

Metabolic and Lipidomic Markers for Early Mortality Prediction in COVID-19

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Abstract

The COVID-19 pandemic has presented a global health crisis, affecting millions of lives worldwide. While most individuals experience mild to moderate symptoms, severe cases can lead to Acute Respiratory Distress Syndrome (ARDS) and multi-organ failure, resulting in mortality. Early identification of patients at risk of severe outcomes is crucial for providing timely interventions and improving survival rates. Recent research has focused on exploring metabolic and lipidomic markers as potential predictors of early mortality in COVID-19 patients. This article examines the significance of these markers in predicting COVID-19-related mortality and their potential implications for clinical practice. Metabolomics is the study of small-molecule metabolites present in biological systems. These metabolites play essential roles in various cellular processes, and their altered levels can reflect changes in the body's physiological state. Similarly, lipidomics focuses on analyzing lipid molecules and their role in cellular function and signaling pathways. In the context of COVID-19, the dysregulation of metabolic and lipidomic pathways has been linked to disease severity and outcomes. Emerging evidence suggests that specific metabolic markers can serve as early indicators of poor prognosis in COVID-19 patients.

Keywords: COVID-19 • Lipidomic • Signaling pathways

Introduction

Elevated lactate levels in COVID-19 patients have been associated with a higher risk of mortality. Lactate is a byproduct of anaerobic metabolism, indicating tissue hypoxia and impaired cellular function. Dysregulated glucose metabolism, often characterized by high blood glucose levels, has been observed in severe COVID-19 cases. Hyperglycemia is linked to worse outcomes and may promote inflammation and immune dysfunction. CRP is a marker of inflammation and is often elevated in severe COVID-19 cases. Persistent high levels of CRP may predict a more severe disease course. Lipids are essential components of cell membranes and are involved in energy storage and signaling. Lipidomic profiling has revealed several lipid classes with potential prognostic value in COVID-19. Altered sphingolipid levels have been associated with severe COVID-19. Ceramides, a type of sphingolipid, can induce inflammatory responses and endothelial dysfunction, contributing to disease severity. Changes in phospholipid metabolism have been observed in COVID-19 patients, and certain phospholipids have been linked to lung injury and inflammation. Regular monitoring of metabolic and lipidomic markers during a patient's hospitalization can provide insights into disease progression and response to treatment. Adjusting interventions based on these markers may lead to better patient outcomes. Understanding the metabolic and lipidomic alterations associated with severe COVID-19 can inform the development of targeted therapeutics to mitigate the disease's progression [1].

Literature Review

There is a need for standardized protocols and analytical techniques for

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metabolic and lipidomic profiling to ensure consistency and comparability of results across studies. Large-scale studies are required to validate the predictive value of these markers robustly. Collaboration among research institutions and data sharing are essential to achieve this. Long-term follow-up studies are necessary to determine the stability and reliability of metabolic and lipidomic markers over time. Integrating metabolic and lipidomic data with clinical information, such as age, comorbidities, and disease severity, is critical for establishing comprehensive predictive models. Metabolic and lipidomic markers hold tremendous promise as early predictors of mortality in COVID-19 patients. Their inclusion in clinical practice could enable timely interventions and improve patient outcomes. However, further research is needed to validate their predictive value and establish standardized protocols for their implementation. As we continue to battle the COVID-19 pandemic, these markers offer hope for better risk assessment and targeted therapies, ultimately saving lives and mitigating the impact of the disease on a global scale [2].

COVID-19, caused by the Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), has emerged as a global health crisis since its outbreak in late 2019. While the majority of individuals infected with SARS-CoV-2 experience mild to moderate symptoms, a significant number of patients develop severe respiratory distress, leading to high mortality rates. Early identification of patients at risk of severe disease is crucial for timely intervention and improved outcomes. In recent years, researchers have turned their attention to metabolic and lipidomic markers as potential indicators for early mortality prediction in COVID-19. Understanding these biomarkers could help identify high-risk patients and personalize treatment strategies to reduce fatalities. This article explores the current understanding of metabolic and lipidomic markers in predicting early mortality in COVID-19. Metabolomics involves the study of small molecules, such as metabolites, within biological systems. These molecules play a crucial role in various cellular processes and can serve as valuable indicators of disease progression and severity. In COVID-19, metabolic dysregulation has been linked to severe outcomes, making metabolomics markers potential candidates for early mortality prediction. Lipids act as signalling molecules and modulate immune responses. In COVID-19, specific lipid mediators, such as prostaglandins and leukotrienes, may influence the magnitude and duration of the inflammatory response. Understanding the lipidomic profile can provide insights into the immune dysregulation observed in severe COVID-19 cases [3].

Discussion

Severe COVID-19 cases are often characterized by an exaggerated immune response known as the "cytokine storm." This hyper inflammatory state leads to metabolic disturbances, including alterations in glucose and lipid metabolism. Studies have shown that elevated levels of pro-inflammatory cytokines, such as Interleukin-6 (IL-6), are associated with adverse outcomes in COVID-19 patients. Moreover, IL-6 is linked to insulin resistance and impaired glucose metabolism, which can exacerbate pre-existing conditions like diabetes and increase mortality risk. Oxidized lipids, such as oxidized phospholipids and oxidized cholesterol, are known to trigger inflammation and oxidative stress. In COVID-19, the increased oxidative stress due to a hyper inflammatory state can lead to lipid oxidation. Elevated levels of oxidized lipids have been associated with lung injury and severe disease outcomes. Sphingolipids are bioactive lipids that regulate cell proliferation, apoptosis, and inflammation. In COVID-19, dysregulation of sphingolipid metabolism has been linked to endothelial dysfunction, which plays a crucial role in the pathogenesis of severe disease. Sphingosine-1-Phosphate (S1P) and ceramides are among the sphingolipids implicated in endothelial dysfunction and vascular complications in COVID-19 [4].

Lipids play essential roles in cell membrane structure, signaling and energy storage. COVID-19 patients, especially those with severe disease, often exhibit abnormal lipid profiles. Increased levels of triglycerides, Low-Density Lipoprotein Cholesterol (LDL-C), and decreased levels of High-Density Lipoprotein Cholesterol (HDL-C) have been reported in severe cases. Dysregulated lipid metabolism can contribute to endothelial dysfunction and the formation of blood clots, potentially leading to critical complications like thrombosis and organ damage. Metabolic shifts, such as increased production of ketone bodies, have been observed in severe COVID-19 patients. Ketone bodies, like β -hydroxybutyrate, serve as alternative energy sources during metabolic stress. However, prolonged elevation of ketone bodies may indicate a state of metabolic imbalance, leading to worsened outcomes. Lipidomics is a subset of metabolomics that focuses on the comprehensive analysis of lipids in biological samples. As lipids play critical roles in inflammation, immunity, and cell signaling, lipidomic profiling can offer valuable insights into disease progression and patient outcomes in COVID-19 [5].

The integration of metabolic and lipidomic markers with clinical and demographic data can enhance early mortality prediction in COVID-19 patients. Machine learning algorithms and artificial intelligence-driven analyses can help identify robust biomarker patterns associated with severe disease outcomes. By analyzing the metabolic and lipidomic profiles of COVID-19 patients upon admission, healthcare providers can stratify patients into low, moderate, and high-risk groups. This risk stratification can guide personalized treatment approaches and resource allocation, especially in settings with limited medical resources. Dynamic monitoring of metabolic and lipidomic markers during the course of COVID-19 can provide valuable information about disease progression and response to therapies. Changes in these markers may help predict potential clinical deterioration, prompting early interventions to prevent adverse outcomes. Metabolic and lipidomic markers can reveal potential drug targets and pathways for therapeutic intervention. Drugs that modulate dysregulated metabolic pathways or restore lipid homeostasis may hold promise for improving patient outcomes in severe COVID-19 cases.

Early mortality prediction is of paramount importance in managing severe COVID-19 cases and reducing fatalities. Metabolic and lipidomic markers offer valuable insights into the dysregulation of cellular processes in COVID-19 patients and can serve as indicators of disease severity. Integrating these biomarkers with clinical data and employing advanced analytics can enhance risk stratification, disease monitoring, and the development of targeted therapies. Continued research and validation are essential to unlock the full potential of metabolic and lipidomic markers for early mortality prediction in COVID-19, ultimately improving patient outcomes and mitigating the impact of the pandemic [6].

Conclusion

Early mortality prediction is of paramount importance in managing severe

COVID-19 cases and improving patient outcomes. Metabolic and lipidomic markers offer a promising avenue for such predictions, as they provide insights into the underlying physiological changes associated with disease severity. By integrating these markers and utilizing advanced AI algorithms, healthcare professionals can identify high-risk patients early on and implement targeted interventions to mitigate adverse outcomes. However, further research and validation are necessary before implementing these markers in routine clinical practice. As the scientific community continues to deepen its understanding of COVID-19 pathogenesis, metabolic and lipidomic markers are likely to play an increasingly crucial role in predicting early mortality and refining patient management strategies. These predictive models can take into account a wide range of variables, including demographic information, comorbidities, and disease progression, alongside metabolic and lipidomic markers.

Integrating AI with clinical data can enhance the accuracy of mortality prediction, enabling healthcare professionals to make informed decisions about patient care. Machine learning and Artificial Intelligence (AI) algorithms have gained traction in various medical applications, including disease prediction. By leveraging large datasets of metabolic and lipidomic profiles from COVID-19 patients, AI can be used to develop predictive models that accurately identify patients at a higher risk of early mortality. The integration of metabolic and lipidomic markers could provide a more comprehensive approach to early mortality prediction in COVID-19. Combining markers such as LDH, glucose levels, CRP, ferritin, and D-dimer with lipidomic profiles may offer a more robust predictive model. This multi-marker approach can provide valuable insights into the complex interplay between metabolism, inflammation, and immune response in COVID-19 pathogenesis.

Acknowledgement

None.

Conflict of Interest

None.

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