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Deterioration of Left Ventricular Diastolic-Systolic Coupling in Healthy Humans due to Myocardial Steatosis

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Abstract

A growing body of evidence points towards the involvement of myocardial steatosis in the development of left ventricular diastolic dysfunction, although conclusive proof in humans is hampered by complicating coexisting conditions. To address this, we employed a 48-hour food restriction protocol to induce an acute elevation in Myocardial Triglyceride (mTG) content quantified using 1H magnetic resonance spectroscopy in a cohort of 27 young and healthy volunteers (comprising 13 men and 14 women). The results of the fasting regimen exhibited a remarkable over threefold rise in mTG content (P<0.001). Interestingly, the early diastolic circumferential strain rate (CSRd), a marker of diastolic function, remained unaffected following the 48-hour fasting intervention. However, a noteworthy elevation in systolic circumferential strain rate was observed (P<0.001), indicating a decoupling between systolic and diastolic phases. This phenomenon of uncoupling was further substantiated by an additional experiment involving 10 individuals, where the administration of low-dose dobutamine (2 µg/kg/min) resulted in a similar alteration in systolic circumferential strain rate as the one observed during the 48-hour food restriction. Moreover, this change was accompanied by a proportionate increase in CSRd, thereby preserving the synchronization between the two metrics. The collective findings of this study underscore the role of myocardial steatosis in instigating diastolic dysfunction by disrupting the coupling between diastole and systole in the context of healthy adult subjects. Furthermore, these findings postulate a potential contributory role of steatosis in the progression of cardiovascular ailments.

Keywords: Preclinical evidence • Heart disease • Myocardial steatosis

Introduction

Cardiovascular diseases continue to be a leading cause of morbidity and mortality worldwide. Among these, myocardial steatosis, characterized by excessive accumulation of fat within the heart muscle, has gained attention due to its potential implications on cardiac function. Emerging research suggests that myocardial steatosis may play a significant role in impairing left ventricular diastolic-systolic coupling in healthy individuals, thereby affecting overall cardiac performance [1].

Understanding myocardial steatosis

Myocardial steatosis refers to the deposition of fatty acids within the myocardial cells. While some fat within the heart is normal and even necessary for energy storage, an excessive accumulation of fat can lead to structural and functional abnormalities. This phenomenon has long been associated with obesity, insulin resistance, and metabolic syndrome [2].

Diastolic-systolic coupling and cardiac function

The normal functioning of the heart relies on a delicate balance between the two main phases of the cardiac cycle: diastole and systole. During diastole, the heart relaxes and fills with blood, allowing for proper oxygen and nutrient delivery to the myocardium. Systole, on the other hand, involves the contraction of the heart muscle to eject blood into the systemic circulation. The coupling of these two phases is crucial for maintaining efficient cardiac output and overall cardiovascular health [3].

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Description

Impairment of diastolic-systolic coupling

Recent studies have shed light on the potential negative impact of myocardial steatosis on left ventricular diastolic-systolic coupling. This impairment disrupts the coordination between relaxation and contraction phases of the cardiac cycle, leading to inefficient blood ejection and inadequate filling of the heart chambers during diastole [4].

Several mechanisms contribute to the impaired diastolic-systolic coupling observed in individuals with myocardial steatosis:

Altered calcium handling: Excessive fat accumulation within myocardial cells interferes with calcium handling, a critical process that regulates myocardial contraction. Dysregulated calcium levels can lead to impaired contractility and relaxation of the heart.

Mitochondrial dysfunction: Myocardial steatosis is associated with mitochondrial dysfunction, which disrupts energy production within the heart. This energy deficit can impact the heart's ability to contract and relax effectively.

Inflammation and fibrosis: Fatty infiltration triggers an inflammatory response and promotes fibrotic tissue remodeling within the heart. These changes can alter the architecture of the myocardium, further compromising cardiac function.

Cellular oxidative stress: Excess fat in the myocardium can lead to oxidative stress, causing damage to cellular structures and impairing overall heart function.

Clinical implications: The impairment of left ventricular diastolic-systolic coupling due to myocardial steatosis has significant clinical implications. Individuals with this condition may experience reduced exercise tolerance, increased susceptibility to heart failure, and an elevated risk of arrhythmias. Furthermore, this impairment may serve as an early indicator of cardiovascular dysfunction even in apparently healthy individuals, making it a potential target for preventive interventions [5,6].

Conclusion

Myocardial steatosis, characterized by the accumulation of fat within the heart muscle, can exert a detrimental impact on left ventricular diastolic-systolic coupling in healthy humans. The disruption of this crucial coupling mechanism can compromise cardiac function, leading to a range of cardiovascular complications. As research continues to unveil the intricate relationship between myocardial steatosis and cardiac performance, healthcare professionals must remain vigilant in identifying and managing this condition to promote better heart health and prevent the onset of cardiovascular diseases.

Acknowledgement

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Conflict of Interest

None.

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