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A Case Study of Spontaneous Osteoporotic Vertebral Compression Fracture in an Elderly Man, Misdiagnosed in Initial Visits: Case Report

Alyaa Alouthmani[1], Ashraf ALakkad[2*], Alia Al Sakkal[3]

[1]Family Medicine Department, Al Mizher Health center, Dubai, UAE
[2]Internal Medicine Department, Madinat Zayed Hospital, AL Dhafra region, UAE
[3]Family Medicine Department, Al Barsha Health center, Dubai, UAE

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***Corresponding author:** Ashraf ALakkad
2 Internal Medicine Department, Madinat Zayed Hospital, AL Dhafra region, UAE

Abstract

***Background*:** Compression fractures are common among osteoporotic patients and can be a significant source of pain and disability. Patients who suffer a compression fracture are most often treated conservatively. ***Case Presentation*:** This report presents the case of an 80-year-old non-hypertensive, non-diabetic male who presented to the hospital with severe acute lower back pain for one week previous. There was no associated past history of trauma. Initially, the patient was misdiagnosed as a case of simple musculoskeletal lower back pain, for which he received treatment, but the pain intensified over the following two weeks, prompting consultation with a neurologist. A physical examination and lumber x ray indicated signs of lumbar spondylosis with degenerative disc disease, for which the patient was prescribed paracetamol and non-steroidal anti- inflammatory drugs. However, no improvement was noted. Later, after three weeks, an MRI investigation was conducted. The findings revealed multiple disc narrowing levels throughout the lumbar spine. Based on these investigations, treatment was initiated with pregabalin. Tramadol was discontinued and visible improvements in the patient's condition were noted within a week. Later, the patient sought consultation from an orthopedic specialist. The doctor recommended additional imaging scans. Magnetic resonance imaging with contrast and computerized tomography scans were performed. Based on these imaging scans, a diagnosis of lumbar spine scoliosis and osteoporotic compression fracture at D12 was made, prompting the initiation of osteoporosis treatment with zoledronic acid. ***Conclusions*:** Initial diagnosis and management of osteoporotic vertebral compression fractures are important in preventing continued pain and preserving the quality of life in older patients. This case highlights the need for comprehensive evaluation, including MRIs, to discriminate acute fractures from other pathologies, facilitating appropriate intervention and osteoporosis management.

Keywords: Vertebral Compression Fracture, Osteoporosis, lower back pain.

INTRODUCTION

Vertebral compression fractures (VCFs) are the most prevalent complication of osteoporosis, impacting over 700,000 Americans each year [1]. As individuals age, their fracture susceptibility rises, with four out of ten white women over 50 experiencing a hip, spine, or vertebral fracture during their lifetime [2]. While high-energy trauma is the leading cause, it's essential to recognize that other mechanisms can also lead to VCF[3]. A less obvious mechanism involves tetanic muscle spasms triggered by an electric shock. While most fractures in this scenario result from subsequent falls rather than the electric injury itself, it's crucial to be vigilant when assessing patients after a low-voltage (LV) shock [4].

VCFs can result in persistent pain, height reduction, limitations in daily activities, pneumonia, physical deformity, heightened risk of pressure ulcers, and emotional distress [5]. Symptoms of an acute VCF may include sudden back pain triggered by movement, sneezing, lifting, or coughing [6]. While physical exams often yield normal results, they may reveal tenderness and kyphosis along the midline of the spine [7]. Interestingly, over two-thirds of patients are asymptomatic and are diagnosed incidentally through routine plain radiography [8].

Acute VCFs can be managed with various analgesics such as non-steroidal anti-inflammatory drugs, acetaminophen, calcitonin, and narcotics [9]. Careful consideration of adverse medication effects is essential, especially in older patients [10]. Conservative treatment options include limited epidural injections, nerve root blocks, physical therapy, bracing, and bed rest [11]. Percutaneous vertebral augmentation, such as kyphoplasty and vertebroplasty, remains controversial but may be considered for patients who do not receive adequate pain relief with nonsurgical approaches or when persistent pain significantly impacts their quality of life [12]. Family physicians play an important part in preventing vertebral fractures by addressing risk factors and dealing with osteoporosis [13].

Moreover, degenerative scoliosis is frequently observed in older patients, especially in the lumbar spine, and is typically managed through surgery, including a multilevel laminectomy combined with instrumented posterior fusion [14]. In certain cases, an additional level of interbody fusion may be included [15]. Late complications may include hardware failure, pseudarthrosis, and adjacent segment degeneration [16]. This report presents the unique case of an 80-year-old patient who was diagnosed with a case of compression vertebral fracture and was successfully managed with conservative treatment.

Case Presentation

An eighty-year-old non-hypertensive, non-diabetic male presented to the hospital with a complaint of severe lower back pain. The patient reported that he had suffered with it for one week and that it worsened with movements of the spine. Specifically, the patient complained about experiencing the worst lower back pain during changing positions from sitting to standing and sitting to lying down.

Physical Examination

The patient's vital signs were observed during the examination, revealing a blood pressure of 105/70 mmHg, a pulse rate of 78 beats per minute, a tympanic temperature of 36°C (96.8°F), and a respiratory rate of 18 breaths per minute. Anthropometric measurements showed a height of 165 cm (64.9 inches) and a weight of 95kg. The patient's oxygen saturation (SpO2) level was recorded at 96% and the calculated body mass index (BMI) was 34.9 kg/m².

The patient's overall constitutional examination exhibited a normal appearance. The head, eyes, ears, nose, and throat (HENT) examination was normal, cardiovascular and pulmonary evaluations showed a normal pulse and normal pulmonary effort, and the skin and abdominal investigation were unremarkable.

The musculoskeletal examination showed tenderness at the central and right lower back, along with an abnormal range of motion. However, the leg raise test was normal on both sides. Neurologically, the patient did not display focal deficits or remarkable muscular weakness, which is noteworthy considering the patient's age. Sensation, gait, and deep tendon reflexes were normal. The patient's mental status was reported as alert and oriented to person, place, and time. The psychiatric evaluation indicated a normal mood, affect, and behavior. Overall, the systemic examination was considered unremarkable.

Review of Systems

Table 1

System	Positive Signs	Negative Signs
Constitutional		Chills, diaphoresis, fever
HENT		No congestion, no sore throat
Respiratory		No cough
Cardiovascular		Unremarkable findings
Genitourinary		Nocturia, no dysuria
Gastrointestinal		No abdominal pain, no anorexia, no changes in bowel habits, no nausea, no vomiting
Musculoskeletal	Severe lower back pain when changing the position of the spine, from sitting to standing and from sitting to lying down	No gait problems, no joint swelling, no myalgias, no neck pain
Skin		
Neurological		No vertigo, no weakness, no numbness, no headaches

Laboratory Results

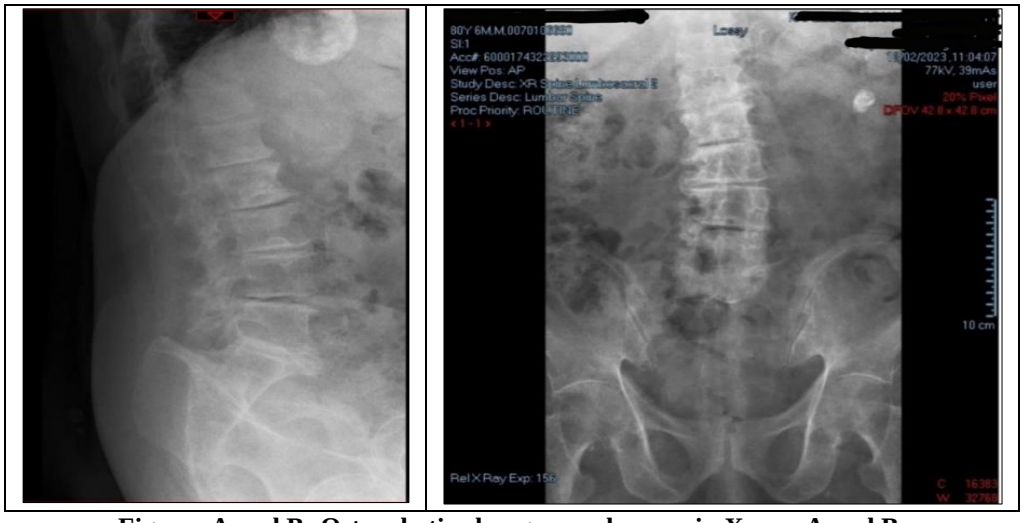
Laboratory results revealed a slightly low red blood cell (RBC) count of 4.39 million cells per microliter. The Red Cell Distribution Width (RDW) was elevated at 14.7%, suggesting variability in RBC size. A high Gamma-Glutamyl Transferase (GGT) level of 57 units per liter was observed. Additionally, the Erythrocyte Sedimentation Rate (ESR) was notably elevated at 38 millimeters per hour. The patient exhibited a low vitamin D level of 22.2 nanograms per milliliter. The rest of the findings were unremarkable.

Table 2

Monocytes Absolute	**0.32**	**0.20 - 1.00 10^3/uL**
LYMPHOCYTE %	35	20% to 40%
NEUTROPHIL%	58	40% to 60%
MONOCYTE %	05	2% to 10%
EOSINOPHIL %	02	1% to 6%
BASOPHIL %	00	0.1% to 1%
GAMMA GT	57 H	<55 U/L
SGOT	24	< 34 U/L
Alkaline Phosphatase	85	40 - 129 U/L
CALCIUM TOTAL	9.5	8.7 - 10.4 mg/dL
Bilirubin, Total	0.5	0 - 1.2 mg/dL
Total Protein	6.9	6.6 - 8.7 g/dL
SGPT(ALT)	21	< 49 U/L
GLOBULIN	2.1	2.0 - 3.8 gm/dL
Albumin	4.8	3.4 - 4.8 g/dL
TSH	1.79	0.27 - 4.2 uIU/mL
Creatinine	0.63	0.70 - 1.20 mg/dL
Urea	34.2	10-50 mg/dl
Uric acid	6.2	3.7-7.7 mg/dl
ESR	38 H	2 - 28 mm/1hr
C-Reactive Protein	<0.5	<5.0 mg/L
VITAMIN D (25-OH)	22.2 L	Deficiency: <20 Insufficiency: 20 - 29 Sufficiency: 30 - 100
Vitamin B-12	335	197 - 771 pg/mL
HbA1C	5.0	<5.7 %
Fasting Glucose	97	<126 mg/dL

Imaging Results

Upon consultation with the neurologist, a lumbar X-ray was ordered. Radiographic examination of the lumbo sacral spine revealed osteophytic changes involving the L1-L5 vertebral bodies, along with a reduction in the intervertebral disc spacer, indicating degenerative disc disease from L1-L2 to L5-S1 levels. Normal lumbar lordosis was maintained, while calcified nodular lesions were noted in the left renal region and the region of the thoracic vertebrae. Additionally, vascular calcification of the abdominal aorta was observed. Overall, findings revealed evidence of lumbar spondylosis with degenerative disc disease at the L1-L2 and L5-S1 levels.



Figures A and B: Osteophytic changes can be seen in X rays A and B.

Initially, in the first week, the patient sought medical attention from an orthopedic specialist, who diagnosed the condition as muscular lower back pain and prescribed simple, non-opioid analgesics such as paracetamol and non-steroidal anti-inflammatory drugs. However, despite treatment, the pain progressively worsened over the following two weeks, prompting the patient to seek a second opinion from a neurologist.

In response to the worsening pain during the third week, the neurologist introduced opioid analgesics (Tramadol) alongside the ongoing non-opioid analgesics and scheduled a follow-up appointment.

With no improvement observed after a week and the pain exacerbating upon any movement of the spine, further investigation was warranted. An MRI of the lumbar region was conducted.

The MRI findings revealed Lumbar spondylo-degenerative changes.-Modic type I marrow edema of D12 vertebra. Central compression of above vertebra with multiple levels of disc narrowing throughout the lumbar spine. At the L2/L3 level, there was a symmetrical posterior disc bulge causing severe indentation of the ventral aspect of the thecal sac, along with a narrowing of bilateral neural exit pathways and impingement of bilateral traversing exit nerve roots. Similarly, at the L3-L4 level, there was a posterior disc bulge leading to effacement of ventral subarachnoid cerebrospinal fluid (CSF) with a preference for attenuation of the right neural exit pathway, as well as touching of the right traversing exit nerve roots. Additionally, at the L4-L5 and L5-S1 levels, symmetrical posterior disc bulges caused a mild indentation of the ventral subarachnoid CSF with preferential left lateral recess attenuation, and encroachment upon the left traversing exit nerve roots at these levels. In light of these findings, comprehensive laboratory investigations and MRI scans of the thoracic and lumbar regions with contrast were ordered to rule out secondary causes of vertebral fracture and malignancy.

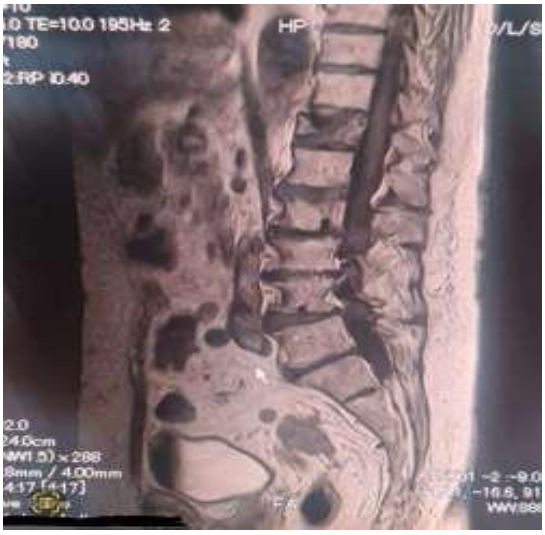


Figure C: The MRI findings revealed multiple levels of disc narrowing with a compression fracture at D12 with features favoring a benign osteoporotic compression fracture showed in MRI with contrast

In light of these findings, in the fourth week, the neurologist initiated treatment with analgesic (celebrex+ paracetamol and pregabalin 75 mg daily, discontinued Tramadol tablets, and advised the patient to wear a Taylor brace for eight weeks. Notably, the patient demonstrated visible improvements within a week of commencing conservative treatment.

Figure D: Patient wearing a Taylor brace

In the seventh week, the patient sought consultation with an orthopedic specialist. The orthopedic doctor ordered the patient to undergo a DEXA scan, an MRI with contrast, and a local CT scan to rule out secondary causes for the fracture. These revealed several significant findings.

Firstly, there was evidence of scoliosis of the lumbar spine, tilting to the left side. Lumbar spondylotic changes, characterized by a loss of normal signal intensity and height at multiple intervertebral discs (IVD), were observed, along with vertebral osteophytes.

Additionally, diffuse disc-osteophyte bulges were noted at multiple levels, resulting in the narrowing of lateral neural recesses, exit neural foramina, and the spinal canal. Hypertrophy of apophyseal joints with thickening of the ligamentum flavum was most prominent at the L3-4 level, with signs of indentation of the theca on the right side. Moreover, compression of the D12 vertebral body was observed, along with diffuse changes in signal intensity, suggestive of bone marrow edematous changes, accompanied by focal fluid signal adjacent to the superior end plate.

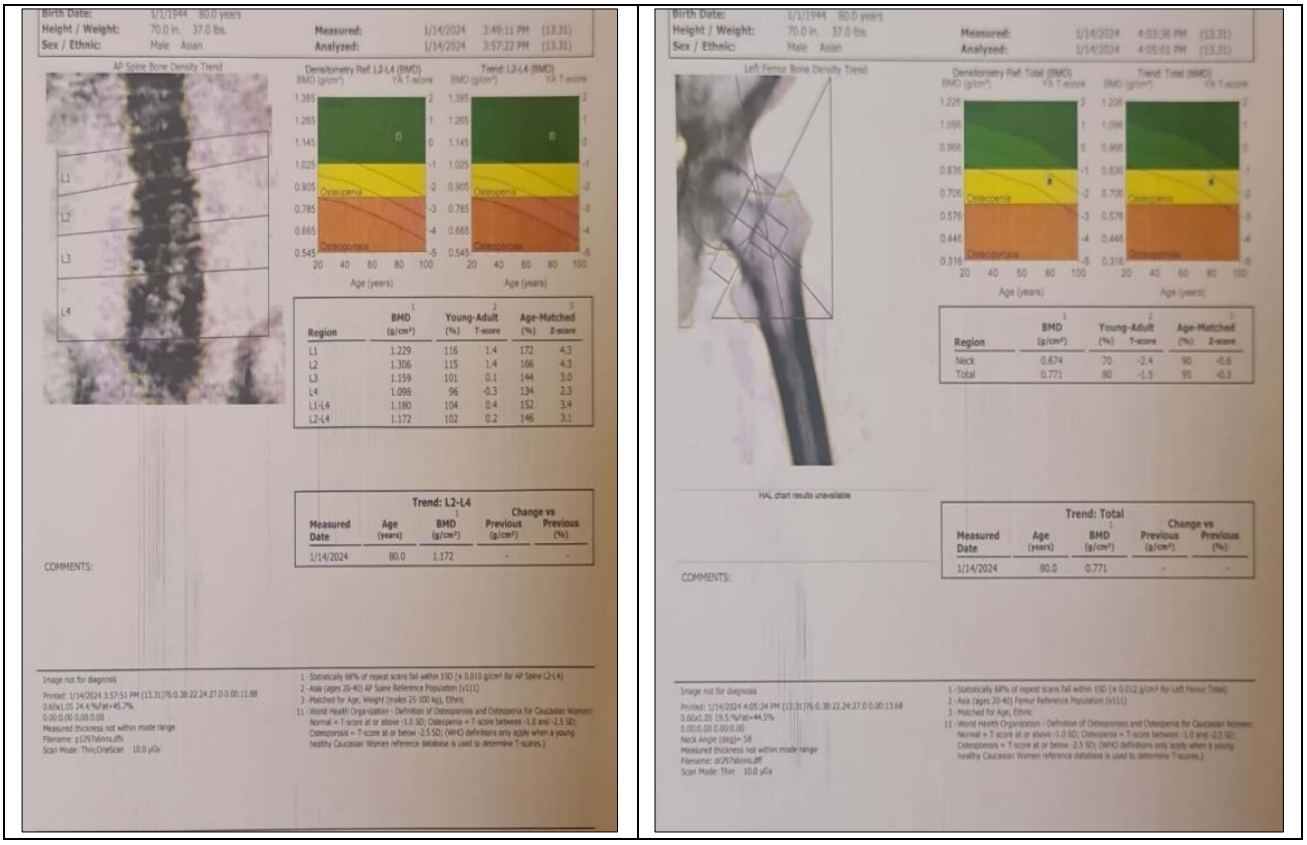


Figure E: Dexa scan revealed a T-score of -2.4 at the femoral neck.

The complementary CT scan showed evidence of a compression fracture at D12 with features favoring a benign osteoporotic compression fracture over secondary changes, as evidenced by the presence of vacuum phenomena.

Additionally, a Dexa scan revealed a T-score of -2.4 at the femoral neck, prompting the initiation of osteoporosis treatment per established guidelines.

During the eighth week, the patient was prescribed Aclasta (zoledronic acid) as treatment for osteoporosis, which is to be taken annually.

DISCUSSION

This case of an 80-year-old male patient presents several key clinical and diagnostic challenges commonly encountered in the management of acute low back pain in the elderly.

Vertebral compression fractures are prevalent among older adults due to the gradual decline in spine bone mineral density as individuals age. By the time they reach their 80s, people have typically lost nearly half of their axial bone mass [17]. In our case, the patient was also 80 years old and presented with VLC, even in the absence of trauma history. This occurrence likely stemmed from a gradual decline in spine bone mineral density due to age-related factors.

Over two-thirds of individuals with vertebral compression fractures are asymptomatic and are typically diagnosed incidentally [18]. Symptomatic patients often present with back pain, with fractures

typically detected on radiographs. These are most frequently between the T8 and L4 vertebrae [18]. Those experiencing an acute fracture may report sudden onset pain triggered by changes in position, coughing, sneezing, or lifting [19]. Although physical examination findings are frequently unremarkable, they may reveal kyphosis and tenderness along the midline of the spine [19]. Our patient exhibited severe back pain aggravated by spinal movements, particularly when transitioning from a seated to standing position, as well as from sitting to lying down. Additionally, tenderness was noted at the mid-spine region, correlating with the fracture site at T12.

Magnetic resonance imaging plays an important part role in distinguishing between benign and malignant fractures and evaluating fracture timing, as new fractures often show symptoms of edema [14, 15]. In these cases, both MRI and computed tomography are important for recognizing supposed retropulsion, fractures encompassing the posterior column, and involvement of the spinal cord [17]. Additionally, in patients who do not respond well to conservative treatment or experience worsening of their symptoms, magnetic resonance imaging or computerized tomography should be considered [17]. Dual-energy x-ray absorptiometry can also be performed following the diagnosis of a VCF to investigate osteoporosis and determine disease severity [17]. In our case, the patient underwent a thorough diagnostic evaluation, including DEXA, MRI, and CT scans.

Conservative treatment is the best option in these cases. This can lead to prolonged bed rest, reduced activity, significant pain, and related comorbidities. Recovery is possible for these patients with conservative measures like physical therapy and the administration of analgesics. Additionally, they may need bracing for eight to twelve weeks. Our patient was given analgesics and advised to wear a brace for eight weeks [1].

CONCLUSION

Initial diagnosis and management of osteoporotic vertebral compression fractures are important in preventing continued pain and conserving quality of life in older patients. This case highlights the need for comprehensive evaluation, including MRI scans, to discriminate acute fractures from other pathologies, facilitating appropriate intervention and osteoporosis management.

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