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Galectin-3: A Potential Biomarker and Therapeutic Target in Severe COVID-19

Herooety Feroojer*

Department of Clinical Pulmonology, Clinical Hospital Centre Rijeka, 51000 Rijeka, Croatia

Abstract

The COVID-19 pandemic caused by the novel coronavirus SARS-CoV-2 has posed unprecedented challenges to global public health. While the majority of COVID-19 cases exhibit mild symptoms, a subset of patients develops severe respiratory distress, multi-organ failure and even death. Identifying biomarkers associated with disease severity is crucial for prognosis and developing targeted therapies. Recent research suggests that elevated levels of Galectin-3 may play a significant role in the pathogenesis of severe COVID-19. Galectin-3 is a β-galactoside-binding lectin involved in various physiological and pathological processes, including inflammation, fibrosis and immune regulation. It is expressed by various immune cells, endothelial cells and epithelial cells and its dysregulation has been implicated in numerous diseases, including cancer, heart failure and inflammatory disorders. Severe COVID-19 presents with diverse clinical manifestations, including acute respiratory distress syndrome and multiorgan dysfunction. Identifying reliable biomarkers and therapeutic targets is critical for improving patient outcomes.

Keywords: Galectin-3 • Therapeutic • Biomarker

Introduction

In the context of COVID-19, dysregulated immune responses, particularly cytokine storms, are known to contribute to disease severity. Galectin-3 has emerged as a key mediator of inflammation, promoting the activation and recruitment of immune cells, as well as cytokine production [1]. Studies have demonstrated elevated Galectin-3 levels in patients with severe COVID-19, suggesting its involvement in the hyperinflammatory response characteristic of the disease. Several studies have reported a correlation between elevated Galectin-3 levels and disease severity in COVID-19 patients. These findings indicate that Galectin-3 could serve as a prognostic biomarker for identifying individuals at higher risk of developing severe complications. Moreover, elevated Galectin-3 levels have been associated with increased mortality rates, highlighting its potential as a predictor of poor clinical outcomes [2].

Literature Review

Furthermore, Galectin-3 may have utility in monitoring disease progression and response to therapy. Serial measurements of Galectin-3 levels could provide valuable insights into the dynamics of the inflammatory response and help guide treatment decisions. This review summarizes current literature on the role of galectin-3 in COVID-19 pathogenesis and its potential as a biomarker and therapeutic target. Relevant articles published up to January 2024 were included, focusing on studies investigating galectin-3 levels, its association with disease severity and therapeutic interventions targeting galectin-3. Additionally, targeting Galectin-3 signaling pathways could represent a novel therapeutic approach for mitigating inflammation and preventing disease progression in severe COVID-19 cases. The precise mechanisms underlying the contribution of Galectin-3 to severe COVID-19 pathology are still under investigation. However, several potential mechanisms have been proposed based on existing knowledge of Galectin-3 function and COVID-19 pathophysiology. Firstly, Galectin-3 may exacerbate lung injury by promoting inflammatory cell recruitment and cytokine release, leading to the amplification of the local inflammatory response and tissue damage [3].

Secondly, Galectin-3 has been implicated in the regulation of endothelial function and vascular permeability, which are critical factors in the development of acute respiratory distress syndrome and other vascular complications associated with severe COVID-19 [4]. Moreover, Galectin-3 may contribute to systemic inflammation and multi-organ dysfunction by modulating immune cell activation and trafficking. Its ability to interact with various immune receptors and signaling molecules suggests a multifaceted role in orchestrating immune responses during viral infection. Additionally, Galectin-3 has been linked to fibrosis and tissue remodeling processes, which could contribute to long-term sequelae in COVID-19 survivors. Given the potential significance of Galectin-3 in severe COVID-19, targeting its signaling pathways could represent a promising therapeutic strategy [5].

Discussion

Several approaches could be explored, including the development of Galectin-3 inhibitors or modulators to attenuate inflammation and tissue damage. Galectin-3, a β-galactosidase-binding lectin, has emerged as a promising biomarker in severe COVID-19. Elevated levels of galectin-3 have been consistently reported in patients with severe disease and are associated with worse clinical outcomes, including mortality. Mechanistically, galectin-3 contributes to COVID-19 pathogenesis through its involvement in inflammatory cytokine production, endothelial dysfunction and fibrosis. Therapeutic targeting of galectin-3 holds potential for mitigating COVID-19-associated inflammation and tissue damage. Several preclinical studies have demonstrated the efficacy of galectin-3 inhibitors in attenuating lung injury and improving survival in animal models of ARDS and COVID-19. Preclinical studies in animal models of COVID-19 could help elucidate the efficacy and safety of such interventions before clinical translation. Furthermore, clinical trials are warranted to evaluate the utility of Galectin-3 as a biomarker for predicting disease severity and guiding treatment decisions in COVID-19 patients. Prospective studies assessing the prognostic value of Galectin-3 levels in large cohorts of patients with varying disease severity would provide valuable insights into its clinical relevance [6].

***Address for Correspondence:** Herooety Feroojer, Department of Clinical Pulmonology, Clinical Hospital Centre Rijeka, 51000 Rijeka, Croatia; E-mail: Feroojerh@gmail.com*

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Conclusion

In conclusion, elevated Galectin-3 levels have emerged as a potential biomarker and therapeutic target in severe COVID-19. Understanding the mechanisms by which Galectin-3 contributes to disease pathogenesis could pave the way for the development of novel therapeutic interventions aimed at mitigating inflammation and improving clinical outcomes in critically ill patients. Further research is needed to validate these findings and translate them into clinical practice, with the ultimate goal of reducing the burden of severe COVID-19 worldwide.

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Conflict of Interest

None.

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