Non-traumatic Thoracic Vertebral Compression Fractures Occurred in a Young Epileptic Patient: A Case Report

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What to Learn from this Article?
It is important to prevent bone mineral density loss in patients taking long-term anti-epileptic drugs.

Introduction: The occurrence of non-traumatic vertebral compression fractures (VCFs) in a healthy young male is very rare. We present a rare case of non-traumatic thoracic VCFs in a young epileptic patient.

Case Report: A 19-year-old healthy male experienced severe back pain. There had been no significant traumatic event. A radiograph of the spine showed collapsed vertebra at Th6 and Th7 and magnetic resonance image of the spine showed intensity changes at Th6, Th7 and Th8. Bone mineral density (BMD) at the radius was low and urine N-terminal telopeptide (NTx) was very high. The patient was diagnosed with VCFs caused by low BMD. The patient had a medical history of epilepsy and had taken valproate for thirteen years. We instructed the patient to stop taking valproate and to begin taking bisphosphonate. As a result, urine NTx became normal.

Conclusion: It was previously reported that valproate reduced BMD in epileptic children and reduction in BMD increased with the duration of valproate therapy. We propose that regular BMD screening and measurement of bone metabolic markers should be conducted for all patients taking long-term antiepileptic drugs to prevent BMD loss and associated fractures.

Key word: non-traumatic, vertebral compression fractures, young, epilepsy, valproate, bone mineral density
and resorption [5]. Bone turnover, resulting in a balance between bone formation activity in order to compensate for increased osteoclastic activity [5]. VPA increase of these bone formation markers was caused by increased osteoblast NTx, etc compared with the control group [5]. The authors postulated that the significant increase of bone specific alkaline phosphatase (BAP), osteocalcin, vitamin D metabolism because VPA is not an inducer. In an experimental vitamin D levels. However, the effect of VPA cannot be readily explained by some AEDs, such as carbamazepine, phenytoin, and the resultant reduction remain elusive in spite of many reports. Abnormal calcium metabolism was considered to result from the cytochrome P450 enzyme-inducing properties of carbamazepine and VPA on BMD in epileptic children [1]. They concluded that VPA significantly reduces BMD in epileptic children and reduction in vitamin D levels. Many studies have demonstrated a significant reduction of BMD and an increased risk of fractures in patients taking antiepileptic drugs (AEDs) [1-4]. Sheth reported the effect of carbamazepine and VPA on BMD in epileptic children [1]. They concluded that VPA significantly reduces BMD in epileptic children and reduction in BMD increased with the duration of VPA therapy [1]. Kafali [2] and Ecevit [3] also reported VPA reduced BMD in children. Vestergaard assessed the fracture risk associated with antiepileptic drug therapy and suggested that VPA was significantly associated with the risk of fracture [4]. We consider loss of BMD is commonly asymptomatic; therefore, patients aren’t usually aware of the condition until they suffer fractures. In addition, we regret many clinicians don’t have knowledge that AEDs can reduce BMD in children. The mechanisms of the adverse effects of AEDs on calcium metabolism remain elusive in spite of many reports. Abnormal calcium metabolism was considered to result from the cytochrome P450 enzyme-inducing properties of some AEDs, such as carbamazepine, phenytoin, and the resultant reduction in vitamin D levels. However, the effect of VPA cannot be readily explained by vitamin D metabolism because VPA is not an inducer. In an experimental study, oral administration of VPA to epileptic rats for 6 months resulted in a significant increase of bone specific alkaline phosphatase (BAP), osteocalcin, NTx, etc compared with the control group [5]. The authors postulated that the increase of these bone formation markers was caused by increased osteoblast activity in order to compensate for increased osteoclastic activity [5]. VPA may facilitate bone turn over, resulting in a balance between bone formation and resorption [5].

#### References

1. Sheth RD, Wesolowski CA, Jacob JC, Penny S, Hobbs GR, Riggs JE, Bodensteiner JB. Effect of carbamazepine and valproate on bone mineral density. J Pediatr 1995; 12: 256-62.
2. Kafali G, Erselcan T, Tanzer F. Effect of antiepileptic drugs on bone mineral density in children between age 6 and 12years. Clin Pediatr 1999;38:93-8.
3. Ecevit C, Aydogan A, Kavakli T, Altinoz S. Effect of carbamazepine and valproate on bone mineral density. Pediatr Neurol 2004;31:279-82.
4. Vestergaad P, Rejnmark L, Mosekilde L. Fracture risk associated with use of different antiepileptic drugs. Epilepsia 2004;45:1330-7.
5. Elwakkad AS, El Elshamy KA, Sibaii H. Fish liver oil and propolis as protective natural products against the effect of the anti-epileptic drug.
valproate on immunological markers of bone formation in rats. Epilepsy 2008; 80: 47-56.

6. Takahashi A and Onodera K. Valproic acid induced osteopenia and its prevention with alfalcacidol and alendronate. J Hard Tissue Biology 2005;14: 275-6.

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