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DUANKAN YAYONG: PREVALENCE AND FACTORS ASSOCIATED  
WITH FOLATE DEFICIENCY IN EPILEPTIC THAI PATIENTS. THESIS  
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The study was performed to evaluate the prevalence and factors associated with folate deficiency in epileptic Thai patients at epilepsy clinic, Prasat Neurological Institute from 1 August 1998 to 31 August 1999. Eighty-seven patients and 25 healthy volunteers who met the inclusion criteria were enrolled in our study. Their mean ages were  $35.69 \pm 1.34$  and  $34.48 \pm 2.47$  years, respectively. All patients or their relatives were directly interviewed using a questionnaire concerning dietary habits, nutritional history, social history and self-medication. All information was considered together with information obtained from patient charts. A microbiological assay using *Lactobacillus casei* was used to determine fasting serum and red blood cell folate levels and a radiodissolution assay was used to measure vitamin B12 in age-matched control subjects and in epileptic patients who were being treated with conventional antiepileptic drugs (phenytoin, phenobarbital, carbamazepine and valproic acid). The prevalence of folate deficiency depends on the criteria used. Folate deficiency based on a serum folate level of less than 3 ng/ml occurred in 37% (32/87) of the patients. Based on a RBC folate level below 140 ng/ml, 37% (32/87) epileptic patients were deficient. However, based on the criteria in our proposal (requiring both serum folate level  $< 3$  ng/ml and RBC folate level  $< 140$  ng/ml), only 14% (12/87) patients were identified with a folate deficiency. Macrocytic RBC was detected in 2 patients. The antiepileptic drugs which induced RBC folate deficiency in epileptic patients on monotherapy were phenytoin (7/18), phenobarbital (5/16), carbamazepine (4/12) and valproic acid (2/7). The comparisons of serum and RBC folate levels between subgroups of patients were demonstrated. There were not significant differences between subgroups of patients receiving monotherapy except for valproic acid subgroups which the mean serum and RBC folate levels were significant higher than phenytoin ( $p < 0.01$ ), phenobarbital ( $p < 0.05$ ) and carbamazepine ( $p < 0.01$ ) subgroups. In addition, folate deficiency was not associated with age, sex, education level and duration of treatment. Serum and RBC folate levels were not correlated with serum antiepileptic drug concentration. In conclusion, folate deficiency resulting from antiepileptic drug therapy is a common occurrence and may be progress to a macrocytic anemia. Therefore, the folic acid supplementation of folic acid should be recommended in epileptic patients especially in patients receiving phenytoin, phenobarbital and carbamazepine. However, serum antiepileptic drug should be monitored because folic acid may increase the metabolism of these antiepileptic drugs.