IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 17, Issue 11 Ver. 1 (November. 2018), PP 64-66
www.iosrjournals.org

"Non-traumatic vertebral body compression fracture in a young patient on antiepileptic treatment: A rare case report"

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Abstract:
Background: *Non traumatic compression fracture are normally seen in old and osteoporotic patient but rarely seen in young adult. We are presenting a rare case of a-traumatic thoracic vertebral body fracture in a young adult on antiepileptic treatment.*
Case characteristic: *A 24 Years old male patient presented to us with chief complain of backache for 2 months. He was taking antiepileptic for last 6 month. There was no history of any other significant illness or trauma. On examination, mild localized tenderness with minimal round kyphosis over dorsal spine was present. On radiological evaluation, there was mild collapse of D5 and D9 vertebrae with T2 signal changes of adjacent vertebrae suggestive of osteoporotic compression fracture. Bone mineral density was less and both T and Z score were -1.9.*
Outcome: *Patient was advised to stop antiepileptic drugs and bisphosphonate along with calcium and vitamin D3 were prescribed simultaneously resulted incomplete relief of pain as well his BMD was also improved after 2 month of therapy.*
Osteoporosis and loss of bone mass is known side effect of long term antiepileptic treatment but decrease in bone mineral density leading to non-traumatic vertebral body fracture in a short course (only 6 month) of antiepileptic therapy had not been document in literature.
Conclusion: *Regular screening for bone mineral density along with supplementation of bisphosphonate, calcium and vitamin D to maintain bone health should be done in all patients with antiepileptic treatment even for short course.*
Keywords: *Vertebral, Compression, Fracture, Young, Antiepileptic therapy*

Date of Submission: 20-10-2018 Date of acceptance: 03-11-2018

I. Introduction

Compression fracture of vertebral body in young adult is rare but can be seen in high velocity trauma like road traffic accident and fall from height; However common in old and osteoporotic patient in a clinical practice with reduced bone mineral density. We are presenting a rare case of non-traumatic spontaneous onset thoracic vertebral compression fracture in a young epileptic patient.

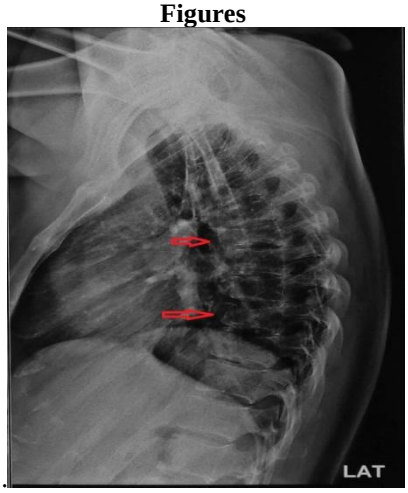
II. Case Report

A 24 year old male presented to us with upper back pain which is constant, non-radiating without any history of recent trauma, fever, loss of weight or poor nutritional status with history of hospitalization 6 months back for an episode of epilepsy for which he was treated at that time and was on anti-epileptic therapy (valproic acid) since then. Within this period of two months he has taken treatment for the back pain in the form of analgesic and muscle relaxation but not being relieved.

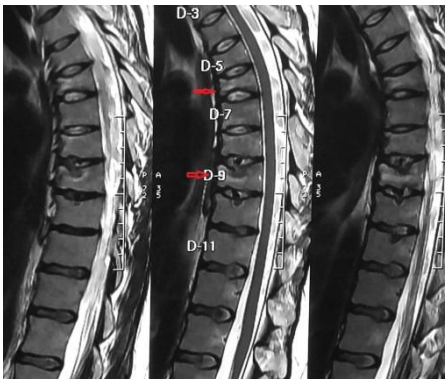
The patient was evaluated thoroughly clinically, hematologically, bio-chemically and radio-logically. In clinical examination, patient has mild localized tenderness over upper back with mild round kyphosis without local rise of temperature or neurological deficit. Level of vitamin D3 was very low. On radiological evaluation, there is gross osteoporosis of vertebral column with fracture of D-6 and D-9 vertebral body (Fig 1). On further evaluation on MRI we noticed signal changes in D-5, D-6, D-8 and D-9 vertebra without any pre or paravertebral collection/soft tissue shadow without any compression of neural canal suggestive of osteoporotic compression (Fig 2). On DEXA scan, BMD of the patient was very low with T- score and Z score -1.9. Then patient was advised to stop anti-epileptic therapy on consultation with neurologist, and prescribed with anti-

osteoporotic treatment in the form of this bisphosphonates (alendonate 70 mg) per week along with vitamin D and calcium therapy along with proper rest. After two month of getting treatment, patient improved clinically in the form of complete relief from back pain and simultaneously his BMD improved.

Figures



"Fig.1": Lateral radiograph of dorsal spine showing destruction of the D6 and D9 vertebral bodies without decreased intervertebral disc spaces.



"Fig.2": Magnetic resonance imaging (Sagittal T2image) showing compression fracture of D6 and D9 vertebral body with marrow edema inside adjacent vertebral bodies.

III. Discussion

Epilepsy is a group of neurological disorders characterized by epileptic seizures and is associated with several co-morbidities such as developmental disorder, movement disorder, mental retardation and bone health. Long term medical therapy with antiepileptic drugs has detrimental effect on bone mineralization. There is increasing evidence suggesting that epilepsy and its treatment can effect bone mineralization and calcium metabolism. Use of anti-epileptic drugs has been associated with increased bone turn-over and occasionally with mineralization defect leading to decreased bone mineral density and decreased bone strength and increased fracture risk[1-3]. Many studies has demonstrated significant reduction of BMD with increased risk of fracture in the patients taking anti- epileptic therapy. Sheth et. al. reported the effect of carbamazepine and valproic acid on BMD in epileptic children they concluded that valproic acid significantly reduces bone marrow density in children and reduction is increased with the duration of the therapy. Kafali and Ecevit also reported VPA reduce BMD in children[4]. Vestegaard assessed the fracture risk associated with anti-epileptic drug therapy and suggested that VPA was significantly associated with increased risk of fracture[5]. We considered loss of BMD is commonly asymptomatic and therefore patients are not usually aware of the condition until they suffer fractures. In addition many physicians do not have adequate evidence that antiepileptic medication can cause reduction of BMD associated with increased risk of fractures. Mechanism of the adverse effects of the epileptic therapy on calcium metabolism remaining illusive inspite of many reports. Abnormal calcium metabolism was considered to result from P cytochrome P-450 enzyme inducing property of some anti-epileptics such as carbamazepine , phenytoin and resultant reduction in vitamin D level. However of effect of valproic acid cannot be readily explained by reduction in vitamin D level because it is not an inducer of enzymes. Experimental studies oral administration of valproate to epileptic rats for six months resulted in a significant increase in bone specific

alkaline phosphatase as compared to control group. To prevent BMD loss vitamin D-3 and/or bisphosponate were recommended for high risk patients who are receiving long term anti-epileptic therapy. Takahashi reported the co- administration of activated vitamin D3 or bisphosphonate (alendronate) with valproate did not induced osteopenia in growing rats[6]. On this basis, we prescribed bisphosphonate along with vitamin D3 resulting insignificant clinical improvement and increase in vitamin D and BMD level.

In summary we presented a rare case of non-traumatic vertebral compression fractures that had occurred in the young epileptic patient who was on anti-epileptic therapy only for a period of 6 months. So we proposed that significant reduction of BMD may occur even in a short duration therapy of anti-epileptic drug (valproic acid) that can lead to osteoporotic fractures.

IV. Conclusion

Regular screening for bone health by measuring bone mineral density and vitamin D estimation along with supplementation of bisphosphonate, calcium and vitamin D to maintain bone health should be done in all patients on antiepileptic treatment even for short course.

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