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Case Report

MISSING AN OSTEOPOROTIC VERTEBRAL FRACTURE

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ABSTRACT
This case illustrates an 80-year-old lady who presented with acute low back pain and finally succumbed from complications of osteoporotic vertebral fracture. Among the contributing factors are delayed diagnosis and a lack of continuity of care. It reminds the practitioners of the importance to look out for red flag symptoms and to have a high index of suspicion for vertebral fracture in high risk patients presenting with low back pain.
Keywords: Low back pain, osteoporotic vertebral fracture
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INTRODUCTION

Low back pain is a common symptom encountered in primary care practice. Acute low back pain is the fifth most common reason for all physician office visits.[1] Most of the causes are benign and can be managed conservatively. However, when assessing a patient with low back pain, a comprehensive history taking and physical examination are needed so that important and life threatening causes are not missed, and appropriate interventions can be taken to prevent morbidity and mortality. The following case illustrated the painful journey of a patient who suffered acute low back pain resulting from a late diagnosis of vertebral compression fracture.

CASE SUMMARY

Madam KAS, an 80-year-old lady, presented with acute back pain following a fall in the bathroom. Her family members brought her to see a general practitioner (GP) on the same day and was informed that the pain was musculoskeletal in nature. She was treated with analgesic. Her pain did not improve and 4 days later, she was brought to the Accident and Emergency Unit of a teaching hospital for further assessment. Blood investigations were done and she was informed her results were normal and she was discharged home. The next day she was brought to the GP again, who gave her more analgesic. Throughout these periods, the patient was unable to move after the incidence due to pain.

Two weeks later, Madam KAS was brought to the outpatient clinic at another teaching hospital with a complaint of generalized lethargy and bilateral ankle oedema. Her chest radiography and ECG were normal and she was treated symptomatically with Frusemide and Slow K. A month later Madam KAS returned to the clinic with a complaint of passing out loose stools and was unable to control her bladder. She was then found to be hyponatremic, hypokalemic and hypochloraemic. At that time, she was given oral rehydration salts and frusemide was stopped. She was then sent home. Two months after her fall, she was again brought to the outpatient clinic; this time she had bedsores over pressure areas.

A review of her past history revealed she had type 2 diabetes mellitus for 30 years and had blindness in the right eye since 1988 after a cataract operation. Prior to the fall, she could walk slowly with support and was able to feed, dress, bathe and go to toilet herself.

She was brought in on a wheelchair. Physical examination revealed a thin and pale lady. There was no ankle oedema, peripheral or central cyanosis. Her blood pressure was 130/80 mmHg and her pulse rate was 92 beats per minute with regular rhythm. There was corneal opacity in her right eye and presence of cataract in her left eye. She was totally blind in the right eye and the visual acuity in the left eye was down to finger counting. Examinations of her cardiovascular, respiratory and abdomen were normal.

Neurological examination findings showed that in the lower limbs, the tone was reduced with power of grade 0 bilaterally. Reflexes were absent bilaterally, and plantar response was down going. In the upper limbs, the tone was normal with power of grade 4 bilaterally while the reflexes were diminished on both sides. Pin prick sensation in the legs was inconsistent and her anal tone was lax.

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There were multiple pressure sores: (1) A sore over the sacral region measured 15 x 10 cm, with necrotic skin; (2) At the greater trochanteric regions there were sores measuring 2 x 3 cm with skin abrasions bilaterally; (3) Over the heel regions the sores measured 2 x 2 cm with skin abrasions bilaterally.

The glucometer reading was 19.8 mmol/L. Her haemoglobin count was 92.0 g/L, and total white count was $16.6x10^9$/L. Lumbosacral X-ray (antero-posterior and lateral view) showed compressed fracture of L5 and osteopenia of the lumbosacral spine.

Madam KAS was admitted to the orthopaedic ward. She was co-managed by the spine team, endocrinologist, and the rehabilitation physician for wound and pressure sores management. She was referred to dietician, and nasogastric tube feeding was commenced as she was not tolerating orally. Further, MRI scan of the thoracolumbar spine was done from T5 to S3 which showed fractures of T12, L1 and L5. Retropulsion of bone was seen causing spinal canal stenosis. There was distal cord contusion. There was no epidural or paravertebral mass seen and these fractures were reported to be probably trauma related. Further blood investigations were performed in the ward to look for secondary causes for vertebral fractures i.e. thyroid function test, serum cortisol and protein electrophoresis. The results were all normal. A diagnosis of paraplegia secondary to osteoporotic vertebral fracture complicated by multiple pressure sores with underlying uncontrolled type 2 diabetes mellitus was made. Wound debridement was subsequently done in the ward. After staying in the ward for 6 weeks, Madam KAS was discharged home with advice given on nursing care that included NG tube feeding, bowel training, wound dressing, stretching exercise and frequent turning in bed. Her medications on discharge included S/C Actrapid 10 unit tds, S/C Insulatard 10 units at night, multivitamins, tramadol 50mg tds and alendronate 70mg weekly.

Two weeks after discharge, Madam KAS presented to the accident and emergency unit with complaints of pus discharging from the surrounding area of the sacral pressure sore and she was readmitted. Her blood culture grew methicillin resistant coagulase-negative Staphylococcus. Her condition then deteriorated with multiple infections while she was in the ward, including mixed organism from the pressure sores (*Acinetobacter baumanii*, *Pseudomonas aeruginosa* and *Staphylococcus aureus*), urinary tract infection (*Pseudomonas aeruginosa*), and nosocomial chest infection. She was discharged 4 weeks later when her vital signs were stable although she was still weak and fully dependent. Two weeks later, she passed away at home.

DISCUSSION

Vertebral compression fracture is common in the older adults. It is usually caused by osteoporosis. The prevalence of this condition increases with advancing age, reaching 40% in women aged 80 years old.[2] Serious fracture can cause significant pain and neurological deficit with resulting increase in morbidity and mortality. Osteoporotic vertebral fracture is often missed. Only one third of vertebral fractures are actually diagnosed,[3] because many patients and families consider back pain as “rheumatism”, or symptoms of aging.

Vertebral fracture may present with acute episode of severe backache.[4-7] In patients with severe osteoporosis, simple trauma such as stepping out of bathtub, vigorous sneezing or lifting a trivial object[7] can cause vertebral fracture. A study by Patel *et al* looking at the clinical profile of acute vertebral compression fractures in osteoporotic patients reported that up to 30 percent of the patients first noticed pain on attempting to get out of bed in the morning. However, it was unclear whether the fracture resulted from the trivial effort of getting out of bed or it occurred whilst the patient was at complete rest or sleeping.[6] Fractures resulting from this type of trauma are almost always stable. It is seldom associated with neurologic loss but can be associated with severe and prolonged backache.[5] The pain persisted over the affected area or could radiate to the flanks and anteriorly, but radiation to the leg was uncommon.[4,6] In patients of moderate osteoporosis, more force or trauma is required to cause a fracture, such as falling off a chair, tripping, or attempting to lift a heavy object.[7] Alternatively, an asymptomatic thoracic wedge or lumbar compression fracture of the spine may be noted on a radiographic examination performed for an unrelated purpose. The macroscopic fracture observed on radiography represents integration over time of a series of small, individually insignificant microfractures.[4]

For patients with vertebral fractures presented with acute back pain, only 43% of the cases had the diagnosis made at first visit to the doctor.[6] The lack of a traumatic precipitating event often leads the patients and doctors to suspect muscle strain.[6] Therefore, compression fracture should be suspected in any patient over the age of 50 years with acute onset of sudden low back pain. Careful history and examinations need to be carried out for these patients. Although secondary causes are rare, it should not be missed and red flags symptoms should alert the doctor of more serious underlying causes. Table 1 illustrates the red flags symptoms and signs.[8,9]

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Table 1: Red flags for acute low back pain[8,9]

History
Age greater than 50 years
Fever
Weight loss
Pain that is increased or unrelieved by rest
Significant trauma related to age*
Bladder and bowel incontinence
Cancer, immunosuppresion, prolonged used of steroids, intravenous drug use
Physical signs
Fever
Vertebral tenderness
Motor weakness in lower extremities
Saddle anesthesia
Loss of anal sphincter tone

*e.g. fall from a height or motor vehicle accident in a young patient, minor fall or heavy lifting in a potentially osteoporotic or older patient or a person with possible osteoporosis

Plain radiography is rarely useful and is not recommended for initial evaluation of patients with acute onset of low back pain without red flags symptoms or signs. However, if the red flags symptoms and signs are present, anteroposterior and lateral radiographs are then indicated. The criteria for radiographic investigation is listed in Table 2.[8,9] Adequate X-rays are important and should include several vertebra above and below the area of pain because the site of pain and pathology may be distant and multiple fractures are not always adjacent.[6] Radiographically, a decrease in vertebral height of 20 percent or more, or a decrease of at least 4mm compare with baseline height, is considered positive for compression fracture.[7] Osteoporotic vertebral fracture can occur anywhere from the occiput to the sacrum, however certain vertebras, namely T8, T12, L1 and L4 are more prone to fracture.[6] Some studies suggest that a significant proportion of fractures may be missed initially because radiographic evidence of vertebral deformation may not appear until weeks or months after the initial symptoms.[10] A radionuclide bone scan is useful in this case to pick up fresh fractures.[6,7] In the further assessment and investigations of fracture in the hospital, computed tomography (CT) and magnetic resonance imaging (MRI) are used to rule out other causes of back pain e.g. a complex fracture, spinal canal narrowing, spinal cord compression, and malignancy as the cause for pathologic fracture.[7]

Table 2: Indications for radiography in acute low back pain[8,9]

Age >50 years
Significant trauma
Neurological deficits
Temperature greater than 38ºC
Unexplained weight loss
Medical history: cancer, corticosteroid use, drugs or alcohol abuse
Ankylosing spondylitis suspected
Recent visit (within 1 month) for same problem and no improvement

In this case, doctors had missed the diagnosis despite the presence of red flag symptoms, until Madam KAS presented with obvious neurological deficits and pressure sores two months after the fracture, the sixth visit to the doctors after her fall. A failure of resolution of symptoms coupled with presence of red flag symptoms and signs should alert doctors of serious cause that was unfortunately been missed not once but several times in this patient. Madam KAS has also seen several doctors for her illness. This doctor shopping behaviour and lack of continuity of care might contribute to the delay in diagnosis. Should madam KAS be followed up by the same doctor, one wondered her problem could have been detected and managed earlier. In this case, it could be possible that a combination of factors had contributed to her fracture such as the fall, osteoporosis, her vision, and poor mobility; while the doctors' missed diagnosis and the lack of continuity care could account for the delay in diagnosis. In addition, the lack of continuity in this patient's care could have led to the poor care she received. Thus, eliciting a good history and examination as well as be aware of the red flag symptoms and signs are of utmost importance in assessing patient in primary care to identify problem early to reduce morbidity and mortality. The provision of continuous care will also facilitate early problem identification for better quality of care.

Osteoporosis and falls are two important risk factors of bone fracture in the elderly.[11,12] Treating osteoporosis and preventing fall can reduce the risk of fracture. However, there are still patients with undiagnosed osteoporosis presenting with fragility fracture that were missed by orthopaedic surgeons, emergency room physicians and primary care providers.[12] Therefore, it is important to detect and treat osteoporosis in high risk patients and to provide strategies to prevent falls in the elderly.

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CONCLUSIONS

Acute low back pain is a common presentation in primary care. Although majority of patients follows a benign course, red-flags symptoms and signs should alert the physician to look for more serious causes e.g. vertebral fracture in an elderly. A high index of suspicion can prevent the patient from sustaining further morbidity and mortality. Continuity of care is important in the management of patient for early identification of high risk patients for fracture and subsequent treatment.

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Canadian study verified the "cardiac equivalent" of diabetes

Dagenais GR, St Pierre A, Gilbert P, *et al*. Comparison of prognosis for men with type 2 diabetes mellitus and men with cardiovascular disease. *CMAJ*. 2009;180(1):40-7

4376 men aged 35-64 years from Quebec in Canada. During the 24-year follow-up, new diabetes without cardiovascular disease (myocardial infarction, unstable angina or stroke) was documented in 137 men. A first cardiovascular event without diabetes was documented in 527 men. When compared to controls without diabetes or cardiovascular disease,

- The relative risk of cardiovascular mortality in those with diabetes was 3.11 (95%CI 1.96-4.92)
- The relative risk of cardiovascular mortality in those with cardiovascular disease was 4.46 (95%CI 3.15-6.30)

When compared to those with diabetes (within the first five year after diagnosis), men with isolated cardiovascular disease has higher cardiovascular mortality (RR 2.03, 95%CI 1.01-4.08)