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Exploring the Potential of Hyperbaric Oxygenation Therapy as an Intervention in Post-Myocardial Infarction Patients

Jeffrey V. Esteron*, Aylwin S. Geanga

College of Nursing , Angeles University Foundation, Pampanga, Philippines

DESCRIPTION

Myocardial Infarction (MI), commonly known as a heart attack, necessitates immediate attention due to its severity. In recent years, Hyperbaric Oxygen Therapy (HBOT) has emerged as a promising treatment option for MI. HBOT involves administering pure oxygen in a pressurized environment and has shown potential benefits in various medical conditions, including MI. This paper explores the effects of HBOT on cardiac function, oxidative stress, inflammation, and autophagy in cardiomyocytes post-MI. HBOT has been found to reduce inflammatory markers, improve cardiac function, balance oxidative stress, modulate autophagy, and promote angiogenesis. These combined effects contribute to the cardio protective benefits of HBOT in the context of MI. The paper suggest that HBOT holds promise as an adjunctive therapy for enhancing recovery and improving outcomes in patients who have experienced an MI.

Myocardial Infarction (MI), frequently referred as a heart attack, is a severe medical condition that necessitates prompt and immediate attention. The aftermath of an MI presents survivors with various obstacles on their path to recovery, such as compromised cardiac function and diminished quality of life. Standard treatment for MI includes medications to dissolve blood clots, reduce blood pressure, and prevent further damage to the heart. In certain instances, it may be required to utilize procedures like angioplasty or bypass surgery to reinstate proper circulation of blood to the heart. Although these treatments can effectively decrease the immediate risk of death, many patients continue to experience long-term complications including heart failure, arrhythmias, and reduced quality of life. Recently, a ray of hope has emerged in the form of Hyperbaric Oxygenation Therapy (HBOT). This groundbreaking treatment offers the possibility of rejuvenating the heart and restoring vitality to those who have experienced the devastating effects of an MI.

The utilization of Hyperbaric Oxygen Therapy (HBOT) involves the administration of pure oxygen in a controlled, pressurized environment and has been the focus of research due to its potential benefits in treating array of medical conditions, including Myocardial Infarction (MI). HBOT reduces the inflammatory response in cardiocytes post-myocardial infarction by decreasing pro-inflammatory proteins, apoptosis, and oxidative stress, while increasing growth factors, angiogenesis, and neovascularization. Hyperbaric Oxygen Therapy (HBOT) successfully decreases the concentrations of inflammatory markers in individuals who have suffered from a myocardial infarction (MI). Specifically, HBOT decreases the levels of inflammatory cytokines such as IL-6, IL-1β, TNF-α, and CRP in both the serum and myocardial tissues. Moreover, HBOT achieves this reduction by modulating the inflammatory response. It achieves this by inhibiting the TLR4-NF-κB pathway, which plays a crucial role in the inflammatory process [1,2].

HBOT leads to cardiac function improvements in myocardial infarction (MI) models. Specifically, HBOT has been shown to enhance metrics such as left ventricular Ejection Fraction (EF) and Fractional Shortening (FS), which are important indicators of cardiac performance. Additionally, HBOT plays a role in reducing myocardial enzyme levels and apoptosis of cardiomyocytes [2]. These effects contribute to better cardiac outcomes, as they help protect and preserve the health of the heart muscle. Likewise, HBOT has been found to impact oxidative stress and antioxidant balance. By reducing oxidative markers and increasing antioxidant potential, HBOT indirectly supports the reduction of inflammation [3,4]. This

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Corresponding author: Jeffrey V. Esteron, College of Nursing, Angeles University Foundation, Philippines; E-mail: esteron.jeffrey@auf.edu.ph

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balance in oxidative stress can have significant implications for overall cardiac health and function. Apart from influencing cardiac function and oxidative stress, HBOT also modulates autophagy in cardiomyocytes [1]. By activating the mTOR pathway, HBOT helps to reduce excessive autophagy [1-3]. This modulation of autophagy is particularly beneficial in managing Myocardial Ischemia-Reperfusion Injury (MIRI), a condition that can occur following reperfusion therapy after a heart attack.

CONCLUSION

Hyperbaric oxygen therapy significantly impacts the inflammatory response in cardiocytes post-myocardial infarction by reducing inflammatory markers, improving cardiac function, balancing oxidative stress, modulating autophagy, and promoting angiogenesis. These combined effects contribute to the overall cardio protective benefits of HBOT in the context of myocardial infarction.

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