Healthcare and nursing

Your Name (First M. Last)

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Beneficial effects of Ketogenic Diet

**Introduction**

It is asserted that metabolic therapies and dietary fibers play a significant role in overcoming different kinds of disease such as epilepsy, neurological diseases, neurotrauma, Alzheimer’s disease, brain disorders, headache, sleep disorders pain, autism, and other multiple scleroses. The treatment of disease through diary fires signifies a natural essence to treatment highlighting that gaps and barriers in adequate intake of nutrients and other ingredients required by the body are necessary to live healthy lives. The role of diet in treating and ameliorating symptoms of different diseases asserts that these diseases stem from the lack of effectiveness of certain pharmacological therapies along with an intrinsic appeal that could facilitate the implication of natural treatment (Soeters, et al., 2019).

Research has highlighted that any shift and change in the diet pattern may cause an interference in the impact of the nutrients on the body organs leading to a difference in the effect of diet. One of the diets that can help to overcome and mitigate several health issues is Ketogenic diet. It is defined as a diet that is very low in carb with the comparatively high fact that shares a lot of similarities with diets such as Atkins and other low carbohydrate diets. When an individual takes a Ketogenic diet, the human body becomes more efficient at burning fat for attaining energy (Gasior, et al., 2006). KD is responsible for the conversion of fats into ketones in the liver that could supply energy for the adequate functioning of the brain. It would not be wrong to say, that the aim of having a Ketogenic diet is to get more calories from the fats and protein and less from the carbohydrates.

An analysis of the Ketogenic diet highlights and asserts that when an individual eats less than 50 runs of carb in a single day, and if body lack sugar, then this diet would be quickly used. After that, the protein and fat for energy would start to break that will help to reduce weight and this process is called ketosis (Klement, et al., 2017). It is more added that the Ketogenic diet is a very short-term diet that is more concerned with weight loss rather than the pursuit of other health benefits (Dashti, et al., 2007). It is asserted that Ketogenic diet is responsible for causing a massive reduction in the insulin labels as well as the blood sugar, take into account that it is more like an illustration of health befits. There are three different versions of Ketogenic diet.

**Standard ketogenic diet**

It is a very low carb diet having moderate proteins and a high-fat diet. It comprises of about 75% fat with 5% carbon and 20% protein (Gasior, et al., 2006).

**Cyclic Ketogenic Diet**

This diet involves comparatively high periods of carbon feeds such as a normal proportion of 5 lactogenic days that are followed by 2 high carb days (Gasior, et al., 2006).

**Targeted ketogenic Diet**

This diet allows an individual to add carbohydrates to diet around workouts. High protein ketogenic diet is much similar to standard ketogenic diet because it has a high proportion of protein. This ratio comprises of about 60% fat, 5% carb, and about 35% proteins (Gasior, et al., 2006).

These ketones are then paired and united to form two different categories, one of the categories comprising of standard and high protein ketogenic diet is extensively studied and the other category comprises of cyclic and alerted ketogenic diet that is used by athletes’ other bodybuilders.

**Discussion**

Ketogenic Diet is now rated as one of the proven therapy that is used for drug-resistant epilepsy. It is asserted that the underlying mechanism of the ketogenic diet is assumed because of the anticonvulsant effects that are not understood by now, still a number of experiments are mounted for the analysis of neuroprotective properties and the collection of data that could support this use of ketogenic diet in different neurological disease. It is asserted that the patients who are suffering from refractory epilepsy have remained seizure-free on the Ketogenic diet for a time span of 2 years are observed to have a quick recovery. It is very uncommon for the clinics to observe that diet and the anticonvulsant medications are successfully discontinued without any recrudescence of seizures (Dashti, et al., 2007). Moreover, there are a number of review articles that explores the ideology that ketogenic diet can be used to treat neurological disorders outside of epilepsy. It would not be wrong to say that different roles are assigned to the analysis of the ketogenic diet at is in turn used to treat a number of diseases.

**Addressing current challenges with the help of KD Nutrition Strategy**

Since the 19 century, it was found that the diets having low carbohydrate and the ketogenic diet have been studied and then implemented to observe their role in the neurological disease where it was found that KD is capable of addressing major challenges of time. It is found effective in neurological diseases, weight loss, insulin resistance, cardiovascular disease, and cancer. Moreover, it was found that the evidence in support of implementing KD can act as a therapy to address various pathophysiological and chronic disease challenges in human health. It was revealed that these aspects have made a dramatic disaster in the past 15 to 20 years because all of the population determinants such as healthy, unhealthy, untrained individuals and athletes are increasingly aware of the research related to the use of KDs (Dashti, et al., 2007). A significant body of the research also illustrated that the nutritional and psychological ketosis that is also called circulating ketones between 0.5-5.0 mM has a clinical and performance application because of the certain signaling challenges and potent metabolic complications in humans. Moreover, a significant body of the work highlighted that there are much military relevant health and other reformate challenges that are affected by the nutritional ketosis (Klement, et al., 2017)**.**

**Body Compositions, Overweight and the rate of Obesity**

It is found that the high rates of obesity and overweight present numerous obstacles in military recruitments and physical readiness. Research has revealed that about two-thirds of the Americans in the military who have an age of 17 to 24 years are found to be unfit to be enlisted. This issue has been termed as national security concern because of the DoD recruitment and homeland defense (Klement, et al., 2019). Moreover, it is found that adequate diet plan and consultation with food insulators can help to mitigate the issues that are acting as a serious threat in adequate body regulation with an intention to improve nutrient addition in military personal. Moreover, body composition improvements such as a reduction in body fat can be one of the most consistent outcomes that are reported in the context of KD intervention. Moreover, primarily published researches have concluded that the KD is more superior to a low-fat diet that can help to lose weight with any explicit instruction that can reduce calories (Dashti, et al., 2007).

**Neuroprotective role performed by the ketogenic diet**

The research on a diet over last few years has highlighted that there are numerous mechanisms through which neuroprotective activity is performed by the ketogenic diet. A comprehensive discussion on the neuroprotective role has asserted that ketogenic diet is responsible for raising the production of ketones in the human body by reducing glucose level in blood. The progression and increase in the ratio of ketones is the product of fatty acid oxidation taking into account that polyunsaturated fatty acid regulates the excitability of neural members. As a result of excitability, the voltage channels of sodium and calcium block and as a result there is a reduction in inflammation because peroxisome proliferator receptors are activated. It also induces the expression of mitochondria that results in uncoupling of protein it reduces the oxygen species production. According to biomedical research, it is found that the ketone bodies are themselves found to be comprised of neuroprotective properties and as a result of such attributes ATP levels are risen along with a reduction of Reactive Oxygen Species. It is also found that ketogenic diet is also used to stimulate biogenesis of mitochondria that may result in the stabilization of the synaptic functions (Gasior, et al., 2006).

**Reduction in glycolytic flux**

It is significant to note that the glycolytic flux is another major significant feature of Ketogenic diet. Any reduction in glycolysis is one of the essentials features of calorie restriction that plays a vital role in the suppression of seizures. Moreover, it is also responsible for prolonging the lifespan of a number of species such as primates, although the link between calorie restriction and ketogenic mechanism is controversial still it is quite effective in the reduction of glucose in blood along with a prominent reduction in the glycolytic flux (Soeters, et al., 2019). It is asserted that there are several other significant mechanisms that can contribute to the neuroprotective consequences associated with calorie restriction. It also includes an improved and efficient function of mitochondria along with a reduction of oxidative stress and a decrease in the activity of pro-apoptotic factors (Klement, et al., 2017).

Several other factors are found to be associated with KD and they would result in the protective properties, numerous of such mechanism are related to the anticonvulsant effect of Ketogenic diet. These mechanisms could result in the prevention of neural injury, any neural dysfunction, and cellular homeostasis. It is important to note that this mechanism can also facilitate disease that are outside epilepsy such as examination of pleiotropic effects of metabolism-based therapies (Klement, et al., 2017).

**Ketogenic Diet as a barrier to epilepsy**

It is asserted that ketogenic diet can play a significant role in preventing the body from seizures in patients especially children who are diagnosed with refractory epilepsy. In 19 century, after the introduction of Ketogenic diet, it was used for treating children epilepsy. Although anticonvulsant medicine was already induced still, waned ketogenic diet was used to know if it treats epilepsy or not. In the early 1990s, a large number of efforts were made by the parents of the children who were suffering from epilepsy in the form sticking to kite genic diet and in past few years, surprising results were achieved (Klement, et al., 2017). According to research, it is highlighted that research by life scientists and the effort of clinical particles and rules have proved that a ketogenic diet can be used for the treatment of epilepsy. Today, the Ketogenic diet has become as one of an integral part of the armamentarium of critical epilepsy centers all over the world (Klement, et al., 2019).

**Mitigation of Aging**

Research has highlighted that gaining results in a decreased body function, in a number of cases and over many time, it out rate degeneration of neural circuits and neurons. It was assumed that an alteration in the energy metabolism with the Ketogenic Diet can help to get a control over the rate of degeneration of numerous neural structures and a lot of bodily functions would be slowed (Klement, et al., 2019). It is also asserted that Ketogenic Diet is capable of inducing certain differential morphological effects in the structures of the body, particular brain such as the hippocampus, Moreover, it was found that aging process is actually a consequence of the neuron vulnerability at a specific region of the brain. Research has shown that the medium-chain triglycerides, a type of ketogenic groups can facilitate the induction of detrimental synaptic changes in CA1 stratum molecular, side by side, and many beneficial effects can be added to the outer molecular layer of dentate gyros. Although some risk factors are found in the implication of the ketogenic diet, still it is found to play a significant role in overcoming aging by rescuing dysfunction that is associated with age-related mitochondria (Dashti, et al., 2007). It is found that ketogenic diet is capable of reducing oxidative stress and the downstream consequences that are associated with it by providing a valid rationale for mitigating the consequences of aging. It is also found that the ketogenic diet is also responsible to involve certain neuroprotective mechanisms that are able to ameliorate anthological aging, specifically in the context of neurodegeneration.

**The ketogenic diet is used for the treatment of Alzheimer Disease**

It is a common statement that ketone has greater neurological excitability in patients who are suffering from Alzheimer’s disease (Vidali, et al., 2015). In accordance with the information collected tom essential anthological processes of Alzheimer disease, it was found to involve neural degeneration along with the accumulation of certain abnormal cellular product such as tangles, and fibrillary plaques (Vidali, et al., 2015). However, recent evidence has highlighted that there is an alteration in the function of neural circuits and homeostasis of mitochondria because of Alzheimer’s disease. Moreover, there is a higher ratio of seizure in the patients who are suffering from Alzheimer’s disease as compared to the unaffected population and it is used as a rationale to justify that Ketogenic diet can play a positive and significant role in overcoming the process of aging. It is asserted that if ketone bodies are the primary sites for catering to aging and neurodegeneration in Alzheimer’s than the implementation of the Ketogenic Diet should be affected by age-related differences in the extraction as well as the production of ketones (Klement, et al., 2017).

Clinical studies have played a significant role in determining and judging the fact taking into account that MCT ketogenic Diet resulted in excelled cognitive abilities in patients having APOE4-negative patients of Alzheimer’s disease. In accordance with this research, it was found that the primary cognitive end point measures were the mean change from the baseline of the assessment of Alzheimer’s disease in terms of Global Scale and Scale Cognitive Subscale. Although it was a significant clinical improvement, still it was given secondary importance because ketogenic diet comprising of beta-hydroxybutyrate have shown to be strong protection against the toxic effects of beta-amyloid in the neurons (Klement, et al., 2019). It was more added that ketogenic diet was more capable of decreasing beta depiction. It is significant to note that other diets such as Mediterranean also showed a significant approach in dealing with Alzheimer’s disease because it promotes a reduction in the systematic inflammation thus it resulted in improved metabolic profiles (Klement, et al., 2017).

In recent research, a much closer link is found between epilepsy and Alzheimer’s disease. For example, it was found that the animal models of Alzheimer’s disease exhibited neural hyper excitability and it enhanced the propensity of seizure. Moreover, it was found that the animal models can play signified role in determining the anticonvulsant and cognitive exact of the ketogenic diet or some other dietary phenomena for example calorie restriction. According to a pathophysiological mechanism that was used to hypothesize the lineages in Alzheimer’s disease have the capacity to alter glucose metabolism and mitochondrial function, taking into account the accumulation of advanced glycan. Age accumulation is defined as a process that involves normal aging, which is accelerated in any individual who is suffering from Alzheimer's disease because the proteins are glycosylated none enzymatically and the cross-linking of proteins accentuates their dysfunction (Vidali, et al., 2015). There is another proposition made in terms of increased ROS and the formation of freedom that hampers mitochondrial function. It is asserted that in intriguing possibility refers to AFE inhibitors such as tenilsetam, aminoguanidine, and carnosine that could act in collaboration with a ketogenic diet or other antioxidants that can help in retarding the progression of Alzheimer’s disease that remained speculative at that time (Klement, et al., 2016).

In a nutshell, it is found that the Ketogenic Diet is one of the most effective treatments for the treatment of Alzheimer’s disease with the support of metabolism induced mechanism that is able to reduce oxidative stress along with neuro-inflammation. Moreover, ketogenic diet can enhance biogenetic profiles that are largely enhanced with mitochondrial functioning. It is also asserted that there are some cautions to be addressed as well such as discrepancies in terms of the unwanted and unpredicted side effects and the clinical efficacy.

**Controlling Parkinson Disease**

Research has highlighted that the Parkinson’s disease is defined as an excitotoxic degeneration of the dopaminergic neurons that are found in the substantia nigra which may lead to abnormal movements of different body parts to such an extent that it may distort cortical and other cognitive functions (Boison, et al., 2017). Adhering to the fact that ketone bodies are able to bypass the defects in mitochondrial complex in activity that has been implicated under the impression of Parkinson’s disease. A small study was conducted that comprises of 5 to 7 patients who were affected with Parkinson’s Disease showed an improved score when measured on standard Parkinson Disease rating scale, taking into account that placebo effect cannot be ruled out. The research on animals’ models highlighted that Parkinson Disease that was produced by 1 methyl 1 phenyl and 1236 tetrahydropyridine, BHHB administration ameliorated taking into account mitochondrial respiratory chain damage that resulted from that toxic (Ma, et al., 2018). Some additional evidence proved that ketogenic diet is much helpful in treating Parkinson’ s disease, that was proved by the in vitro experiments that demonstrate the protective effects of the substrates against mitochondrial respiratory chain dysfunction that was actually induced exogenously but complex I and II inhibitors rotenone and 3 nitro propionic acid. It is also found that there are certain anti-inflammatory actions of Ketogenic diet on the MPTP induced neurotoxicity (Klement, et al., 2019).

**Use of Ketogenic Diet in Amyotrophic Lateral Sclerosis**

ALS is termed as one of the diseases that is spreading rapidly due to the degeneration of motor neurons of the cortex and the anterior horn of spinal cord. It is found that in this disease the voluntary motor activity of the brain gradually deteriorates where an individual is left extremely weak, despite large cognitive functioning (Klement, et al., 2016). It is asserted that essential pathophysiological mechanism is not fully audited that is responsible for this relentless disorder. However, it is found to be similar to other neurodegenerative disorders along with the involvement of energy-producing systems that play a central role in this disease as well as mitochondrial dysfunction. Here KD is found to be one of the disjunctive treatments that can care about this devastating disease. However, the mouse model of ALS asserted that this disease is produced by the knocking out of gene encoding, the dismutase of copper/zinc superoxide that can cause progressive witness in the muscles. It may even cause death because of the respiratory failure. It’s important to note that the administration of KD to these mutant mics paved the way for both, functional improvements and histological empowerments. Here histological improvements are concerned with the high motor neuron counts while functional improvements are concerned with preserved motor function on the totored sets. Research has proven that 20% of cases of ALS have SOD1 mutations and thus there is a great possibility that KD may benefit the patients who are suffering from ALS (Ma, et al., 2018).

It is also important to note that there is no direct evidence of the positive effect of KD on all types of degenerative disease, taking into account the fact whether opting for this intervention is necessary for the required and protective effect of KD treatment or not (Ismayilova, et al., 2018).

**Curing Cancer**

According to a research, it has been proven that there are certain cells that exhibit most metallic rates and they are the ones who are more sensitive to the lack of energy that could facilitate metabolism in order to fuel and reengage their activity. It is asserted that a well-recognized biochemical phenomenon known as the Warburg effect defined the phenomena. In the theoretical context, highly metabolic cancer cells are deprived of usual fuel supply because of rapid division, taking into account that glucose is one of the energy supply that could be clinically therapeutic. Despite a detailed account on the cellular observation, it is found that KD has not been considered as a clinical treatment in the field of oncology (Klement, et al., 2017).

Research on animals has illustrated that the animals who got brain tumor under experimental conditions were provided by KD and it resulted in the fact that the growth of tumors got reduced under the impact of ketogenic diet (Ismayilova, et al., 2018). It was found that the reduction in growth rate of tumors was the result of calorie restriction rather than the induced KD ketosis (Gasior, et al., 2006). Many other researchers have found the same ideology under the effect of ketogenic diet taking into account that its induction can cause a reversion in the patterns in the non-tumor specimens and there is a reduction on the expression of a gene that is encoded by following single transduction pathway. However, there are certain growth factors that are found to be involved in the glioma growth. One of the interesting facts is to note that PPAR alpha gets activated by nutrients such as fatty acids that serve as a target for developing anti-cancer drugs that are capable of targeting metabolism in mitochondria (Soeters, et al., 2019).

**Carbohydrate restriction as a protective mechanism**

Research has highlighted that the carbohydrate restriction is one of the key aspects of the ketogenic diet. It is asserted that the role of decreased carbohydrates in the neuroprotection has been investigated by the use of 2 deoxy-D-glucose, termed as one of the glucose analogs that is not metabolized by glycolysis. One of the researchers has found that the administration of 2-DG in animals, i.e. adult rats in any nontoxic dose that is being imparted for almost 7 days will result in dramatic protection against the hippocampus damage and the neurological functional deficits that are induced by the seizure-inducing excitotoxin kainite (Vidali, et al., 2015). In addition, in this research, 2-DG was more protective against the glutamate-induced and other oxidative stress-induced neuronal death in the interior culture of cell. The authors of the research have also found that the reduced availability of glucose has induced and added stress protein that includes GRP78 and HSP70 that was meant to suppress ROS production and it will stabilize the intracellular calcium along with the maintenance of mitochondrial function (Klement, et al., 2019).

**Reducing anti-inflammatory effects**

It is asserted through research that the inflammatory mechanism has played a central role in the pathophysiology of both chronic and acute neurogenerative disorders. The research asserted that inflammation has been hypothesized to contribute and play a central role in the development of chronic epilepsy taking into account the fact that feeding at a high-fat diet has been associated with the effects on inflammatory mechanisms of the cells. Research has formulated and promoted a link between the anti-inflammatory diet, ketogenic diet and the modification of neurological disease that is found to be hugely tentative (Dashti, et al., 2007). Moreover, it is found that the intermittently tested rats have an increased expression of the cytokine interferon-y in the hippocampus. Moreover, it was further shown that the cytokine has conferred protection against the excitotoxic cell death. The high fatty acid load of ketogenic diet may also activate an anti-inflammatory mechanism such as the activation of peroxisome proliferator activated receptor alpha which may in return cause inhibitory effect in the proinflammatory transcription factors, nuclear factor-KB and the activation of protein-1(Ismayilova, et al., 2018).

**Regulation of programmed cell death**

It is asserted that ketogenic diet plays a significant role in the protection of various forms of cell death. One of the examples of this aspect is the fact that diet was protective against apoptotic cell death in mice that were induced by the glutamate receptor and the excitotoxins kainate. It was also evidenced by the reduction of markers of the apoptosis that included terminal deoxynucleotidyl transferase, mediated deoxyuridine triphosphate-biotin nick-end labeling and the caspase-3 staining in the neurons that are found in the regions of CA1 and CA3 in the hippocampus (Ma, et al., 2018). It is also proved that the activation of caspase-3, belonging to one of the larger family of cysteine protease has been already implicated in the neural cell death that was produced by different brain disorders included both ischemia and seizures (Boison, et al., 2017). However, the apoptosis in seizure models has proceeded with the help of numerous molecular pathways in which one of the molecules can play role of calbindin which is increased in mice who is served with the ketogenic diet. Calbindin is believed to have some neuroprotective activity because of its capacity to buffer intracellular calcium that is a mediator of cell death. Moreover, the protection by the ketogenic diet is mediated by the prevention of kainic acid- induce mechanism that is marked by the accumulation of protein named as clusterin that can act as a predeath signal (Vidali, et al., 2015).

**Regulation of antioxidant mechanism**

The enhancement of antioxidant mechanism portrays and represents an additional potential mechanism of neuroprotection. A significant example of this fact is ketone bodies that have shown activity in such a way that it can reduce the amount of Q semiquinone, an enzyme that is meant for decreasing free radical production. It is more added that the enzyme that is involved in the control of ROS information is glutathione peroxidase (Vidali, et al., 2015). It is a peroxidase that is found in erythrocytes that can help to prevent lipid peroxidase by a reduction in the lipid hydroperoxides or the corresponding alcohols and also reduces free hydrogen peroxide to water, H2O. The ketogenic diet is capable of inducing a glutathione peroxidase cavity in the hippocampus of a rat. Moreover, the ketogenic diet is also capable of increasing the production of specific mitochondrial uncoupling protein also called UCPs. An example of mice was used for practical approach illustrating that a mouse was fed with ketogenic diet (Klement, et al., 2017). As a result, UCP2, UCP4, and UCP5 were increased especially in the dentate gyrus. Moreover, UCPs serve to dissipate the mitochondrial membrane potential that will in return decrease the formation of ROS. Thus, the juvenile mice who were fed with the ketogenic diet were found to have maximum and comparatively higher mitochondrial respiration rates as compared to those mice who were fed with a controlled diet. Oligomycin induced ROS production was also found to be lowered in the groups who were fed with ketogenic diet-fed group. It is proved that the ketogenic diet is more likely to induce UCP production with the help of fatty acids, where the level of many of polyunsaturated fatty acids was elevated in the human patients just because of the ketogenic diet (Klement, et al., 2017).

**Effect of ketogenic diet on γ-aminobutyric acid systems**

There are a number of ways in which the ketogenic diet is found to confer with the neuroprotection facilitated through the enhancement if γ-aminobutyric acid (GABA) levels. However, this phenomenon was supported with a consequent increase in the GABA-mediated inhibition (Gasior, et al., 2006). The ketone bodies have a structure that has the power to increase the GABA content in the brain synaptosomes of rat by using an in-vivo protein, also called as a two-dimension double quantum spin-echo spectroscopy. However, the ketogenic diet was associated with an elevated level of GABA in a few of human subjects as well that were studied by research. In contrast, the rats fed with ketogenic diet did not show an increase in cerebral GABA (Soeters, et al., 2019).

**Effect on Energy Metabolism**

It is found that the ketone bodies that include both beta-hydroxybutyrate, which is reduced as a result of consumption of the ketogenic diet can serve as an alternative source of energy in the state of metabolic stress, taking into account that it can contribute to the neuroprotective activity of the diet. However, B-hydroxybutyrate is also capable of providing a more efficient source of energy for brain per unit oxygen as compared to glucose. Recently, microarrays were used to define the pattern of gene expression where Bough, 2006 made an astonishing discovery that revealed that ketogenic diet is capable of causing coordinated regulation of the hippocampal genes that could encode energy metabolism and then mitochondrial enzymes (Klement, et al., 2016). The illustration of Electron micrographs has resulted in a debate because half region of the hippocampus that showed an increase of 46% in the mitochondrial profiles in the rats were fed with the ketogenic diet. Moreover, the ketogenic diet appears to stimulate the mitochondrial biogenesis taking into account that there was a greater phosphocreatine. It is defined as certain ratio in the hippocampal tissues that can indicate an increase in the cellular energy reserves as experienced and observed from the abundance of mitochondria. In a nutshell, it was found that during the consumption of ketogenic diet, there were two prominent and major factors to consider that could contribute to the ability of neurons to resist and react against the metabolic stress (Klement, et al., 2017). It was found that if there would be a greater mitochondrial load and more energy-efficient fuel. In addition to these combinations, these factors are also found to account for the enhanced ability of the neurons that can help to withstand the metabolic challenge of a degree that could ordinarily enhance the reliance of the neurons and it may result in cellular demise. (Vidali, et al., 2015).

**Dravet syndrome**

There is a comprehensive and short account of effect of the ketogenic diet in treating homogenous epilepsy syndrome, taking that account that it can also cause a benefit on the patients who are suffering from DS when they are given gold standard therapy, accompanied by an unsatisfactory control. It is found that KD is capable of reducing the clinic seizure frequency up to 75% and it is capable of reducing and decreasing myoclonic fits and other atypical absences (Dashti, et al., 2007). However, in about fifty percent of patients, it was found that they were Responders to the syndrome within the time span of 3 and 6 months while remaining 40% were at a long term such as 9 months. The researches shared that they tried to maintain efficacy of one third for more than 1 year and the responders were given a follow up of less than 12 months who were still on the KD diet (Vidali, et al., 2015). It was asserted that the efficacy scale seems much inferior to the range that was reported in past few observations. However, in the first study, observations were made after 1 year and it demonstrated that 13 out of 20 patients will remain on the diet. Here 50 % of the patients achieved about 50% decrease in frequency of seizures where two patients were free of seizures. In another study, it was highlighted that 11 out of 16 patients stuck to diet for the time span of 12 months however among them only 30 of the patient achieved less than 90% seizures reduction and it was then considered that the patients who experienced a decrease of 75% in seizures frequency since classical cutoff were less than 50% and they were considered as insufficient for the patients who were on polytherapy (Boison, et al., 2017). In a shell, the service reflected that about 5 patients were the consistent respondent for 1 year of KD where one patient was seizure-free, and it reflected that KD can control the DS (Klement, et al., 2019).

**Dealing with Brain Trauma**

It is evident that the incidents of brain trauma are increasing in both military context as well as civilians. It is asserted that brain injury is the product of any penetrating injury or trauma that can lead to major cognition or motor consequence. Moreover, it is found that the epilepsy is also one of the causes of brain tumor because epilepsy adds to the morbidity of individual who are effaced and it is a sign to the emergence of hypoexcitable neuron circuits over the curse of time (Vidali, et al., 2015). It is asserted that the clinical problems of past traumatic epileptogenic and the ideology that KD can reduce the seizure activity are treated with dietary therapy because it has the potential to ameliorate brain injury and the long-term consequences such as epilepsy (Dashti, et al., 2007).

This idea is also supported by animal studies that have a prime focus on ketone bodies. The research was supported by controlled cortical impact injury model that was correlated with maturation-dependent differences in the ketone utilization and cerebral metabolism. In the context of causes that were followed by anti-epileptogenesis followed by head injury, it was found that the data regarding KD are mixed. The implication of the KD diet before and after fluid percussion injury in rats was found to be not altered by seizure sensitivity (Klement, et al., 2017). However, the degree of hippocampal cell loss was reduced by the inclusion of post-treatment. Moreover, it was found that the lithium pilocarpine model of temporal lobe epilepsy was also treated with KS treatment that was made a compulsion prior to the induction and it led to morphological neuroprotection in the hippocampus of brain. It did not affect latency to the onset of a spontaneous recurrent seizure. In contrast, Jiang, 2012 reported that an increase in KD that was increased after discharged thresholds alpha with the reduced generalized seizure occurs in the amygdala of the rat. Although it was not associated with believing later, the recent findings asserted that the KD inhibits that target of the pathway that is linked to the modulation of somitogenesis where further studies in another animal models are warranted (Klement, et al., 2019).

**Role of KD in Psychiatrist Disorder**

Research has proven that KD is capable of mood shifting, the mood-stabilizing properties of KD have been hypothesized by research taking into account that there is no record of clinical studies that affirm this assumption. However, the potential role of the KD in depression as well as in Autism has been studied by using the forced-choice model of depression in ways that asserted and proved the beneficial effects of the diet similar to effects that are the product of antidepressants (Klement, et al., 2016).

**KD in Autism**

Research proved that using KD can help to overcome autism, proved by a research study that was conducted on the island of Crete. There is a far greater ratio of autism patients in Crete as compared to other regions and as results of longitudinal studies revealed that the potential involvement of adenosine an endogenous neuromodulator and anticonvulsants along with ameliorating autistic behaviors have the potential to raise possibility of laps with the mechanism of KD (Boison, et al., 2017).

Another research study has proven that migraine is also one of the paroxysmal neurological disorder that has the clinical phenotype overlap with epilepsy. Although there are the intrinsic mechanism underlying migraine attack and seizure attacks differ in fundamental aspect equally there are theoretical reasons to the consideration of ideology that KD can deal with chronic migraine. It is important to note that the first report comprising of information regarding the use of ketogenic diet in the treatment of Migraine was for the first published in 1928, followed by another report on which twenty-eight patients reported that they are feeling much better because of the change in diet and diet was highly ketogenic in nature (Ismayilova, et al., 2018).

 It is also asserted that there is a clinical record that could prove this fact but there are several general and subject oriented research reports under which people were treated and experimented with the particular diet for overcoming a disease (Ismayilova, et al., 2018). According to a researcher, it has been highlighted that the laboratory investigations have confirmed that long-chain triglyceride form of the Ketogenic Diet results in a significant reduction in the measured velocity of cortical spreading depression velocity in the immature rats. Another aspect of this study highlighted that tri-heptanoate substrate that enhances and empowers the tricarboxylic acid cycle function plays a significant role in mitigating and overcoming the pentylenetetrazol tonic seizure threshold and the delayed development of corneal kindled seizures (Ismayilova, et al., 2018).

**Conclusion**

Ketogenic diet is one of the best and alternative way to treat certain conditions that can accelerate and defend health circumstances. Although it is hard to follow still it is one of the major aspects that can cater to the needs of patients by addressing highly medicine-oriented diseases. Although there is a relative lack of clinical information, there is an emerging literature that supports the use of KD in dealing with both major and minor health issues. It is more asserted that the preliminary studies are largely and broadly based on the fundamental ideology of metabolic shifts that may lead to neuroprotective actions. It is significant to note that the alteration in diet is the result of a product that is meant for treating different diseases. There are diverse aspects associated with the Ketogenic diet, taking into account that the common and final pathways are mechanistically shared. Moreover, ultimate details revealed that the altered metabolism has the potential to reduce neuronal excitability along with an abrogation of ongoing neurodegeneration that could mitigate functional disability and are still left unknown. However, there is a lot of space for further studies that could explore other benefits of a ketogenic diet because it is something that can play a significant role in sustaining life as well as the treatment of major diseases.

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