Noncontiguous Multiple Compression Fractures After a Grand mal Seizure: Case Report

Abolfazl Rahimizadeh, Mohammad Saghri, Arghvan Soheili, Leila Janani, Ava Rahimizadeh

Pars Hospital, Neurospinesociety of Iran, Tehran, Islamic Republic of Iran

ABSTRACT

The violent forces generated by the strong paraspinal muscles during generalized tonic-clonic seizures are known to be associated with numerous musculoskeletal injuries and mostly with vertebral fractures. Vertebral fractures following epileptic seizures are typically a single compression fracture, mostly occurring in the mid-thoracic region and usually without resultant neurological deficit. Despite the relative frequency of single level vertebral compression fractures complicating seizures, less frequently two or three adjacent vertebrae might be affected by the seizure. However, non-contiguous multiple compression fractures after grand mal epilepsy have been not reported previously.

Herein, a 40-year-old male experienced witnessed grand mal seizure resulting in 3-level compression fracture at 3 levels at thoracic spine and two levels at thoracolumbar region the patient was managed conservatively with an appropriate orthosis. The current case might be the first seizure induced non-contiguous multilevel compression fractures reported in the literature.

KEY WORDS: Compression fracture, convulsion, epilepsy, osteoporosis, seizure

INTRODUCTION

Forceful muscle contractions during convulsive seizures can result in vertebral fractures without any external trauma (1-3,5-11,17,18,21-26). Lehndorff in 1907 was the first to suggest that strong muscular contraction during forceful epilepsy can collapse a vertebra, since then, vertebral fractures became a well known complication of convulsive seizures (1-3, 7-9, 13, 14, 20-25). Grand mal epilepsy is the most common cause of seizure induced vertebral fractures. Majority of the seizure-related vertebral fractures are compression fractures, which are mostly located at mid-thoracic region from T3 to T8 and usually without neurological deficit (1-3, 7-9, 13, 14, 20-25). Seizure related compression fractures of the thoracic spine less frequently might occur in two or more contiguous levels.

Herein, a 38-year-old male who suffered seizure-induced non-contiguous multi-level compression fractures is presented. Convulsion was a witnessed. nocturnal tonic-clonic seizure which had occurred for the first time. To the best of our knowledge, this the first example of non-contiguous multi-level compression fractures following grand mal epilepsy.

CASE REPORT

A 38-year-old man suffered a witnessed severe tonic-clonic seizure while he was sleeping. The seizure lasted about 3 minutes and once it seized his wife noticed tongue bite and urination. Post-ictally, he was drowsy but became alert gradually. The patient was taken by an ambulance to the nearest regional hospital where diagnosis of grand mal seizure was made. Blood sugar and serum Ca were normal. The patient and his family denied any similar attack, but he mentioned that he has experienced 2 to 5 daily attacks of absence, which interrupted him for a few seconds in the last 6 months. He had neither care about this short attacks nor he was treated with any anti-convulsive medication up to the time of admission.
EEG done on admission was compatible with post ictal state where brain MRI showed no apparent abnormality.

Initially he was treated with IV phenytoin but simultaneously oral sodium valproate, 200 mg three times daily was administered. He was discharged 3 days later. However, the following day, he noticed pain of increasing intensity at his mid-thoracic and upper part of low back region. Therefore, he was admitted to our institute. His examination revealed no abnormality except sensitivity of the mid thoracic and thoracolumbar spine to palpation.

Dorsal spine x-ray films showed compression fractures of the T3,T-4, T-5, and T-6 as well as at T12 and L-1 vertebrae (Figure 1). Subsequent evaluation by MRI led to the diagnosis quadruple-level compression fracture at thoracic spine and double-level at thoracolumbar region affecting respectively without any compressive effect on the spinal cord (Figure 2). Bone mineral density revealed severe osteoporosis. The patient was managed with a Jowett body jacket orthosis and painkiller (Figure 3). For osteoporosis was managed with calcium, strontium mg Calcitonin and D3. He was discharged a week after admission. He is doing well at 12-months follow up.

**DISCUSSION**

Epileptic seizures are relatively common, affecting approximately 0.2–0.5% of the general population. Epileptic patients are at risk for seizure-related injuries and fracture either directly or accidental consequences of epileptic seizures (9,14,20,25).

The incidence seizure-induced vertebral fracture stated in the literature varies from 1–16% in patients suffering from epilepsy (9, 14, 20, 25). Surprisingly, in 25% of the cases, it might be as a consequence of the first seizure.
episode. Grand mal induced vertebral fractures are mostly nocturnal and occur during the sleep hours. However, a few percentage of vertebral fractures related to seizure are due to brain pathologies, hypoglycemia, electroshock and tetanus or are drug induced such as isoniazide and tramadol (7,8,15,16,21). Less than 5% of the seizure-induced vertebral fractures are due to external traumas such as fall from the chair, from the height or during car driving. The male -female ratio of seizure related vertebral fracture is 3 to 1 and they are more frequently seen in adults than in children. The volume of the paraspinal muscles is probably the major cause of its relative frequency in adult males (1-3, 7-9, 13, 14, 20-25).

The mechanism of vertebral fracture is usually powerful contractions of the paraspinal muscle and less frequently due to sudden fall during the convulsion (1-3,7,8,11). With muscular tension of the posterior spinous and abdominal muscles, the spine flexes forward and is subjected to axial loading and flexion compressive forces (1-3,7-9,13,14,20-25). Actually, violent contractions result in a loss of the anterior height of the vertebral body while the posterior aspect remains intact where the final scenario is the compression of the vertebral body (1-3,7-9,13,14,20-25). Seizure-related vertebral fractures are usually a single level compression fracture and are generally located at mid–thoracic spine between T3–T8 vertebrae. Multiple compression fractures of the thoracic spine are less frequent and if this occur it is limited in two or three contiguous vertebrae Intensive contractions concentrated in the anterior column of the vertebral bodies of the thoracic spine seems to be responsible for this type of fractures.

Thoracic burst fractures in seizure attacks remain a very rare entity because the axial skeletal contractive forces are usually insufficient to burst the vertebral body (10).

Thoracolumbar junction, which is the point of transition from rigid kyphotic thoracic spine to more mobile lordotic lumbar spine, is the second most common region being affected by seizure.

Strong muscular forces generated by axial skeletal contractions during seizure activity are implicated as the cause of fractures of the anterior and middle columns or burst fracture (6, 11, 17, 18, 26).

All types of convulsion-related fractures are most frequent in patients whose attacks occur during sleep and in the patients with history of status epilepticus (1). Risk of vertebral fracture increase with age, associated osteoporosis, weak muscularity, increased duration of isolated seizure and recurrent convulsions (4,9).

However, the long-standing use of antiepileptic drugs has emerged as an independent risk factor for fracture. The underlying mechanism may be drug -induced decreased mineral bone density with increasing fracture risk. Several studies show that anti-convulsive medication reduces bone mineral density and increases the risk of bone fracture, especially in the vertebral bodies (4,19). The majority of authors report on patients treated with sodium valproate and carbamazepine (2,13). Sheth et al reported a decrease in the bone mineral density of 14% of children prescribed carbamazepine, and 10% in those taking sodium valproate (19).

Contiguous multiple vertebral compression fractures which result from violent contractions or status epilepticus are more seen in patients with underlying anticonvulsant -related osteoporosis.
Post-seizure compression fractures are usually asymptomatic without any neurological deficit. Therefore, these fractures might be easily overseen and missed (1-3,7-9,13,14,20-25). Vasconcelos suggests that 15% of primarily asymptomatic fractures also known as idiopathic are actually caused by seizure (23). There should be a tendency towards a higher incidence of kyphosis in the patients with contiguous multilevel compression fractures compared to the single level fractures. Furthermore, the spinal cord compromise in non-contiguous fractures is only possible if there is combination of compression and burst fractures.

Diagnosis of post-ictal patients with vertebral fracture should maintain a high degree of suspicion and conduct a detailed imaging survey. Patients who present in a post-ictal state should be carefully monitored for any neurological or skeletal compromise. Although, plain radiographs as a preliminary imaging study may demonstrate the vertebral fracture, but MRI remain the most sensitive diagnostic tool for detection of site, type and the number of the fractures (12). Further, with application of axial and reconstructed CT scan differentiation between compression and burst fracture can be made.

Compression fractures usually respond to conservative treatment and rarely need surgical intervention. However, in multiple contiguous compression fractures, the possibility of late kyphosis should be born in mind. Non-contiguous multilevel compression fractures should be considered as a more complicated spinal injury where thoughtful judgment between surgical intervention and conservative management is necessities.

Recently, treatment with percutaneous kyphoplasty was introduced in a patient suffering from non-traumatic compression fracture of the thoracic spine following seizure. Besides preventing late kyphosis, this procedure can be used in those with intractable pain (5).

Ultimate, outcome of seizure-induced multiple compression fractures should be good or acceptable in compliant patients (1-3, 7-9, 13, 14, 20-25). In non-compliant subjects, this type of injury might be complicated with severe kyphosis; the scenario of late kyphosis will require correction with a long fixation in association different type of osteotomy including PSO and Ponte. Undoubtedly, a deformity caused by a non-contiguous multiple compression fracture will require a more extensive surgery with consideration of sagittal balance.

In summary:
1. A patient sustaining seizure induced compression fracture might remain asymptomatic where thorough examination of the spinal column in particular palpation of the spine is recommended after any kind of epilepsy.
2. In the case of doubt, obtaining appropriate whole length spine radiographs to rule out a vertebral compression fracture is mandatory.
3. Seizure-induced compression fractures usually respond to conservative treatment but careful periodic follow-up is necessary in order to recognize late kyphosis earlier.

REFERENCES