

Case Reports

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SEVERE NEUROLOGIC AND CARDIAC SEQUELA FROM OSTEOPOROTIC VERTEBRAL FRACTURES

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ABSTRACT

Objective: To report two adult Filipino patients with osteoporotic vertebral fractures who developed severe neurologic and hemodynamic complications.

Case 1: V.G. is a 74-year old postmenopausal female who was admitted for nape pain and progressive bilateral leg weakness of one month duration. There was no reported trauma. Somato-sensory testing showed a spinal level of L1. Spine radiographs revealed compression fractures of C2-C3, T7-T8 and L3 vertebrae. Diagnosis of multiple osteoporotic vertebral fractures was made and she was kept flat on bed and started on intra-nasal calcitonin. On the second hospital day, she developed sudden onset dyspnea, diaphoresis and hypertension, the latter unresponsive to combination of metoprolol and clonidine. She was admitted to the ICU and subsequently intubated. She later developed episodes of hypotension. Dysautonomia was considered when blood pressure swings, tachycardia and diaphoresis persisted. Acute coronary syndrome was likewise considered. Cardiac enzymes were elevated and electrocardiogram showed acute myocardial injury pattern. She sought discharge against medical advice and expired on the ninth hospital day.

Case 2: L.S. is a 66-year postmenopausal female was admitted for back pain accompanied by numbness of the lower extremities of two weeks duration following a low-impact fall. Thoracic spine CT scan showed compression deformity at T8-T9 vertebra probably secondary to osteoporosis. She was given calcitonin and kept flat on bed. She developed chest pain, dyspnea and hypertension with tachycardia and diaphoresis on the second hospital day. Cardiac enzymes were within normal. On the third hospital day she had urinary bladder atony and constipation. She was transferred to

the ICU where IV verapamil and clonidine were started. The patient's tachycardia and hypertension eventually resolved on the fifth ICU day. She was able to start physical rehabilitation, given alendronate and was eventually discharged on the sixteenth hospital day.

Conclusion: Vertebral fractures are often asymptomatic. Here, we present two cases where serious neurologic complications and a cardiac event developed following osteoporotic vertebral fractures. Occurrence of these fractures in the thoracic spine predisposed these patients to severe hemodynamic instability, cardiac symptoms and life-threatening situations.

Keywords: Osteoporosis, vertebral fracture, autonomic dysreflexia, dysautonomia

INTRODUCTION

Osteoporosis is a common and important health problem that accounts for up to two million fractures per year in postmenopausal women.³ The spine, hip, and distal radius are most commonly fractured. The true incidence of osteoporotic vertebral fractures is not well defined because many of these fractures are asymptomatic.⁴ Osteoporotic fractures of the spine cause pain, kyphosis, and loss of height and are not as devastating as those in the hip.³ Likewise, the true incidence of neurological compromise as a result of osteoporotic vertebral fractures is not known and is thought to be low.⁵

Objective

To report two adult Filipino patients with osteoporotic vertebral fractures who developed severe neurologic and hemodynamic complication.

Case 1

V.G., a 74 year old postmenopausal Filipina, was admitted for nape pain and bilateral leg weakness of one month duration. The paraparesis was associated with decreased sensation, progressive clumsiness and difficulty in ambulation, prompting admission. She denied back pain and trauma. She was diagnosed

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with hypertension five months prior to admission and was maintained on metoprolol. There was no dizziness, nausea, vomiting, nor blurring of vision.

On examination, she was ambulatory with assistance, BP was 130/90, heart rate was 86/min, respiratory rate was 18/min. Motor strength of the right leg was grade 3/5, left leg was 5/5. She had 50 percent sensory deficit at L1 level. The rest of the physical examination findings were normal. Cervical and thoracolumbar spine x-rays showed compression fracture of C2-C3, T7-T8 and L3 vertebrae. She was managed as a case of spinal cord compression secondary to osteoporosis, with bed rest and nasal calcitonin spray.

On her second hospital day, she developed dyspnea, diaphoresis and hypertension, unresponsive to metoprolol and clonidine. She was transferred to the Intensive Care Unit (ICU). Dysautonomia was considered but acute coronary syndrome could not be totally ruled out. Soon after, she was intubated and hooked to a ventilator. She then had episodes of hypotension. Cardiac enzymes were elevated (Trop I and CPK MB) and electrocardiogram showed anterior wall acute injury pattern. She was managed as a case of acute myocardial infarction. Hypotension was persistent, and she continued to have diaphoresis and cold clammy skin despite stabilization of ECG changes. Patient subsequently expired on ninth day during the trip home against medical advice.

Case 2

L.S., a 66 year old female, was admitted for upper back pain radiating to the lower back of two weeks duration. Pain intensified three days prior to admission, prompting admission. This was associated with body weakness and numbness of both lower extremities. She had a history of a low impact fall a month PTA.

On examination, she was asthenic, wheel chair borne, with a BMI of 17, BP of 140/90, heart rate, 78/min and respiratory rate, 20/min. She had a right facial palsy since a year prior to admission. The thoracic vertebral spines down to the sacrum were tender. There was gibbus deformity at the level of T7-T8. Motor and sensory functions were normal. Thoracic spine CT scan showed compression fracture at T8-T9. Chest x-ray showed a stable right upper lung fibrosis and an left ventricular form cardiomegaly with atherosclerotic aorta. Hematologic exam, serum creatinine and blood sugar were normal. She was managed as a case of thoracic spine compression

fracture secondary to osteoporosis. She was given tramadol, calcitonin nasal spray, gabapentin.

On the second hospital day, she developed chest pain, dyspnea, diaphoresis, hypertension ranging from 160-190/100-110mmHg and tachycardia of 110 -120/min. Cardiac enzymes were normal. ECG showed tachycardia with no signs of acute myocardial infarction or ischemia. She developed urinary bladder atony and constipation on the third hospital day. She was transferred to the ICU and managed as a case of possible autonomic dysreflexia (AD). Intravenous verapamil and clonidine were given for the control of tachycardia and hypertension. Diaphoresis resolved on the fifth ICU day. She continued to have upper back pain radiating down to the lower extremities and bladder atony. Tachycardia was controlled on the 5th ICU day and blood pressure was down to 130 - 140/90. She was transferred to room after five days in ICU. Further improvement in pain allowed her to tolerate physical therapy. She developed urinary tract infection, catheter-related, and was given a course of ceftriaxone. She was discharged on the sixteenth HD, wheelchair-borne with indwelling foley catheter.

A month later, patient was ambulatory, with complete resolution of bladder atony, normal blood pressure while on maintenance metoprolol and without pain in the spine.

DISCUSSION

Osteoporotic vertebral fractures are often asymptomatic and go undiagnosed.^{3,4,17} Vertebral fractures usually manifest with loss of height, globular abdomen, kyphosis and half the time, back pains. They are usually not associated with serious complications. Here we present two cases of osteoporotic vertebral fractures manifesting with severe neurologic and cardio-respiratory sequelae.

The true incidence of neurological compromise as a result of osteoporotic vertebral fractures is not known and is thought to be low⁵. Parfitt and Duncan⁶ suggested the complete absence of risk of spinal cord involvement in cases of vertebral fracture secondary to osteoporosis. In contrast, Shikata *et al*⁷ found that 12.1% of fractures required operative treatment because of neurological sequelae. Our patients manifested with neurologic, hemodynamic complications and possible autonomic dysreflexia (AD) - lability of blood pressure, tachycardia and diaphoresis in the setting of multiple vertebral compression fractures. The first case was complicated by acute myocardial infarction although

the persistence of symptoms of diaphoresis and hypotension may have been caused by a concurrent dysautonomia. The second case manifested with hypertension, tachycardia, diaphoresis and bladder atony. Although both cases had radiographic evidence of thoracic spine compression fractures lower than T6- the area of the sympathetic chain, they manifested with signs of sudden hypertension, tachycardia, diaphoresis and bladder atony. Tests to establish dysautonomia were not completed due to the severe unstable symptoms in Case 1 and the early resolution of symptoms in Case 2.

Osteoporotic vertebral fractures can present with back pain. These symptoms improve in three to six weeks, but severe back pain may persist for many weeks to months.⁸⁻¹⁰ Because the middle column is intact in compression fractures, the neural tissue is usually not affected. However, in burst fractures, because the middle column is fractured, bone fragments may cause compression on neural structures posteriorly. In addition, spine angulation cause intervertebral foramina constriction, resulting in root compression on the associated segment. Compression of the cord is generally located at the upper or lower part of the vertebral body with angulation or kyphosis. Additional factors in compression, such as discal herniation and a narrowed spinal canal, have also been encountered. Ischemia of the neural elements due to the compression on the vascular structures may aggravate the condition.¹¹ The frequency of occurrence of a burst-type fracture in atraumatic osteoporotic vertebral fracture is unknown.³

The neurological condition may vary according to the level of lesion; it may manifest as paraplegia, various sensory or motor deficits associated with cauda equina or root compression, and as rectal or urinary incontinence.¹² Compression may develop on the roots according to the localization of the retropulsed fragment separated from the vertebral body, but bilateral root plegia is a rare pathological condition.⁴

Autonomic dysreflexia (AD) on the other hand, is a condition of uncontrolled sympathetic response secondary to a precipitant that generally occurs in patients with injury to the spinal cord at levels of T6 and above.¹ It has been reported following traumatic spinal cord injury and is thought to occur with burst-type of vertebral fracture.³ The most prominent component of an AD episode is a dramatic rise in blood pressure.¹ The cases we reported both manifested with sudden hypertension, refractory to initial treatment. Burst fracture that can affect the

sympathetic chain along the thoracic spine may have occurred in these cases. While both cases had thoracic involvement in T7-8, lower than the caudal tip of the sympathetic chain, the symptoms of AD may still be explained by the surrounding tissue edema that may have affected the structures cephalad to the actual site of fracture.

Numerous theories have been proposed to explain AD.² Most of these involve an element of aberrant sympathetic nervous system overactivity. The basis for the AD response seems to be related to an exaggerated reaction of sympathetic pre-ganglionic neurons to afferent stimuli, however the part that spinal interneurons play has yet to be determined.^{14,15} A hypothesized "autonomic imbalance" cause splanchnic and peripheral vasoconstriction. Compensatory mechanisms to reduce the hypertension secondary to parasympathetic activity result above the level of the lesion, hence patients experience sweating, nasal congestion, and flushing.¹⁶

Osteoporotic vertebral fractures are often effectively treated by non-operative means.^{3,4,18} Cases with accompanying neurological deficit can be managed surgically using decompression and stabilization by fusion and instrumentation^{11,12}. Medical management has an important role in the initial treatment and in time, symptoms resolve, as seen in Case 2.

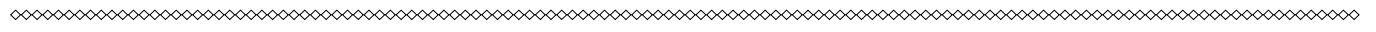
CONCLUSION

Vertebral fractures usually cause pain and are often not serious. In this report, we presented two cases with serious neurologic and cardiac complications. Occurrence of osteoporotic vertebral fractures in the thoracic spine may not be entirely benign and may lead to potentially life-threatening neurologic sequelae.

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