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IS Balanced Diet Has Effect On Epileptics?

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This study the effect of balanced diet on the cognition, growth, biochemical and electroencephalogram changes among epileptic children and adolescents, this study was carried on 100 epileptic children and adolescents of both sexes, their ages ranged between 11 -14 years, food consumption for all cases was calculated. All cases were grouped into two groups, 50 cases were under special balanced diet program (group I) and 50 cases were left on their regular diet (group II). All cases were subjected to complete clinical and neurological examination, anthropometric assessment and behavior assessment. Biochemical assessment for serum calcium, zinc, copper and hemoglobin were assessed. Electroencephalogram was done for all cases at the start and by the end of the study. Present results showed a significant changes in psychometric behavior between both groups. Patients of group II show highly significant decrease in levels of hemoglobin, copper, zinc and calcium, in comparison to patients of group I. A highly significant increase in anthropometric measurements among patients of group I, as compared to patients of group II. Regarding changes in electroencephalogram there was an improvement in 22% of cases of group I as compared with group II which showed an improvement in only 6% of cases. Then we concluded that children and adolescents with epilepsy are often more sensitive to the world around them than others, therefore it is important to ensure that their nutrition is as well balanced as possible, for better life, improvement and efficacy for antiepileptic drugs.

Key words: Epilepsy, child health, electroencephalogram, nutrition

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INTRODUCTION

Epilepsy is a neurological disorder that affects people in every country throughout the world. Epilepsy is also one of the oldest conditions known to mankind (WHO, 2001). It is a common chronic neurological disorder that is characterized by recurrent unprovoked seizures (Blume *et al.*, 2001). These seizures are transient signs and/or symptoms due to abnormal, excessive or synchronous neuronal activity in the brain (Fisher *et al.*, 2005). About 50 million people worldwide have epilepsy at any one time (WHO, 2001). Epilepsy is usually controlled, but not cured, with medication. However, over 30% of people with epilepsy do not have seizure control even with the best available medications (Cascino, 1994).

Epilepsy's approximate annual incidence rate is 40-70 per 100,000 in industrialized countries and 100-190 per 100,000 in resource-poor countries; socioeconomically deprived people are at higher risk. In industrialized countries the incidence rate decreased in children but increased among the elderly during the three decades prior to 2003, for reasons not fully understood (Sander, 2003).

Seizures occur when there is an excess of excitatory processes in the brain compared with inhibitory processes. Changes in afferent excitation, disinhibition, shifts in extracellular ion concentrations, voltage-gated ion-channel opening and enhanced neuronal synchrony are all important in the initiation and propagation of seizure activity. Neuronal activity is regulated by the concentration of ions in the extracellular and intracellular spaces and the selective flux of these ions across the neuronal membrane. Voltage-gated or ligand-gated ion-channel genes are, therefore, attractive candidate genes for the epilepsies. Mutations in such genes could lead to channel dysfunction, which could alter ion concentrations across the cell membrane, resulting in reduced or increased neuronal excitability (Fisher, 1995).

The actual causes are still relatively unknown. The spontaneous discharges characteristic of epilepsy may occur for no apparent reason, or may be triggered by a wide range of things, including exposure to an allergen; congenital defects; lead poisoning or injury during childbirth; use of or intoxication from alcohol or drugs; drug or alcohol withdrawal, fever, flashing lights, hunger, hypoglycemia, hypocalcemia, uremia, hypoxia, infection, vascular disturbances, lack of sleep; metabolic or nutritional imbalances, physical or emotional trauma (Duffy, 2000). Genetic factors are thought to contribute to the aetiology in up to 60% of cases. Various molecular and cellular mechanisms give rise to epilepsy and epilepsy

genes fall into several distinct categories. They include genes in which mutations cause abnormal ion-channel function, disordered brain development, progressive neurodegeneration and disturbances of cerebral energy metabolism (Fisher, 1995).

Common nutritional deficiencies quoted with epilepsy are manganese, zinc and magnesium, so that nutritional analysis and supplementation where necessary is vital to successful treatment. Vitamin E supplementation alone has been quoted as reducing seizure rates by as much as 50%. Selenium supplementation may also be of benefit (Osiecki, 1998).

The ketogenic diet is an effective therapy for all types of difficult-to-control seizures. It has been mainly studied in patients who have failed therapy with 2 anticonvulsant medications. There are several different forms of the ketogenic diet, including the classic diet (Freeman *et al.*, 2007) and, more recently, a modified Atkins-like diet (Eric *et al.*, 2008), they all are approximately equally effective, at Johns Hopkins, they have primarily used the classic ketogenic diet.

It is essential that anyone taking anticonvulsant medications take adequate supplementation of nutrients that are likely to be depleted by both the condition and the interventional drug under the supervision of a qualified health care professional. Equally, it will be important to continually monitor liver and other bodily functions through regular medical checkups as these drugs play havoc with the body's homeostatic mechanisms. Unfortunately, a large number of medical practitioners demonstrate remarkable incompetence when it comes to clinical nutrition (Galland, 1997).

There are many potential causes of seizures. Those related to nutrition are hunger, hypoglycemia, hypocalcemia and nutritional imbalances, so, it is essential to try to remove these possible problems. In this study we studied the effect of balanced diet on the cognitive functions, growth measurements, biochemical changes and electroencephalogram (EEG) changes among epileptic children and adolescents under antiepileptic treatment.

MATERIALS AND METHODS

This study was carried on 100 epileptic children and adolescents of both sexes, having the same daily activity (≥ 60 min) and lifestyle, their ages ranged between 11 up to 14 years and all of them were under calculated doses of antiepileptic treatment by either valproate and/or carbamazepine, from those patients attending the outpatient clinic of Pediatric Neurology El Mounira Outpatient Clinic, of Student Hospital, over a period of

eighteen months December, 2006 till June, 2008. Cases were grouped into two groups. Group I 50 cases were put under controlled balanced diet with average 2250 calories/day (2000-2500 calories/day), designed by the clinical nutritionist specially for this study, according to their ages and socioeconomic status including three variable forms (Breakfast, Lunch and Dinner) and the other 50 cases were left on their regular ordinary diet (Group II).

All cases involved in this study were subjected to full nutritional, clinical and neurological history, as well as complete general and neurological examination, including the assessment of nutritional status and anthropometric assessment.

Assessment of dietary consumption: using the nutritional questionnaire sheet prepared for those cases, the amount of food consumed were calculated, then estimation of nutrient intake which based on the repeated 24 h dietary recall method, then by using the food composition tables and Software World Food 2 copyright 1996, the total intake of calories, iron, zinc, copper, calcium, vitamin B₁₂ and vitamin E, all were calculated.

Controlled balanced diet with average 2250 calories/day (2000-2500 calories/day), including three variable forms breakfast, lunch and dinner was designed specially for this study and given to cases of group I.

Assessment of weight, height and calculation of BMI at the start of the study and by the end of the study, according to the National Centre For Chronic Disease prevention and Health Promotion.

Psychometric studies including tests of behavior using Revised Behavior Problem Checklist (RBPC), according to Herbert and Donald (1986), which is translated into arabic by El-Khloly and Abo-Elala (1996), were done for all cases of the study at the start of the study and by the end of the study then the differences between both groups were examined.

Biochemical assessment: Each case in the study a venous fasting blood sample was withdrawn for analysis of serum calcium colorimetrically using Kit brushed from Bio Merieux Company, zinc and copper according to method of Homster and Zac (1985) and hemoglobin concentration and random blood sugar were done.

Plasma vitamin B₁₂ was estimated by radioimmunoassay method using Kits obtained from Diagnostic Product Corporation. Estimation of alpha tochoferol (Vitamin E) was determined using the method of Desia and Machlin (1985).

Electroencephalogram was done for all cases at the start of the study and by the end of the study for each case.

Statistical analysis: Data was analyzed using the Microsoft Excel and SPSS 11.5 for Windows software packages.

RESULTS

Table 1 shows that the average food consumed by all cases at the start of the study as compared with the Recommended Daily Intake (RDA), was high regarding caloric intake and carbohydrate intake, while on the other hand there was a decrease in the intake of iron, calcium, zinc, copper as well as vitamin B₁₂ and E.

Table 2 and 3 show the recorded weight, height and BMI of each group at the start of the study and by the end of the study. In group I, there was a highly statistical significant increase in weight and a statistical significant increase in BMI (56.086±3.95 kg, 24.25±3.56 kg m⁻²) respectively, as compared to their recorded measures at the start of the study (48.9±3.04 kg, 21.04±3.65 kg m⁻²) respectively, with no statistical significance regarding their height, While in cases of group II there was no statistical significance differences regarding their height, weight and BMI by the end of the study (153.19±4.55 cm, 50.47±2.97 kg and 21.32±3.15 kg m⁻²), respectively, as compared to their recorded measures at the start of the study (151.2±3.65 cm, 49.2±2.85 kg, 21.52±2.96 kg m⁻²), respectively.

Regarding the difference between both groups by the end of the study (Table 4), we found that there was a highly statistical significant difference in the form of increase in weight and BMI in cases of group I (56.086±3.95 kg, 24.25±3.56 kg m⁻²), respectively, as compared to cases of group II (50.47±2.97 kg and 21.32±3.15 kg m⁻²), respectively.

Regarding the psychometric results by the end of the study, Table 5 shows that, there was a significant high

Table 1: The food consumption by all cases in the start of the study as compared to the RDA according to their ages

| Nutritional supply | Total energy (Kcal day ⁻¹) | Total CHO (g day ⁻¹) | Iron ----- | Calcium (mg day ⁻¹) | Zinc ----- | Copper ----- | Vit. B ₁₂ | Vit. E |
|--------------------------------|---|-------------------------------------|---------------|------------------------------------|---------------|-----------------|----------------------|-----------|
| Range | 2765-3896 | 359-453 | 8.78-9.34 | 875-993 | 9.7- 10.6 | 1.9- 2.3 | 0.87 -0.94 | 6.02 -6.9 |
| Mean (n= 100) | 3215.2/3000 | 418.6 | 9.03/13 | 921/1250 | 10.03/15 | 2.1/2 | 0.85 | 6.45 |
| RDA according to age range (%) | 107.17 | 117.17 | 69.46 | 73.68 | 66.87 | 105 | 70.833 | 92.143 |

scores of conduct disorders, socialized aggression and anxiety withdrawn scale in cases of group II compared to scores achieved by cases of group I. While, a significant

Table 2: Mean value±SD of weight, height and BMI of cases of group I at the start of the study as compared with their measures by the end of the study

| Variables | Height (cm) | Weight (kg) | BMI (kg m ⁻²) |
|---|-------------|-------------|---------------------------|
| Cases of group I at the start of the study (n= 50) | | | |
| Range | 145.2-159.7 | 44.2-57.8 | 17.33-27.42 |
| Mean | 152.04 | 48.9 | 21.04 |
| ±SD | 4.05 | 3.04 | 3.65 |
| Cases of group I by the end of the study (n= 50) | | | |
| Range | 146.8-158.9 | 50.8-62 | 20.12-28.77 |
| Mean | 153.346 | 56.086 | 24.25 |
| ±SD | 3.87 | 3.95 | 3.56 |
| p-value | 0.12 | 0.001*** | 0.001*** |

***Significant at p = 0.001

Table 3: Mean value±SD of weight, height and BMI of cases of group II at the start of the study as compared with their measures by the end of the study

| Variables | Height (cm) | Weight (kg) | BMI (kg m ⁻²) |
|--|-------------|-------------|---------------------------|
| Cases of group II at the start of the study (n= 50) | | | |
| Range | 145.5-158.5 | 44-58.6 | 17.6-28.3 |
| Mean | 151.2 | 49.2 | 21.52 |
| ±SD | 3.65 | 2.85 | 2.96 |
| Cases of group II by the end of the study (n= 50) | | | |
| Range | 147.3-161.5 | 46-55 | 17.64-25.35 |
| Mean | 153.19 | 50.47 | 21.32 |
| ±SD | 4.55 | 2.97 | 3.15 |
| p-value | 0.2 | 0.06 | 0.1 |

Table 4: Mean value±SD of height, weight and BMI of cases of group I as compared with cases of group (I) by the end of the study

| Variables | Height (cm) | Weight (kg) | BMI (kg m ⁻²) |
|--|-------------|-------------|---------------------------|
| Cases of group I by the end of the study (n= 50) | | | |
| Range | 146.8-158.9 | 50.8-62 | 20.12-28.77 |
| Mean | 153.346 | 56.086 | 24.25 |
| ±SD | 3.87 | 3.95 | 3.56 |
| Cases of group II by the end of the study (n= 50) | | | |
| Range | 147.3-161.5 | 46-55 | 17.64-25.35 |
| Mean | 153.19 | 50.47 | 21.32 |
| ±SD | 4.55 | 2.97 | 3.15 |
| p-value | 0.3 | 0.001*** | 0.001*** |

***Significant at p = 0.001

Table 5: Percentage and number of cases with psychometric studies including tests of behavior using revised behavior problem checklist for cases of group I as compared with cases of group II by the end of the study

| Variables | Aggression | Restlessness | Retardation | Hyperactivity | Lack of concentration | Distraction | Depression | Speech disorders |
|---|--------------|--------------|-------------|---------------|-----------------------|--------------|--------------|------------------|
| Cases of group I by the end of the study | 18% 9 cases | 40% 20 cases | 2% 1 case | 42% 21 cases | 42% 21 cases | 36% 18 cases | 20% 10 cases | No cases |
| Cases of group II by the end of the study | 20% 10 cases | 36% 18 cases | 4% 2 cases | 38% 19 cases | 64% 32 cases | 46% 23 cases | 28% 14 cases | No cases |

Table 6: Mean values±SD of hematological and biochemical results for cases of group I as compared with cases of group II by the end of the study

| Variables | Hb | Copper | Zinc | Calcium | Vit. B ₁₂ (Pg mL ⁻¹) | Vit. E (mg dL ⁻¹) |
|-----------------|-----------|-------------|-----------|-----------|---|-------------------------------|
| Group I | | | | | | |
| Range | 11-12.45 | 102.9-115.9 | 92.75-100 | 4.3-4.9 | 305-651 | 1.64-2.31 |
| Mean | 11.72 | 109.4 | 96.2 | 4.63 | 512.3 | 2.09 |
| ±SD | 0.65 | 6.05 | 3.08 | 0.05 | 36.2 | 0.015 |
| Group II | | | | | | |
| Range | 9.5-11.35 | 98-110 | 88-92.5 | 3.75-4.85 | 294-582 | 0.9-1.73 |
| Mean | 10.49 | 104.25 | 90.2 | 4.02 | 408.54 | 0.98 |
| ±SD | 0.79 | 5.67 | 2.15 | 0.23 | 31.6 | 0.02 |
| p-value | 0.05* | 0.05* | 0.05* | 0.02* | 0.003** | 0.001*** |

difference between both groups with higher frequency in group I regarding restlessness while regarding the psychomotor retardation, distractibility and depression showed higher frequency in group II in comparison to patients of group I.

Regarding the laboratory results by the end of the study, Table 6, show that cases of group II have highly significant low levels of hemoglobin, copper, zinc and calcium as 10.49±0.79 g dL⁻¹, 104.25±5.67 µg dL⁻¹, 90.2±2.15 µg dL⁻¹ and 4.02±0.23 mg dL⁻¹, respectively, as well as lower levels of vitamin B₁₂ and vitamin E as 408.54±31.6 Pg mL⁻¹ and 0.98±0.02 mg dL⁻¹, as compared with their levels among cases of group I, 11.72±0.65 g dL⁻¹, 109.4±6.05 µg dL⁻¹, 96.2±3.08 µg dL⁻¹ and 4.63±0.05 mg dL⁻¹, respectively and 512.3±36.2 Pg mL⁻¹ and 2.09±0.015 mg dL⁻¹, respectively.

Regarding the neurological findings, Table 7 shows that the recurrence of epileptic fits among cases of group I was less frequently occurred by 83% of cases as compared with those of group II 60% of cases, also comparison between both groups as evident by EEG changes by the end of the study, we found that there was an improvement in the EEG findings in 11 cases (22%) of group I, as compared with the improvement in EEG of cases of group II which showed an improvement in only 3 cases (6%).

The neurophysiological examination for all cases at the start of the study and by the end of the study in the form of EEG done for all cases of the study we found that the abnormal EEGs done at the start of the study had either an epileptogenic activity in the form of slow wave and spike followed by secondary generalization, or subcortical epileptogenic activity, while the EEGs for the same patients done by the end of the study, shows normal EEG findings.

Table 7: No. of cases with abnormal EEG at the start of the study and by the end of the study in both groups of cases

| Group | No. of cases with abnormal EEG | |
|-------------|--------------------------------|-------------------------|
| | At the start of the study | By the end of the study |
| I (n = 50) | 38/50 | 27/50 |
| II (n = 50) | 36/50 | 33/50 |

DISCUSSION

Epilepsy is the most common serious neurological condition in the world. Traditional epilepsy management includes pharmacological treatment, epilepsy surgery and vagal nerve stimulation. Despite these therapies, the Ketogenic Diet (KD), which has been in use since 1921, is a treatment option for many of these children. Estimates of the overall efficacy of the KD in controlling seizures were reported as follows: 16% became seizure free, 32% had a >90% reduction in seizures and 56 had a >50% reduction, through an unknown mechanism (Beth *et al.*, 2008). From this point of view we asked the question, is balanced diet have an effect on epileptic children and adolescents, or not?

In this study we stated that adequate balanced diet can help in improving the clinical conditions of epileptic patients guided by EEG improvement and nutritional support may help in adjusting and minimizing the doses of antiepileptic drugs, as the recurrence of epileptic fits among cases of group I was improved clinically in (83% of cases), as compared with those improved clinically among cases of group II (60% of cases) and the EEG findings of cases of group I shows an improvement in 11 cases (22%), as compared with the improvement in EEG of cases of group II which showed an improvement in only 3 cases (6%), which means that regulation of diet has an improving effect on the EEG findings and these findings are agreed with Galland (1997), who stated that it is essential that anyone taking anticonvulsant medications take adequate supplementation of nutrients that are likely to be depleted by both the condition and the interventional drug under the supervision of a qualified health care professional. Equally, it will be important to continually monitor liver and other bodily functions through regular medical checkups as these drugs play havoc with the body's homeostatic mechanisms. Unfortunately, a large number of medical practitioners demonstrate remarkable incompetence when it comes to clinical nutrition (Galland, 1997). Also, our findings can be explained and agreed by Hongkui *et al.* (2008), who stated that diet differentially modifies the development of brain physiological functions.

The average food consumed by all cases at the start of the study was low as compared with the Recommended Daily Intake (RDA). The intake of calcium and zinc was

low and by the end of the study laboratory results showed that cases of group II have highly significant low levels of zinc and calcium than those of group I which means that adjustment of the food intake by giving balanced diet improve the levels of that elements which had a good effect on the clinical and EEG findings and this is agreed by Osiecki (1998), who stated that common nutritional deficiencies quoted with epilepsy are manganese, zinc, calcium and magnesium, so that nutritional analysis and supplementation where necessary is vital to successful treatment of epilepsy. Osiecki (1998) stated also that, vitamin E supplementation alone has been quoted as reducing seizure rates by as much as 50%, which is agreed with our study as the levels of vitamin E was low by the start of the study and improved by the end of the study which was reflected on the results of the EEG findings. Also Selenium supplementation may also be of benefit.

In this study we found that levels of vitamin B₁₂ was low at the start of the study and adjusted by the end of the study and was reflected on the clinical picture of patients as well as on their EEG findings and this is agreed by Bland (1999), he stated that supplementation of the vitamins A, B, C, and E and other vitamins/minerals, that may be depleted by the condition or drug is indicated.

In this study we found that supplementation of adjusted balanced diet had a good effect on the clinical as well as EEG findings of epileptic patients which means that we must think about the alternatives to treatment in order to minimize or adjust the amount of medications given because of the potential side effects and interaction with foods and this is agreed with Bland (1999), he stated that functional and nutritional considerations must be put into our mind with epileptic patients, he stated that eating smaller and more frequent balanced meals, not drinking large quantities of liquids all at once and taking two tablespoons of olive oil daily, increasing intake of beet greens, chard, eggs, green leafy vegetables, raw cheese, raw milk, raw nuts, seeds and soybeans (GM free and provided of course that the individual does not have an allergy to these) and drinking live, fresh juices made from beets, carrots, green beans, green leafy vegetables, peas, red grapes and sea-weed for concentrated nutrients, all has a beneficial effect on the condition of epileptic patients. He stated also that specifically working toward self-care and working toward becoming as free from drugs and seizures as possible. Make it a point to learn about your condition and the alternatives to treatment. Being aware of the drug you are taking. Know its potential side-effects and interaction with foods, herbal preparations, or other drugs you may be taking. How does this drug interact with body and mind? (Bland, 1999).

We may explain our results that adequate balanced diet supplied their bodies with the essential elements and vitamins which help and enhance the effect of the antiepileptic drugs as well as it is known that balanced diet modifies the development of brain physiological functions.

CONCLUSION

Children and adolescents with epilepsy are often more sensitive to the world around them than others, therefore it is important to ensure that their nutrition is as well balanced as possible, for better life, better improvement and better efficacy for antiepileptic drugs, as evidenced by improvement changes of EEG in about 22% of cases in the study. So, we can stated that adjustment of dietary intake by balanced diet can help in adjusting not only the dose of the anti-epileptics but also improving the general conditions as well as reduce the recurrence of epileptic fits.

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