Among type 2 diabetic mice, a ketogenic diet improves cardiac dysfunction by balancing mitochondrial dynamics and inhibiting apoptosis.

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Introduction

Although the ketogenic diet (KD) has been widely utilised in clinical studies been proved to have an anti-diabetic effect, the underlying processes are still unknown. Their goal was to look into the effects of the KD on cardiac function in mice, as well as the underlying mechanisms. In this study, mice were fed either a normal diet (ND) or a high-fat diet (KD). Eight weeks after KD treatment, fasting blood glucose, cardiac function and morphology, mitochondrial dynamics, oxidative stress, and apoptosis were all examined. The KD improved glycemic control and protected against diabetic cardiomyopathy, and better mitochondrial activity and lower oxidative stress are found in hearts when opposed to the ND. Diabetes mellitus (DM) is a metabolic disease caused by a combination of hereditary and environmental variables those results to persistent hyperglycaemia as a result of impaired insulin production, insulin resistance, or both. It is commonly acknowledged that the rising prevalence of diabetes mellitus is just a severe public health issue. DCM is a major consequence of diabetes mellitus that is marked by cardiac remodelling and dysfunction but not by coronary artery disease, hypertension, or substantial stroke [1].

Animals

All tests on animals were carried out in accordance with the National Institutes of Health Guidelines for the Care and Use of Laboratory Animals, and the Chongqing Medical University Committee on Animal Care authorised all methods. After 8 weeks of nutritional treatment, the cardiac and mitochondrial functioning were evaluated. Serum glucagon and insulin levels were measured in fasting blood samples.

The architecture and function of the heart are examined

Histological examinations was carried out as described earlier [2]. With hematoxylin and eosin staining, cardiac tissues were paraffin embedded and sectioned at a thickness of 5 m. A Vevo 770 High-Resolution Micro-Imaging System was used to perform echocardiography on the mice to measure heart function.

ATP, BHB, serum glucagon, insulin, and lipid profile measurements

A BHB level in the ventricle were determined using -Hydroxybutyrate Assay Kit kits and the cardiac ATP content was assessed with a luciferase assay kit (Promega) according to the manufacturer's instructions. The commercial kit was used to measure serum glucagon and insulin. Specific commercial kits were used to measure free fatty acid, triglyceride, and total cholesterol levels [3].

Growth of cardiomyocytes and assessment of viable cells

Myocytes from 1-day-old SD rats were extracted and cultivated as described before. In a humid chamber with 95 percent air and 5 percent CO_2, cells were maintained in DMEM supplemented with 10% FBS at 37 °C in a humidified incubator with 95 percent air and 5 percent CO_2 [4]. An MTT cell Cytotoxicity Assay kit was used to determine the vitality of the cell. Around 570 nm, the absorbance was measured. 6 different experiments were carried out [5].

Discussion

Glitches in the endocrine and metabolic systems play a key role in the initiation of diabetes. Through the PI3K pathway, the ketone body BHB was discovered to have an anti-apoptotic impact on diabetic myocardial. Such data, along with those showing that feeding mice the KD preserved mitochondrial function and reduced peroxidation, point to the possibility of using ketone bodies as a pro treatment. In a recent animal study, it was discovered that the KD increases the lifespan and health span of adult mice.

Conclusion

Dietary intervention has been advocated as a primary technique in the clinical management of diabetes mellitus. Another study indicated that KD enhances memory and decreases midlife mortality in ageing rats. In mice, providing a high-fat diet (45 percent of calories from fatty acids) for two months after transverse aortic constriction (TAC) surgery improved fatty acid consumption and avoided the development of heart failure, according to prior research.

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