Clinical Cardiology Reserch and Reports

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Case Report **Clinical Cardiology Research and Reports** **Open Access**

A Cross Sectional Study of Echocardiography on Myocardial Metabolism

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Abstract

Coronary Heart Disease patients remain at a high risk for reduced ejection fraction and heart failure many years after the onset. Heart failure is a common cause of hospitalization among coronary heart disease patients. Early interventions for the associated risk factors are crucial. Previous studies have reported that diabetes, hypertension, obesity, and multi-vessel disease are all independent risk factors for cardiac insufficiency in the late stages of coronary heart disease. It has also been reported recently that coronary heart disease patients with hypertriglyceridemia may be at a higher risk for new cardiac events. However, it is unknown whether hypertriglyceridemia alters the myocardial structure in coronary heart disease patients in the early stages of heart failure.

Keywords: Essential hypertension; echocardiography; diastolic dysfunction; atherosclerosis

Introduction

Modern environment and lifestyle, such as increased light exposure and activities during night and widespread use of electronic media and mobile devices, not only deprive humans of sufficient sleep but also considerably disturb the regularity of sleep behaviors. An adequate amount of sleep, which is essential for global rejuvenation of the human body, plays a central role in normal functioning of metabolism and energy homeostasis. As a result, reduced quantity of sleep has been associated with higher risk of obesity, metabolic syndrome, and diabetes in numerous previous studies. However, less is known regarding the impact of irregular sleep (i.e., high day-to-day variability in sleep duration and timing), which could negatively influence metabolic health through sleep debt caused by nights of sleep deprivation not compensated for by nights of extended sleep and/or by disruption of circadian rhythms.

To date, a majority of epidemiologic studies on sleep health and cardiometabolic outcomes has examined average sleep duration, with less research on variations in sleep duration and timing. It is now well established that an inherent circadian rhythmicity is a universal mechanism underlying various biologic processes, including metabolism. Clear circadian patterns are exhibited from gene expressions to downstream circulating metabolites (, as well as secretion of hormones involved in metabolic regulation.

The main energy substrate of adult cardiomyocytes for their contractility are the fatty acids. Its metabolism generates high ATP levels at the expense of high oxygen consumption in the mitochondria. Under low oxygen supply, they can get energy from other substrates, mainly glucose, lactate, ketone bodies, etc., but the mitochondrial dysfunction, in pathological conditions, reduces the oxidative metabolism. In consequence, fatty acids are stored into epicardial fat and its accumulation provokes inflammation, insulin resistance, and oxidative stress, which enhance the myocardium dysfunction. Some therapies focused on improvement the fatty acids entry into mitochondria have failed to demonstrate benefits on cardiovascular disorders. Oppositely, those therapies with effects on epicardial fat volume and inflammation might improve the oxidative metabolism of myocardium and might reduce the cardiovascular disease progression. This review aims at explain (a) the energy substrate adaptation of myocardium in physiological conditions, (b) the reduction of oxidative metabolism in pathological conditions and consequences on epicardial fat accumulation and insulin resistance, and (c) the reduction of cardiovascular outcomes after regulation by some therapies.

Diet and Physical Activity

Diet and physical activity are the major factors involved in fatty acids or glucose oxidation. Fasting conditions increase released fatty acids by adipose tissue and their oxidation by cardiomyocytes. This process is associated with the inhibition of glucose oxidation. However, after postprandial state, the high insulin and glucose increment improve the intracellular glucose through insulin-dependent or independent glucose transporters (GLUT) [and there is an inhibition of fatty acids oxidation. Glucose can either be degraded into lactate, stored into glycogen, or oxidized for getting energy.

Circadian Rhythm

The center of circadian clock is in the hypothalamus, specifically in the suprachiasmatic nucleus. It is a 24-hour cycle which helps cells to anticipate the needs of the time of day and is regulated through feedback loops and zeitgebers as light or temperature. The gene expression oscillations in normal intact hearts can be modulated by extracellular (i.e., neurohumoral, workload, and circulating nutrients) and/or intracellular (i.e., circadian clock) stimulus. During the night, there is a reduction of vascular tone, oxygen demand, heart rate, and cell death. The circadian cycle determines the expression of some genes.

Hormonal Regulation

During the postprandial period, there is an increase in glucose, and in consequence, insulin is secreted by the pancreas. After insulin binding to its receptor, the kinase signaling (PKB/Akt) is activated and glucose transporter is translocated from cytosol into membrane. Then, glucose goes inside of cells.

Ageing

Adult hearts need a high mitochondria number due to the oxidative phosphorylation activity. During ageing, fatty acids transporters in the sarcolemma are enhanced, but the inability to oxidize them causes lipid accumulation and lipotoxicity, such as palmitic acid, acylcarnitine, unesterified cholesterol, lysolecithin, ceramide, and diacylglycerides. These

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lipids can trigger apoptosis and inflammation and can emphasize mitochondrial dysfunction.

Conclusion

Cardiac abnormalities were common in subjects with metabolic syndrome, predominantly affecting the left ventricular mass, diameter and left atrial volume. Early life style modifications are essential to prevent these complications.

References

1. Orfali K.A., Fryer L.G.D., Holness M.J., Sugden M.C. Interactive effects of insulin and triiodothyronine on pyruvate dehydrogenase kinase activity in cardiac myocytes. J. Mol. Cell. Cardiol. 1995;27:901–908.
2. Portman M.A. Thyroid Hormone Regulation of Heart Metabolism. Thyroid. 2008;18:217–225.
3. Barros R.P.A., Gustafsson J.Å. Estrogen receptors and the metabolic network. Cell Metabol. 2011;14:289–299.
4. Walejko J.M., Antolic A., Koelmel J.P., Garrett T.J., Edison A.S., Keller-Wood M. Chronic maternal cortisol excess during late gestation leads to metabolic alterations in the newborn heart. Am. J. Physiol. Metab. 2019;316:E546–E556.
5. Baggio L.L, Yusta B., Mulvihill E.E., Cao X., Streutker C.J., Butany J., Cappola T.P., Margulies K.B., Drucker D.J. GLP-1 Receptor Expression within the Human Heart. Endocrinology. 2018;159:1570–1584.
6. Lesnefsky E.J., Chen Q., Hoppel C.L. Mitochondrial Metabolism in Aging Heart. Circul. Res. 2016;118:1593–1611.

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