Vascular Aging and Stroke

Cardiovascular disease (CVD) and cerebrovascular disease are considered the most common causes of morbidity and mortality among the elderly in developed communities [1], with stroke being the second cause of death and the leading cause of disability worldwide [2].

Aging is defined as a progressive, deleterious, universal, and irreversible decline of an organism’s functional and structural aspects. It is characterized as an essential not-modifiable risk factor for the development of atherosclerotic CVD and, in specific atherosclerotic ischemic stroke [3, 4].

To a great extent, but not always, aging is abetted by increased mortality. Specifically, changes associated with a generalized increase in mortality in older people without comorbidities may exhibit important potential biomarkers and qualify as better potential predictors of specific disease-independent mortality than chronological age per se [5]. However, despite the substantial number of promising biomarkers of aging that have been proposed, none has been validated or universally accepted [6].

Several studies considered aging as “the persistent decline in the age-specific fitness components of an organism” and correlated telomere length with this phenomenon. Subsequently, it seems worthwhile to investigate telomere length as a biomarker for aging. Furthermore, given that telomere length shortening is observed in advanced age, telomere length could serve as a potential risk factor indicator for the occurrence and progression of the most common age-related diseases [7]. Several cardio-cerebrovascular essential risk factors exist, such as hypertension, diabetes, obesity, dyslipidemias, smoking, atrial fibrillation, and history of cardiovascular or/ and peripheral arterial disease event.

Vascular aging is characterized by inflammation, endothelial dysfunction, and gradual development of arterial stiffness [1,8,9,10]. It is also associated with increased mitochondrial reactive oxygen species production, decreased nitric oxide bioavailability, and endothelial nitric oxide synthase uncoupling [1, 8, 9, 10].

Atherosclerosis seems to exhibit an autoimmune component with adaptive and innate immunity, responding to noxious stimuli and conditions, such as tissue injury, non-septic infections and triggering of inflammatory responses [11]. Both elements of immunity demonstrate a crucial role in the expansion of atherosclerosis as it has been considered a chronic non-septic low-grade inflammation of the arterial wall.

An interplay exists between vascular aging and atherosclerosis that is also associated with premature vascular aging, which the aforementioned cardiovascular risk factors can induce.

In the context of primary and secondary prevention of vascular aging and atherosclerotic CVD, various recommendations exist regarding the adoption of a healthy lifestyle, dietary habits, use of natural products that are rich in antioxidants, and the existing pharmacological approaches such as the use of antihypertensives, antidiabetics, antithrombotic agents, hypolipidemic drugs and anti-inflammatory therapy.

In this current thematic issue, the journal highlights and focuses on the disparities between the data that refer to aging and CVD and how these affect the quest for a causal link between vascular aging and stroke. 4 articles are included in this issue; “Telomere Length as a marker of biological aging: A critical review of recent literature,” as an introductory thematic review, “Inflammation, Oxidative Stress, Vascular Aging, and Atherosclerotic Ischemic Stroke” and “Immunity, Vascular Aging, and Stroke,” and the last but not least as an epilogue “Inflammatory Molecular Mediators and Pathways Involved in Vascular Aging and Stroke: A Comprehensive Review.”

Keywords: Vascular aging, atherosclerosis, cardiovascular disease, stroke.

REFERENCES


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