Antiepileptic Drugs and Bone Health in Thai Children with Epilepsy

Sudathip Paticheep MD*,
Chanisa Chotipanich MD**, Khanittha Khusiwilai MD*,
Anuttara Wichaporn BSc***, Subsai Khongsaengdao MD****

** Division of Neurology, Department of Pediatric, Faculty of Medicine, Thammasat University, Pathumthani, Thailand*
*** Division of Nuclear Medicine, Chulaphorn Hospital, Bangkok, Thailand*
**** Division of Radiology, Thammasat Hospital, Pathumthani, Thailand*
***** Division of Neurology, Department of Medicine, Rajavithi Hospital, Department of Medical Services, College of Medicine, Rangsit University, Bangkok, Thailand*

Background: *Epilepsy is the most common neurological disease in children. The patient must take antiepileptic drug for controlling the seizure at least 2 years. Many previous studies show the effect of antiepileptic drug to vitamin D status and bone health.*
Objective: *To study the prevalence of vitamin D deficiency and bone mineral density in the children who are taking antiepileptic drug at least 6 months.*
Material and Method: *Thirty epileptic children who are 3-18 years old with taking antiepileptic drug at least 6 months and 30 healthy children in the same age were performed to investigate serum 25-hydroxyvitamin D, calcium, phosphorus, magnesium, creatinine, alkaline phosphatase, albumin, parathyroid hormone, spot urine calcium, spot urine phosphorus, spot urine creatinine and bone mineral density between October 2012 to September 2013.*
Results: *Seven epileptic children (23.3%), eight healthy children (26.7%) have vitamin D deficiency. Only 3 epileptic children who are cerebral palsy also have low bone mineral density. There is only statistical significant of decreased serum albumin (p-value = 0.03) and corrected serum calcium (p-value = 0.04) that reveal in epileptic children group.*
Conclusion: *Evaluation of serum 25-hydroxyvitamin D status and bone metabolism is essential in the treatment of childhood epilepsy.*

Keywords: *Bone mineral density, Vitamin D, Calcium metabolism, Antiepileptic drug, Epilepsy, Children*

J Med Assoc Thai 2015; 98 (6): 535-41
Full text. e-Journal: *http://www.jmatonline.com*

Epilepsy is a common worldwide problem in all ages. Estimates show that a quarter of epileptic patients are children[1]. The various prevalences range from 1.5 to 14 per 1,000 population worldwide[2]. There are relatively few data regarding epidemiology of epilepsy in Thailand. A community-based study in the rural parts of Thailand estimated the prevalence of epilepsy at 7.2 per 1,000 population. The two highest peaks of age groups were 5-9 and 25-34 years. This report may underestimate the accurate prevalence in Thailand[3].

All epileptic patients must be treated with antiepileptic drug(s) for at least for 2 years of seizure control. Many previous studies showed an association between the adverse effects of the antiepileptic drugs and bone health, for instance decreased bone mineral density (BMD), reduced vitamin D level and calcium metabolism changes[4-10]. Although, there were no studies explain exact mechanism of antiepileptic drug affected on bone metabolism[11-16]. Moreover, some studies show no association between antiepileptic drugs and BMD[17-19].

However, maintain normal metabolism of bone need various factors especially vitamin D, calcium, and parathyroid hormone. Interestingly, the active metabolite of vitamin D was decreased in the patients who taking antiepileptic drug[14]. Vitamin D function is to maintain skeletal calcium balance by promoting calcium absorption in the intestines, promoting bone resorption by increasing osteoclast number, maintaining calcium and phosphate levels for bone formation, and allowing proper functioning of parathyroid hormone to maintain serum calcium level. Long-term antiepileptic drug administration may be effect to bone metabolism, especially in childhood

Correspondence to:
Paticheep S, Division of Neurology, Department of Pediatric, Faculty of Medicine, Thammasat University, 99/10 Moo 18, Paholyothin Road, Klongluang, Pathumthani 12120, Thailand.
Phone: +66-2-9269514
E-mail: sudathipp@yahoo.com

and adolescence which are critical periods for bone mineralization.

In Thailand, there was only one publication of prevalence and risk factors of low BMD and 25-hydroxyvitamin D status in young healthy epileptic adult taking antiepileptic drugs. The result was a significant correlation between long-term use of antiepileptic drugs and low BMD[20].

The objectives of this research were to assess the prevalence of vitamin D deficiency, the prevalence of abnormal bone mineral density, and factors that may influence bone health in Thai epileptic children, who administered at least for 6 months of antiepileptic drug(s), compared with healthy children.

Material and Method

This case controlled study was conducted at Thammasat University hospital between October 2012 and September 2013. The study included thirty healthy Thai children aged 3 to18 years old and 30 Thai epileptic children of similar age who had received antiepileptic drugs for at least 6 months. Children who 1) had taken any vitamin or calcium supplement; 2) had underlying diseases of kidney, liver, metabolic or bone diseases; 3) had a family history of metabolic bone disease; and 4) had a history of bone fracture were excluded from the study. Informed consent was obtained from both their parents and the patients aged 10-18 years. This study was approved by the Human Research Ethics Committee of Thammasat University, project number MTU-EC-PE-2-060/55.

Clinical assessments, previous diseases, capability of ambulation, cerebral palsy and medication history were recorded in all patients. Laboratory data, including serum 25-hydroxyvitamin D level, total calcium, phosphorus, magnesium, albumin, creatinine, parathyroid hormone, spot urine calcium, spot urine phosphorus and spot urine creatinine, were collected between October 2012 and September 2013.

BMD was determined in whole body and lumbar spine (L1-L5 level) by dual-energy X-ray absorptiometer (DEXA) scan (Hologic, software version 12.6, Model Discovery W, S/N 81495). BMD values were analyzed by APEX system software version 3.2 Model: Discovery A, S/N 81165 and represented as Z scores adjusted by sex and age with reference to the Asian population.

Statistical analysis

Descriptive statistics was used for the demographic data. Analytical statistics compared the mean and standard deviation between control and epileptic groups, by using nonparametric two independent binomial statistical analyses, Wilcoxon-Mann-Whitney test by sing StatXact® Cytel® studio license number 2060107. Poisson variables of mean prevalence with 95% confidence interval cii (poisson variable) of factors that may influence bone health were analyzed by STATA® version 11. The prevalence of each antiepileptic drug used in children with significant factors that may influence bone health and prevalence of factors that may influence bone health between control children, children who treated with antiepileptic drug(s) between 6 to 12 months or more than 12 months were analyzed by using Poisson regression analysis with STATA® version 11. A significant p-value was $p<0.05$.

Results

Thirty epileptic children, 15 boys and 15 girls, aged between 42 to 215 months, mean (SD) of 104 (9) months were enrolled in this study. The thirty healthy children in the control group were 13 boys and 17 girls, aged between 44 to 179 months, means (SD) of 108 (7) months. There was no statistical difference of demographics such as age, weight, height between epileptic children and control groups. The demographic data is shown in Table 1.

Table 1. Demographic data of normal children and epileptic children

Demographic data	Control group	Epileptic group	p-value
Total number	30	30	-
Gender (male:female)	15:15	13:17	-
Age (month), mean ± SD (range)	108.20±7.61 (44-179)	104.90±9.31 (42-215)	0.63
Body weight (kilograms), mean ± SD (range)	33.33±3.35 (13.7-83.9)	34.32±3.96 (12.2-102.8)	0.75
Height (centimeters), mean ± SD (range)	133.50±4.13 (97-178)	129.20±3.95 (102-171)	0.38

p-value were estimated by Wilcoxon-Mann-Whitney test, significant p-value <0.05.
p-value were calculated by using StatXact® Cytel® studio license number 2060107.

In the epileptic group, 3 of 30 children were diagnosed with cerebral palsy with quadriplegia, paraplegia or hemiplegia respectively. The mean duration of antiepileptic drug therapy was 23.04±16.33 months (mean ± SD). The polypharmacy split of antiepileptic drugs used was 56.7 percent on monotherapy and 43.3 percent on polytherapy. These comprised 14 percent of old generation, 3 percent of new generation and 13 percent either old or new antiepileptic medications, as shown in Table 2. Seven children in the epileptic group and eight children in the control group were found to have vitamin D deficiency (Table 3). The prevalence of vitamin D deficiency were 23.3 percent (95% CI 9.3-48.0) and 26.6 percent (95% CI 11.5-52.5) in epileptic and control children respectively (p>0.05). Only 3 children in the epileptic group also had low bone mineral density. The prevalence of abnormal bone mineral density were 10.0 percent (95% CI 2.0-29.2) and 0 percent in epileptic and control children respectively (p<0.001). All of children with abnormal bone mineral density were cerebral palsy and the effect showed after receiving antiepileptic drugs for more than 12 months (p<0.001). There were no statistically significant differences in serum 25-hydroxyvitamin D level, phosphorus, magnesium, parathyroid hormone, creatinine, alkaline phosphatase, urine calcium, urine phosphorus, urine creatinine and bone mineral density between the epileptic and control groups. Serum albumin and corrected serum calcium were significantly different between two groups (Table 4).

Table 2. Characteristics of epileptic children

	Number of patient
Cerebral palsy, n (%)	
Quadriplegia	1 (3.3)
Paraplegia	1 (3.3)
Hemiplegia	1 (3.3)
Ambulatory, n (%)	
Bed ridden	1 (3.3)
Wheelchair	1 (3.3)
Walk	28 (93.3)
Duration of antiepileptic drug therapy (month), mean ± SD (range)	23.04±16.33 (6-69)
Antiepileptic drug therapy, n (%)	
Monotherapy	17 (56.7)
Polytherapy	
2 drugs	9 (30.0)
3 drugs	4 (13.3)
Type of antiepileptic drug*, n (%)	
Old	14 (46.7)
New	3 (10.0)
Both	13 (43.3)

* Old type: phenobarbital, phenytoin, carbamazepine, benzodiazepine, valproate; New type: oxcarbamazepine, topiramate, levetiracetam, lamotrigine

Table 3. Result of vitamin D status and bone mineral density between normal children and epileptic children

Result	Control group (n = 30)	Epileptic group (n = 30)
Vitamin D deficiency	8	7
Bone mineral density: low for age	0	3

In subgroup analysis of epileptic children who had vitamin D deficiency, low normal limited of serum calcium (<8.5 mg/dL), low normal limited of serum albumin (<3.5 g/dL) and low bone mineral density has shown correlation with the duration of antiepileptic drug therapy, there were overall abnormalities in 5 and 14 children who administered antiepileptic drug for 6-12 months and more than 12 months, respectively (Table 5).

In subgroup analysis of epileptic children who had vitamin D deficiency, low normal limited of serum calcium (<8.5 mg/dL), low normal limited of serum albumin (<3.5 g/dL) and low bone mineral density, the prevalence of abnormalities increased in children who treated with multiple antiepileptic drugs in both old generation (Phenobarbital, Phenytoin, Carbamazepine, Benzodiazepine, Valproic acid) and newer generation (Topiramate, Oxcarbazepine, Levetiracetam) (p<0.001). Interestingly, there was no prevalence of abnormalities in lamotrigine treated children (Table 6).

Discussion

The present study, there is no difference in vitamin D status between children who were administered antiepileptic drug and the healthy children group in this study. This result is similar to the Weinstein et al study which found that antiepileptic drugs do not affect 25-hydroxyvitamin D level[21]. However, we found a higher prevalence of vitamin D deficiency in children than in the past same as the previous Thai children studied from 10.3 percent in 2012[22] to 40 percent in the South East Asia Nutrition Survey (SEANUTS), in 2012-2013[23]. This finding may be due to the change of lifestyle, sunlight exposure, daily activity, nutritional intake and further used ultraviolet sunscreen. The authors believe that prolonged vitamin D deficiency will be effect to bone metabolism in the future.

Table 4. Laboratory of normal children and epileptic children

Result	Control group	Epileptic group	*p*-value
Albumin (g/dL)	3.927±0.07	3.76±0.06	0.03
Calcium (mg/dL)	8.990±0.09	8.793±0.06	0.06
Corrected calcium (mg/dL)	9.014±0.09	8.815±0.06	0.04
Phosphorus (mg/dL)	4.473±0.13	4.35±0.11	0.43
Magnesium (mg/dL)	2.087±0.05	2.087±0.04	0.43
Creatinine (mg/dL)	0.562±0.02	0.571±0.04	0.95
Parathyroid hormone (pg/mL)	49.07±5.51	41.67±2.98	0.48
Alkaline phosphatase (mg/dL)	232.60±15.7	208.8±13.8	0.16
Vitamin D level (ng/mL)	22.25±0.79	25.44±1.29	0.92
Spot urine calcium (mg/dL)	6.37±1.05	5.917±0.86	0.52
Spot urine creatinine (mg/dL)	98.94±10.9	89.75±11.1	0.57
Spot urine phosphorus (mg/dL)	52.78±8.99	54.22±7.44	0.89
Urine calcium/creatinine ratio	0.098±0.02	0.087±0.01	0.75
Urine FE PO_4	8.025±0.91	8.005±0.79	0.83
BMD lumbar Z-score	-0.093±0.18	-0.22±0.28	0.26
BMD whole body Z-score	-0.473±0.22	-0.19±0.23	0.34

FE PO_4 = fractional excretion of phosphorus; BMD = bone mineral density
p-value were estimated by Wilcoxon-Mann-Whitney test, significant *p*-value <0.05.
p-value were calculated by using StatXact® Cytel® studio license number 2060107.

Table 5. Poisson prevalence of abnormal factors that may influence bone health in children who treated with antiepileptic drug(s) for 6-12 months, more than 12 months, compare to normal control children

Abnormalities	Poisson prevalence with 95% confidence interval			*p*-value
	Controlled children	Children with AED 6-12 months	Children with AED >12 months	
Vitamin D deficiency	26.6 (11.5-52.5)	50.0 (10.3-100.0)	16.6 (4.5-42.6)	<0.001
Corrected serum calcium	6.6 (0.8-24.0)	16.6 (0.4-92.8)	12.5 (2.5-36.5)	<0.001
Serum albumin	10.0 (2.0-29.2)	33.3 (4.0-100.0)	12.5 (2.5-36.5)	<0.001
Low bone mineral density	0 (0-12.2)	0 (0-61.4)	12.5 (2.5-36.5)	<0.001

There were increased prevalence of vitamin D deficiency, low corrected serum calcium, low serum albumin in children who treated with antiepileptic drug (AEDs) 6-12 months ($p<0.001$) and increased prevalence of low bone mineral density in children who treated with antiepileptic drug (AEDs) for more than 12 months. The *p*-value were calculated. Poisson regression analysis with STATA version 11 was used to analyze the regression model.

The authors did not find the statistical significant difference in biochemical markers of bone health between two groups. In subgroup analysis of epileptic children who had vitamin D deficiency, the authors find the statistical significance of low normal limited of corrected serum calcium (<8.5 mg/dL), low normal limited of serum albumin (<3.5 g/dL) and low bone mineral density correlate with administered antiepileptic drug for 6-12 months and more than 12 months respectively. This result is the interesting point that may support the theory that antiepileptic drug may alter calcium-albumin binding mechanism for a short period after 6-12 months of antiepileptic drug administration. After the calcium homeostasis functioning go on, the values will return normally, but it will have late effect to bone metabolism and finally represent low bone mineralization.

In the present study population, polytherapy of antiepileptic drug, duration of antiepileptic drug administration and reduced physical activity such as cerebral palsy were significantly correlate with increased prevalence of vitamin D deficiency, low

Table 6. Poisson prevalence of each antiepileptic drug in abnormal factors that may influence bone health in children who treated with antiepileptic drug(s)

Abnormalities	Poisson prevalence with 95% confidence interval									*p*-value
	PHB n = 3	PHT n = 4	CBZ n = 4	OXC n = 2	VPA n = 18	TPM n = 14	LEV n = 4	LMG n = 1	BZD n = 2	
Vitamin D deficiency	1 (33.3)	1 (25.0)	1 (25.0)	1 (50.0)	4 (22.0)	4 (28.5)	2 (50.0)	0	0	<0.001
Corrected serum calcium	0	0	1 (25.0)	0	2 (11.0)	2 (14.2)	1 (25.0)	0	0	<0.001
Serum albumin	0	0	0	0	5 (27.0)	3 (21.4)	0	0	0	<0.001
Low bone mineral density	1 (33.3)	0	1 (25.0)	0	1 (5.0)	3 (21.0)	2 (50.0)	0	1 (50.0)	<0.001

PHB = phenobarbital; PHT = phenytoin; CBZ = carmabazepine; OXC = oxcarbamazepine; VPA = valproate; TPM = topiramate; LEV = levetiracetam; LMG = lamotrigine; BZD = benzodiazepine

There were increased prevalence of vitamin D deficiency, low corrected serum calcium, low serum albumin, and low bone mineral density in children who treated with antiepileptic drug (AEDs), especially polytherapy AED. The *p*-value were calculated. Poisson regression analysis with STATA version 11 was used to analyze the regression model.

normal limited of corrected serum calcium, low normal limited of serum albumin and low bone mineral density. Despite of no definitive explanation for this result, Coppola et al[24] study found this similar effect that antiepileptic drug administration, duration of treatment and motor impairment seems to play a significant role in abnormal bone mineral density.

The authors conclude that low bone mineral density may be effect from multifactorial such as vitamin D status, polytherapy of antiepileptic drug, duration of antiepileptic drug administration and decreased physical activity. We essentially realize to screen the bone health status when administer antiepileptic drug for 6-12 months or in case of refractory epilepsy.

Due to limitation of small sample size and shortterm follow-up in this study, others factor that may determine the results of bone health in children should be further studies by using larger study population in long-term follow-up.

Conclusion

In the present case controlled study, vitamin D deficiency was found in children who take antiepileptic drugs without a low bone mineral density correlate. The higher incidence of vitamin D deficiency and abnormal bone mineral density was found in children with multiple antiepileptic drugs treatment. Increased prevalence low bone mineral density found in children with cerebral palsy who treated with long-term antiepileptic drugs. The decreased serum albumin and calcium may be influence in bone metabolism, especially with long-term antiepileptic drugs more than twelve months. Finally, screening of DEXA scan, serum albumin, serum calcium as well as vitamin D status may be useful in the epileptic children especially after treatment with antiepileptic drugs for 6-12 months.

What is already known on this topic?

Several theories have mentioned the effect of antiepileptic drug to bone metabolism but there is inconclusively definite effect.

What this study adds?

Multiple antiepileptic drug therapy more than 6 months and unable ambulation are the risks of low bone mineral density.

Acknowledgement

The authors are grateful to the Thammasat University in Pathumthani, Thailand, for the grant support. The authors wish to thank Thammasat University Hospital, King Chulalongkorn Memorial Hospital for provision of examination and analysis data. The authors also thank Prof. Stephen D Martin, MBBS, MRCPsych, MEWI, Brandon Lane Neuropsychiatry Clinic, Durham, United Kingdom for draft proof reading. Finally, we wish to acknowledge and thank all the patients who participated in this study.

Potential conflicts of interest

None.

References

1. Guerrini R. Epilepsy in children. Lancet 2006; 367: 499-524.
2. Mac TL, Tran DS, Quet F, Odermatt P, Preux PM, Tan CT. Epidemiology, aetiology, and clinical management of epilepsy in Asia: a systematic

review. Lancet Neurol 2007; 6: 533-43.
3. Asawavichienjinda T, Sitthi-Amorn C, Tanyanont W. Prevalence of epilepsy in rural Thailand: a population-based study. J Med Assoc Thai 2002; 85: 1066-73.
4. Kumandas S, Koklu E, Gumus H, Koklu S, Kurtoglu S, Karakukcu M, et al. Effect of carbamezapine and valproic acid on bone mineral density, IGF-I and IGFBP-3. J Pediatr Endocrinol Metab 2006; 19: 529-34.
5. Kim SH, Lee JW, Choi KG, Chung HW, Lee HW. A 6-month longitudinal study of bone mineral density with antiepileptic drug monotherapy. Epilepsy Behav 2007; 10: 291-5.
6. Pack A. Bone health in people with epilepsy: is it impaired and what are the risk factors? Seizure 2008; 17: 181-6.
7. Pack AM, Morrell MJ. Adverse effects of anti-epileptic drugs on bone structure: epidemiology, mechanisms and therapeutic implications. CNS Drugs 2001; 15: 633-42.
8. Foss MC, Meneghelli UG, Tabosa Verissimo JM. The effect of the anticonvulsants phenobarbital and diphenylhydantoin on intestinal absorption of calcium. Acta Physiol Lat Am 1979; 29: 223-8.
9. Misra A, Aggarwal A, Singh O, Sharma S. Effect of carbamazepine therapy on vitamin D and parathormone in epileptic children. Pediatr Neurol 2010; 43: 320-4.
10. El Hajj FG, Dib L, Yamout B, Sawaya R, Mikati MA. Predictors of bone density in ambulatory patients on antiepileptic drugs. Bone 2008; 43: 149-55.
11. Kuo CC. A common anticonvulsant binding site for phenytoin, carbamazepine, and lamotrigine in neuronal Na+ channels. Mol Pharmacol 1998; 54: 712-21.
12. Tsukahara H, Kimura K, Todoroki Y, Ohshima Y, Hiraoka M, Shigematsu Y, et al. Bone mineral status in ambulatory pediatric patients on long-term anti-epileptic drug therapy. Pediatr Int 2002; 44: 247-53.
13. Babayigit A, Dirik E, Bober E, Cakmakci H. Adverse effects of antiepileptic drugs on bone mineral density. Pediatr Neurol 2006; 35: 177-81.
14. Sato Y, Kondo I, Ishida S, Motooka H, Takayama K, Tomita Y, et al. Decreased bone mass and increased bone turnover with valproate therapy in adults with epilepsy. Neurology 2001; 57: 445-9.
15. Feldkamp J, Becker A, Witte OW, Scharff D, Scherbaum WA. Long-term anticonvulsant therapy leads to low bone mineral density—evidence for direct drug effects of phenytoin and carbamazepine on human osteoblast-like cells. Exp Clin Endocrinol Diabetes 2000; 108: 37-43.
16. Fitzpatrick LA. Pathophysiology of bone loss in patients receiving anticonvulsant therapy. Epilepsy Behav 2004; 5 (Suppl 2): S3-15.
17. Akin R, Okutan V, Sarici U, Altunbas A, Gokcay E. Evaluation of bone mineral density in children receiving antiepileptic drugs. Pediatr Neurol 1998; 19: 129-31.
18. Ala-Houhala M, Korpela R, Koivikko M, Koskinen T, Koskinen M, Koivula T. Long-term anticonvulsant therapy and vitamin D metabolism in ambulatory pubertal children. Neuropediatrics 1986; 17: 212-6.
19. Tekgul H, Serdaroglu G, Huseyinov A, Gokben S. Bone mineral status in pediatric outpatients on antiepileptic drug monotherapy. J Child Neurol 2006; 21: 411-4.
20. Phabphal K, Geater A, Leelawattana R, Sathirapunya P, Sattawatcharawanich S, Limapichat K. Prevalence and risk factors of low bone mineral density and 25-hydroxyvitamin D status in young healthy epileptic adult patients in a tropical Asian country taking antiepileptic drug. Bone 2009; 45: 232-7.
21. Weinstein RS, Bryce GF, Sappington LJ, King DW, Gallagher BB. Decreased serum ionized calcium and normal vitamin D metabolite levels with anticonvulsant drug treatment. J Clin Endocrinol Metab 1984; 58: 1003-9.
22. Poomthavorn P, Saowan S, Mahachoklertwattana P, Chailurkit L, Khlairit P. Vitamin D status and glucose homeostasis in obese children and adolescents living in the tropics. Int J Obes (Lond) 2012; 36: 491-5.
23. Rojroongwasinkul N, Kijboonchoo K, Wimonpeerapattana W, Purttiponthanee S, Yamborisut U, Boonpraderm A, et al. SEANUTS: the nutritional status and dietary intakes of 0.5-12-year-old Thai children. Br J Nutr 2013; 110 (Suppl 3): S36-44.
24. Coppola G, Fortunato D, Auricchio G, Mainolfi C, Operto FF, Signoriello G, et al. Bone mineral density in children, adolescents, and young adults with epilepsy. Epilepsia 2009; 50: 2140-6.

การศึกษาผลกระทบต่อกระดูกในเด็กไทยโรคลมชักที่ได้รับยากันชัก

สุดาทิพย์ ผาติชีพ, ชนิสา โชติพานิช, ขนิษฐา คูศิวิไลส์, อนุตตรา วิชาพร, สืบสาย คงแสงดาว

ภูมิหลัง: *โรคลมชักเป็นปัญหาทางระบบประสาทที่พบบ่อยที่สุดในเด็ก การรักษาโรคลมชักจำเป็นต้องรับประทานยากันชักต่อเนื่องเป็นระยะเวลานานอย่างน้อย 2 ปี ผลข้างเคียงจากการรับประทานยากันชักเป็นเวลานานอาจส่งผลต่อระดับวิตามินดี และการสร้างมวลกระดูก*

วัตถุประสงค์: *เพื่อหาความชุกของภาวะขาดวิตามินดี และความหนาแน่นของกระดูกบางในผู้ป่วยเด็กโรคลมชักที่รับประทานยากันชักอย่างน้อย 6 เดือนขึ้นไป*

วัสดุและวิธีการ: *การศึกษานี้ได้ทำการตรวจและวิเคราะห์หาระดับวิตามินดี แมกนีเซียม alkaline phosphatase อัลบูมิน พาราไทรอยด์ฮอร์โมนในเลือด และปริมาณแคลเซียม ฟอสฟอรัส creatinine ทั้งในเลือดและปัสสาวะ ประกอบกับการตรวจวัดความหนาแน่นกระดูกในกลุ่มเด็กโรคลมชักอายุระหว่าง 3-18 ปี ที่รับประทานยากันชักอย่างน้อย 6 เดือนขึ้นไป เปรียบเทียบกับเด็กปกติที่ไม่มีโรคประจำตัวในวัยเดียวกันอย่างละ 30 คน ระหว่างเดือนตุลาคม พ.ศ. 2555 ถึง 30 กันยายน พ.ศ. 2556*

ผลการศึกษา: *พบว่ามีภาวะขาดวิตามินดีในกลุ่มผู้ป่วยเด็กโรคลมชักและกลุ่มเด็กปกติร้อยละ 23.3 และ 26.7 ตามลำดับ พบมีผู้ป่วยเด็กโรคลมชักที่มีภาวะสมองพิการร่วมด้วยทั้งหมด 3 ราย ที่มีภาวะความหนาแน่นกระดูกต่ำ ไม่พบความแตกต่างอย่างมีนัยสำคัญทางสถิติของระดับวิตามินดี ฟอสฟอรัส แมกนีเซียม พาราไทรอยด์ฮอร์โมน creatinine และ alkaline phosphatase ในเลือดรวมถึงปริมาณแคลเซียม ฟอสฟอรัส creatinine ในปัสสาวะและความหนาแน่นกระดูก แต่พบความแตกต่างของระดับอัลบูมิน (p-value = 0.03) และค่า corrected calcium ในเลือด (p-value = 0.04) โดยพบระดับที่ต่ำกว่าของค่าดังกล่าวในกลุ่มผู้ป่วยเด็กโรคลมชักอย่างมีนัยสำคัญทางสถิติ*

สรุป: *การประเมินสภาวะวิตามินดีและความแข็งแรงของกระดูกเป็นสิ่งที่แพทย์ควรทำในระหว่างการรักษาผู้ป่วยเด็กที่เป็นโรคลมชัก*