Research Article

The Ketogenic Diet For Epilepsy and Autism in Children: A Review

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Abstract

For over 80 years, the ketogenic diet has been employed in clinical practice, largely to treat epilepsy symptoms. There is currently good evidence that the ketogenic diet prevents seizures in children with difficult-to-treat epilepsy. In theory, In childrens, the ketogenic diet is more beneficial than in adults, because younger brains have greater capacity to transfer ketones. This review highlights the mechanism of action, applications, beneficial and undesirable consequences of ketogenic diet in epileptic and autistic children.

Keywords:

Epilepsy; Autism; Ketogenic foods; ketogenic diet epileptic and autistic children

Introduction

The ketogenic diet is a low-carbohydrate, high-fat, high-protein diet used to treat children who have epilepsy that is difficult to control (refractory).

As a result of the diet, the body is pushed to burn fats rather than carbohydrates. Meal carbs are normally converted to glucose, which is then transported throughout the body and plays a critical role in brain activity.

The liver, on the other hand, when there is relatively little carbohydrate in the diet, fat is converted to fatty acids and ketone bodies. Ketone bodies enter into brain and take place of glucose as a source for energy [1].

Ketosis, or a rise in ketone bodies in the blood, causes epileptic seizures to become less frequent.

The ketogenic diet is a non-pharmacological therapeutic option for pharmacoresistant epilepsies, as well as children with difficult-to-treat catastrophic epilepsy syndromes and early epileptic encephalopathies.

Although the diet is beneficial to people of all ages, clinical evidence suggests that it may be more beneficial to children because adults have greater difficulty sticking to it.

Long-chain triglycerides make up the majority of dietary fat. Medium-chain triglycerides are more ketogenic than long-chain triglycerides because they are made up of fatty acids with shorter carbon chains.

The medium-chain triglycerides ketogenic diet is a variant of the traditional ketogenic diet that uses MCT-rich coconut oil to provide around half of the calories.

Materials and Methods

The Ketogenic diet causes fat metabolism in the liver, three ketone bodies are formed as a result of this process: beta-hydroxybutyrate, acetoacetate, and acetone.

Ketone bodies are transported over the blood-brain barrier via the monocarboxylic acid transporter, where they become the brain's primary energy source. The brain's principal excitatory neurotransmitter, Glutamate, is removed more efficiently synoptically during ketosis.The glutamate/gamma-aminobutyric acid–glutamine cycle converts glutamate to gamma-aminobutyric acid, a major inhibitory neurotransmitter, which inhibits neurons.

BHB and Acetoacetate block neurotransmission of glutamate by suppressing vesicular glutamate transporter activity [2]. Diet has a significant impact on neuronal function in the hippocampus. A high-fat, calorie-restricted diet stimulates mitochondrial biogenesis in the hippocampus, which promotes metabolic gene expression and boosts energy storage. The Ketogenic diet's anticonvulsant properties may be due to energy conservation, which may be especially relevant for more metabolically active GABA interneurons. The benefits of dietary therapies like Ketogenic diet could be extended to the treatment of neurodegenerative illnesses since the better ability of neurons to regulate metabolic issues following ketone bodies likely promotes neuronal survival and function under stressful settings.

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Discussion

French physicians, Guelpa and Marie, author first scientific report of fasting for epilepsy

Phenytoin discovered; decline in use of KD

Medium-chain triglyceride diet introduced

Resurgence of KD due to success in Charlie Abrahams

Charlie, the son of a Hollywood producer, was two years old when he began having intractable myoclonic, generalised tonic, and tonic-clonic seizures.

Gained national attention for successful use of KD to control his seizures

The Charlie Foundation created for KD and epilepsy

Made-for-television movie based on Charlie's experience with KD

First randomized, controlled trial of KD

mechanism of action

The Ketogenic diet causes fat metabolism in the liver, three ketone bodies are formed as a result of this process: beta-hydroxybutyrate, acetoacetate, and acetone. Ketone bodies are transported over the blood-brain barrier via the monocarboxylic acid transporter, where they become the brain's primary energy source. The brain's principal excitatory neurotransmitter, Glutamate, is removed more efficiently synoptically during ketosis. The glutamate/ gamma-aminobutyric acid–glutamine cycle converts glutamate to gamma-aminobutyric acid, a major inhibitory neurotransmitter, which inhibits neurons. BHB and Acetoacetate block neurotransmission of glutamate by suppressing vesicular glutamate transporter activity [3]. Diet has a significant impact on neuronal function in the hippocampus. A high-fat, calorie-restricted diet stimulates mitochondrial biogenesis in the hippocampus, which promotes metabolic gene expression and boosts energy storage. The Ketogenic diet's anticonvulsant properties may be due to energy conservation, which may be especially relevant for more metabolically active GABA interneurons. The benefits of dietary therapies like Ketogenic diet could be extended to the treatment of neurodegenerative illnesses since the better ability of neurons to regulate metabolic issues following ketone bodies likely promotes neuronal survival and function under stressful settings.Classical Ketogenic Diet - Any ratio can be used, however the most common is 3-4:1 (90 percent fat, 4% carbohydrate, and 6 percent protein).

Any ratio is acceptable. Based on 4:1 ratio

(Ten percent LCT fat, sixty percent MCT fat, twenty percent carbohydrate, and ten percent protein)

Atkins with a twist (Modified Ketogenic Diet)-

Approximately a 1.1:1 ratio

(65 percent fat, ten percent carbohydrate, and two-fifths of a pound of protein)

Diet with a low glycemic index-

Approximately a 0.6:1 ratio

(Sixty percent fat, ten percent carbohydrate, and thirty percent protein)

Healthy fats and non-starchy vegetables make up 75% of diet.

Leafy greens and vegetables - spinach, amarathus, broccoli, cauliflower, asparagus, zucchini, and Capsicum .

Egg yolks,

Nut butter- hazelnut butter, peanut butter, almond butter, cashew butter.

Oils- avocado oil, almonds oil, flaxseed oil, coconut oil, olive oil etc.

Proteins should make up 20% of diet.

Animal protein: e.g. chicken, grass-fed beef, turkey, duck, and pork etc.

Supplements containing collagen and gelatin powder

Dairy products- Paneer, sour cream, cream cheese without sweeteners, heavy cream etc.

Berries - Raspberries, blueberries, strawberries etc. (small quantities)

Legumes - Green peas, beans etc.

Ayurveda and yoga are said to be able to heal epilepsy in some Indian cultures. In India, fasting has both medical and religious significance. Onion, garlic, spices, rice, chapatti (flat bread), leafy vegetables, root vegetables, paneer, cream are all classics in Indian cuisine [4]. Ghee and coconut oil are two commonly used fats in India. Any of the four Keto diets can be effective. "The KD is not a new therapy, it is as old as Ayurveda" according to Dr. Janak Nathan. 34 gram chicken, 40 gram onion, 93 gram tomato, 1 gram ginger, 1 gram garlic, 34 gram ghee. Added in very small quantities: clove, bay leaf, green chili, red chili powder (394 kcal, 1.8:1 ratio).

Ketogenic Diet - implemented in three phases.

Pre-counseling and investigations

Initiating the Diet

Fine Tuning.

After the treatment is finished, the patient can resume his regular diet.

Not a "natural" treatment for epilepsy

Parents should not start the diet without medical guidance

Should be initiated and monitored by a physician working with the health-care team

Difficult to tolerate

40-50% will discontinue diet within first 6 months

Requires accurate measurement of all components of the diet

Hidden sources of carbohydrates in diet and other products

Strict compliance is necessary for benefits of the diet

Small lapses are enough to stop ketosis

Not finishing a meal

Consuming too much carbohydrate or protein

Non-compliance may lead to seizures

More palatable diets available

Less restrictions and adverse effects

Modified Atkins diet

MCT diet

All-liquid formulations of the diet are available for infants and enterally fed children

Easier to implement

More compliance and greater efficacy

When attempted, should be tried for at least 3 months

Patients who respond to and tolerate diet should continue for at least 2 years

The ketogenic diet has been used to treat persistent childhood epilepsy since the 1920s. The ketogenic diet fell out of favour as new anticonvulsant drugs were developed, beginning with phenytoin in 1938, but it has resurfaced in recent years, particularly to treat refractory epilepsy. Epilepsy is a dangerous neurological condition that knows no racial, social, or geographic barriers. Recurrent seizures characterise this illness, which has a wide range of symptoms. Fasting has long been linked to a decrease in seizure frequency. Ketones are created when you fast for a long time. The ketogenic diet mimics this fasting condition while also produces ketones. Ketones are a sign of a significant metabolic shift, but they cannot explain the anti-seizure impact by themselves. Almost 50% of adolescents and adults with drug-resistant epilepsy may benefit from the KD and its variations. when surgery is not an option for refractory epilepsy or epileptic encephalopathies, the ketogenic diet is considered. Most refractory and widespread epilepsies, such as Dravet, Doose, Lennox-Gastaut, and West syndromes, benefit from the KD as a coadjuvant therapy. On the ketogenic diet, people with focal epilepsy may improve, although they may not attain complete seizure independence.7 Traditionally, children's diets are stopped after two years, especially if they are seizure-free. The probability of seizure recurrence after a seizure-free treatment term with the ketogenic diet appears to be equivalent to surgery and slightly lower than after quitting anticonvulsant medicines. In that situation, the likelihood of seizures becoming intractable is also low.

Ketogenic Diet in Children with Autistic Behavior: Seizures (5% to 46%), epileptiform activity (upto 60%) are commonly present in autism spectrum disorders. Children with autism spectrum disorders have been observed to have greater immune problems in response to diet. Early childhood, puberty, adolescence is the most prevalent times for seizure onset in autism spectrum disorders.16 There have also been accounts of patients with Rett syndrome being treated with a Ketogenic diet. Ketogenic diets were initially prescribed for these people to help them control their seizures, but it has now been discovered that they can also help with mental behaviour and hyperactivity. Some evidence shows that the ketogenic diet can be used as an additional or alternative therapy for autistic behaviour. Gluten-free, casein-free medium-chain-triglyceride-rich ketogenic diets may be a better option for children with autism spectrum disorders than traditional ketogenic diets. Furthermore, in children with autism spectrum disorders who have seizures, a gluten-free casein-free ketogenic diet may be more effective than pharmacological agents alone, and may alleviate some of the undesirable responses of antiepileptic drugs that are common in children with autism spectrum disorders. Traditional KDs have more tolerability issues than the modified variants. The traditional diet's "extremely restrictive character" is often cited as a reason for its cessation.

Chronic complications

- constipation,
- Renal stones
- Weight loss
- Anorexia
- Specific vitamin and mineral deficiencies
- Higher chances of infection
- Cardiomyopathy
- Retarded growth
- Progressive bone mineral content loss
- Increased cholesterol levels
- Low albumin and carnitine levels
- Rarely fatal pancreatitis in those with abnormal lipid metabolism.

Nephrolithiasis, Hypertriglyceridemia, Significant reduction in height and weight after prolonged use, Catch-up growth observed after diet discontinuation, Increased risk of bone fracture, BMI reduction and diet effectiveness may be correlated [5]. Hypoglycemia is particularly common in newborns and young children who are unable maintaining enough caloric intake and who vomit often. The most prevalent reason for early diet cessation, especially in adults, is intolerance owing to gastrointestinal side effects. Improvements in attention, well-being, and fitness, as well as more erect posture, fluent communication, and increased mood, have all been noted.

CONTRAINDICATIONS

Epilepsy surgery candidates, Failure to thrive/poor nutritional status, Renal stones and hyperlipidemia, Special diet needs/ preferences, Parent/caregiver noncompliance, Medical conditions aggravated by diet are the relative contraindications of ketogenic diet. Primary carnitine deficit, Carnitine palmitoyltransferase I or II deficiency, Carnitine translocase deficiency, Fatty oxidation defects, Porphyria, and Pyruvate carboxylase insufficiency are absolute ketogenic diet contraindications.

KD EFFICACY AND SAFETY

Improved concentration, well-being, and fitness, as well as more erect posture, fluent pronunciation, and improved mood, have all been noted. A better seizure outcome was connected to seizure onset at a later age, female gender, a higher ketogenic diet ratio, and non-fasting induction.18 According to a recent meta-analysis of studies published between 1925 and 1998 that included both traditional and MCT diets, 37 percent of patients had a seizure reduction of > 90 percent, and another 30 percent had a seizure reduction of 50-90 percent.

Conclusion

KD is a tried-and-true method of treating seizures in children. It does, however, requires meticulous growth and laboratory parameter monitoring. Dietary adjustments may be necessary to help with growth and nutrition while maintaining seizure control. Pediatric neurologists, paediatricians, and dieticians working together to monitor children on KD is critical to the efficacy of the therapy as well as the prevention, identification, and management of adverse effects.

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