Case report

Lumbar spine burst fracture as a result of hypoglycaemia induced seizure

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1. Introduction

It has been reported in literature that vertebral fractures can occur as a consequence of seizures. Muscle forces alone during tonic–clonic seizures can result in severe musculo-skeletal injuries which include vertebral fractures, neck of femur fractures, proximal humeral fractures and dislocations of shoulder.

We report the case of a 42 years old lady, who sustained burst fracture of second lumbar vertebra with mild retropulsion as a consequence of hypoglycaemia induced seizure.

2. Case

A 42-year-old lady presented to accident and emergency department with a history of five days of low back pain. Significant past medical history included poorly controlled insulin dependent diabetes mellitus, celiac disease. She is a non-smoker and worked as a shop assistant until recently, independently mobile, managing activities of daily living without support.

Questioning revealed a hypoglycaemia induced seizure five days ago. The seizure was not related with any obvious trauma to the back or fall during the episode. She had attended the department on the same day for treatment of her hypoglycaemia induced seizure which was controlled with dextrose infusion and better control of her insulin regime. She had complained of difficulty in breathing at the same time on presentation. A chest radiograph was taken, but no obvious cause for her respiratory distress was identified. The lung fields were clear and no rib injury or pneumothorax identified. She was discharged home with analgesia for her back pain and advice on insulin regime.

The second presentation five days later was due to persistent low back pain. At this point she had a thorough clinical examination of her spine which revealed a tender L2 spine. Plain radiograph revealed a 50% L2 anterior wedge compression fracture. She was then transferred to the regional orthopaedic unit for further management.

Her American Spinal Injury Association (ASIA) examination was normal with no signs of cord compression. She had full control over her bowel and bladder function. She had reduced power in the lower limbs bilaterally which was attributed to pain. The focal tenderness was confirmed, with no other obvious tender points over the spinous processes. Computer tomogram demonstrated an isolated burst fracture at the level of L2 involving the anterior and middle column with retropulsion into the spinal canal. The canal was reduced to 8 mm diameter. Her case was discussed with the specialist spinal unit for second opinion and treatment was by conservative management. She was fitted with a lumbar spine support brace (C37) and mobilised next day with analgesic support and physiotherapy guidance. She made good recovery and was discharged in two days with adequate independent mobilisation with analgesic control for her pain.

3. Discussion

Various collateral injuries have been reported as a result of seizures, including fractures of the vertebrae, femoral neck, pelvis and dislocation of shoulder.1,4 These fractures usually result from fall or accident caused by the seizure.5 But in our case, fracture occurred as a consequence of hypoglycaemia induced seizure in bed, without any evidence of fall or trauma. In literature, the incidence of vertebral fracture associated with convulsive seizures varies from 0.95 to 16%,5,7 although it is difficult to identify purely atraumatic fractures, as seizures are frequently unreported.

The fracture incidence has been reported as 2.4% in patients undergoing electroconvulsive therapy.8 Vertebral fractures occurred in 0.95% of cases and thoracic compression fractures [T3–T8] were quantitatively the most common fractures sustained.5 The seizure-induced vertebral fractures appear to occur more frequently in male patients, suggesting a direct relationship between the risk of vertebral fractures and muscle bulk.9 During seizure tonic–clonic muscle activity can cause fractures that are associated with only mild pain, 15% of primary asymptomatic fractures are attributed to seizure.11

This emphasises the importance of a critical musculo-skeletal examination of patients admitted after tonic–clonic seizures even if an event of fall or trauma is not reported in the history. Special emphasis should be made towards spine or limbs as well as major joints. In case of doubt, radiographic assessment should be performed to rule out skeletal involvement like fractures or dislocations.

There are mainly two hypotheses suggesting the mechanism of fracture:
1. In the absence of external trauma, the mechanism of seizure-induced vertebral burst fractures appears to involve a violent muscle contraction producing either simple axial compression or flexion-compression. This theory has been put to the question with equivocal answers.\(^6\)

2. A decrease in measured internal pressure from slow to high speed loading groups suggests that the nucleus entering the vertebral body may act as a wedge thereby splitting the vertebral body and enabling the retropulsion which is commonly noted in these fractures by pushing the bony fragments to enter the canal.\(^6\) For the same energy and direction of impact, a high loading rate produces fractures with significant canal encroachment (47.6%)\(^6\) which would explain the high incidence in these fractures due to the high volume of forces produced during seizure. Conversely, minimal encroachment (6.8%) into the canal was noted at low loading rates.\(^6\)

Spinal canal impingement of over 50% cases of seizures have been noted in literature.\(^12\) In order to produce spinal canal impingement, seizure has to generate a high loading rate. There has been one more case which has been reported previously for atraumatic lumbar burst fracture\(^8,12\) and proximal humeral fracture caused by nocturnal seizure precipitated most likely by hypoglycaemia in an insulin dependent diabetic patient.

Vertebral fractures caused without the presence of trauma related to seizures follow a different fracture pattern in comparison to spinal fracture following direct trauma. Seizure induced atraumatic vertebral fractures are usually located in mid-thoracic region between T3 to T8. Traumatic injury to the spine leading to fracture is usually located in the cervical spine, thoraco-lumbar or lumbo-sacral junction.\(^12\)

Mid lumbar fracture is a very rare entity specially made rarer in the absence of trauma.

Diabetes and iron overload are associated with mineral bone disease.\(^10\) There could have been an underlying mineral bone disease component. This was not investigated as she did not fit the criteria other than the single risk factor of diabetes, there was no other obvious site involvement to suggest generalised mineral bone disease.

The treatment of neurologically intact patients with thoraco-lumbar burst fractures is still contentious. However, there is a growing body of evidence suggesting that operative management of burst fractures in the absence of abnormal neurological findings provide no major long-term advantage compared with non-operative treatment.\(^13,8\)

Conservative management of thoraco-lumbar burst fracture is followed by marked degree of spontaneous redevelopment of the deformed spinal canal, providing further argument in favour of non-operative management.\(^2\) This patient was managed non-operatively and achieved excellent symptomatic recovery. There was no neurological deficit noted at further follow-up.

4. Conclusion

In conclusion we can say that tonic-clonic seizures produce a high rate of loading to cause an atraumatic burst fracture in a vertebral body leading to retropulsion in majority of cases.

There is a need for high degree of clinical suspicion in post-seizure patients with subtle symptoms to diagnose musculoskeletal injury. If in doubt a radiological investigation is always advocated to rule out potential bony injury, especially in the presence of symptoms.

The presence of diabetes in this patient, the aetiology for the seizure, could be a potential underlying cause for mineral bone disease. Without radiological investigation, it is impossible to diagnose and treat patients in this category.

Conservative management in a lumbar burst fracture led to good symptom relief and full recovery of function, further adding to the evidence of good recovery from conservative management.

Appendix 1

Roentgenogram of lumbar spine of a burst fracture of the second lumbar vertebra of a patient with hypoglycaemia induced seizure.
CT views of L2 burst fracture of a patient with hypoglycaemic-induced seizures.
References


Appendix 2

Follow-up roentgenogram of lumbar spine at six months.