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# Covid-19 New-Onset Hypertension and Role of Spike Proteins and Variants

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## Introduction

The COVID-19 pandemic has disrupted lives across the globe, not only with its acute respiratory symptoms but also with its seemingly far-reaching impact on various organ systems. While much attention has been focused on respiratory issues, emerging evidence suggests a specific effect of the SARS-CoV-2 virus on blood pressure during and after the acute phase of infection. In this article, we explore the connection between COVID-19 and hypertension, shedding light on the higher incidence of new-onset hypertension in patients with COVID-19 compared to contemporary controls. Hypertension, or high blood pressure, is a well-known risk factor for severe outcomes in COVID-19 patients. However, recent research has unearthed a more intricate relationship between SARS-CoV-2 and blood pressure.

## Description

One striking revelation is the heightened incidence of new-onset hypertension in COVID-19 patients. Studies have shown that approximately 9% of individuals infected with the virus develop hypertension during or after their battle with COVID-19, a notable contrast to the 5% rate observed in contemporary controls. This increased risk of hypertension is a cause for concern, as hypertension is associated with a plethora of cardiovascular complications, including heart attacks and strokes. To understand this phenomenon, researchers have delved into the virus's mechanisms. SARS-CoV-2, like other coronaviruses, utilizes spike proteins to enter human cells. The spike proteins bind to ACE2 (angiotensin-converting enzyme 2), a receptor found on the surface of many cell types, including those lining blood vessels [1].

During COVID-19 infection, the interaction between spike proteins and ACE2 may have a specific effect on blood pressure. This interaction potentially triggers a cascade of events that leads to the constriction of blood vessels and an increase in blood pressure. While more research is needed to pinpoint the exact mechanisms, this theory provides a compelling insight into the connection between COVID-19 and hypertension. The evolution of SARS-CoV-2 has given rise to new variants, some of which have developed mutations that may increase their adhesivity to ACE2 receptors. These mutations could heighten the risk of new-onset hypertension in infected individuals. The implications of these variants in terms of cardiovascular health are an evolving area of study that warrants close monitoring and research [2].

The connection between COVID-19 and blood pressure is multifaceted and still not fully understood. However, the evidence is mounting, and the relationship between SARS-CoV-2, new-onset hypertension, spike proteins, and ACE2 receptors is becoming increasingly clear. As the pandemic unfolds, medical professionals and researchers are working tirelessly to unravel the intricacies of this viral impact on our cardiovascular health. For individuals who have contracted COVID-19 or are at risk, it is crucial to monitor blood pressure and maintain a healthy lifestyle. Early detection and management of hypertension can significantly reduce the risk of cardiovascular complications. As researchers continue to explore the complex relationship between the virus and blood pressure, knowledge and vigilance remain our best tools in navigating these uncertain times [3].

The SARS-CoV-2 virus, responsible for the COVID-19 pandemic, has proven to be a complex adversary with a wide range of effects on the human body. Beyond its well-known respiratory symptoms, this virus has exhibited a potential impact on blood pressure through an intriguing mechanism involving spike proteins and ACE2 receptors. Moreover, the emergence of new variants adds another layer of complexity, as some of these mutations may enhance the adhesivity to ACE2 receptors, thus increasing the risk of new-onset hypertension. In this article, we delve into the evolving understanding of how the interaction between spike proteins and ACE2 remains a plausible mechanism underlying the rise in blood pressure, with a focus on the role of viral variants [4].

To infect human cells, SARS-CoV-2 employs spike proteins, which protrude from its surface and bind to angiotensin-converting enzyme 2 (ACE2) receptors found on the surface of various cells, including those lining blood vessels. ACE2 is a critical component of the renin-angiotensin-aldosterone system, which plays a crucial role in regulating blood pressure. The renin-angiotensin-aldosterone system (RAAS) helps maintain blood pressure and fluid balance within the body. ACE2 is responsible for converting angiotensin II, a potent vasoconstrictor that narrows blood vessels, into angiotensin (1-7), a peptide that promotes vasodilation and lowers blood pressure. The balance of these two components is essential for cardiovascular health [5].

When the SARS-CoV-2 spike proteins bind to ACE2 receptors, they may disrupt the equilibrium of the RAAS. This interaction could lead to an increase in angiotensin II levels, which results in vasoconstriction and a rise in blood pressure. Additionally, inflammation and oxidative stress induced by the viral infection further contribute to this effect. The evolving nature of the virus has given rise to new variants, some of which have developed mutations in their spike proteins. These mutations can influence the virus's ability to bind to ACE2 receptors. Certain mutations in spike proteins may increase their adhesivity to ACE2 receptors. This heightened affinity can lead to a more effective invasion of human cells. As a result, individuals infected with these variants may experience more significant disruptions to the RAAS, potentially resulting in elevated blood pressure.

## Conclusion

The risk of new-onset hypertension in individuals infected with these variants is a growing concern. As research continues to unveil the implications of these mutations, it is evident that monitoring the interaction between viral variants and blood pressure is crucial to understanding the full scope of COVID-19's effects on the cardiovascular system. The interaction between spike proteins and ACE2 receptors remains a plausible mechanism underlying the increase in blood pressure observed in some COVID-19 patients. As the virus evolves and new variants emerge, the risk of new-onset hypertension becomes more complex. Researchers continue to explore the intricate

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relationship between SARS-CoV-2, ACE2, and hypertension, seeking a more comprehensive understanding of these effects. This knowledge is vital for healthcare professionals and individuals alike, as it can lead to more effective strategies for managing and preventing COVID-19-related cardiovascular complications.

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## Conflict of Interest

None.

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