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Galectins in Cardiovascular Health and Cirrhotic Cardiomyopathy: A Comprehensive Review

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Abstract

Galectins are a family of β-galactoside-binding proteins involved in various physiological and pathological processes, including inflammation, fibrosis and cancer. In recent years, emerging evidence has implicated galectins in the pathophysiology of cardiovascular diseases, including cirrhotic cardiomyopathy-a condition characterized by cardiac dysfunction in patients with liver cirrhosis. This comprehensive review explores the multifaceted roles of galectins in cardiovascular health and the specific mechanisms underlying their involvement in cirrhotic cardiomyopathy. We discuss current understanding of galectin biology, their impact on cardiac structure and function and their potential as therapeutic targets in managing cardiovascular complications associated with liver disease.

Keywords: Galectins • Cardiovascular diseases • Cirrhotic cardiomyopathy • Fibrosis

Introduction

Galectins constitute a diverse family of carbohydrate-binding proteins implicated in modulating immune responses, cell adhesion and signaling pathways in various physiological and pathological conditions. Their ability to bind β-galactoside-containing glycoconjugates on cell surfaces and in the extracellular matrix facilitates interactions that regulate cell proliferation, apoptosis and inflammation. In cardiovascular medicine, galectins have gained attention for their roles in myocardial remodeling, vascular inflammation and endothelial dysfunction-key processes in the pathogenesis of cardiovascular diseases such as atherosclerosis, heart failure and hypertension. Specifically, galectins have been implicated in the development of cirrhotic cardiomyopathy, a condition observed in patients with advanced liver cirrhosis characterized by impaired cardiac contractility, diastolic dysfunction and arrhythmias [1]. The pathophysiology of cirrhotic cardiomyopathy involves multifactorial mechanisms, including hemodynamic alterations, neurohormonal dysregulation, oxidative stress and systemic inflammation, all of which contribute to structural and functional cardiac abnormalities. This review aims to synthesize current knowledge on the role of galectins in cardiovascular health and their specific implications in cirrhotic cardiomyopathy. We will examine the molecular mechanisms through which galectins influence cardiac structure and function in the context of liver cirrhosis, highlighting their potential as diagnostic biomarkers and therapeutic targets. By elucidating the intricate interplay between galectin biology and cardiovascular pathology, this review seeks to provide insights into novel strategies for managing cardiovascular complications associated with chronic liver disease [2].

Literature Review

Galectins are a family of β-galactoside-binding proteins that play diverse roles in inflammation, fibrosis and cancer, with emerging significance in cardiovascular diseases and cirrhotic cardiomyopathy. In cardiovascular health, galectins contribute to myocardial remodeling, vascular inflammation and endothelial dysfunction, processes central to conditions like atherosclerosis, heart failure and hypertension. Specifically, in cirrhotic cardiomyopathy-a complication of advanced liver cirrhosis-galectins are implicated in mediating cardiac dysfunction through multiple pathways. Research indicates that galectins, particularly galectin-3, are elevated in patients with cirrhosis and correlate with the severity of liver disease and cardiac dysfunction. Galectin-3 promotes myocardial fibrosis by stimulating cardiac fibroblast activation and collagen deposition, contributing to impaired diastolic function and increased susceptibility to arrhythmias. Moreover, galectin-3 interacts with inflammatory cytokines and oxidative stress pathways, exacerbating systemic inflammation and oxidative damage in both the liver and the heart [3].

Experimental studies have highlighted galectin-3 as a potential biomarker for predicting cardiovascular complications in cirrhotic patients, offering insights into disease progression and prognosis. In addition to galectin-3, other galectins such as galectin-1 and galectin-9 have been implicated in modulating immune responses and tissue remodeling in cardiovascular and liver diseases, suggesting broader roles beyond fibrotic pathways. Understanding the intricate roles of galectins in cardiovascular health and cirrhotic cardiomyopathy provides a foundation for exploring novel therapeutic strategies. Targeting galectin-mediated pathways could potentially mitigate myocardial fibrosis, improve cardiac function and reduce cardiovascular morbidity and mortality in patients with liver cirrhosis [4].

Discussion

The involvement of galectins in cirrhotic cardiomyopathy underscores their dual role in modulating cardiac and hepatic fibrosis. Galectin-3, in particular, emerges as a key mediator of fibrotic processes in both organs, linking liver dysfunction with cardiac complications through shared inflammatory and fibrotic pathways. Elevated galectin-3 levels correlate with worse outcomes in cirrhotic patients, highlighting its potential as a prognostic biomarker and therapeutic target. Therapeutically, targeting galectin-3 with pharmacological inhibitors or monoclonal antibodies has shown promise in preclinical studies for reducing fibrosis and improving cardiac function in various cardiovascular and liver diseases. Clinical trials investigating the efficacy of galectin-3 inhibitors in attenuating cirrhotic cardiomyopathy and reducing cardiovascular events are underway, offering hope for novel therapeutic interventions in this challenging patient population. However, challenges remain in translating preclinical findings into clinical practice, including the need for robust biomarkers to identify patients at highest risk for cirrhotic cardiomyopathy and monitor treatment response. Furthermore, elucidating the specific roles of different galectins and their interactions in cardiovascular and hepatic pathophysiology requires further investigation through integrated translational

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research approaches [5,6].

Conclusion

In conclusion, galectins play pivotal roles in cardiovascular health and the pathogenesis of cirrhotic cardiomyopathy. Elevated galectin levels, particularly galectin-3, contribute to myocardial fibrosis, inflammation and oxidative stress in patients with liver cirrhosis, exacerbating cardiac dysfunction and increasing cardiovascular risk. The identification of galectins as key mediators of fibrotic pathways in both the liver and the heart highlights their potential as diagnostic markers and therapeutic targets for managing cirrhotic cardiomyopathy. Moving forward, continued research efforts are essential to deepen our understanding of galectin biology in cardiovascular and hepatic diseases, refine diagnostic strategies and develop targeted therapies to improve outcomes for patients with cirrhotic cardiomyopathy. Integrating multidisciplinary approaches that encompass basic science, clinical research and translational studies will facilitate the development of personalized treatment strategies aimed at mitigating cardiovascular complications and improving overall prognosis in this vulnerable patient population.

Acknowledgment

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Conflict of Interest

No conflict of interest.

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