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Cellular and Clinical Understanding of the Aging Cardiovascular System

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Introduction

Cardiovascular disease (CVD) is a significant burden on aged people, caregivers, and health-care systems. Vascular structural and functional changes occur over time, increasing the risk of cardiovascular disease (CVD). The world's ageing population underscores the need to better understand how ageing increases CVD so that new solutions can be developed to combat the problem. This study illustrates some key unresolved clinical issues that we experience in daily cardiovascular practise when caring for older patients. The authors then go over the present state of knowledge on the mechanisms involved in cardiovascular ageing, as well as the possibility of targeting novel pathways linked to endothelial dysfunction, mitochondrial oxidative stress, chromatin remodelling, and genomic instability. Finally, the authors discuss important features of vascular healing, such as autologous bone marrow-derived stem cell transplantation in older individuals.

Risk factors for cardiovascular disease (CVD) are dominated by age 1, 2. Indeed, modern treatments for acute coronary syndromes and stroke have aided in the extension of life expectancy. Although a huge accomplishment on an individual level, the resulting demographic change poses one of the world's most significant challenges to social and health care systems. The number of people aged 65 and up will treble from 12% in 2010 to 22% in 2040. Indeed, by 2020, the number of persons aged 60 and up will surpass that of children under the age of five. The rate of population ageing is rapidly growing over the world, especially in low- and middle-income countries (e.g., Chile, China, Iran, and Russia) [1].

Description

Despite the fact that ageing is associated with a variety of illnesses, cardiovascular disease (CVD) is the leading cause of death and disability among the elderly, their caregivers, and health-care systems. Coronary heart disease (CHD) is the leading cause of death in Europe and the United States, and it is strongly linked to age. 7, 8, and 9 CVD prevalence rises in persons over 65, especially in those over 80, and is expected to rise by 10% in the next 20 years. Due to the rapid accumulation of elders, an additional 27 million people will develop hypertension, 8 million will develop coronary heart disease, 4 million will have a stroke, and 3 million will develop heart failure between 2010 and 2030 [1].

Frailty, a state of greater vulnerability to stresses, interacts with higher CVD prevalence. CVD was related with an odds ratio (OR) of 2.7 to 4.1 for prevalent frailty and an OR of 1.5 for incident frailty in those without frailty at baseline in a meta-analysis involving 54,250 older adults. According to current forecasts, spending on CHD and heart failure will climb by 200 percent over the next 20 years, with stroke accounting for the biggest proportional increase in yearly medical costs, at 238 percent. These factors underscore the importance of knowing why age is so important in the development of CVD in order to cope with the population's ageing. The clinical and experimental evidence presented in this review backs up the prevailing theory [2].

Aspects of CVD in elderly patients from a clinical perspective

A number of age-related disorders pose unique obstacles to our cardiovascular (CV) patients' clinical management. The growing number of aged people in our population emphasises the importance of CV ageing to practitioners [3].

Hypertension in the systolic chamber and a widening of the pulse pressure

Because of increased collagen and decreased elastin, the aorta stiffens with age. Increased transforming growth factor (TGF) activity promotes collagen build-up in the aorta wall. The activity of different elastases, such as matrix metalloproteinases (MMPs) like MMP-9 and MMP-12, as well as overexpression of cysteine proteinases like cathepsins S, K, and L, and the serine proteinase neutrophil elastase, all contribute to elastin depletion. These changes in the extracellular matrix of the aorta play a significant role in its loss of distensibility. Increased stiffness causes reflected waves to rise and systolic pressure to rise. However, as people become older, their diastolic pressure tends to drop. Pulse pressure rises as aortic pulse wave velocity rises. Pulse pressure is, in fact, an independent risk factor for cardiovascular events. The majority of uncontrolled hypertension in Americans over 50 years of age is due to isolated systolic hypertension 14, 15 [2].

As diastolic pressure falls, the drive for coronary perfusion, which occurs predominantly during diastole, reduces, encouraging the development of myocardial ischemia. With increasing systolic pressure, left ventricular (LV) afterload increases, which is a primary predictor of myocardial oxygen demand. Chronic exposure to high systolic pressure causes LV hypertrophy, which raises myocardial oxygen demand even more. TGF-, angiotensin II, and the mineralocorticoid aldosterone are mediators that contribute to hypertrophy and fibrosis in the pressure-overloaded LV in a mechanistic way. As a result of the systolic hypertension and decreasing diastolic pressure associated with age, a "perfect storm" of reduced oxygen supply vs increased oxygen demand occurs. Because coronary atherosclerosis worsens with age, this additional restriction on oxygen flow often adds to the already high oxygen demand [1].

Chronic hypertension causes modification of the myocardial microvasculature in addition to impaired major artery function. The tunica medium of myocardial arterioles can thicken, obstructing LV perfusion and impairing vasomotion. Myocardial ischemia is caused by the effects of increased systolic and decreased diastolic pressure that occur with age. The same pathways implicated in the production of aortic stiffness are likely involved in the regulation of arteriolar remodelling in myocardial subjected to increased systolic and decreased diastolic pressure. We don't know enough about the fundamental age-related triggers for these pathophysiological processes, which play a big role in CV problems in the elderly [4].

Definition, pathogenesis, and consequences of vascular ageing

As people age, changes in the structure and function of their arteries

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occur, increasing their chances of getting CVD 9, 30. As a result, knowing the mechanisms by which age affects the vasculature should enable us to prevent or reduce the increased risk of CVD in the elderly.

A number of recent (pre-) clinical studies have identified significant vascular changes associated with ageing 31, 32, with two basic features: widespread endothelial dysfunction and central arterial stiffness. The function of the endothelium, the cells that line the lumen of blood arteries, is altered by vascular ageing in the first case. Reduced vasodilatory and antithrombotic characteristics, as well as an increase in oxidative stress and inflammatory cytokines 33, 34, 35, encourage atherogenesis and thrombosis, and predispose to CVD, are all signs of endothelial dysfunction. Furthermore, ageing may enhance NO degradation due to higher levels of reactive oxygen species (ROS), which are mediated in part by chronic inflammation, resulting in a vicious cycle that depletes NO [4].

Heart failure with a preserved ejection percentage but a lower ejection fraction

Heart failure with preserved ejection fraction (HFpEF) is a condition that affects the elderly, primarily women, and is caused by the development of aortic stiffness as well as remodelling of the myocardial, its extracellular matrix, and its microvasculature. At the ages of 62 and 63, the risk of having HFpEF increases dramatically. Indeed, lusitropy can be harmed by LV hypertrophy and fibrosis in the elderly. Practitioners are usually aware of the rising prevalence of HFpEF, its greater prevalence in senior patients, and its negative consequences in our ageing patient population, including increased morbidity, poorer quality of life, and increased resource use [5].

Calcification of the valve and cardiac skeleton

Calcium in the axial skeleton declines with age, but calcium accumulates in CV structures. The understanding that disorders like calcific aortic stenosis are the result of a systemic process has grown. Inflammation has been implicated as a possible physiological component to CV calcification. Lipoprotein (a) is also implicated in the pathophysiology of aortic valve calcification based on strong human genetic findings [6].

Conclusion

Increased oxidative stress and inflammation cause cardiovascular ageing, according to more than two decades of dedicated research. However, universal antioxidant supplements, such as vitamin E and -carotene, failed to decrease CV events in both asymptomatic and high-risk patients 154, 155. Anti-inflammatory therapy, such as anti-TNF medications, has not been shown to reduce morbidity and mortality in patients with chronic heart failure. Finally, lifestyle changes such as calorie restriction or regular endurance exercise may slow the progression of vascular ageing by raising SIRT1 levels, PGC-1-dependent mitochondrial biogenesis, eNOS functionality, and antioxidant response via increased Nfr-2 activity 41, 158. The application of the basic and clinical science discussed here should better prepare us to deal with the burden of CVD in our ageing population.

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