

Bone Mineral Density in Ambulatory Children with Epilepsy

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Abstract

Objective To elucidate the effects of antiepileptic drugs (AEDs) on bone health status of ambulatory epileptic children. **Methods** A total of 120 epileptic children aged 2–15 y were enrolled in three groups. The first group was on therapy with carbamazepine, phenobarbital or primidone. The second was treated with valproic acid and the third group was untreated. Serum calcium, phosphorous, total alkaline phosphatase, and parathyroid hormone levels were compared between groups. Bone mineral density tests were also performed at four sites of the lumbar spine and three sites of femoral neck and results were compared between the groups.

Results Of all enrolled subjects, 67 patients (55.8 %) were vitamin D deficient. The three groups were not significantly different in terms of vitamin D, calcium, phosphorus, total alkaline phosphatase, and parathyroid hormone levels. While patients in first group had lower Z-score of femoral neck and lumbar spine compared to those on valproic acid, these values were also significantly different than that of the third group. **Conclusions** It can be concluded that both enzyme-inducing AEDs and non enzyme-inducing AEDs decrease bone mineral density (BMD). Also alkaline phosphatase (ALP) is affected in ambulatory epileptic children on enzyme-inducing AEDs. Nevertheless, valproic acid (a non-enzyme-inducing agent) does not have the mentioned side effects.

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Introduction

About one percent of children worldwide suffer from various types of epilepsy and usually require long-term treatment with antiepileptic drugs (AEDs) [1]. The association of skeletal abnormalities with chronic antiepileptic therapy was first described about three decades ago and most of the available data is from adults [2].

AEDs associated osteopathy includes decreased bone mineral density (BMD), increased fracture risk, and overt osteomalacia [2].

Although the skeletal consequences of antiepileptic drugs may vary, the AEDs, especially enzyme-inducing drugs such as phenobarbital, primidone, phenytoin, and carbamazepine are generally known to alter bone metabolism [3, 4].

Subjects on enzyme-inducing AEDs are expected to have lower BMD compared to those on non-enzyme-inducing medicines such as valproic acid in which the hepatic cytochrome P450 enzyme system remains intact [3, 4].

Unpleasant side effects of AEDs on calcium metabolism and bone density were primarily reported in institutionalized