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Predictive Value of Elevated Cardiac Troponin I for Mortality in Hospitalized COVID-19 Patients

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Introduction

The coronavirus disease 2019 (COVID-19) pandemic has presented unprecedented challenges to global healthcare systems, with severe cases often involving respiratory distress and multiorgan involvement. Emerging evidence suggests that COVID-19 can significantly impact the cardiovascular system, leading to myocardial injury and elevated cardiac biomarkers such as troponin I. This report investigates the predictive value of elevated cardiac troponin I levels as a mortality predictor in hospitalized COVID-19 patients, exploring the underlying mechanisms, clinical implications, and implications for patient management.

COVID-19 is caused by severe acute respiratory syndrome coronavirus 2, which primarily targets Angiotensin-Converting Enzyme 2 (ACE2) receptors on respiratory epithelial cells. However, ACE2 receptors are also expressed in cardiovascular tissues, including cardiomyocytes and endothelial cells. Direct viral invasion, cytokine storm, and systemic inflammatory responses can lead to myocardial injury, myocarditis, and microvascular dysfunction, contributing to elevated cardiac troponin I levels. Elevated cardiac troponin I levels (>99th percentile upper reference limit) indicate myocardial injury and are associated with adverse clinical outcomes in COVID-19 patients. Studies have shown that patients with elevated troponin I levels on admission or during hospitalization have increased mortality rates compared to those with normal troponin levels. The severity of troponin elevation correlates with the extent of myocardial damage and is often a marker of more severe disease progression and poor prognosis [1].

Description

Measuring cardiac troponin I levels in hospitalized COVID-19 patients provides valuable prognostic information beyond traditional risk factors. Elevated troponin levels can help risk-stratify patients, guide clinical management decisions, and prompt timely interventions to mitigate adverse outcomes. Serial monitoring of troponin levels may indicate disease progression or response to treatment, influencing therapeutic strategies such as Intensive Care Unit (ICU) admission, ventilatory support, or initiation of cardiac-specific therapies. The presence of elevated cardiac troponin I levels in COVID-19 patients warrants a comprehensive clinical approach. Regular cardiac monitoring, including electrocardiography and echocardiography, to assess myocardial function and detect complications such as myocarditis or myocardial infarction. Tailoring treatment strategies to address myocardial injury, including management of fluid balance, oxygenation, and hemodynamic stability [2].

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Prompt initiation of antiviral therapies, anti-inflammatory agents, and, when indicated, immunomodulatory treatments to mitigate systemic inflammation and reduce myocardial damage. Understand the specific mechanisms by which SARS-CoV-2 induces myocardial injury and elevates cardiac troponin I levels. Identify optimal treatment approaches targeting COVID-19-associated myocardial injury to improve patient outcomes. Investigate the long-term cardiovascular consequences of COVID-19, including the potential for post-infection cardiac sequelae and chronic myocardial dysfunction. Elevated cardiac troponin I levels serve as a valuable biomarker for predicting mortality and assessing myocardial injury severity in hospitalized COVID-19 patients. Incorporating troponin measurement into routine clinical practice enhances risk stratification and informs targeted therapeutic interventions to improve patient outcomes. Continued research efforts are essential to enhance our understanding of COVID-19-associated cardiovascular complications and optimize management strategies accordingly [3].

Elevated cardiac Troponin I (cTnI) levels are indicative of myocardial injury or damage and serve as a critical biomarker in clinical practice, particularly in the context of Acute Coronary Syndromes (ACS), Myocardial Infarction (MI), and various other cardiac conditions. Troponins are regulatory proteins found exclusively in cardiac muscle cells and are released into the bloodstream following myocardial injury or stress. Here, we explore the significance of elevated cardiac troponin I levels, their clinical applications, interpretation, and implications for patient management. Elevated cTnI levels signify myocardial injury, which can result from conditions such as myocardial infarction (heart attack), myocarditis (inflammation of the heart muscle), heart failure exacerbation, pulmonary embolism, and other cardiac insults.

Cardiac troponins, including cTnI, are specific to cardiac muscle tissue. Their release into the bloodstream occurs when there is damage to cardiomyocytes, making them highly sensitive markers for myocardial injury. Timing of cTnI measurement is crucial for clinical interpretation. Serial measurements are often used to assess changes over time, helping to differentiate acute from chronic myocardial injury and guide treatment decisions. In the context of chest pain or symptoms suggestive of ACS, elevated cTnI levels aid in diagnosing acute Myocardial Infarction (MI) and guiding the appropriate management, including invasive procedures such as coronary angiography and revascularization [4].

Elevated cTnI levels are associated with worse outcomes in various cardiac conditions, including higher mortality rates and increased risk of major adverse cardiovascular events , such as recurrent MI, stroke, and heart failure. In patients with known cardiovascular disease or those at risk, baseline and serial cTnI measurements help stratify the risk of future cardiovascular events and guide preventive strategies and treatment intensification. Elevated cTnI levels are typically defined as values above the 99th percentile upper reference limit in a healthy population. The magnitude of elevation often correlates with the extent of myocardial damage and prognosis. Non-cardiac conditions, such as renal failure, sepsis, and pulmonary embolism, can also lead to elevated cTnI levels due to impaired renal clearance or direct myocardial injury from systemic inflammation or hypoxia. Careful clinical correlation is necessary to differentiate cardiac from non-cardiac causes of troponin elevation. Elevated cTnI levels influence therapeutic strategies, including initiation of antiplatelet therapy, anticoagulation, beta-blockers, and statins, aimed at reducing myocardial ischemia, inflammation, and subsequent cardiovascular events [5].

Conclusion

Serial monitoring of cTnI levels helps assess response to treatment interventions, guiding adjustments in therapy and providing prognostic information regarding disease progression or resolution. Advances in high-sensitivity troponin assays offer greater sensitivity and precision in detecting minor elevations in cTnI levels, potentially allowing earlier detection of myocardial injury and improving risk stratification. Combining cTnI measurements with novel biomarkers and imaging modalities holds promise for enhancing diagnostic accuracy, prognostication, and personalized management strategies in cardiovascular medicine.

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Conflict of Interest

Authors declare no conflict of interest.

References

1. Fairweather, DeLisa, Danielle J. Beetler, Damian N. Di Florio and Nicolas Musigk, et al. "COVID-19, myocarditis and pericarditis." *Circ Res* 132 (2023): 1302-1319.
2. Devaux, Christian A. and Laurence Camoin-Jau. "An update on angiotensin-converting enzyme 2 structure/functions, polymorphism, and duplicitous nature in the pathophysiology of coronavirus disease 2019: Implications for vascular and coagulation disease associated with severe acute respiratory syndrome coronavirus infection." *Front Microbiol* 13 (2022): 1042200.
3. Lindner, Diana, Antonia Fitzek, Hanna Bräuninger and Ganna Aleshcheva, et al. "Association of cardiac infection with SARS-CoV-2 in confirmed COVID-19 autopsy cases." *JAMA Cardiol* 5 (2020): 1281-1285.
4. Bearse, Mayara, Yin P. Hung, Aram J. Krauson and Liana Bonanno, et al. "Factors associated with myocardial SARS-CoV-2 infection, myocarditis, and cardiac inflammation in patients with COVID-19." *Mod Pathol* 34 (2021): 1345-1357.
5. Ghantous, Eihab, Yishay Szekely, Yael Lichter and Erez Levi, et al. "Pericardial involvement in patients hospitalized with COVID-19: Prevalence, associates, and clinical implications." *J Am Heart Assoc* 11 (2022): e024363.

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