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Heart Attacks are Gender and Race Biased

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ABSTRACT

Heart diseases are the leading cause of death for women world over. The risk factors for heart disease are high blood pressure, high LDL (low-density lipoprotein) cholesterol and smoking. Researches and epidemiological surveys indicated that cardiovascular disorders are gender and race biased. This may be due to differences in genetic, hormonal and life style, parameters in males and females and white and blacks. Sex steroids such as estrogen, progesterone and testosterone modulate many endocrine functions involved in atherosclerosis and other cardio-vascular disorder in women. Certain risk factors increase around the time of menopause; in the last decade, newer hormones, including leptin, adiponectin, and vitamin D, those in turn also regulated by sex steroid hormones have also been linked to different phases of vascular dysfunction.

Keywords: Sex Steroid Hormones, Genetics, Cardio Vascular Disorders, Leptons

Introduction

With age health status changes and therefore we more prone to diseases. During early childhood boys have a worse health profile than girls. This scenario changes in puberty, simply because the hormones produced by the endocrine glands in each gender are different also take part in the physiological processes and start modulating the cardiovascular system. The genders start showing different physiological reactions. Women are more frequently ill than men, but with relatively mild problems. By contrast, men feel ill less often, but their illnesses and injuries are more serious; men may be less willing and able to restrict their activities when ill or injured. The increased relative risk for cardio vascular diseases (CHD) death in men (2.5- to 4.5-fold) compared with women is seen in countries with high or low rates of heart disease [1]. This male surplus of CHD in diverse populations with very divergent lifestyles, eating patterns, and disease rates is only compatible with an intrinsic female advantage or a male disadvantage [2]. The female advantage has naturally been attributed to estrogen.

CVD in Females

There are four main types of CVD namely, coronary heart disease, stroke, peripheral arterial disease and aortic disease. A common perception that “heart disease is a man’s disease” is no truer. Recent epidemiological survey data painted altogether different picture. The endocrine glands are the same in both (male and females), the reproductive glands, and the testes in males and ovaries in females that change whole scenario of heart ailments [3]. The incidence of CVD in women is usually lower than in men, women have a higher mortality and worse prognosis after acute cardiovascular events.

Endocrine glands in the reproductive system produce sex hormones that are responsible for secondary sex characteristics in men and women [4,5]. Exposure to sex hormones throughout an individual's lifespan modulates many endocrine factors involved in atherosclerosis [6]. The relationships between cholesterol, atherosclerosis and cardiovascular risk remain controversial; elevated cholesterol is always linked to heart disease. When a woman enters peri-menopause, the lead up to menopause, there is a reshuffling in hormonal milieu. Hormones play a key role in maintaining cardiovascular health and during menopause significant decrease in estrogen production. As such menopause does not cause cardiovascular diseases. However, in general, certain risk factors increase around the time of menopause. In the last decade, newer hormones, including leptin, adiponectin, and vitamin D, have also been linked to different phases of vascular dysfunction [7-10].

From teen age to menopause adequate amount of estrogen is present in the system and it is said to be the cardio-protective [11,12]. However, during and post-menopausal conditions in women’s life the estrogen levels decline and low estrogen levels are responsible for cardio-vascular disorder in women [3,12-13].

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Estrogen Alter Cardiac Cell Physiology

Increased HDL cholesterol decreases LDL cholesterol promotes blood clot formation, and also causes some changes that have the opposite effect. Estrogen relaxes, smoothes and dilates the blood vessels so blood flow increases. Estrogen is also a vasodilator and hypotensive agent, and can induce vascular relaxation by stimulating release of endothelium-derived vasodilatory substances (e.g., nitric oxide [NO]) or by acting directly on the vascular smooth muscle [14]. Estrogen has been reported to prevent development of cardiac hypertrophy in females. The cellular effects by which 17β-estradiol (E2) inhibits angiotensin II (AngII)-induced cardiac hypertrophy in vivo. Estrogen receptors (ERs) act by regulating transcriptional processes [15,16]. The effects of estrogen on cardiovascular function are mediated by estrogen receptors (ER). Estrogen (E2) binds to ER, resulting in dimerization and recruitment of co-regulators. The estrogen–ER complex binds to estrogen response elements (ERE) on the DNA (A), resulting in altered gene transcriptions. Estrogen can also alter gene transcription by binding to transcription factors (TF) such as AP1 (B). In addition, ER can be phosphorylated by growth factors and other plasma membrane estrogen receptors that are coupled to kinase signalling [17]. Phosphorylated ER can activate gene transcription in a ligand-independent manner (C and D).

CVD in Males

Comparisons between the sexes also reveal gender differences in psychosocial and behavioural coronary risk factors, including excessive alcohol consumption and smoking, favouring women. CHD mortality rates between industrialised countries are larger than those between men and women, suggesting that biological factors are not the sole influences on the gender gap in CHD. Overall, it appears that men's coping with stressful events may be less adaptive physiologically, behaviourally, and emotionally, contributing to their increased risk for CHD [11].

Ethnic Variation Too

Blacks were more likely than whites to have shortness of breath and left-sided chest pain as the presenting symptoms of coronary heart disease. Long QTc is associated with increased risk of CHD and CVD mortality in black and white healthy men and women [18]. The QT interval is a measurement made on an electrocardiogram used to assess some of the electrical properties of the heart [19]. The gender difference in CHD mortality was more pronounced in whites than in blacks at younger ages. This discrepancy was not explained by adjustment for CHD risk factors and suggests that other factors may be responsible for the ethnic variation in the gender gap [20]. The relatively small number of blacks may account for the lack of observed racial differences in both initial symptoms and in delay in seeking treatment. Inevitably from the economic disadvantage blacks also suffered [21].

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