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BURST FRACTURE OF THE FIRST LUMBAR VERTEBRA AND CONUS-CAUDA SYNDROME COMPLICATING A SINGLE CONVULSIVE SEIZURE: A CHALLENGE OF DIAGNOSIS IN THE EMERGENCY DEPARTMENT

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□ **Abstract—Fractures of the thoracic and lumbar vertebrae as a direct consequence of generalized epileptic convulsions are the most common non-traumatic type of fracture complicating epileptic seizures. The majority of these fractures are compression fractures that occur with minimal symptoms and virtually no permanent neurological sequela. Nevertheless, muscle contractions generated during generalized motor seizures can result in severe axial skeletal trauma and grave neurological complications. We describe the case of a 35-year-old man who suffered a burst fracture of the first lumbar vertebral body and acute conus medullaris-cauda equina syndrome as a direct consequence of a single grand mal seizure. The aim of this report is to draw attention to this serious complication of generalized convulsive seizures and alert readers to epilepsy-related vertebral fractures. Diagnosis and management of acute cauda equina-conus medullaris syndrome caused by lumbar fracture are reviewed.** © 2006 Elsevier Inc.

□ **Keywords—Fracture; cauda equine; conus medullaris; epileptic seizure**

INTRODUCTION

Vertebral fractures occurring as a complication of generalized convulsive (grand mal) seizures are commonly overlooked, perhaps because these fractures frequently tend to be asymptomatic (1,2). We describe a unique case of a burst fracture of the first lumbar (L1) vertebral body, which occurred during a single incidence of grand mal seizure, and caused compression of both the conus medullaris and cauda equina, resulting in acute conus medullaris-cauda equina syndrome. Early diagnosis of this condition is imperative as this syndrome may constitute a medical emergency (3–5).

CASE REPORT

A 35-year-old man was admitted to the Long Island College Hospital Emergency Department (ED) due to fever, confusion, seizure, and lower limbs weakness. According to his mother, he was well until 2 days before admission, when he developed a "flu-like" syndrome. On the day of admission, while sitting on a chair and talking to his mother, the patient suddenly became unresponsive. His upper and lower limbs then became stiff and he started to shake for a period of approximately 30–60 s. At this time his mother helped him slide down to the floor without fall or trauma and brought him to the ED. There was no history of recent travel outside New York City. He had no history of epileptic seizures, back pain, headache, chills, night sweat, urinary urgency, diarrhea, sphincter problems, or insect bite. There were no risk factors for infection with human immunodeficiency virus. He did not use alcohol, tobacco, or illicit drugs.

On physical examination, the patient seemed to be in no acute distress. The patient's oral temperature was 38°C, the pulse was 107 beats/min, and the respiratory

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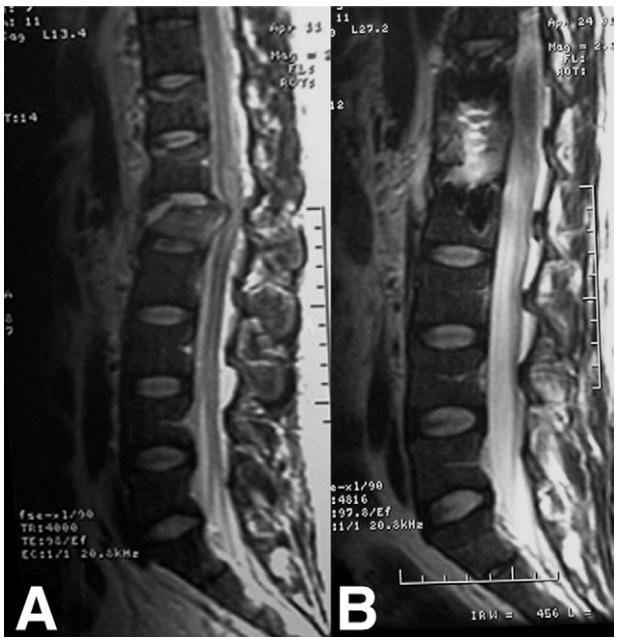

Figure 1. MRI T_2- weighted image of the thoracolumbar spine demonstrating burst fracture of the L1 vertebra with bone retropulsion into the spinal canal (A). The same region after surgical decompression (B).

rate was 20 breaths/min. The blood pressure was 155/90 mm Hg. The conjunctivae were clear, the neck was supple, and there were no enlarged cervical or axillary lymph nodes. Examination of heart, lungs, and abdomen revealed no abnormalities. No skin rash, tongue laceration, pharyngitis, or signs of external trauma were appreciated.

On neurologic examination, the patient was noted to be confused and obtunded with fluent speech. He obeyed one-step commands. Pupils were equal and reactive to light. Eye movements were full and his face was symmetrical. His arms had normal muscle tone and strength. The muscle tone was decreased in both legs and he could not lift them from the bed. No dysmetria was detected on the finger-to-nose test. The deep-tendon reflexes were normal in the upper extremities and absent in the lower extremities. The plantar responses were minimally flexor. No ankle clonus was observed. The patient could not stand or walk. The patient's post-ictal alteration in mental status precluded a reliable assessment of the sensory and motor systems. Rectal examination was deferred at this time.

The results of a urinalysis were normal. The blood chemistry values, including serum electrolytes, serum glucose, liver function tests, and complete blood count were unremarkable with the exception of a white blood cell count of 14.6 thousand per cubic millimeter without bandemia.

An electrocardiogram disclosed a sinus tachycardia at a rate of 107. A roentgenogram of the chest and a non-enhanced computed tomography (CT) scan of the brain were both normal. A lumbar puncture was performed. Spinal fluid analysis was remarkable only for elevated protein (188 milligrams/deciliter).

As a routine procedure for non-ambulatory patients, a Foley catheter was inserted and the patient was admitted to the intensive care unit with the diagnosis of generalized convulsive seizures, post-ictal state, bilateral Todd's post-epileptic paralysis, and possibly acute viral encephalomyeloradiculopathy. Treatment was begun with acyclovir, intravenous fluids and phenytoin.

On the second hospital day, the patient became more alert. He denied back or leg pain. Neurologic examination at this time showed severe bilateral lower extremity weakness (Medical Research Council grade 0–3/5). Quadriceps muscles, knee flexors, and hip flexors demonstrated the greatest degree of weakness. Rectal tone was diminished. Superficial pain and touch sensation were impaired throughout the lower extremities and saddle region. Proprioception was intact. Lower extremity tendon reflexes were absent. No pyramidal tract signs were evident. An acute lower spinal cord disorder was suspected and emergency magnetic resonance imaging (MRI) of the spine and brain were performed. MRI of the brain was normal. MRI of the spine revealed a burst fracture of the first lumbar vertebra with compression of the conus medullaris and the superior region of the cauda equina (Figure 1). A thoraco-lumbar CT scan also revealed a burst fracture of the L1 vertebral body and violation of the spinal canal (Figure 2).

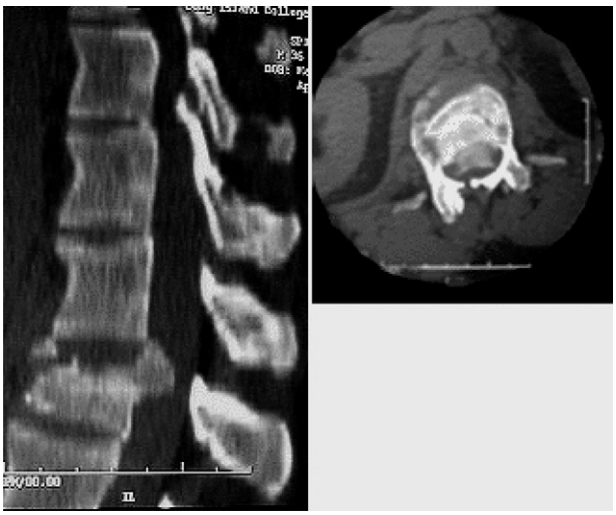

Figure 2. CT scan of thoracolumbar spine demonstrating an L1 burst fracture with bone retropulsion into the spinal canal. Sagittal reconstruction (left) and axial (right).