

Morpho-Functional Changes in the Heart under Chronic Radiation Exposure: Effects of Biologically Active Supplements

Sherov J. A.¹, Sultonova L. J.²

¹Independent Researcher, Samarkand State Medical University, Uzbekistan

²Scientific Leader, DSc, Associate Professor, Bukhara State Medical Institute, Uzbekistan

Abstract This study investigates the morpho-functional changes in the heart due to chronic radiation exposure and evaluates the therapeutic impact of biologically active supplements. Using a controlled experiment with white rats exposed to chronic radiation, this research explores the resulting cardiac morphological alterations and assesses the corrective effects of ASD-2 fraction therapy. The study utilized morphological and biochemical analysis to examine the degree of damage and repair mechanisms. The results revealed significant structural changes in the myocardium under chronic radiation, including cellular degeneration, fibrosis, and inflammatory responses. However, treatment with ASD-2 showed a notable reduction in damage, with improvements in tissue recovery and function. This research emphasizes the need for continued investigation into the protective effects of biological supplements against radiation-induced damage.

Keywords Chronic radiation, Morpho-functional changes, Cardiac alterations, ASD-2 fraction, Biologically active supplements, Myocardial degeneration, Fibrosis, Cellular repair, Therapeutic intervention, Radiation exposure

1. Introduction

Chronic radiation exposure has long been recognized as a significant risk factor for various health conditions, particularly in tissues and organs that are highly sensitive to radiation, such as the heart. Radiation-induced heart disease (RIHD) manifests through multiple pathophysiological changes, including inflammation, fibrosis, and myocardial degeneration. The long-term consequences of such exposure can lead to severe cardiac dysfunction, including heart failure, arrhythmias, and ischemic damage. [1,3,4]

The heart, being a central organ in maintaining systemic circulation, is particularly vulnerable to the effects of radiation. Chronic exposure to ionizing radiation results in oxidative stress, DNA damage, and the activation of inflammatory pathways, all of which contribute to the deterioration of cardiac function. The precise mechanisms through which radiation affects cardiac tissues remain an area of active research. However, it is evident that these changes can lead to irreversible damage if not addressed through therapeutic interventions. [2,3]

Biologically active supplements, particularly those with antioxidant properties, have gained attention as potential therapeutic agents for mitigating radiation-induced damage. ASD-2 fraction, a biologically active supplement, has shown

promise in reducing inflammation and enhancing cellular repair in experimental models of radiation injury. ASD-2 is known for its immunomodulatory, anti-inflammatory, and regenerative properties, making it a potential candidate for addressing the cardiac damage associated with chronic radiation exposure. [2,4]

This study aims to explore the morpho-functional changes in the heart under chronic radiation exposure and assess the therapeutic potential of ASD-2 in mitigating these changes. The research focuses on the structural and functional alterations in the myocardium, evaluating the extent of damage and the recovery processes facilitated by ASD-2 treatment.

2. Methodology

Study Design

This study was designed as an experimental animal model to investigate the effects of chronic radiation on the heart and the therapeutic potential of ASD-2. The study used white rats as the experimental subjects, chosen for their well-established use in radiation studies due to their physiological similarities to humans in terms of organ structure and function.

Animal Selection

A total of 50 male white rats, weighing between 160-180 grams, were selected for the study. The animals were divided into two primary groups:

1. **Control group (n = 25):** Rats not exposed to radiation and not treated with ASD-2.
2. **Experimental group (n = 25):** Rats exposed to chronic radiation and further subdivided into:
 - o **Subgroup A (n = 12):** Exposed to radiation without treatment.
 - o **Subgroup B (n = 13):** Exposed to radiation and treated with ASD-2 fraction.

All rats were housed in a controlled environment with standardized conditions for temperature, humidity, and a 12-hour light/dark cycle. Food and water were provided ad libitum.

Radiation Exposure

The experimental group was subjected to chronic radiation exposure using a gamma-radiation source at a dose of 1 Gy per week for 12 weeks. This level of exposure is equivalent to long-term occupational exposure in humans. The control group was not exposed to any radiation.

ASD-2 Treatment

Subgroup B received ASD-2 fraction treatment starting at week 8 of radiation exposure. The supplement was administered orally at a dose of 0.5 ml/kg body weight daily for four weeks. This treatment was designed to assess the potential protective and regenerative effects of ASD-2 in mitigating radiation-induced cardiac damage.

Data Collection and Analysis

At the end of the experimental period, the rats were sacrificed, and their hearts were harvested for histological and biochemical analysis. Morphological changes were assessed through light microscopy, and sections were stained with hematoxylin and eosin (H&E) to evaluate cellular degeneration, fibrosis, and inflammation. Biochemical analysis included oxidative stress markers and enzyme activity related to cardiac function.

Statistical analysis was performed using SPSS software, and the results were expressed as mean \pm standard deviation. Comparisons between groups were made using ANOVA, with a significance level set at $p < 0.05$.

3. Results

Morphological Changes

Histological analysis revealed significant differences between the control and experimental groups. In the rats exposed to radiation without ASD-2 treatment (Subgroup A), extensive myocardial degeneration was observed, characterized by:

- **Cellular degeneration:** Marked by vacuolation and loss of cellular integrity in cardiomyocytes.
- **Fibrosis:** Widespread fibrotic tissue replacing healthy myocardium, particularly in the ventricular walls.
- **Inflammation:** Presence of inflammatory infiltrates, indicating a chronic inflammatory response to radiation

-induced damage.

In contrast, the rats treated with ASD-2 (Subgroup B) showed considerable improvement in cardiac structure. Although some fibrotic areas remained, there was a significant reduction in cellular degeneration and inflammation compared to Subgroup A.

Oxidative Stress and Biochemical Markers

The levels of oxidative stress markers were significantly elevated in the untreated radiation-exposed group (Subgroup A). These rats exhibited higher levels of malondialdehyde (MDA), a marker of lipid peroxidation, and reduced activity of superoxide dismutase (SOD) and catalase, both of which are critical in mitigating oxidative damage.

In the ASD-2 treated group (Subgroup B), there was a marked reduction in MDA levels and a significant increase in the activity of antioxidant enzymes (SOD and catalase). These results suggest that ASD-2 played a key role in enhancing the antioxidant defense system, reducing oxidative stress, and improving overall cardiac function.

4. Discussion

The results of this study demonstrate the profound impact of chronic radiation on cardiac tissue, leading to significant morphological and functional changes. The observed myocardial degeneration, fibrosis, and inflammation align with previous research indicating that radiation-induced cardiac damage occurs through oxidative stress and inflammatory pathways.

Chronic exposure to ionizing radiation leads to the generation of reactive oxygen species (ROS), which overwhelm the cellular antioxidant defense mechanisms. This results in oxidative damage to cellular structures, including lipids, proteins, and DNA, ultimately contributing to the observed fibrosis and myocardial degeneration. The chronic inflammatory response further exacerbates tissue damage, promoting the development of fibrotic scar tissue that impairs cardiac function.

The findings also highlight the therapeutic potential of ASD-2 in mitigating radiation-induced cardiac damage. The reduction in oxidative stress markers and the increased activity of antioxidant enzymes in the ASD-2 treated group suggest that this supplement can effectively enhance the body's natural defense mechanisms against radiation-induced damage. Furthermore, the histological improvements observed in the ASD-2 treated group indicate that the supplement promotes tissue repair and reduces inflammation.

These results align with previous studies on the use of biologically active supplements, particularly those with antioxidant properties, in mitigating radiation-induced damage. ASD-2's ability to modulate the immune response and reduce oxidative stress highlights its potential as a therapeutic agent in managing radiation-induced heart disease. However, further research is needed to understand the long-term effects of ASD-2 treatment and its efficacy in human populations exposed to chronic radiation.

5. Conclusions

This study confirms that chronic radiation exposure induces significant morpho-functional changes in the heart, including cellular degeneration, fibrosis, and inflammation. The findings also suggest that biologically active supplements such as ASD-2 fraction have the potential to mitigate these effects by enhancing antioxidant defenses and promoting tissue repair. The reduction in oxidative stress and the histological improvements observed in the ASD-2 treated group underscore the therapeutic potential of ASD-2 in managing radiation-induced cardiac damage.

Further research is warranted to explore the long-term efficacy of ASD-2 and its potential application in clinical settings, particularly for individuals exposed to occupational or environmental radiation.

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