

Spinal Fractures Caused by Hypoglycemic Convulsion: Beware of the Distracted Injury

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Convulsions are known to dislocate joints and can cause fractures from falls or due to the thrust caused by seizures. Vertebral compression fractures have been described in tetanus and epilepsy [1]. Only a few cases of convulsion-induced vertebral fractures after hypoglycemic attacks in diabetes mellitus have been reported. We present the case of a patient who suffered a hypoglycemic convulsion causing non-contiguous vertebral fractures. These fractures presented gradually, causing neurological symptoms, and led to two surgical interventions.

PATIENT DESCRIPTION

A 53 year old healthy, well-controlled insulin-dependent diabetic man was brought to the emergency department after his wife found him in bed convulsing. His initial glucose levels were 15 mg/dl. An intravenous line was inserted and he was treated with a solution of 25% dextrose until normoglycemia returned.

Upon regaining consciousness, the patient complained of pain in his upper thoracic back and right shoulder. On physical examination, tenderness over the upper thoracic midline was noted and he had a limited range of motion of his right shoulder without any neurological deficit. He was able to walk and had no other complaints. A thoracic computed tomography scan demonstrated vertebral compression fractures of D5 and D6 vertebrae and a fracture of the right scapula. The patient was placed in a body brace;

however, the pain continued and robotic guided kyphoplasty of D5 and D6 was performed [Figure A]. The next day the patient’s upper back pain disappeared, but he started to complain of radicular pain in his left leg according to the L4 dermatome and could not walk. A CT scan of the lumbar spine demonstrated two previously undiagnosed fractures – a burst fracture of L4 and a compression fracture L3 [Figure B].

In the attempt to avoid another surgical intervention, an epidural catheter was placed remitting the patient’s pain. When it was removed, the pain recurred and the patient lost ambulation. He underwent decompression of the left L4 nerve root and cement augmentation of L3 and L4 vertebrae [Figure C]. He returned to work and daily activities 3 weeks later. All pain medications were discontinued within 3 months. A diagnosis of primary osteoporosis was made after a full metabolic

[A] Sagittal reconstruction of the CT after kyphoplasty of D5 and D6. We can see the cement is contained in the body of the vertebrae, with full reconstruction of the vertebral height



[B] Sagittal reconstruction of the CT scan showing a burst fracture of L4 and a compression fracture L3



[C] Lateral X-ray after decompression of the left L4 nerve root and cement augmentation of L3 and L4 vertebrae



bone workup, and the patient was put on bisphosphonates, calcium and vitamin D.

COMMENT

Seizures are a known cause of fractures. The most common bones to break are vertebrae, distal radius, and proximal femurs. Interestingly, as in the case presented here, seizures during sleep cause more fractures, indicating that the muscle spasm is strong enough to cause fractures. Most of the vertebral fractures are compression fractures that respond to non-operative treatment (analgesia, rest, bracing). However, burst fractures have also been documented, narrowing the spinal canal, causing neurological symptoms requiring urgent surgical decompression [2].

Hypoglycemia (defined as a measured blood sugar below 70 mg/dl) is a known cause of seizures. When blood glucose levels drop the brain does not receive the basal amounts of glucose needed for normal function, eventually leading to confusion, disorientation, seizures, coma and death. Insulin-dependent diabetic patients suffer from hypoglycemia 10% of the time, and 62–170 episodes of severe hypoglycemia per 100 patient years were reported to occur [3]. Insulin-dependent diabetes has been associated with low bone density. The Nord-Trondelag Health Survey showed a significant increase in hip fracture rates among female diabetic patients compared to non-diabetic female patients [4]. The longer the patient has diabetes, the higher the chances of suffering a fracture. The exact cause of higher fracture risk is not known; however, animal models suggested a deleterious effect

of insulin-like growth factors and other cytokines on diabetic bone metabolism. In addition, the fact that the disease usually starts at a young age, when bones are still growing, may influence maximal bone density and bone mineralization. There is an association between diabetes and celiac disease, gastropathy and neurogenic bowel syndrome – all factors that lead to osteomalacia and osteoporosis and increase the risk of fractures.

Despite the high prevalence of osteopenia and hypoglycemic events in diabetic patients, there are singular reports in the English literature of vertebral fractures caused by hypoglycemia-induced epileptic seizures in diabetic patients. In his article, Nabarro [5] states that in his long career as a diabetologist he encountered only four cases of vertebral fractures caused by hypoglycemia, and that these fractures are most likely missed, indicating the need for a thorough evaluation of diabetic patients complaining of back ache after suffering from a nocturnal hypoglycemic event.

The incidence of concomitant vertebral fractures is as high as 10% in traumatic and osteoporotic fractures. The fractures can be in adjacent levels or skipping vertebrae. These fractures can be easily missed, as the patient refers to the primary cause of pain and the second injury may be masked at that time. As in the case presented here, only after undergoing treatment for the T5-T6 fractures did the patient begin to suffer from the lumbar burst fracture. In trauma cases, ATLS (Advanced Life Trauma Life Support) recommends a whole-body CT scan to avoid missing injuries in stable patients as part of

the secondary survey. The case described here raises the question whether convulsion-induced trauma should be managed in the same way. These fractures need to be sought with a high index of suspicion when the patient complains of sensorineural changes after the initial diagnosis.

In conclusion, vertebral fractures are an elusive complication and should be looked for in patients suffering backache after hypoglycemic convulsions. If a fracture is noted, a complete survey of the spine is recommended to rule out other vertebral lesions. These patients must be treated for their acute injuries followed by a full evaluation and treatment of their metabolic bone disease.

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