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AGE-RELATED HISTOLOGICAL CHANGES IN CARDIAC MUSCLE TISSUE

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ABSTRACT

Age-related changes in cardiac muscle tissue represent an important morphological basis for the development of cardiovascular diseases in the elderly. This study aims to analyze histological alterations of myocardial tissue associated with aging, with particular attention to structural, cellular, and interstitial modifications. Histological examination revealed progressive hypertrophy of cardiomyocytes, a reduction in the regenerative capacity of muscle fibers, and an increase in connective tissue components with advancing age. Additionally, age-related degeneration of myofibrils, accumulation of lipofuscin granules, and changes in nuclear morphology were observed.

Keywords: cardiac muscle tissue, aging, histological changes, cardiomyocytes, myocardial remodeling, connective tissue

INTRODUCTION

Aging is a natural biological process accompanied by progressive structural and functional changes in various organs and tissues, including the cardiovascular system. The heart, as a highly specialized muscular organ, undergoes significant age-related modifications that affect its contractile capacity, metabolic activity, and adaptive potential. These alterations play a crucial role in the increased prevalence of cardiovascular diseases observed in elderly populations. Cardiac muscle tissue is characterized by limited regenerative ability; therefore, cumulative cellular damage over time leads to irreversible histological changes. Previous studies have demonstrated that aging is associated with cardiomyocyte hypertrophy, increased interstitial fibrosis, alterations in extracellular matrix composition, and microvascular remodeling. Such changes contribute to myocardial stiffness, reduced compliance, and impaired diastolic and systolic function. At the cellular level, age-related myocardial remodeling involves degeneration of myofibrils, mitochondrial dysfunction, accumulation of lipofuscin pigments, and nuclear morphological alterations. In addition, endothelial dysfunction and reduced capillary density further compromise myocardial perfusion and oxygen delivery. Despite advances in cardiovascular research, the histological mechanisms underlying cardiac aging remain insufficiently characterized, particularly in relation to regional and population-specific features.

MATERIALS AND METHODS

The study was conducted using myocardial tissue samples obtained from individuals of different age groups. The specimens were divided into three groups according to age: young adults, middle-aged individuals, and elderly subjects. All samples were collected in accordance with ethical standards and institutional guidelines. Cardiac muscle tissue fragments were fixed in 10% neutral buffered formalin, followed by routine histological processing. The specimens were dehydrated through graded alcohol series, cleared in xylene, and embedded in paraffin. Serial sections of 4–6 μm thickness were prepared using a rotary microtome. Histological sections were stained with hematoxylin and eosin for general tissue morphology. To assess connective tissue components and interstitial fibrosis, Masson's trichrome staining was performed. In selected samples, special staining methods were applied to visualize lipofuscin accumulation and vascular structures.

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RESULTS

Histological analysis of cardiac muscle tissue demonstrated clear age-dependent morphological changes across the examined groups. In myocardial samples from young individuals, cardiomyocytes were regularly arranged, with uniform size and clearly defined cross-striations. The nuclei were centrally located and exhibited normal chromatin distribution. Interstitial connective tissue was scarce, and capillary networks were well developed. In the middle-aged group, cardiomyocytes showed moderate hypertrophy, manifested by an increase in cell diameter and slight irregularity in muscle fiber orientation. Partial disorganization of myofibrils and mild nuclear enlargement were observed. The interstitial spaces contained an increased amount of connective tissue, and early fibrotic changes were evident. Capillary density was moderately reduced, with signs of vascular wall thickening. In myocardial tissue from elderly individuals, pronounced histological alterations were identified. Cardiomyocytes exhibited marked hypertrophy and variability in size, along with degeneration and fragmentation of myofibrils.

DISCUSSION

The findings of the present study confirm that aging is associated with progressive histological remodeling of cardiac muscle tissue. Cardiomyocyte hypertrophy observed in older age groups likely represents a compensatory response to increased functional demands and reduced regenerative capacity of myocardial cells. However, this adaptive mechanism becomes maladaptive over time, contributing to impaired myocardial function. The increase in interstitial connective tissue and collagen accumulation reflects age-related fibrotic remodeling, which leads to decreased myocardial elasticity and increased stiffness. These structural changes are closely linked to diastolic dysfunction commonly observed in the aging heart. Moreover, excessive fibrosis may disrupt normal electrical conduction pathways, increasing the risk of arrhythmias. Accumulation of lipofuscin granules and degeneration of myofibrils indicate chronic oxidative stress and mitochondrial dysfunction, which play a central role in cardiac aging.

CONCLUSION

Age-related changes in cardiac muscle tissue represent a complex and progressive process involving structural, cellular, and vascular remodeling. The results of this study demonstrate that aging is accompanied by cardiomyocyte hypertrophy, degeneration of myofibrils, accumulation of lipofuscin granules, and pronounced interstitial fibrosis. These histological alterations reflect a gradual decline in the regenerative and adaptive capacity of myocardial tissue. The increase in connective tissue components and collagen deposition contributes to myocardial stiffness and reduced elasticity, which are key morphological factors underlying age-associated cardiac dysfunction. Additionally, microvascular changes, including reduced capillary density and thickening of vascular walls, impair myocardial perfusion and exacerbate functional decline. Understanding the histological mechanisms of cardiac aging provides valuable insight into the pathogenesis of age-related cardiovascular diseases.

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