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Cancer Research 2020: Effects of ketogenic diet on brain tumor metabolism- Motta, J.- University of Florida

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Objective: Establish dietary-induced ketosis in one oncology patient to determine if a ketogenic state would decrease glucose availability to certain tumors, thereby potentially impairing tumor metabolism without adversely affecting the patient's overall nutritional status.

Introduction: Human threatening glioma is a consistently lethal illness due, to some degree, to the constraints of as of now accessible medicines which incorporate medical procedure, chemotherapy and radiation treatment. Normal endurance of patients with glioblastoma multiforme (GBM) is 1.5 years, and tumors of the focal sensory system are the most widely recognized strong tumor in the pediatric populace. It is in this way of vital significance that new remedial techniques for mind malignant growth patients be grown, particularly those that can improve the adequacy of current treatment alternatives without harming ordinary cerebrum tissue. Advances in our comprehension of the science of these tumors have prompted an expansion in the quantity of focused treatments in preclinical and clinical preliminaries. While these treatments may demonstrate to some degree successful, the heterogeneity of this tumor regularly blocks the focused on atoms from being found on all cells in the tumor in this manner lessening the viability of these medicines. Conversely, one characteristic shared by for all intents and purposes all tumor cells is adjusted metabolism. The term "metabolic redesigning" has been utilized to depict metabolic changes that can happen in malignant growth cell and oncogene related pathways are presently known to cross with, and modify metabolic pathways.

The KD is a high-fat low protein/starch diet used to treat stubborn epilepsy. It has been appeared to have neuro-protective impacts and there are presently studies to decide its adequacy for various neurological issue, including epilepsy, Alzheimer's infection, Parkinson's sickness, rest issue, migraine, awful mind injury, amyotrophic sidelong sclerosis, torment and chemical imbalance (Masino and Ruskin, 2013; Gano et al., 2014). The KD expands blood ketones and diminishes blood glucose by mimicking the physiological reaction to fasting, consequently prompting high paces of unsaturated fat oxidation and an expansion in the creation of acetyl coenzyme An (acetyl-CoA).

Case report: One pet male with advanced stage brain tumor. Patient was followed as outpatients for 1 year. Ketosis was maintained by consuming a 60% medium chain triglyceride oil-based diet. Some cancers cannot metabolize ketone bodies, due to mitochondrial dysfunction and down-regulation of enzymes necessary for ketone utilization. Tumor glucose metabolism was assessed by RMI, before and following the trial period.

As radiation-initiated tumor slaughtering is known to uncover the resistant framework to a more noteworthy decent variety of tumor antigens, it is conceivable that the KD as an adjuvant attempts to enlarge the impact of radiation partially by upgrading invulnerability against GBM. The assortment of impacts seen when glucose in brought down or potentially ketones are expanded recommends this may likewise potentiate different treatments, including more up to date safe and focused on treatments.

Results: Within 7 days of initiating the ketogenic diet, blood glucose levels declined to low-normal levels and blood ketones were elevated. Results of RMI indicated a 50% average decrease uptake at the tumor site in subject. The patient exhibited significant clinical improvements in seizures, neurological deficits and new skill development during the treatment. He still continues used the ketogenic diet remaining free of disease progression.

Conclusion: While this diet does not replace conventional antineoplastic treatments, these preliminary results suggest a potential for clinical application which merits further research

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