Ketogenic diet therapy for epilepsy in adults

Klaudia Oliwia Socha[1,A–E]

1 Faculty of Health Sciences, Medical University of Warsaw, Poland
A – Research concept and design, B – Collection and/or assembly of data, C – Data analysis and interpretation, D – Writing the article, E – Critical revision of the article, F – Final approval of the article

CORRESPONDING AUTHOR
Klaudia Oliwia Socha
Faculty of Health Sciences, Medical University of Warsaw, Poland; email: klaudiasocha96@gmail.com
Phone: 735531522

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Abstract:

Epilepsy can be controlled with medication in most cases, however, around 30% of adult patients remain pharmacoresistant. Ketogenic diet (KD) and its variants have been used as a therapy in children with epilepsy for over a hundred years and they have been proven to be an effective method of controlling refractory epilepsy. Adult patients could also benefit from this method of controlling seizures, however, the studies evaluating its efficacy are limited and the diet is not as widely used in this group of patients. The diet works by mimicking a fasting state in which ketone bodies become the primary source of energy. However, the exact mechanisms underlying the anticonvulsant effects of the diet are not yet fully understood. This review aims to assess the effectiveness of ketogenic diet and its subtypes in controlling drug-resistant epilepsy (DRE) in adults, discusses possible mechanisms of its anticonvulsant action, potential adverse effects, and applicability of this method in managing epilepsy. It also compares different variants of the diet and provides practical guidelines on introducing and maintaining the KD therapy.

Keywords: adults, diet therapy, ketogenic diet, refractory epilepsy.

Introduction

Epilepsy is one of the most common neurological conditions, affecting around 70 million people around the world. It can be controlled with anti-epileptic drugs (AEDs) in most cases; however, a considerable number of patients remain resistant to pharmacological therapies (1). According to the International League Against Epilepsy (ILAE), the disease is considered to be drug-resistant if two trials of appropriately chosen (tried alone or in combination) anticonvulsants have failed to achieve sustained freedom of seizures (2). It is estimated that around a third of the patients are pharmacoresistant. Since only some of these patients qualify for surgeries, there is a need for alternative treatment options, one of which is ketogenic diet therapy (3). Ketogenic diet has been used for treating drug-resistant epilepsy in children for over a century. This therapeutic approach was first described in 1920s in the Mayo Clinic as an alternative to fasting, which was observed to be helpful in controlling epileptic seizures (4). The diet is very high in fat and low in carbohydrates, which constitute for less than 10% of total daily calories. It acts by simulating a starvation state and forces the body to shift to using fat, rather than glucose metabolism, as a primary source of energy for the cells. This is known as a ketogenic state, in which liver metabolizes fatty acids to ketone bodies on the way of beta-oxidation, producing mainly acetoacetate and β-hydroxybutyrate (5). The KD therapy has been a well-recognised tool for treating refractory epilepsy in paediatric patients

and in recent years an interest in its use has spread to adult patients. Emerging evidence is suggesting that its use can be extended into adult practice as an effective alternative treatment option.

Methodology

This article is a narrative review of the literature focusing on the use of ketogenic diet therapies in drug-resistant epilepsy, with particular emphasis on adult patients. A non-systematic review of the literature was conducted using PubMed and Google Scholar databases. Articles published in English between 2000 and 2020 were considered. The search terms included combinations of: "ketogenic diet", "ketogenic diet therapy", "epilepsy", "drug--resistant epilepsy", "refractory epilepsy", "adults", "modified Atkins diet".

Additional relevant publications were identified through reference lists of selected articles, allowing for the inclusion of studies published prior to 2000. These papers provide historical context for the development of ketogenic diet therapy.

Ketogenic diet therapy for epilepsy in children

The efficacy of the ketogenic diet therapy in controlling intractable seizures in children has been well documented in literature (6,7). A meta-analysis from 2006 reviewed 19 studies and involved 1084 paediatric epileptic patients. According to its results, 52% of those who adhered to the diet experienced a 90% seizure reduction and 24% achieved seizure--freedom. However, 50,9% of the patients discontinued the treatment at various time points. The main reason for the drop-outs was the diet ineffectiveness (8). A randomised controlled trial conducted in 2008 aimed to assess the effectiveness of KD in managing DRE in 145 children. The study showed a 75% reduction in epileptic seizures occurrence in the KD group compared to the control group (9). The KD is also a first-line treatment for specific epilepsy conditions, such as GLUT-1 deficiency syndrome and pyruvate dehydrogenase deficiency in children. It is also very useful in Dravet and Doose syndromes and in treating infantile spasms (5).

Ketogenic diet therapy for epilepsy in adults

There has been an increasing interest in the use of the KD therapies in treating pharmacoresistant epilepsy in adults. Recent evidence shows that KD may indeed be a useful alternative treatment for adult patients suffering from epilepsy that is difficult to control. A meta-analysis published in 2015 reviewed the efficacy of the KD and its variants in adults with the DRE from 12 studies that involved 270 patients. The combined efficacy of all types of KD was 42%, while the compliance rate was 45%. The classical KD seemed to be more effective than the modified Atkins diet (52% vs 34%, respectively), however, the MAD was characterised by a greater patient adherence than the CKD (56% and 38%, respectively). These findings were reported across studies exhibiting significant heterogeneity (10). Another meta-analysis of observational studies from 2018 summarized the findings from 16 studies including 338 patients with intractable epilepsy. The paper looked at the KD and its main variants and it revealed that 13% of patients attained seizure--freedom, 53% had a seizure reduction of 50% or more, and 27% achieved a reduction of less than 50%. The meta-analysis looked at 4 different subtypes of the KD: CKD, MAD, low glycaemic index diet (LGID) and low-dose fish oil diet (LFOD), however, all of the mentioned diets led to a shift in cell metabolism from using carbohydrates to lipids as the main source of fuel. However, there was a considerable heterogeneity across the studies included in the meta-analysis ($I^2 = 80.2\%$, $p = 0.000$) (11). A very recent observational prospective study performed in the United Kingdom evaluated the real-life efficacy of the MKD in 42 epileptic patients. According to the researchers, 60% of patients reported an improvement in seizures frequency, 38% reported a reduction greater than 50%, and 13% reported a period of seizure-freedom. However, 30% of the patients experienced worsening of the seizure frequency at some point during the treatment. Moreover, the retention rates were decreasing over time, from 60% at 3 months to 43% at 6 months, and only 29% at 12 months (12). A study from 2019

assessed the impact of an MKD therapy on seizures frequency, seizures severity and the quality of life in pharmacoresistant epileptic patients. The study results not only showed a significant improvement in seizures frequency (60% of the study participants observed a reduction ≥ 50%), but the researchers also reported that 76% of the participants experienced an improvement in the seizure severity, and increased quality of life scores were observed in 87% of the patients (13). However, the number of studies in the area is still limited and many of the studies published are characterised by small sample size, what affects the quality of the evidence. More studies, preferably multi-centre clinical trials, are required. They should also evaluate adverse effects and their management and a comparison of different KD variations would be valuable.

Different variants of the ketogenic diet

The subtypes of the diet differ in terms of the amount of the energy derived from the three macronutrient groups, nonetheless, all of them remain very high in fat and low in carbohydrates with an adequate amount of protein needed for growth and development. Only then, in the absence of glucose, the body is forced into a ketogenic state in which the metabolism shifts to using fat as a primary source of fuel for the cells. In this state, the energy is generated by fatty acids oxidation in mitochondria, what leads to a production of large amounts of acetyl-CoA. The build-up of acetyl-CoA triggers the production of ketone bodies in the liver, which are then released into the circulation and act as a primary fuel source that replaces glucose (14). The classic Ketogenic Diet (CKD) is typically composed of a 4:1 ratio of fat (in grams) to protein and carbohydrates (in grams), it is very rich in lipids which provide 90% of the energy in the diet. Although this ratio is considered to be the gold standard for the CKD, it is characterised by a low compliance due to its low palatability and strict restrictions. Moreover, strict dietary calculations and exact weighing of all foods is required. Therefore, multiple new subtypes of the KD have emerged. Medium-chain triglyceride (MCT) ketogenic diet was designed as an alternative to the CKD in 1970s. MCTs absorption and transport is more efficient than that of other types of lipids and therefore they are more ketogenic per unit of dietary energy. Thus, the MCT KD diet allows higher proportion of protein and carbohydrates to fats. Originally, 60% of the energy in the MCT KD was derived from the MCTs, however, such high amount of MCT was reported to lead to multiple gastrointestinal adverse effects and the MCT oil is considered to be less palatable. Thus, a modified version of the MCT KD was designed, comprising 30% energy from MCT oil and 30% energy from long-chain triglycerides (15). The exact amount of the MCT oil in the diet depends on the patient's individual tolerability and it can be modified throughout the therapy. The MCT oil can also be used as a supplement on the CKD to improve ketones production per calorie of fat and to help with constipation due to its laxative effect (16,17).

Another less restrictive subtype of the KD is the modified Atkins diet (MAD), which is a combination of the CKD and Atkins diet, limiting carbohydrate

Table 1. Composition of different KD subtypes

Diet type	Ketogenic ratio	% Carbohydrates	% Protein	% Fat
CKD	4:1	4	6	90
MKD	3:1 – 1:1	5	15-20	75-80
MCT KD	3:1	15-18	10	70-75 (30-60 MCT)
MAD	1:1	5-10	30	60-65
LGID	0.6:1	10 (GI <50)	30	60

Abbreviations. KD = ketogenic diet; CKD = classic ketogenic diet; MKD = modified ketogenic diet; MCT = medium chain triglyceride, MAD = modified Atkins diet; LGID = low glycaemic index diet; GI = glycaemic index.

intake and allowing protein intake to be practically unrestricted, as it is not strictly monitored. However, excessive protein consumption may reduce ketosis, therefore the MAD encourages lipids in place of excess protein. The ratio of MAD is usually around 1:1 and an exact weighing of products is not required; however, the daily amount of carbohydrates consumed is restricted to around 10-20 grams. The MAD is also characterised by a better palatability, and therefore improved compliance then the CKD (18). It is also reported to be more tolerable and has fewer side effects both in children and in adults (10,19). Modified ketogenic diet (MKD) is also a more liberal version of the KD designed to be more flexible and often used when starting the KD therapy. It is a combination of the CKD and the MAD. In the MKD, the diet ratio is between 3:1 and 1:1 and its choice depends on the patient's needs and tolerability (20). A low glycaemic index diet (LGID) is an another KD variety that is considered to be easier to manage in patients. The ratio of LGID is 0.6:1 of lipids to protein and low glycaemic index (GI) carbohydrates (GI lower than 50) and 60% of the energy is taken from fat (21). The choice of the KD therapy type should be tailored to the patients' individual needs and it should take into consideration personal characteristics such as age, lifestyle, epilepsy type, medical condition, etc. It is also possible to switch between different KD subtypes to help manage adverse effects and improve compliance and/or therapy efficacy (22).

Possible mechanisms of action – why does the diet work?

After a century of clinical use, the mechanisms underlying the seizure-controlling action of the diet remain poorly understood, however, multiple theories attempting to explain its anticonvulsant effects have emerged. Some of them suggest that the elevated levels of circulating ketone bodies in chronic ketosis are the main mediators underlying the anti-seizure mechanisms of the KD. The evidence for this hypothesis comes mostly from animal models (19,23,24).

Other hypotheses focus on the role of inhibitory mediators, like adenosine (25), produced in the brain or compounds modulating ion channels, such as polyunsaturated fatty acids (26,27).

A recent study in rodents suggests a novel epigenetic mechanism involving DNA methylation, which persisted after the diet was discontinued (28,29). Moreover, there is growing interest in the role of the gut microbiome as a potential mediator of the anti-seizure effects of KD therapy (30). Notably, Olson et al. (2018, *Cell*) proposed that KD-associated shifts in microbial composition can modulate hippocampal GABA/glutamate levels, which has become the most widely cited microbiome-brain interaction model in the context of the ketogenic diet. Animal studies have demonstrated that the KD leads to alterations in mice microbiota composition (31).

In contrast, human data are largely observational, with persistently elevated levels of ketone bodies being correlated with changes in gut microbiota composition (32), without direct evidence of a causal relationship. Nevertheless, further research is required to clarify the role of the gut microbiome in human epilepsy and to determine how KD-induced microbial changes contribute to seizure control. The anticonvulsant effects of the KD therapy are likely to be a combination of various mechanisms and their better understanding is crucial for optimising the ketogenic therapy but also to develop more effective AEDs to manage refractory epilepsy.

Potential side effects, compliance, and clinical practice guidelines

Although the KD is a generally well-tolerated treatment option for epilepsy, there are some potential adverse reactions associated with its use. The most common ones mentioned in the literature include unfavourable lipid profile (increased low--density lipoprotein and total cholesterol levels), hypoglycaemia, and gastrointestinal symptoms such as vomiting, constipation, nausea, diarrhoea and abdominal cramps (3). However, the gastrointestinal symptoms can be managed with simple diet adjustments, such as decreasing meals size, increasing fibre intake, drinking more water, as well as exercising more (33). Long-term KD use may also lead to micronutrient deficiencies (such as selenium,

zinc, and magnesium) and reduced bone mineral density (17,34). Other reported side effects include weight changes, menstrual irregularities in females, fatigue, lethargy, hunger, weakness, headaches, infections, kidney stones, and acne (3,11). Since KDs are very high in fat, their impact on a lipid profile and cardiovascular risk is particularly controversial. The evidence is not consistent however, some studies report increased levels of total and LDL cholesterol, as well as triglyceride levels (13), while others show an association between the KD therapies and improved HDL cholesterol levels and no changes in LDL cholesterol (35). The composition of the diet seems to be of a key importance. A very low-carbohydrate diet that was higher in unsaturated fatty acids and lower in saturated fatty acids had a beneficial impact on the lipid profile (36). Thus, focusing on the KD quality and its fatty acids composition is likely to be an important part of the KD therapy management and the role of an experienced dietitian in this process is vital. Nevertheless, most of the side effects of the KD diets can be prevented or minimized by close monitoring of the diet and early appropriate interventions (34). Therefore, a careful patient supervision by a team involving a neurologist and a dietitian is an essential part of the KD therapy. In adult populations, the indication for the KD diet should additionally take into account potential contraindications, including severe dyslipidaemia, liver and kidney failure, eating disorders, and inherited disorders of fatty acid oxidation, which may limit its use or necessitate strict medical supervision (37). One of the main issues related to the KD therapies in adult epilepsy treatment is very low patients' compliance. The meta-analysis of 11 studies by Ye et. al. reported a combined rate of patient adherence to the KD of 45% (10). An observational study of 139 patients starting the KD therapy reported a 48% retention rate and the main reasons mentioned for stopping the diet were problems with compliance and strict restrictions (38). The low adherence numbers are consistent with previous reports (21). The retention rates in adults are much lower than they are in children, which has been suggested to be one of the reasons why the diet is reported to be more effective in seizure control in paediatric patients (10). Multiple reasons have been suggested to cause problems with KD adherence in adults. The most common one that is mentioned in the literature is lack of efficacy, leading to increased early drop-out rates. Since the KD requires a period of minimum 3 months for efficacy, it is particularly important to support and encourage the patients during the first weeks of the therapy (39). Moreover, the patients complain about low palatability, high restrictiveness of the diet and the need for an exact weighing of foods. Adverse effects occurrence, especially the short-term ones, such as gastrointestinal symptoms, may also lead to lower retention rates (40). Since low adherence rates remain one of the main challenges of the KD therapy in adults, finding ways to improve compliance, and thus the effectiveness of the KD therapies is crucial. Multiple things can be done to achieve this goal and there is a need for a multidisciplinary approach in the area (41). The team providing a ketogenic diet service should consist of at least one neurologist and an experienced dietitian, whose role in the KD therapy is of a key importance. Other potential members of a ketogenic team include nurses, pharmacists, speech and language therapists, psychologists, and social workers (42). Before the KD therapy is started, there is a list of recommended minimum baseline diagnostic and laboratory studies that should be performed, provided by the International League Against Epilepsy (ILAE). They include basic counselling, weight and height, a history of food allergies and intolerances, as well as food availability and preferences. The mandatory laboratory studies are sodium, potassium, bicarbonate, chloride, blood urea nitrogen, creatinine, and glucose. Moreover, metabolic testing to identify aetiology is recommended (43). A very important part of implementing the KD therapy is patients' education. Initial teaching session (group or individual) should precede the KD implementation. The session should involve instructing the patients on the diet's principles, macronutrient content of foods and ways of planning and preparing meals appropriately to meet the prescription goals. It should also teach them how to manage possible adverse effects.

Moreover, patients should receive training on how to monitor daily ketone levels and control blood glucose. Other strategies useful in ensuring optimal compliance involve, but are not limited to, using electronic applications such as Keto Diet Calculator, My Keto Planner, Electronic Ketogenic Manager, providing patients with ketogenic food recipes and meal plans, organizing culinary workshops, webinars, etc. Such events would also be a great opportunity for patient peer support, which could also play a role in improving diet adherence. Close supervision, individualized counselling and follow--up visits with a neurologist and a dietitian to ensure adequate nutrition and diet compliance are of a great importance. If a patient lives far from the ketogenic diet centre, telephone consultations and/or electronic communication should be available (33). There is also some evidence for the use of early supplementation with ketogenic formulas (44).

Conclusions

The KD is an overall well-tolerated treatment for intractable epilepsy in adults. There is an increasing evidence for promising results of its use in managing the disease, however, more multi-centre clinical trials in the area are needed. While proposed mechanisms of action, including microbiota-mediated effects, are supported mainly by preclinical and theoretical models, clinical evidence in adults currently remains limited and warrants cautious interpretation. It should also be noted that much of the high-quality evidence on the efficacy of KD comes from paediatric populations, and extrapolation to adults should be made carefully.

Some adverse effects are observed, but they are usually mild and can be managed with individual diet adjustments. Concerns remain regarding the diet adherence in adults. In order to improve compliance, and thus the efficacy of the KD therapy in managing refractory epilepsy in adults, it is crucial to minimise the likelihood of side effects occurrence and to possibly alter the diet should they arise. Therefore, it is very important to closely monitor the KD therapy and the role of a dietitian in this process is essential.

More liberal KD variations are also available, and they bring satisfactory seizure controlling effects for many adult individuals. The choice of the diet subtype should be based on the patient's individual needs and preferences to avoid drop-out within the first weeks of treatment, which is significantly higher in adults than it is in paediatric patients.

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