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Optimizing the Effectiveness of the Ketodiet in Parkinson`S Disease

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INTRODUCTION: The goal of this review was to assess the effectiveness of ketogenic diets on the therapy of neurodegenerative diseases. The ketogenic diet is a low-carbohydrate and fat-rich diet. Its implementation has a fasting-like effect, which brings the body into a state of ketosis. The ketogenic diet has, for almost 100 years, been used in the therapy of drug-resistant epilepsy, but current studies indicate possible neuroprotective effects. Thus far, only a few studies have evaluated the role of the ketogenic diet in the prevention of Parkinson's disease (PD) and Alzheimer's disease (AD). Single studies with human participants have demonstrated a reduction of disease symptoms after application. The application of the ketogenic diet to elderly people, however, raises certain concerns. Persons with neurodegenerative diseases are at risk of malnutrition, while food intake reduction is associated with disease symptoms. In turn, the ketogenic diet leads to a reduced appetite; it is not attractive from an organoleptic point of view, and may be accompanied by side effects of the gastrointestinal system. Parkinson disease is the most common form of parkinsonism, a group of neurological disorders with Parkinson disease-like movement problems such as rigidity, slowness, and tremor. More than 6 million individuals worldwide have Parkinson disease. Parkinson's disease (PD) is the second most prevalent neurodegenerative disease, and oxidative stress and mitochondrial dysfunction play a major role in the pathogenesis of PD. Since conventional therapeutics are not sufficient for the treatment of PD, the development of new agents with anti-oxidant potential is crucial. Parkinson's disease (PD) is a common neurodegenerative disease characterized by motor symptoms and non-motor symptoms, and affects millions of people worldwide. Growing evidence implies β-Hydroxybutyrate (BHB), one of the ketone bodies generated by ketogenesis, plays a neuroprotective role in neurodegenerative diseases. We aimed to verify the anti-inflammatory effect of BHB on PD and further explore potential molecular mechanisms. A growing body of evidence supports the idea that mitochondrial dysfunction might represent a key feature of Parkinson's disease (PD). Central regulators of energy production, mitochondria, are also involved in several other essential functions such as cell death pathways and neuroinflammation which make them a potential therapeutic target for PD management. Interestingly, recent studies related to PD have reported a neuroprotective effect of targeting mitochondrial pyruvate carrier (MPC) by the insulin sensitizer MSDC-0160. Numerous studies indicate a high level of abnormal glucose tolerance in patients with Parkinson's disease [Sandyk R. The relationship between diabetes mellitus and Parkinson's disease //Int. J. Neurosci., 69 (2018), pp. 125-130]. At the same time, some authors report that clearly impaired glucose metabolism occurred in

approximately 20% of patients [Marques A., Dutheil F., Durand E., et al. Glucose dysregulation in Parkinson's disease: too much glucose or not enough insulin? Parkinsonism Relat. Discord. 2018; 55: 122–127]. The ketogenic diet (KD) is a high-fat, low-carbohydrate and adequate-protein diet that has gained popularity in recent years in the context of neurological diseases (NDs). The complexity of the pathogenesis of these diseases means that effective forms of treatment are still lacking. Conventional therapy is often associated with increasing tolerance and/or drug resistance. Consequently, more effective therapeutic strategies are being sought to increase the effectiveness of available forms of therapy and improve the quality of life of patients. For the moment, it seems that KD can provide therapeutic benefits in patients with neurological problems by effectively controlling the balance between pro- and antioxidant processes and pro-excitatory and inhibitory neurotransmitters, and modulating inflammation or changing the composition of the gut microbiome. In this review we evaluated the potential therapeutic efficacy of KD in epilepsy, depression, migraine, Alzheimer's disease and Parkinson's disease. In our opinion, KD should be considered as an adjuvant therapeutic option for some neurological diseases.

Aim: to study the role of ketogenesis on the clinical progression of PD and the effect of a low-carbohydrate ketogenic intervention on the cognitive functions of patients with PD.

MATERIALS AND METHODS: 103 patients with PD were selected, of which 73 patients, after explanations and with their consent, were prescribed an 8-week keto diet along with the traditional method of treatment; 30 patients receiving conventional drug therapy. Inclusion criteria included either a total score of 20 to 25 on the Montreal Cognitive Assessment (MoCA), reflecting mild overall cognitive decline, or a score of <5 on the MoCA Delayed Recall Test, the latter being indicative of hippocampal volume loss as has been observed in verbal memory deficits in the early stages of PD.

In addition, patients were required to be on a stable antiparkinsonian drug regimen for at least six weeks and no dose adjustments were required during the study period. Each participant was also required to have a study partner (usually a spouse) who provided education on the assigned diet and assisted in adherence to the protocol.

Exclusion criteria included a diagnosis of dementia, a second neurological disorder, past or current substance abuse, unstable mental health conditions, diabetes, or regular use of glucose- or insulin-modulating medications, or medications that may affect cognitive outcome measures such as benzodiazepines and stimulants.

Research methods. To achieve the goal of the study and solve the assigned problems, general clinical, laboratory and instrumental research methods were used

DISCUSSION AND RESULTS: From scientific research

the expected innovation is for the first time the development of ketone bodies in Parkinson's disease and the role of markers of the development of cognitive disorders in these patients is proven;

the relationship between metabolic disorders and the development of PD in patients is determined;

a prognostic matrix is developed for the development of the clinical course of PD in patients taking into account gender characteristics, anthropometric data, the level of ketone bodies, glucose and insulin; A prognostic matrix for the progression of the clinical course of PD in patients will be developed, taking into account gender characteristics, anthropometric data, levels of ketone bodies, glucose and insulin;

An assessment will be made of the effectiveness of prescribing a keto diet in the complex treatment of patients with PD.

Conclusion: Ketogenic therapies have promise in PD, AD, and MCI for symptom improvement although larger studies are needed to support their implementation in clinical practice

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