Seizure-Induced Thoracolumbar Burst Fracture Secondary to Tramadol Abuse

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ABSTRACT

Abstract: Tramadol is a novel centrally acting opioid analgesic used for the treatment of mild to severe pain. This drug is the most prescribed opioid worldwide. It has been approved as an analgesic in Iran since 2002. Unfortunately Tramadol dependency has become very common in Iran in various age groups and genders but in particular is abused among the young generation. Overdose of Tramadol is a well-known cause of seizure.

On the other hand, seizures regardless of etiology can induce fractures of the vertebral column by violent contractions of skeletal muscles. Seizure-induced vertebral fracture are mostly due to grand mal and are compression fractures located at mid thoracic region. Burst fractures of the spine secondary to seizure have rarely been reported in the literature.

Herein, we describe a patient with thoracolumbar burst fracture secondary to violent contraction induced by severe seizure resulted from Tramadol overdose. This seems to be the first of such example in the literature.

KEY WORDS: Burst fracture, Cauda equina, Conus medullaris, Seizure, Tramadol

INTRODUCTION

Forceful muscle contractions during convulsive seizures can result in vertebral fractures without any external trauma (1,2,21,22). The incidence of symptomatic vertebral fractures in epileptic patients is estimated to be 1% (13,18). However, reported prevalence of asymptomatic fractures associated with seizure is as high as 15% (18). Therefore, demonstration of several old compression fractures in uncontrolled epileptic subjects is not unusual particularly if we accept that more than 40% of these fractures occur during sleep and are actually un Witnessed (1,13,18).

Risk of vertebral fracture increase with age, associated osteoporosis, muscularity, increased duration of isolated seizure and recurrent convulsions. The majority of the seizure-related vertebral fractures are compression fractures which are mostly located at mid-thoracic region from T3 to T8 (1,21,22). Seizure-induced burst fracture is a rare but a well established clinical entity and only seven cases have been reported in the literature two of which had occurred at the thoracic spine and the remaining four at thoracolumbar/lumbar region (5,9-11,15,17,23). The rarity of convulsion-induced thoracolumbar burst fracture might be explained by this fact that a very violent contraction with combination of both axial and flexion forces are necessary for occurrence of this type of vertebral fracture (11,15,17).

Clinically, in contrast to seizure-related compression fractures which are usually asymptomatic, thoracolumbar burst fractures usually result in canal compromise and as the result are mostly associated with conus medullaris-cauda equina syndrome (9,11,17,23).

The etiology of seizure as a primary cause of a vertebral fracture is usually grandmal while tumors, infection, vascular malformations, metabolic disorders, traumas and drugs have been also reported as the causative factor in seizure-induced vertebral fractures (6).
Tramadol is a potent opioid drug introduced and approved in our country since 2002 and become the most prescribed opioid since then (19,20). In Iran, Tramadol has been reported to be increasingly abused by opioid dependent subjects. It seems that the higher prevalence of Tramadol abuse as tablets in our country may be because of less availability of other legal opioid such as Hydrocodone or Oxycodone and its availability in all Iranian private pharmacies (19,20).

Overdose of this drug can cause seizure in one third of Tramadol abusers and this is supported by a number of papers (3,14). However, there is no report on vertebral column fracture secondary to Tramadol-induced seizure in the literature. Therefore a young abuser with severe witnessed convulsion resulting in thoracolumbar burst fracture is presented which is certainly the first example in the literature.

CASE REPORT

This 20-year-old male had severe convulsion while sitting in the rear seat of a car. The event was witnessed by his friend who reported that the seizure had lasted about 20 minutes. He was transferred to the nearest hospital where he regained consciousness about 3 hours after the attack. Seizure work up including EEG and brain MRI were done which were both within normal limit. He was discharged 2 days later. A week after seizure he was referred to our institution because of back pain and feeling of numbness in his lower extremities. On examination, the thoracolumbar spine palpation result in pain, neurological examination was normal except for vague sensory disturbance in the lower extremities. Radiological survey of the spinal column disclosed Denis type B T12 burst fracture (Figure 1) The radiologist commented on osteopenia in this young male radiographs. MRI Confirmed burst fracture with cord compression but no myelomalacia could be depicted (Figure 2). Later the patient confessed that he has been addicted to Tramadol ingestion since a year ago. He had ingested 8 tablets of 100 mg Tramadol in the day of seizure. Diagnosis of Tramadol induced–seizure was made. Osteopenia had probably placed him at increased risk of seizure induced vertebral burst fracture. Because of the delay in diagnosis, ligamentotaxis seemed ineffective. Moreover, because of associated osteopenia, corpectomy was considered to be with risk of settling and subsequent failure. Therefore shortening of the spine through pedicle subtraction osteotomy (PSO) was decided and this was done after pedicle screw rod instrumentation two level above and below the fracture site. Subsequent to laminectomy of involved vertebrae and achievement of PSO, the retropulsed fragment was pushed back into the osteotomy site from both sides Figure 3, Figure 4) Post-operative plain radiograph showed normal curvature of the thoracolumbar spine, with T12 being shortened (Figure 5). The patient tolerated the procedure very well and was discharged within 4 days in a Jewett brace. Two weeks after surgery he was seen with full recovery and started to attend university within a month. He was encouraged to wear the brace for eight weeks Now four years after surgery, we know that he is doing well through phone calls but he is reluctant to come to our institution and has refused even to undergo an X-Ray and CT.

DISCUSSION

The majority of vertebral fractures secondary to seizures are compression type and mostly occur at the mid-thoracic region (1,2,21,22). However, the cervical, cervicothoracic and thoracolumbar regions are seldom affected in tonic clonic contractions of a seizure. The
number of affected vertebrae might be more than one in one convulsive contraction. Seizure-associated vertebral fractures occur primarily in adult males, although fractures in children receiving anti-epileptic drugs have been reported (1,6,21).

Seizure-induced thoracolumbar burst fracture is a clinical entity and only five cases had been reported in medical literature (5,10,11,15,17,23). The seizure was
due to grand mal epilepsy in all of these burst fractures. Thoracolumbar burst fractures might result in the conus or quad equina syndrome (11,17). Therefore, its recognition is crucial for prevention and treating neurological deficit. Unfortunately, the diagnosis of the vertebral burst fracture is usually delayed from a few days to a week or two with a long duration from diagnosis to proper management, making their proper management more complicated (5,10,11,15,17,23). The mechanism of this type of vertebral injury is combination of forward flexion of the spine and forceful axial skeletal contraction forces generated during seizure.

On the other hand, Tramadol is an opioid analgesics that is use and abused in our country (19,20). Unfortunately, Tramadol abuse is quite common among the young generation. The most serious complication of intoxication with this analgesic is seizure (19). It is presumed that 15% to 35% of patients with Tramadol overdose experience seizure (3,19,20). The exact mechanism of Tramadol in induction of seizure remains to be elucidated yet. Research indicates that in high concentrations Tramadol exerts an inhibitory effect on Gamma Aminobutyric Acid (GABA) receptors (14). Inhibition of GABA receptors has been found to potentiate the severity of seizures in animal models. In addition, GABA receptor inhibition induced by Tramadol can be secondary to opioid receptor agonist activity and continuing this agonist activity on opioid receptor has been proven to precipitate seizure due to inhibition of GABA pathways (14,19).

About 85% of subjects with Tramadol overdose experience a single attack of seizure, while multiple seizures are seen in the remaining 15% of the subjects. The length of time between ingestion and seizure varies from 20 minutes to 12 hours. However, 95% of the seizures occurs within the first 6 hours after ingestion (3,19,20).

Forceful and violent contraction induced by seizure in Tramadol overdose can induce vertebral fracture.

Osteopenia and osteoporosis seen in the majority of these abusers who are mostly heavy smokers with low appetite eventually predisposes the vertebral column to fracture.

The rarity of the reports of on vertebral fracture secondary to Tramadol overdose does not truly indicates its rarity. There might be a few more cases that are either underdiagnosed or never reported.

Figure 5: Final plain X-Ray demonstrating the ideal curvature and shortening of T12.
Management of seizure-induced compression fractures of thoracic region might be both conservative or with kyphoplasty in middle age group and older (4). Once the condition is diagnosed late with considerable kyphosis, pedicle subtraction osteotomy is advised. In the case of marked osteoporosis one can augment the pedicle screws with cement.

Thoracolumbar burst fractures can be managed conservatively or by surgery according to the severity of the burst fracture assessed by load sharing classification, absence or presence of a retropulsed fragment, degree of canal compromise and the status of neurology. Conservative management with postural reduction and appropriate brace can be carried out in those with acceptable load sharing score from 1 to 3, moderate size retropulsed fragment and normal neurology, particularly if we accept that canal clearance and remodeling of retropulsed fragment occurs with time. However, with a large retropulsed fragment with considerable canal compromise and in particular in the presence of neurological deficit with load sharing scores of 7 or more, corpectomy followed by either anterior double rod construct or simultaneous staged posterior screw–rod construct is the choice (7,8,12). By application of this technique, decompression of the thecal sac, restoration of height, acceptable arthrodesis and stabilization can be achieved (7). Recently, posterior stabilization combined with posterior vertebral column resection plus reconstruction (PVCR) has become another option (16). In both of these procedures, accompanying osteoporosis might hinder reconstruction of corpectomized vertebral body because of the possibility of settling and failure.

In those cases with load sharing of about 4 to 6 and moderate size retropulsed fracture, ligamentotaxis with application of posterior long segment pedicle screw–rod construct is a good option. This procedure can replace the retropulsed fragment and correct the kyphosis. However, ligamentotaxis is prevailing in thoracolumbar burst fractures and is unable to alleviate the canal compromise ideally. The mean reduction of canal compromise at T12 and L1 region in the best situation are 66% and 55% retrospectively. However, these can be achieved only if the injury is diagnosed early, within a few days after the injury.

Vertebral column shortening is another option as we used in our case who was diagnosed with a considerable delay. Vertebral shortening can be actually achieved through pedicle subtraction osteotomy. The procedure is started with long posterior pedicle screw–rod construct, two levels above and two levels below the fractured vertebra. Subsequently PSO of the affected vertebra should be done. The retropulsed fragment can be removed with reverse angle curette or can be pushed in to the osteotomy site.

In conclusion, forceful muscle contractions during a convulsive seizure may cause burst fracture at mobile segments of the thoracolumbar region. Clinical signs of such unstable fracture might be subtle and absence of severe pain can hinder early diagnosis of these serious injuries. This type of fracture is more common in young males and after the first episode of seizure. With the absence of external trauma and post-ictal impairment of consciousness, a seizure-induced fracture may be easily overseen. Acute low back pain with or without neurological signs after an attack of seizure or in a patient already suffering from epilepsy should be regarded as serious and a whole spine imaging study is necessary for the assessment of the vertebral column. Moreover, in societies where Tramadol abuse is common, Tramadol-related seizure should be suspected when the convulsion occurs for the first time in a young adult individual. More control on the availability of this analgesic is necessary for the prevention of such untoward complications.

REFERENCES