

Seizure-induced lumbar burst fracture associated with conus medullaris-cauda equina compression

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ABSTRACT

A 36-year-old male developed a burst fracture of the L1 vertebra following a seizure. The patient experienced conus-cauda equina compression resulting in urinary retention and patchy sensory loss. An MRI of the spine revealed short tau inversion recovery (STIR) hyperintensity at L1 with 70% loss of vertebral body height, 8-mm posterior bony retropulsion into the spinal canal and T2-weighted hyperintensity in the cord, which is consistent with cord edema. A thorough physical exam and imaging is critical for immediate diagnosis of spine injuries to prevent grave neurological complications.

Key words: • seizure • burst fracture • vertebra • conus medullaris • cauda equina

A seizure-induced burst fracture of the spine is a rare clinical entity with only five cases reported in the medical literature to date (Table) (1–5). Although rare, it is extremely crucial to recognize this emergent fracture because it predisposes the patient to irreversible neurological injury (4).

Following a seizure, the most common site of a compression vertebral fracture is the midthoracic spine, and the most common site of a burst vertebral fracture is the thoracolumbar junction of the spine (1, 4, 6). A burst fracture is more likely to cause neurological complications due to the fragmentation and comminution of the vertebral body, but a compression fracture remains asymptomatic in the majority of cases (7–9). For appropriate patient management, it is imperative to differentiate these two vertebral fracture types. Other forms of musculoskeletal trauma resulting from generalized seizure include fracture of the humeral head and neck, femur neck, pelvic bones, acetabulum and scapula, as well as dislocation at the glenohumeral joint, manubriosternal joint and femoroacetabular joint (1).

Most of the cases of seizure-induced musculoskeletal trauma occur in patients with an isolated, unprovoked seizure. Such seizure episodes often occur due to electrolyte imbalance, drug overdose and/or drug withdrawal (1). Seizure-induced burst fracture of a vertebra is a non-traumatic fracture and may occur even at rest (1, 4). One such case of a nocturnal double thoracic burst fracture has been reported in the literature by Rupperecht et al. (3).

In our report, we present the case of a 36-year-old male who suffered a seizure-induced burst fracture of the L1 vertebra with associated cord compression and right posterior shoulder dislocation with comminuted humeral head fracture. Diagnostic imaging and case management are also discussed.

Case report

A 36-year-old left-handed male checked into the emergency department with an acute episode of a generalized tonic clonic witnessed seizure during sleep. There was no traumatic fall from a bed during the seizure. The patient appeared confused and developed a headache, but he denied a tongue bite, loss of bladder or bowel function, neck pain and weakness or numbness of the extremities in the immediate post-ictal period. The rest of the systems review was not pertinent. The patient complained of soreness in his right shoulder and lower back in the latter part of the post-ictal period. This patient had a past medical history of scoliosis and denied use of tobacco, alcohol or other recreational drugs. His medication history included the use of dilantin for six years for epilepsy. The patient was otherwise very active and functional, both at work and at home. There was no family history

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Table. Tabulation of the cited literature for seizure-induced burst fractures

Study	Author; year (reference number)	Number of patients	Cause of seizure; symptoms of musculoskeletal trauma	Musculoskeletal trauma	Neurological complication	Pertinent imaging findings	Management and follow-up
1	McCullen et al.; 1994 (1)	1	Tricyclic anti-depressant overdose (amoxapine); post-ictal bilateral shoulder soreness and pain with movement as well as late (day 7) midthoracic back discomfort.	T7 thoracic vertebra burst fx, bilateral humerus head fx	None	<i>X-ray of the shoulder:</i> Comminuted 4 part L. humeral head fx, nondisplaced R. humeral surgical neck fx. <i>X-ray of the thoracic spine:</i> T7 compression fx with 50% height loss, T6 and T8 wedging. <i>CT and MRI of the thoracic spine:</i> Type B T7 burst fx with 15% canal compromise with retropulsed fx segment. <i>Bone scan:</i> Increased uptake from T3–T8.	Anterior decompression of T7, posterior fusion using Cotrel-Dubosset instrumentation. Early mobilization at 5 days with Jewett brace for 8 weeks. Long-term follow-up was not available.
2	Youssef et al.; 1995 (2)	1	Electrolyte imbalance; lower back pain on day 5	L1 and L2 vertebra burst fx	None	<i>X ray of the lumbar spine:</i> Contiguous L1 & L2 burst fx with >50% loss of vertebral body height.	Initially treated with Risser cast but was unsuccessful. Later underwent posterior spinal fusion with Cotrel-Dubosset segmental instrumentation and iliac crest bone grafting. Spine was stable at 1 year.
3	Rupprecht et al.; 2001 (3)	1	Cerebral astrocytoma with an epileptogenic focus; back pain in immediate post-ictal period and shoulder pain on day 7.	Double thoracic vertebra burst fx, unstable fx of humerus and scapula	None	NA	NA
4	Roohi et al.; 2006 (4)	1	Infection with flu like symptoms with no definite cause; denied lower back pain on day 2 but presented with weakness, loss of reflexes, loss of superficial pain and touch sensation in both lower extremities and saddle region along with diminished rectal tone.	L1 vertebra burst fx	Conus medullaris-cauda equina syndrome	<i>CT and MRI lumbar spine:</i> Burst fx of L1 vertebral body with violation of spinal canal. Compression of the conus medullaris and superior region of cauda equina was seen on MRI.	Emergent spinal decompression and stabilization. Patient was placed in an orthotic brace and referred to rehabilitative services. At 6 months post-surgery, patient had full recovery except for intermittent nocturnal incontinence, which was cured completely by 5 years.
5	Mehlhorn et al.; 2007 (5)	1	Epilepsy; mild lower back pain in the immediate post-ictal period.	L2 and L4 vertebra burst fx	None	<i>X-ray and CT of lumbar spine:</i> Unstable burst fx of L2 and L4 vertebra without major spinal canal violation	Fx stabilized with a dorsally instrumented internal fixator from L1 to L5 followed by hemilaminectomy and ventral spondylolisthesis. Long-term follow-up was not available.

fx, fracture; R, right; L, left

of seizures or recurrent fractures and dislocations.

On admission, the patient appeared to be in no acute distress and had stable vital signs. His lungs were clear with no evidence of aspiration pneumonia,

the heart exam was unremarkable and the abdomen was soft, non-tender and non-distended.

The musculoskeletal exam of his right shoulder was consistent with dislocation/fracture and revealed swell-

ing upon inspection and tenderness upon palpation. Restriction of motion was present on both active and passive movements. The lower back examination revealed mild scoliosis, restricted range of motion and tenderness upon

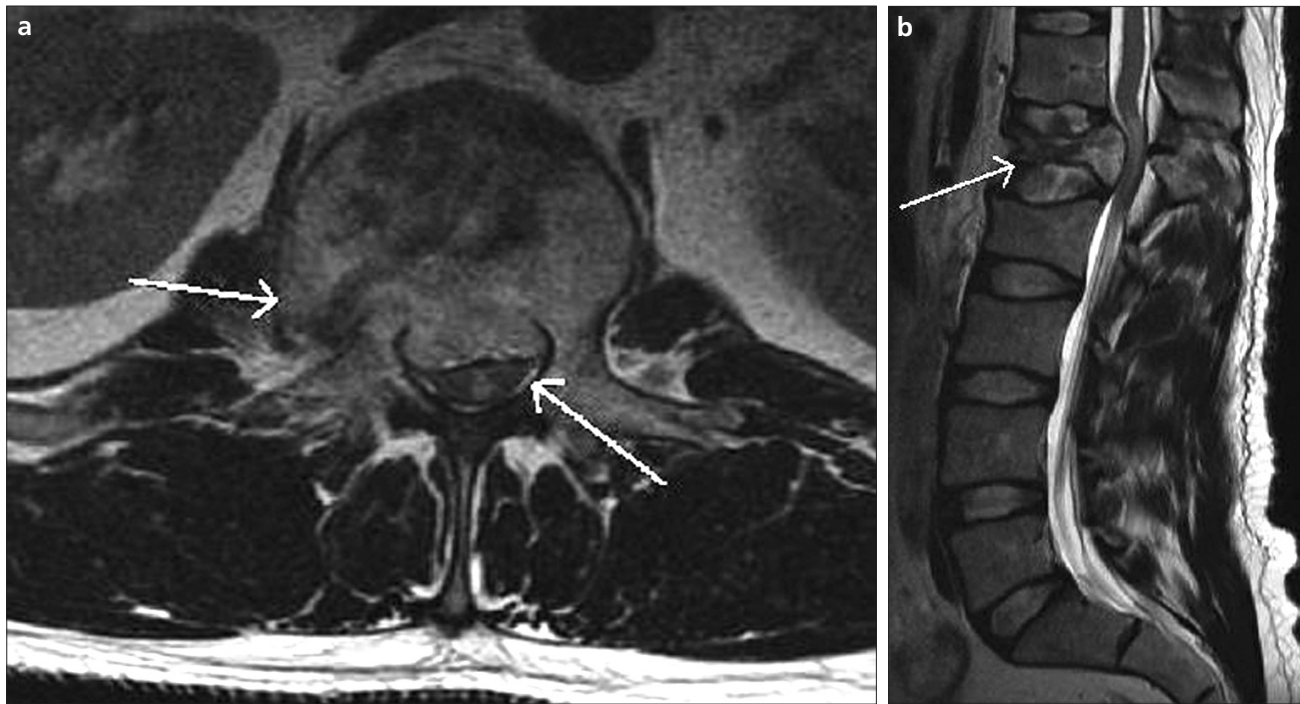


Figure 1. a, b. MRI T2W fast relaxation fast spin-echo sequence (FRFSE) axial (a) and T2W FRFSE sagittal (b) views of the thoracolumbar spine were imaged with parameters of TR/TE, 3800–4550/98–123.6 ms; FOV, 34–36 x 25.5–36 cm; matrix size, 256–512 x 160–256; section thickness, 3 mm; intersection gap, 0.5–1 mm; and echo-train length, 1–4. MRI showed an L1 burst fracture with a 70% loss of vertebral height (arrow, b) and retropulsion of the posterior vertebral elements of up to 8 mm into the spinal canal (arrows, a). Mild compression of the conus medullaris-cauda equina is present. T2W hyperintensity within the cord at the L1 level is consistent with cord edema. Mild loss of anterior vertebral height of L2 represents a chronic compression fracture.

palpation of the spinous processes at the L1 and L2 vertebra, consistent with a possible fracture.

The initial neurological exam was within normal limits until day four post-admission. The patient gradually developed neurological deficits with high residual urine retention (>1000 mL) as well as tingling and paresthesia in the lower extremities and the saddle region. Upon re-examination, the sensory loss exhibited a patchy distribution with loss of superficial pain and touch in the lower extremities. Proprioception, superficial and deep tendon reflexes, motor strength, perianal sensation and rectal tone were intact, with a downgoing plantar response. The routine basic metabolic profile, complete blood counts and urine toxicology screen all tested normal. The lumbar puncture showed a slight elevation of protein. In the interim, the patient underwent an open reduction and internal fixation (ORIF) for the right humeral head fracture along with a reduction of the posterior dislocation of the humerus.

An emergent MRI performed after the appearance of neurological

deficits showed a burst fracture of L1 with STIR hyperintensity consistent with an acute/sub-acute fracture. An approximately 70% loss of vertebral body height was seen with posterior retropulsion of vertebral elements into the spinal canal, resulting in asymmetric severe canal stenosis (right more than left). Posterior displacement and mild compression of the conus medullaris-cauda equina was present. T2-weighted (T2W) hyperintensity within the cord at the L1 level was consistent with cord edema. Severe foraminal narrowing at the L1/L2 level was present. An L1 transverse process displaced fracture was demonstrated. The L2 vertebra anterior compression fracture without STIR hyperintensity suggested a more chronic compression fracture at this level. The lower thoracic cord was normal. Additionally, an MRI also showed an acute compression fracture of the inferior end plate of the T12 vertebra resulting in mild height loss, which was not evident on CT or X-ray (Fig. 1).

The initial X-ray of the lumbar spine revealed an L1 vertebral body compression fracture, with a more than

50% loss of vertebral body height, and a superior end plate compression deformity of L2, with an approximately 25–40% loss of vertebral body height (Fig. 2). A CT of the lumbar spine confirmed the lumbar X-ray findings and helped to diagnose a “burst fracture” of the L1 vertebral body. There was loss of height and retropulsion of bony fragments of up to 9 mm into the spinal canal that caused significant narrowing. The CT also showed an anterior compression fracture of the L2 vertebral body and a displaced L1 transverse process fracture (Fig. 3).

The right shoulder X-ray demonstrated a posterior dislocation of the humerus with a comminuted impacted fracture of the surgical neck and a reverse Hill-Sachs impacted fracture of the humeral head against the glenoid process.

The patient was initially managed with a Keppra and thoracolumbar sacral orthosis (TLSO) brace for the lumbar fracture until neurological deficits developed. On day 4, the MRI of the lumbar spine warranted an immediate surgical intervention. Intravenous steroids were initiated, and a



Figure 2. Lateral view of a lumbar spine X-ray revealing an L1 vertebral body compression fracture with a more than 50% loss of vertebral body height (arrow). In addition to this, superior end plate compression deformity of L2 with an approximately 25–40% loss of vertebral body height is seen.

lumbar spine surgical decompression was successfully performed. A histological sample of the extracted bony fragments revealed poor bone quality with no evidence of cancer, metastasis, osteomyelitis or multiple myeloma. Mobilization was started five days after the lumbar surgery. Overall, the patient responded well to the treatment, and his functional status significantly improved with rehabilitation and physiotherapy.

Discussion

Seizure-induced fracture is an uncommon phenomenon, occurring in only 3% of all epileptic patients (7, 10, 11). Kelly reported an incidence of seizure-induced fracture at 2.4%, but Finelli and Cardì reported an incidence as low as 0.3% (6, 7). Most of these fractures occur at night and have a non-traumatic etiology (1, 3, 4). In 60% of cases, patients remain asymptomatic due to a cloudy sensorium in the post-ictal phase (1, 4). There is a latency period in the diagnosis of frac-

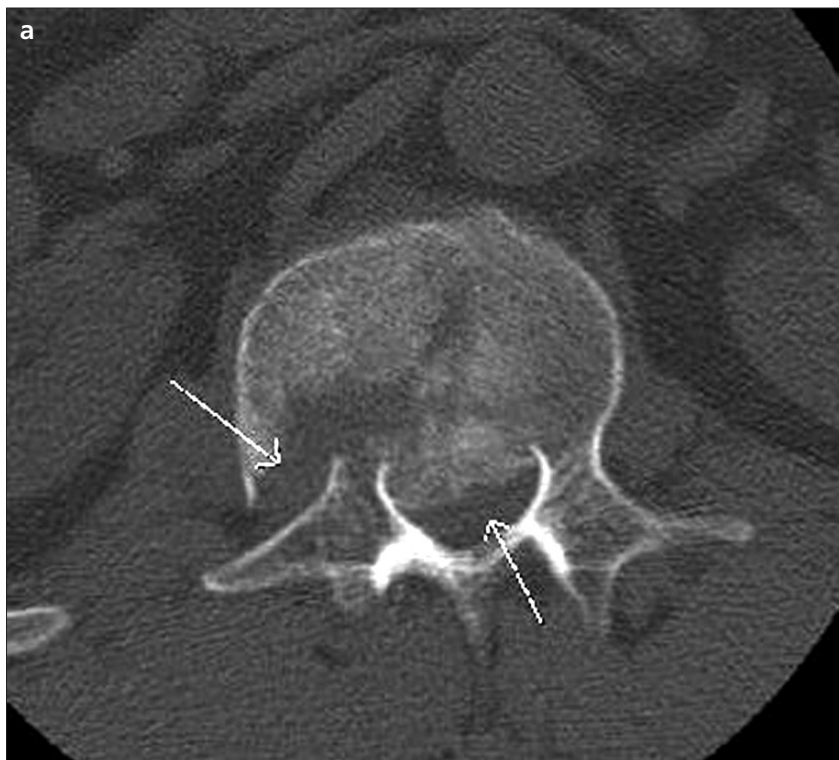


Figure 3. a, b. Axial (a) and sagittal (b) views of the CT lumbar spine show loss of height of the L1 vertebral body (arrow, b) with retropulsion of the bony fragments into the adjacent spinal canal of up to 9 mm (arrows, a), causing narrowing. In addition, the CT spine image shows an anterior compression fracture of the L2 vertebral body. The paravertebral soft tissues are unremarkable.



ture injury that may be delayed by five to seven days in some cases (12). The most common musculoskeletal injuries seen after tonic clonic seizures include compression fractures of the thoracic and lumbar vertebral bodies, humeral head fractures/dislocations/combined injuries, manubriosternal joint disrup-

tion and femoral neck, pelvic, acetabular and scapular fractures (1, 4, 7, 11). Less common fracture sites include the facial bones, odontoid process, ribs, distal radius and ankle (4, 7, 11, 13). Bilateral posterior dislocation of the shoulder is highly suggestive of a seizure-related injury (7).

The spine is the most common seizure-related fracture site and is critical due to the associated irreversible neurological complications (1, 4). The incidence of symptomatic spinal fracture is 1% after seizure (13). However, the incidence of asymptomatic spinal fractures may be as high as 15–16% as per some studies (8, 13). Compression fractures occur more commonly than burst fractures, which account for about 85% of seizure-induced fracture injuries in the spine (9). They cause a decrease in the height of the anterior aspect of the vertebral body without severance of the posterior vertebral elements (7, 8, 10, 14). The mechanism of injury of compression fractures involves the forward flexion of the spine as abdominal and paraspinal muscles contract violently during convulsions (1). Most compression fractures post-seizures are asymptomatic without any major neurological sequelae. They involve the mid-thoracic spine (T3–T8) where the compression forces concentrate along the anterior and middle thoracic kyphotic spine (1, 4, 6). On the contrary, burst fractures are far less prevalent than compression fractures because the axial skeletal contraction forces generated after seizures are insufficient to cause such a massive crush injury (4). A burst fracture following a seizure involves the thoracolumbar vertebral junction (T12 to L2), which is a point of transition of a rigid kyphotic thoracic spine and a more mobile lordotic lumbar spine (15–17). Gross pathological features of burst fractures include the loss of anterior and posterior vertebral body height, comminution of the superior and/or inferior end plate, retropulsion of a bone into the spinal canal, increased interpediculate distance, vertical laminar fracture, sagittal vertebral body fracture, interspinous widening and lateral translation/flexion.

Certain risk factors predispose an individual to seizure-induced fractures. These include osteoporosis, increased muscularity, duration of isolated seizures, repetitive stresses due to recurrent seizures, osteomalacia due to calcium or vitamin D deficiency, bone resorption secondary to primary hyperparathyroidism or secondary hyperparathyroidism, long term use of anti-epileptic drugs (valproate, phenytoin, carbamazepine) and long-standing inflammatory bone disease (1, 4, 6, 18). Appropriately addressing the under-

lying risk factors for seizure-induced pathological fractures might be helpful for preventing them in the future.

The role of imaging in the diagnosis of fracture injuries is very critical. Denis classified burst fractures into five categories on the basis of radiographic appearance (19). Type “B”, involving the superior end plate and retropulsion of the superoposterior cortex, is the most common. Type “C” involves the fracture of the inferior end plate only and is the rarest. Type “A” is comprised of fractures of both the superior and inferior end plates. Types “D” (burst rotation) and “E” (lateral flexion burst) are other burst fracture variations. However, the current and more simplified classification of thoracolumbar fractures includes three categories (A, B and C) based on a progressive scale of morphological bony damage (19). It is targeted toward ascertaining the stability of a spinal fracture. An MRI-based classification of thoracolumbar injuries is warranted in the future to better diagnose discoligamentous injuries and predict outcomes of skeletal and associated neurological injuries.

Initial spine radiographs might be helpful in the diagnosis of compression fractures. However, spine radiographs may miss 25% of burst fractures, hair-line fractures and other non-displaced fractures (20, 21). If a screening spine radiograph reveals a compression fracture, then a spinal CT must always be performed to search for a burst fracture, which may compromise the spinal canal space (1, 4). Spine radiographs give additional information about osteoporosis of the spine contributing to the fracture. Spinal radiographs are efficacious in the routine follow-up of the fracture site post-surgical intervention to look for hardware-related complications.

With the advent of the multidetector CT scan, enabled with advanced reformating and 3D reconstruction techniques, subtle fracture injuries may be diagnosed with high confidence. CT images can demonstrate end plate comminution and sagittal fracture of a vertebral body (19). CT is an excellent tool for diagnosing laminar fracture and fracture/dislocation of the facet joints. Type “D” and “E” burst fractures are better seen with high resolution CT with multiplanar reconstruction (19). Spinal CT is considered the imaging modality of choice to diagnose

any retropulsed bony fragments arising from a burst fracture (17). CT can efficiently demonstrate spinal canal compromise with the sagittal multiplanar reconstruction and gives details of the retropulsed fragment, which is most commonly a single central fragment (19). CT features demonstrating rotation, fracture and migration of the retropulsed segment correspond to a poor surgical outcome. CT helps to predict outcomes of neurological injury by estimating the degree of localized spinal kyphosis from the fracture. Patients with burst fractures resulting in less than 15 degrees of kyphosis have better prognoses and neurological outcomes (19). CT has also been useful for the diagnosis of cauda equina root entrapment into vertical laminar fracture of the lumbar burst fracture, which is a rare complication of the lumbar burst fracture (19). CT is mandatory preoperatively to ascertain that pedicles of the vertebra above and below the burst fracture site are intact, as they are required for screw fixation to stabilize the burst fracture (19).

However, an MRI of the spine is more useful for detecting soft tissue ligamentous injury, particularly the posterior longitudinal ligament (PLL) and interspinous ligament (ISL) (19). MRI can demonstrate spinal cord impingement, neural compression, extradural hematoma or bulging intervertebral discs (20, 22). The PLL rupture is seen as a discontinuous black stripe in a sagittal T2W MRI sequence. The ISL injury appears as a high intensity signal on a T2W sagittal MRI sequence and is better appreciated with MRI than CT (19). Intervertebral disc injury is seen most often in the disc above the fractured vertebra as a high intensity signal in a T2W MRI sequence, as a rupture of a disc into the vertebral body or as a protrusion of the disc into the spinal canal (19). However, in some cases, the disc below the fracture level may be involved. The special MRI-STIR sequences are considered superior for detecting spinal cord edema and, therefore, for predicting an imminent spinal cord compression (22). MRI is extremely useful for differentiating neurological injury patterns: injury to the cord, combined conus medullaris and cauda equina or pure cauda equina (19). This may be clinically relevant as pure cauda equina compression has the best long-term prognosis. Modern

MRI scanners are as efficient as CT for diagnosing burst fracture-related injuries, such as neural arch fractures and wedged compression fractures, which are best observed on T2W spin echo sequences (19). Dynamic MRI is useful for predicting the progressive collapse of an already fractured vertebra (23). MRI also gives information about the acute/subacute/chronic nature of vertebral fractures based on the signal intensity of T1W images (20, 22, 23). Overall, MRI has an increased utility compared to CT in diagnosing burst fractures and their associated soft tissue complications (19).

Neurological injury associated with a seizure-induced burst fracture is extremely rare. Previously, only one case has been reported of a spinal burst fracture causing conus medullaris injury (4). A burst fracture at the L1 vertebra or disc herniation between the L1–L2 vertebrae may cause compression of the conus and/or cauda equina resulting in neurological deficits. “Conus medullaris syndrome” consists of symmetrical saddle anesthesia, bowel and bladder incontinence or sexual dysfunction with relatively normal lower extremity strength (4, 24, 25). The most consistent symptom of cauda equina compression is bowel and bladder dysfunction with the absence of any pyramidal tract signs (4, 24, 26). Due to the close juxtaposition of the conus and cauda equina, a burst fracture at L1 may result in neurological deficits due to compression of either or both of these structures. Our patient presented with urinary retention, bilateral saddle and lower limb anesthesia with intact deep tendon reflexes and sparing of his muscle strength, which suggested a combined conus medullaris-cauda equina syndrome. If there is a confirmation of spinal cord compression upon imaging (MRI/CT) or if there is the presence of an unstable fracture, surgery is the procedure of choice to evacuate the retropulsed bony fragments in addition to spinal fixation with plate and screws (1, 4).

In conclusion, powerful muscle contractions may generate strong axial forces to crush the vertebral body at the thoracolumbar junction that

can result in a burst fracture. These are common in younger males with good musculature; they occur more frequently at the time of the first seizure episode and go unnoticed because patients cannot localize the area of pain. A thorough whole body physical exam is essential for early diagnosis. A screening thoracolumbar spinal X-ray may be included in the routine workup of such patients. When a compression fracture is diagnosed on a plain film, a spinal CT should be performed to confirm the presence of a burst fracture. MRI of the spine is the most useful and comprehensive imaging modality for complete assessment of thoracolumbar burst fractures.

Conflict of interest disclosure

The authors declared no conflicts of interest.

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