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# Efficacy and tolerability of the modified Atkins diet in children with drug-resistant genetic generalized epilepsy

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**ABSTRACT**

***Objective.*** The ketogenic diet therapy is a time-tested and potent non-pharmacologic treatment for epilepsy. However, the study of the ketogenic diet in patients with genetic generalized epilepsy (GGE) is not widely established. The aim of this study was to evaluate the efficacy and tolerability of the modified Atkins diet, a variation of the ketogenic diets, as a treatment for drug-resistant GGE.

***Methods.*** A retrospective chart review was performed in patients with epilepsy treated with the modified Atkins diet at the University of Chicago from 2017 to 2020. For three months following diet initiation, participants were monitored for diet tolerability and effect on seizures. Response to the treatment was recorded by self-reporting patients and guardians.

***Results.*** Thirteen patients with a diagnosis of drug-resistant GGE were identified. An average of 3.8 anti-seizure medications (ASMs) had been tried and 3.4 years had elapsed from seizure onset before dietary therapy was attempted. Patients were receiving a mean of 2.2 ASMs at the time of diet initiation. After undergoing dietary treatment for three months, 12/13 (92%) patients experienced a greater than 50% reduction in seizure frequency, 6/13 (46%) patients became seizure-free, and 7/13 (54%) were able to discontinue at least one ASM. All patients completed at least three months of dietary therapy with an average duration of 9.3 months at the time of report. One patient reported side effects of fatigue which may be attributed to the diet.

***Significance.*** The modified Atkins diet has shown to be an effective and well-tolerated treatment for children with drug-resistant GGE. The diet provides the additional benefit of aiding to discontinue ASMs and, therefore, minimize the side effects from polypharmacy. Given these results, it seems reasonable to consider the modified Atkins diet as an alternative and possibly earlier treatment option for patients with drug-resistant GGE.

**Key words:** ketogenic diet, pediatric epilepsy, non-pharmacological treatment

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The ketogenic diet has long been observed and studied as a non-pharmacologic treatment for epilepsy, especially in drug-resistant epilepsy (DRE). Although dietary therapy has been used for the treatment of seizures since the 1920s, the precise anti-seizure mechanisms, anti-epileptic mechanisms, and when to implement the diet as a treatment modality are only recently becoming better understood and established. Determining which patient populations may benefit from the ketogenic diet therapy (KDT) is an

important step in managing childhood epilepsy as approximately 20-30% of pediatric patients with epilepsy do not achieve sustained seizure freedom from anti-seizure medications (ASMs) alone [1, 2].

Although the ketogenic diet is considered a first-line treatment for certain metabolic epilepsies, such as glucose transporter 1 deficiency syndrome (GLUT1DS) and pyruvate dehydrogenase deficiency, under other circumstances, the KDT is usually considered as an alternative treatment after the failure of traditional ASMs [3, 4]. However, a recent Cochrane review highlights the benefit of KDT in children with DRE as up to 55% of children achieved seizure freedom after three months with a classic ketogenic diet (CKD) and up to 85% of children achieved >50% reduction in seizure frequency, although with low certainty [5]. From the same systematic review, use of the modified Atkins diet (MAD) showed that 15-25% of children with DRE became seizure-free and 56-60% experienced more than 50% seizure reduction [5].

Genetic generalized epilepsy (GGE) is a common group of epilepsy syndromes contributing to approximately 15-20% of all epilepsy syndrome diagnoses, with a typical age at onset in childhood and adolescence [6]. As the name suggests, genetic contribution is suspected with a presumed complex inheritance pattern. Although largely considered polygenetic, a small portion may be attributed to a monogenic variant, for example *SLC2A1* gene variants cause ~10% of early-onset childhood absence epilepsy (CAE) (onset before four years old) and are found in approximately 1% of all patients with GGEs [7]. Although current genetic testing may not carry a high diagnostic yield in GGE, it can sometimes be useful in the case of atypical presentation, such as in early-onset CAE.

The study of KDT, specifically in patients with GGE, is not yet well established. Although some GGE syndromes, such as myoclonic-atonic epilepsy (MAE), have been consistently observed to respond favorably to KDT, other epilepsy syndromes in the same category have not yet been well studied [8]. The signal from these aforementioned studies served as an impetus to further investigate the role of KDT in GGE syndromes.

The goal of this study was to evaluate the efficacy and tolerability of the MAD, a variation of the ketogenic diets, in a pediatric population with drug-resistant GGE. The MAD was selected because of the advantages of being a less restrictive and better tolerated diet than the CKD [9].

## Materials and methods

Retrospective medical chart review was performed in patients with drug-resistant GGE being treated with the MAD at Comer Children's Hospital at the University of Chicago (Chicago, IL) from 2017 to 2020. Thirteen patients with a diagnosis of drug-resistant GGE were identified based on clinical history, physical examination, and EEG characteristics. Epilepsy syndromes represented included CAE, eyelid myoclonia with absences (EMA or Jeavons syndrome), MAE or Doose syndrome, juvenile absence epilepsy (JAE), and generalized epilepsy with generalized tonic-clonic seizure alone (GE-GTCA). Per history, none of the patients with a diagnosis of EMA demonstrated hand-waving episodes suggestive of sunflower syndrome [10].

The MAD was initiated under supervision of a pediatric epileptologist and a ketogenic dietitian, with a daily carbohydrate allowance of 10-20 grams and encouragement of high dietary fat intake. At home, urine ketones were checked daily until ketonuria was achieved. Serum ketones were checked once ketonuria was achieved, at the one-month and three-month follow-up appointments.

For three months following diet initiation, participants were monitored for diet tolerability and effect on seizures. After three months, patients were continued on dietary therapy at the discretion of the patient and the patient's primary epileptologist. Patients' demographic, clinical, and laboratory information were collected during office visits. Response to the treatment including seizure frequency, duration, severity, and non-laboratory side effects were monitored and reported by patient and/or patient guardians. During dietary therapy, discontinuation of previously prescribed ASMs was initiated at the discretion of the patient's primary epileptologist. The main rationale for discontinuation of the medication was due to improvement in seizure control. The specific rational for which medication(s) to discontinue was not well elaborated.

DRE is defined according to the International League Against Epilepsy (ILAE) as the failure to achieve sustained seizure freedom after adequate trials of two tolerated, appropriately chosen, and used ASMs [11]. None of the patients in the study had a diagnosis of GLUT1DS based on the available genetic testing results with an epilepsy gene panel.

## Results

Thirteen patients, seven females (54%), met the study criteria. Mean age at seizure onset was 6.2 years and the mean age at diet initiation was 9.5 years. An average of 3.4 years elapsed between seizure onset and diet initiation. At the time of diet initiation, patients had tried, on average, 3.8 ASMs (range: 2-6). All patients completed at least three months of dietary

therapy with an average duration of 9.3 months at the last follow-up visit. Multiple seizure types, including typical absence, atypical absence, eyelid myoclonia, generalized tonic-clonic, atonic, and myoclonic-atonic seizures were observed (*table 1*). All 13 patients achieved ketonemia within four weeks after diet initiation. Additionally, all 13 patients self-reported compliance with dietary therapy which was supported by laboratory evidence of ketonemia and ketonuria.

After undergoing the KDT for three months, 12/13 (92%) patients experienced greater than 50% reduction in seizure frequency, 6/13 (46%) patients became seizure-free, and 7/13 (54%) were able to discontinue at least one ASM. Average seizure reduction per patient was 85.6%.

Seizure reduction per seizure type and epilepsy syndrome are outlined in *tables 2* and *3*. All seizure types studied demonstrated a greater than 85% reduction in frequency at the three-month follow-up visit. Seizure types with the greatest reduction were generalized tonic-clonic seizures followed by absence seizures (96.6% and 93.5%, respectively). Four out of five GGE syndromes represented in this study showed significant seizure reduction, with JAE and EMA reaching more than 90% seizure reduction for all types of seizures. However, one patient with GE-GTCA did not experience a reduction in seizure frequency or severity (*table 4*).

Despite all patients achieving ketonemia, there was no apparent correlation between level of ketonemia and degree of seizure reduction. For the patients who were able to stop at least one ASM, the discontinued medications did not show any obvious trend and were chosen by prescribers' perceived impression of the drug's efficacy and potential side effects.

While on the dietary therapy, one patient reported an adverse effect of fatigue that may be attributed to the diet. There was no report of significant gastrointestinal disturbances or laboratory evidence of dyslipidemia, nephrolithiasis, liver transaminitis, or excessive weight change.

## Discussion

GGE is a common epilepsy syndrome with a typical age at onset in childhood and adolescence involving many seizure types, with a generalized or bi-hemispheric onset and characteristic EEG findings of a normal background activity with stereotyped generalized spike and wave discharges. GGEs consist of multiple epilepsy syndromes including, but not

▼ **Table 1.** Patient characteristics.

| | |
|---|---|
| Female : Male | 7:6 |
| Age (years) at seizure onset | 6.2 (1-17) |
| Age (years) at diet onset | 9.5 (2 - 20) |
| Number of previously administered ASMs | 3.8 (2 - 6) |
| Patients per epilepsy syndrome: | |
| Childhood absence epilepsy | 4 (31) |
| Eyelid myoclonia with absences (or Jeavons syndrome) | 4 (31) |
| Myoclonic-atonic epilepsy or Doose syndrome | 3 (23) |
| Juvenile absence epilepsy | 1 (8) |
| Generalized tonic-clonic alone | 1 (8) |
| Seizure types represented: | |
| Typical absence / atypical absence | 7 (54) |
| Generalized tonic-clonic | 6 (46) |
| Eyelid myoclonia | 4 (31) |
| Atonic | 2 (15) |
| Myoclonic-atonic | 3 (23) |

ASM: anti-seizure medications. Values are presented as mean (range) or number (%) unless otherwise indicated.

▼ **Table 2.** Seizure frequency per seizure type.

| Seizure type | Frequency at diet initiation | Frequency after 3 months on diet | Percent reduction |
|---|---|---|---|
| Eyelid myoclonia | 47.5 (5 - 150) | 3.5 (1 - 12) | 92.7 |
| Myoclonic-atonic | 25 (20 - 30) | 3.3 (0 - 10) | 86.8 |
| Absence or atypical absence | 13.28 (1 - 30) | 0.86 (0 - 3) | 93.5 |
| Atonic | 12.5 (5 - 20) | 2.5 (0 - 5) | 88.9 |
| Generalized tonic-clonic (per month) | 3.7 (0.2 - 12) | 0.125 (0 - 1) | 96.6 |

Values represent number of daily seizure frequency (range) except for GTC which is reported as monthly frequency.

limited to, benign myoclonic epilepsy of infancy, MAE (Doose syndrome), CAE, JAE, EMA, epilepsy with myoclonic absence, juvenile myoclonic epilepsy, and generalized epilepsy with GTC seizure alone.

Conventional treatment for seizures of GGEs is an ASM such as valproic acid, lamotrigine, levetiracetam, and ethosuximide, with variable response and adverse effects. The overall prognosis of GGEs is relatively favorable. However, approximately 15% of patients with GGEs may develop a DRE [12].

Although KDT is a century-old treatment for epilepsy, the data remains limited regarding its applications for GGE syndromes. Previous studies have demonstrated two syndromes, CAE and MAE, to be well-responsive to KDT [13]. In a cohort from John Hopkins hospital of 21 patients with CAE, 82% of patients demonstrated >50% seizure reduction on either classic the ketogenic diet or MAD [14]. A recent large multicenter retrospective cohort study of MAE found that 79% (131/166) of patients responded favorably to the ketogenic diet, and the dietary therapy was found to be most effective for seizure control [15]. These results are suggestive of a potential therapeutic application of KDT for other GGE syndromes [16].

▼ **Table 3.** Overall seizure reduction per syndrome, per patient, and ability to wean prior anti-seizure medications.

| | |
|---|---|
| Seizure reduction per epilepsy syndrome: | |
| Juvenile absence epilepsy | 100% |
| Absence and eyelid myoclonia (or Jeavons syndrome) | 93.8% |
| Childhood absence epilepsy | 89.8% |
| Myoclonic-atonic epilepsy or Doose syndrome | 74.1% |
| Generalized tonic clonic alone | No change |
| Average seizure reduction per patient | 85.6% |
| Patients with >50% seizure reduction | 92% |
| Patients achieving seizure freedom | 6 (46) |
| Patients able to wean off at least one seizure medication | 7 (54) |

Values are presented as percentage or number (%) unless otherwise indicated.

Various types of ketogenic diets have been used in epilepsy. When comparing the MAD to the CKD, the MAD offers some advantages as a less restrictive diet and, therefore, likely to enhance treatment adherence, allowing for diet initiation in the out-patient setting [9].

In our study, 12/13 patients (92%) experienced greater than 50% reduction in seizure frequency three months after starting the MAD. Six patients (46%) became seizure-free, and 7/13 (54%) were able to discontinue at least one ASM. All patients tolerated the treatment well without significant side effects reported or observed under laboratory surveillance. The treatment response was seen for all seizure types with generalized tonic-clonic seizures and absence seizures demonstrating the greatest response, with more than 90% seizure reduction. Four out of five epilepsy syndromes, CAE, JAE, MAE, and EMA, showed a significant reduction in seizure frequency. The only epilepsy syndrome in the study with unchanged seizure frequency was GE-GTCA. However, despite no change in the seizure frequency, the patient with GE-GTCA was able to discontinue all other ASMs and remain on only MAD monotherapy without worsening of seizures.

The findings in the study are encouraging as they demonstrate that a dietary therapy can be an effective

▼ **Table 4.** Demographics and outcomes of patients diagnosed with drug-resistant GGE treated with the MAD at the University of Chicago, 2017-2020.

| Patient | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Syndrome | CAE | CAE | CAE | CAE | EMA | EMA | EMA | EMA | MAE | MAE | MAE | GE-GTCA | JAE |
| Gender | F | M | F | M | F | F | F | F | M | M | F | M | M |
| Age (yrs) at Sz onset | 7 | 4 | 5 | 6 | 3 | 9 | 11 | 2 | 5 | 1 | 1 | 17 | 9 |
| Age (yrs) at MAD onset | 11 | 6 | 7 | 8 | 15 | 16 | 13 | 6 | 6 | 2 | 3 | 20 | 11 |
| ASMs tried prior to MAD | 5 ACZ CLB ESM LTG VPA | 5 ACZ ESM LEV LTG VPA | 4 ESM LEV LTG VPA | 2 CBD ESM | 6 CBD CLB ESM LEV LTG CBD | 5 ACZ CBD ESM TPM VPA | 2 LMT CLB | 3 CLB LTG VPA | 5 CBD CLB LEV LTG VPA | 4 ESM LEV PB VPA | 3 CLB FBM VPA | 4 LEV LTG TPM VPA | 2 LEV VPA |
| ASMs after 3 months of MAD | 0 | 0 | 1 ESM | 0 | 3 ESM LTG CBD | 1 ESM | 2 LMT CLB | 3 CLB LTG VPA | 0 | 2 ESM LEV | 0 | 2 LEV LTG | 1 VPA |
| Frequency of Sz at initiation (per day) | | | | | | | | | | | | | |
| Absence | 6 | 20 | 3 | 20 | | | | | | 3 | | | 1 |
| Eyelid myoclonia | | | | | 20 | 15 | 5 | 150 | | | | | |
| GTC (per month) | | | | | 20 | 4 | 1 | 12 | 1 | 0.2 | | 1 | |
| Atonic | | | | | 4 | | | | | 5 | 20 | | |
| Myoclonic-atonic | | | | | | | | | 30 | 20 | 25 | | |
| Frequency of Sz at 3 months (per day) | | | | | | | | | | | | | |
| Absence | 0 | 3 | 0 | 2 | 1 | | | | | 0 | | | 0 |
| Eyelid myoclonia | | | | | 1 | 0 | 1 | 12 | | | | | |
| GTC (per month) | | | | | 0 | 0 | 0 | 0 | 0 | 0 | | 1 | |
| Atonic | | | | | | | | | | 0 | 5 | | |
| Myoclonic-atonic | | | | | | | | | 0 | 0 | 10 | | |
| Seizure reduction | 100% | 85% | 100% | 90% | 96% | 100% | 83% | 93% | 100% | 100% | 67% | No change | 100% |

ACZ: acetazolamide; CAE: childhood absence epilepsy; CBD: cannabidiol; CLB: clobazam; EMA: eyelid myoclonia with absences; ESM: ethosuximide; FBM: felbamate; GE-GTC: generalized epilepsy with GTC alone; JAE: juvenile absence epilepsy; LCM: lacosamide; LEV: levetiracetam; LTG: lamotrigine; MAE: myoclonic atonic epilepsy; PB: phenobarbital; TPM: topiramate; VPA: valproic acid; Sz: seizure.

alternative treatment for GGE. Furthermore, it emphasizes that the relatively less restrictive type of dietary therapies, such as the MAD, can be effective in controlling seizures of GGEs, with good treatment adherence and few side effects. The dietary therapy also offers an additional benefit in aiding the ability to discontinue ASMs and, therefore, help to reduce and minimize the side effects that are often inevitable with polypharmacy.

Our results are consistent with previous findings regarding the use of the ketogenic diet for CAE and MAE, and may aide to further expand the spectrum of seizure types and epilepsy syndromes responsive to dietary therapies. It may also be reasonable to assume

that for GGE without DRE, patients would have a similar response to the diet, and this may serve as a reason to consider earlier use of dietary therapy. However, further studies are needed to confirm this postulation. Lastly, in the study, there was an elapsed time of more than three years from seizure onset, with an average of 3.8 previously administered ASMs and ongoing daily seizures before patients were treated with KDT. The delay to treatment may be multifactorial and potentially related to gaps in current knowledge, lack of access to an epilepsy center and specifically a ketogenic diet program, and the conventional thought of employing ketogenic diet therapies as a treatment of last resort. Regardless, we believe that delineating and demonstrating the potential application and efficacy of the diet for many GGE syndromes will help to advance the breadth of knowledge and raise awareness of the earlier use and appropriate selection of KDT in patients with epilepsy.

There are several limitations to this retrospective study which can hopefully be addressed in future investigations. The sample size was small and potentially less generalizable for JAE and GE-GTC syndromes. The full array of GGE syndromes was not represented, for example, no patients in the study carried a diagnosis of juvenile myoclonic epilepsy (JME). Additionally, seizure frequency was self-reported by patients, parents, and guardians which can be challenging to quantify as most patients experienced several seizure types including potentially subtle seizures. The impact of dietary treatment on patient EEG tracings and the serum concentrations of ASMs were not evaluated. Future studies could benefit from a larger sample size with a full spectrum of GGE syndromes, comparison of MAD vs CKD as treatment for GGE, evaluation of serum ASM concentrations in the setting of KDT, and a multicenter prospective study evaluating the efficacy of KDT in comparison to common first-line ASMs for GGEs.

## Conclusion

The MAD is an effective and well-tolerated treatment for children with drug-resistant GGE, with a low side effect profile. The MAD offers an additional benefit in aiding discontinuation of ASMs and minimizing the side effects from polypharmacy treatment. MAD, therefore, should be considered as an alternative treatment for patients with GGE and may be used earlier in the treatment course. A prospective study with a larger number of patients with a full spectrum of GGE syndromes, to compare the efficacy of KDT to current first-line medication treatment, may be considered. ■

**Supplementary material.**
Summary slides accompanying the manuscript are available at www.epilepticdisorders.com.

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None of the authors have any conflicts of interest to disclose.

## Key points

- After undergoing the modified Atkins diet for three months, patients with drug-resistant genetic generalized epilepsy experienced a reduced seizure burden.
- Over 90% of patients experienced greater than 50% reduction in seizure frequency, 46% became seizure-free, and over 50% were able to stop at least one anti-seizure medication.
- The modified Atkins diet appears to be an effective and well-tolerated treatment for children with drug-resistant genetic generalized epilepsy.
- The diet provides additional benefit in aiding discontinuation of anti-seizure medications and, therefore, helps to minimize the side effects from polypharmacy.

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## TEST YOURSELF

**(1) Which of the following epilepsy syndromes is not considered a genetic generalized epilepsy?**
A. Childhood absence epilepsy
B. Myoclonic atonic epilepsy (Doose syndrome)
C. Rolandic epilepsy
D. Dravet syndrome
E. C and D

**(2) What are typical EEG findings commonly seen in a patient with genetic generalized epilepsy?**
A. Normal EEG background without epileptiform discharges
B. Normal EEG background with stereotyped generalized spike and wave discharges
C. Normal EEG with stereotyped focal epileptiform spikes
D. Diffuse background slowing and pleomorphic focal interictal epileptiform discharges
E. Discontinuous background with pleomorphic diffuse interictal epileptiform discharges

**(3) What is the most common side effect of ketogenic dietary therapy?**
A. Gastrointestinal disturbance
B. Tinnitus
C. Nephrolithiasis
D. Alopecia
E. Severe acidosis

*Note: Reading the manuscript provides an answer to all questions. Correct answers may be accessed on the website, www.epilepticdisorders.com.*