example, is one such classical risk factor for CVD risk, and recent studies link at least part of its association with increased vascular inflammation in both animals and humans [4].

Aging is yet another independent risk factor for CVD; biological aging is associated with various inflammatory processes such as increased oxidative stress or more circulating inflammatory cytokines [5]. Clonal hematopoiesis, the process in which increasing numbers of white blood cells derive from one single clone due to specific somatic mutations in hematopoietic stem cells [6], has recently been identified as a pathophysiological mechanism that links aging with atherosclerosis [7]. In atherosclerotic mice, clonal hematopoiesis resulted in a stronger activation of inflammatory pathways and thus accelerated atherosclerosis progres-

sion [8].

Hereditary factors also contribute to the risk of CVD, and a variety of genetic loci have been associated with CVD in large-cohort, genome-wide association studies [9]. Many of these loci appear to be associated with genes that are components of inflammatory pathways [10]. In addition to an intrinsic genetic component to atherosclerosis itself, some classical risk factors like high levels of cholesterol, arterial hypertension, and diabetes also have some degree of genetic predisposition [11], [12].

As outlined above, high cholesterol levels contribute to the initiation and progression of atherosclerosis by acting as an important proinflammatory stimulus. Of note, obesity and unhealthy diet, which are also considered classical risk factors for atherosclerosis [13]–[15], strongly promote high cholesterol levels [16]. Arterial hypertension is another classical risk factor and acts on the integrity of the endothelial cell layer [17]. Ongoing research is intensively investigating the link between atherosclerosis and diabetes, with the general proinflammatory milieu in diabetic patients most likely being a major player in the development of atherosclerosis [18], [19].

Smoking is yet another classical risk factor for cardiovascular inflammation and promotes atherosclerosis via both local and systemic proinflammatory effects, for example, by circulating toxic compounds and increasing hematopoietic stem cell proliferation [20]–[22]. Elevated hematopoiesis contributes to atherosclerosis progression by enhancing inflammatory leukocyte supply [23], [24].

It has become increasingly clear that apart from classical risk factors, additional lifestyle factors and other coexisting pathologies influence CVD. Many of these risk factors have only recently been identified and are thus referred to as nonclassical or nontraditional risk factors. Both viral and bacterial respiratory infections create a higher risk for MI not only during the acute infection but also in the postinfection phase [25], [26].

Acute increases in cardiovascular (CVD) events may result from elevated procoagulant activity, while long-term effects exacerbate vascular inflammation, promoting atherosclerosis progression. Sterile inflammatory conditions like prior myocardial infarction (MI), stroke, or rheumatoid arthritis, as well as nonsterile inflammation, increase cardiovascular risk by elevating proinflammatory leukocyte production in the bone marrow—a mechanism shared by risk factors such as sedentary lifestyles and sleep deprivation [27], [28].

Regular exercise reduces CVD risk through metabolic, antihypertensive, and anti-inflammatory effects. Studies show physical activity lowers leptin levels, induces bone marrow quiescence, and limits proinflammatory leukocyte supply, reducing cardiovascular inflammation and atherosclerotic plaque size [29], [30].

Poor sleep quality similarly raises CVD risk by increasing bone marrow leukocyte production [31]. Sleep fragmentation reduces hypocretin, a hormone that normally limits myeloid cell production, thus promoting cardiovascular inflammation and atherosclerosis [32], [33].

Environmental factors such as air, light, and noise pollution also affect CVD health. Air pollution directly affects inflammatory pathways, whereas noise pollution indirectly increases risk by affecting sleep and stress [34]. Both promote cardiovascular inflammation through mechanisms such as cytokine production, endothelial dysfunction, and leukocyte infiltration [35], [36]. However, the exact pathways of these effects are still under investigation.

3. CHRONIC STRESS EFFECTS ON INFLAMMATION

Inflammation is a pathological process characterized by injury or destruction of tissues caused by a variety of cytological and chemical reactions. The typical signs of inflammation are pain, heat, redness, swelling and loss of function, and inflammation is related to chronic stress [37], [38]. Research shows that inflammation is strongly related to endothelial dysfunction, a preface to AS and thrombotic disease [39], [40]. Inflammatory reactions are generally considered the main causes of AS, and the influence of mononuclear cells, different subtypes of lymphocytes, neutrophils, and other immune and inflammatory cells on the pathological process of AS has been widely studied. However, in chronic stress, inflammation plays a critical role in the pathological process of AS. It is well-known that chronic stress reduces the activity of the hypothalamic–pituitary–adrenal axis and stimulates the sympathetic adrenal medulla, causing an increase in the production of inflammatory cytokines [41], [42]. Symes et al. hypothesized that chronic stress would result in higher alterations in the levels of proinflammatory factors and cell adhesion molecules; however, they observed that the interventions had only a moderate effect on vascular cell adhesion molecule-1 (VCAM-1) [43]. VCAM-1, a member of the immunoglobulin gene superfamily, is mainly expressed in vascular endothelial cells, and its ligands are $\alpha 4\beta 1$ (VLA-4) and $\alpha 4\beta 7$. Its interaction with VLA-4 is involved in the inflammation induced by leukocytes and improves the pathological process of AS [44], [45].

Research shows that the intercellular adhesion molecule-1, acute phase reactant C-reactive protein and proinflammatory cytokine interleukin-6 were significantly elevated in chronic stress-treated apolipoprotein E in knockout mice than in untreated animals [46]. Other inflammatory signals are found in the plasma cluster of differentiation, interleukin-8, 5′-nucleotide ecto, programmed death ligand 1 and plasminogen activator inhibitor PAI-1 [47], [48]. In addition, chronic stress alters the sympathetic and vagal nervous systems homeostasis. Attenuation of the vagal tone contributes to a proinflammatory status, which can help to promote neurotransmitter regulation, particularly the spread of serotonin activation. For example, stress enhances the levels of plasma dipeptidyl peptidase-4 activity and weakens the concentration of plasma glucagon-like peptide-1 and both plasma and adipose adiponectin [49], [50].

4. THE ROLE OF CHRONIC EXPOSURE TO STRESS HORMONES IN CVD

A hormone is defined as a chemical substance that has a specific regulatory effect on the activity of a certain organ or organs. Chronic stress may affect quality of life [51]; however, the role of the stress-related adrenocorticotrophic hormone (ACTH) and cortisol in AS remains to be clarified. Some researches suggested that ACTH and cortisol influence the development of atherogenesis by regulating vascular endothelial action such as driving circulating monocytes to the vascular wall and causing them to disintegrate into macrophages or by controlling the production of inflammatory interleukins [52]. Whereas corticosterone is an anti-inflammatory hormone, it could promote AS in arteries, with increased dyslipidemia [53]. Moreover, one study demonstrated that the level of norepinephrine was increased in a chronic stress experimental group [54]. Thus, these hormones may become a new direction for the treatment and prevention of cardiovascular and cerebrovascular diseases.

Support for this suggestion is provided by the fact that rosiglitazone is connected with the content of cyclic corticosterone; however, experimental data indicate that rosiglitazone does not prevent the occurrence of chronic cardiac angiopathy [55]. In addition, one study showed that the concentrations of cortisol and catecholamines were correlated with socioeconomic development levels, although there was no correlation with hormone levels and education and psychological factors [56]. Levels of cortisol and catecholamine increase as socioeconomic development levels rise and people's lives speed up [56].

The sympathetic–adrenal–medullary system is another important factor in the pathogenesis of hypertension [57]. Under chronic stress, plasma epinephrine and norepinephrine increase rapidly. Past research has shown that the activity of the sympathetic nervous system is enhanced in hypertension; this sympathetic stimulation can result in the constriction of small arteries and veins, thereby elevating the diastolic/systolic blood pressure [58]. Catecholamines are key hormonal mediators of the sympathetic–adrenal–medullary system and play a role in the constriction of peripheral vessels to elevate diastolic blood pressure. The renin–angiotensin–aldosterone system also may play a critical role in chronic stress by increasing levels of angiotensin II, which regulates catecholamine secretion and blood pressure [59], [60]. It is well-known that sympathetic nerve excitement can promote the secretion of renin by stimulating the juxtaglomerular cell and β receptors of local tissues, thus increasing angiotensin II production.

In relation to the role of hormones in the hypothalamic–pituitary–adrenal cortical axis [61], [62], chronic psychological stress triggers the hypothalamus to discharge corticotrophin-releasing hormone and vasopressin that can cause an increase in ACTH secretion. Glucocorticoid is necessary to maintain the normal response of the circulatory system to catecholamine [63]. If the levels of glucocorticoid are low, then the response obviously decreases, the myocardial contraction force weakens, drops, and blood pressure decreases [64], [65]. Some research suggests that there are racial and ethnic differences in the effects of chronic stress on blood pressure [66].

5. CONCLUSION

Chronic stress significantly contributes to inflammation, endothelial dysfunction, and cardiovascular diseases (CVDs), particularly atherosclerosis (AS). It exacerbates AS through immune activation, proinflammatory cytokine production, and dysregulation of the hypothalamic–pituitary–adrenal axis and sympathetic nervous system, leading to hormonal imbalances and vascular dysfunction. Targeting stress-related pathways, such as reducing inflammation, regulating hormones, and managing sympathetic activity, offers promising strategies for CVD prevention. Lifestyle interventions that lower stress and improve autonomic balance may further enhance cardiovascular health. Continued research is essential to refine these approaches and address individual variations, including socioeconomic and ethnic factors.

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