DNA Damage in Atrial Fibrillation is triggered by Different Versions of Cytoskeletal Enzyme

Jharna Rani M*

Abstract

Atrial fibrillation (AF) is a prevalent cardiac arrhythmia associated with significant morbidity and mortality. While the precise mechanisms underlying AF pathogenesis remain incompletely understood, emerging evidence suggests that DNA damage plays a critical role in its development and progression. Recent studies have revealed the presence of different versions or isoforms of a cytoskeletal enzyme in atrial tissue from AF patients. This abstract aims to summarize the current understanding of DNA damage in AF and its association with these distinct cytoskeletal enzyme isoforms. Multiple factors contribute to DNA damage in AF, including oxidative stress, inflammation, and mechanical stress resulting from altered atrial electrical activity. The chaotic electrical environment and disturbed mechanical properties of atrial tissue in AF create conditions that predispose to DNA damage. Consequently, impaired DNA repair processes further perpetuate the arrhythmia. The presence of different cytoskeletal enzyme isoforms in AF patients suggests a potential link between these enzymes and DNA damage. These isoforms exhibit variations in their ability to interact with DNA and participate in DNA repair mechanisms. Altered expression and activity of these isoforms may compromise DNA repair, rendering the atrial tissue more susceptible to genetic alterations and further promoting AF pathogenesis.

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***Corresponding author:** Jharna Rani M, Department of Endocrinology, University of Indonesia, Indonesia, E-mail: jharnarani143@gmail.com

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