Case Report

Pyogenic vertebral osteomyelitis presenting as single spinal compression fracture: a case report and review of the literature

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Study design: A case report of pyogenic vertebral osteomyelitis (PVO) presenting as single collapsed vertebral body without narrowing of the intervertebral disc space, and review of the literature.

Objective: To describe an unusual case of PVO showing atypical radiological change and call attention to this condition so that others may avoid this diagnostic pitfall.

Setting: Japan.

Methods: A 62-year-old diabetic woman with suspected T12 pathological fracture of malignant spinal tumor and neurological involvement received urgent anterior decompression and spinal reconstruction without biopsy.

Results: Anterior decompression and spinal reconstruction was performed, but histological examination of the specimen after surgery unexpectedly revealed PVO. The surgery was followed by therapy with antibiotics for 7 months. A follow-up radiograph at 5 years after surgery revealed that solid consolidation has been achieved.

Conclusions: Diagnosis of PVO presenting with single spinal compression fracture is very difficult. Although the finding of the high signal intensity in the lesion equal to or higher than that of the cerebrospinal fluid on T2-weighted MR image seemed to be the most reliable diagnostic modality retrospectively, diagnosis of this type of PVO is impossible without histology. A needle biopsy before surgery is strongly recommended.

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Introduction

It is well known that pyogenic vertebral osteomyelitis (PVO) manifests as destructive abnormalities of two adjacent vertebral bodies with narrowing of the intervening disc spaces.^{1–4} This narrowing of the intervertebral space seems to be very important in differential diagnosis from compression fracture due to spinal tumor and osteoporosis. However, it is not well known that vertebral infection may present as radiological collapse of a single vertebra without narrowing of the intervertebral disc space and destruction of the endplates. Therefore, this type of PVO is often misdiagnosed and mistreated. We wish to call attention to this condition so that others may avoid this diagnostic pitfall.

Case report

A 62-year-old diabetic woman was transferred lying down on a stretcher for back pain associated with motor weakness of both legs and urinary retention that had lasted 1 month. About 2 months before her admission, she had been admitted to hospital with suspected respiratory tract infection and had received antibiotic therapy. She was not suffering from lower back pain at that time. A month later, she experienced acute lower back pain, motor weakness, and numbness of both legs without any trauma and could not walk. Her previous doctor suspected a pathological compression fracture with neoplasm or osteoporosis. Although the lower back pain and motor weakness were somewhat resolved by bed rest, she still could not sit up because of the back pain. On admission to our hospital, she had no fever but had tenderness on the spinous process in the thoracolumbar region. Neurologic examination revealed a moderate motor weakness of the hip flexors and knee extensors, mild hypoesthesia over both legs below the L1 neurological level, loss

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of bilateral knee and ankle jerks, reduction of anal sphincter control, and 300 ml of residual urine.

The first spinal X-ray in the lateral decubitus position showed a collapse of the T12 vertebral body without narrowing of the adjacent intervertebral disc spaces (Figure 1A). The spinal X-ray in extension showed that the collapsed and wedged vertebral body was corrected and its anterior cortex was unclear with normal pedicles, upper and lower endplates and also normal disc height of adjacent intervertebral discs (Figure 1B). CT of T12 revealed diffuse bone destruction, retroprotrusion of bony fragments into the spinal canal, and slightly diffuse swelling of the soft tissue around the vertebral body (Figure 2). T1-weighted MR image at the extended position showed decreased signal intensity in most of the T12 vertebral body. It also showed retroprotrusion of the inferior part of the vertebral body into the spinal canal with compression of spinal cord, anterior paravertebral extension from T12 to the superior part of L1, and the normal adjacent intervertebral discs without destruc-

tion of the endplates (Figure 3A). T2-weighted MR image showed intensity as high as in cerebrospinal fluid in the entire T12 vertebral body, the posterior bony element of T12 and the small prevertebral extension. The adjacent intervertebral discs and endplates of the T12 vertebra were intact (Figure 3B). Technetium-99m methylene diphosphonate ($^{99m}\text{Tc-MDP}$) bone scintigram showed increased uptake at T12. Laboratory studies gave the following measurements: Erythrocyte sedimentation rate 105 mm/h; hemoglobin 11.3 g/dl; white blood cell count 9900/ml without a left shift; serum albumin 3.4 g/dl and globulin 4.1 g/dl. Protein electrophoresis did not demonstrate a monoclonal spike. Alkaline phosphatase was 7.5 KAU. The tuberculin skin test and controls were nonreactive. The chest X-ray film was normal. Enterococcus and candida was cultured from midstream specimens of catheterized urine. From these findings in diagnostic imagings and laboratory data, neoplasm was suspected. The origin of the neoplasm was looked for mainly in the lung, GI

a

b

Figure 1 (A) Spinal X-ray in the flexed lateral decubitus position in bed shows a collapse of the T12 vertebral body without narrowing of the adjacent intervertebral disk spaces. (B) The lateral roentgenogram on admission shows a diffuse osteolytic lesion in the T12 vertebral body, decreased vertebral height and unclear anterior cortex

Figure 2 CT slice through the T12 vertebral body revealed diffuse bone destruction, the retroprotrusion of bony fragments into the spinal canal, and slightly diffuse swelling of the prevertebral soft-tissue around the vertebral body

tract, kidney, urinary tract, liver, thyroid gland, but no primary lesion was detected.

On account of sudden progression of paraparesis, surgical intervention was performed through a left-sided extrapleural and retroperitoneal approach. On inspection, there was no psoas abscess, but some edema and mild adhesion around the collapsed T12 vertebral body, which had been almost completely replaced by fragile soft tissue mass containing several small bone fragments. The residual bone around the peripheral cortex and bony endplates was like an eggshell. The adjacent intervertebral discs were intact. Anterior decompression by total corpectomy of the involved vertebra and anterior spinal reconstruction from T11 to L1 was performed with a ceramic vertebral spacer, bone cement and anterior spinal instruments, because intraoperative macroscopic finding contradicted metastatic bone tumor. The histology of

Figure 3 (A) Sagittal T1-weighted MR image shows decreased signal intensity in most of the mildly collapsed T12 vertebral body and in the anterior superior part of the L1 vertebral body. It also shows retroprotrusion of the inferior part of the T12 vertebral body into the spinal canal with compression of spinal cord, anterior para-vertebral extension from T12 to the superior part of L1, and a slightly increased disc height of the adjacent intervertebral discs without destruction of the endplates. (B) Sagittal T2-weighted MR image shows signal intensity as high as in CSF in the entire vertebral body and the posterior element of T12, and small prevertebral extension. It also shows the adjacent intervertebral discs are a little bulged, but the end-plates of T12 are almost intact

the surgical specimens disclosed PVO after surgery (Figure 4) and culture of the bone specimen revealed *Escherichia coli*, which was susceptible to aspoxicillin, cefuzonam natrium, and imipenem cilastatin sodium.

Aspoxicillin and cefuzonam natrium were administered for 4 weeks after surgery. The patient could walk without lower back pain with a cane and thoracolumbar-sacral orthotic polypropylene jacket. She was discharged and given therapy with oral antibiotics for another 6 months. Her motor weakness, sensory change and bladder function markedly recovered. One year after the surgery, muscle strength in both legs was normal and she could walk without a cane or any form of back support. Sphincter control was intact. Radiographs 10 years after surgery demonstrated successful consolidation without loosening of vertebral screws and bone cement (Figure 5).

Discussion

Radiological changes of PVO on plain X-ray films are not apparent until several weeks or months after the onset of clinical symptoms.³ The destructive changes in the body usually begin as a lytic area in the anterior aspect of the body adjacent to the disc and diffusely in the endplate. The earliest and most constant radiographic finding of PVO is narrowing of the disc space with end-plate erosion,¹ which was found in 74% of 86 patients.⁴ At the fully developed stage, the most common finding consists of destructive lesions of the surfaces of the two adjacent vertebral bodies with narrowing of the intervertebral disc spaces.¹ With these findings, a diagnosis of vertebral osteomyelitis (VO) including PVO and TVO (tuberculous VO) is not difficult. However, if VO patients without fever or with fever from another origin present with a single collapsed vertebral body with normal disc space radiologically, physicians may misdiagnose the VO as compression fracture in osteoporosis or neoplasm. We couldn't suspect PVO or TVO in the present case. Although fever or increase in WBC count with a left

shift seems to be important for diagnosis of PVO, our patient had neither. According to Sapico and Montgomerie (1979),⁴ it is not rare for PVO to present without fever or increase in WBC count. They reported that fever was not present in 48% of 256 patients and WBC count was not increased in 58% of the cases.

According to McHenry (1988),⁵ this type of PVO was not rare and accounted for 13% of 45 patients with VO, 2.4% of 249 hospitalized patients with compression fracture in osteoporosis, and 1.6% of 372 inpatients with compression fracture. However, there have been few reports of adult PVO with a collapse of the vertebral body and normal disc space (Table 1).⁵⁻¹² According to analysis of 13 cases reported in the literature and our case, this type of adult PVO is predominant in elderly and diabetic patients with osteoporosis; mean age 57 years (40 to 76), diabetes mellitus in five cases (36%), and osteoporosis in seven cases (50%). The most commonly affected spinal level was T12 (36%) followed by the lumbar spine (L1, L2 and L3 in 2

Figure 4 Histology of the specimen at surgery shows a dead bone and inflammatory cell (neutrophils, lymphocytes, histiocytes, and plasma cell) infiltration. H&E, ×400

Figure 5 Plain X-ray 10 years after surgery. X-ray film shows anterior spinal reconstruction performed with a ceramic vertebral spacer, bone cement and anterior instruments, and successful consolidation without loosening of screws and bone cement

Table 1 Reported PVO with a single collapsed vertebral body

Cases	Age/sex	Lesions	Causative organisms	Trauma* ¹	Plain X-ray of the spine* ²	DM* ³	Osteop* ⁴	UT infect* ⁵	Sepsis* ⁶	Steroid therapy	Others
1 ⁵	76/F	L1	<i>Escherichia coli</i>	M	L1 comp Fx	+	+	S	S	—	cardiac transplant
2 ⁵	45/M	L3	<i>Nocardia asteroides</i>	—	L3 comp Fx	—	+	—	—	+	polynuritis, renal calculus
3 ⁵	48/F	L4	<i>Proteus mirabilis</i>	—	L4 comp Fx	—	+	P	P	+	alcoholism, hepatic cirrhosis
4 ⁵	54/F	T12	<i>Escherichia coli</i>	M	T12 comp Fx	—	+	P	P	—	subtotal colectomy, pancreas ca.
5 ⁵	70/F	T12	<i>Klebsiella pneumoniae</i>	m	T12 comp Fx	—	+	S	—	—	peritonitis, pneumonia
6 ⁵	62/F	T12	<i>Staphylococcus aureus</i>	—	T12 comp Fx	+	+	—	—	—	chronic lymph. leukemia
7 ⁸	62/M	L1	<i>Pseudomonas aeruginosa</i>	M	L1 comp Fx	—	—	—	—	+	tooth decay
8 ⁷	60/M	L2	<i>Streptococcus?</i>	M	L2 comp Fx	—	—	—	—	—	diabetic gangrene
9 ¹¹	52/M	L3	<i>Staphylococcus aureus</i>	M	L3 comp Fx	+	—	S	—	—	—
10 ⁶	60/M	T10	<i>Staphylococcus aureus</i>	—	T10 comp Fx	+	—	—	—	—	thoractomy tube
11 ⁹	57/M	T12	<i>Staphylococcus aureus</i>	M	T12 comp Fx	—	—	S	S	—	paraparesis, residual urine
12 ¹⁰	40/M	L2	<i>Serratia marcescens</i>	M	L2 burst Fx	—	—	S	S	—	multiple rib fracture, flail chest
13 ⁴	55/M	T5	<i>Streptococcus pneumoniae</i>	M	T5 comp Fx	—	—	—	—	—	pneumonia?
14 ^{*7}	62/F	T12	<i>Escherichia coli</i>	—	T12 comp Fx	+	+	S	—	—	—

*1: M: major trauma such as a traffic accident, fall from a high place or fall in the bathroom, m: minor trauma such as lifting a bowling ball, —: no trauma; *2: comp Fx: compression fracture; *3: DM: diabetes mellitus; *4: Osteop: osteoporosis; *5: UT infect: urinary tract infection, S: UT infect at the same time as onset of PVO or back pain, P: UT infection prior to the onset of PVO or back pain, —: no UT infection. *6: S: sepsis at the same time as onset of PVO or back pain, P: sepsis prior to the onset of PVO or back pain, —: no sepsis; *7: our case

cases respectively). The causative organisms were staphylococcus aureus in four cases (29%) and *Escherichia coli* in three cases (21%). Eight subjects (57%) had recently had major spinal trauma prior to the onset of disease, one had experienced minor spinal trauma such as acute back pain after lifting a bowling ball. Five cases had no trauma (36%). Urinary tract infection occurred prior to or at the same time as the onset of disease or back pain in eight cases (57%). Sepsis prior to or at the same time as the pain was found in five cases (36%). Three patients were under adrenal corticosteroid therapy for transplantation, polyneuritis or lymphocytic leukemia. Almost all the patients had an immuno-depressive status, focus of infection, or surgical wound. Although this type of PVO has such features, we could not find any basis on which to differentiate this from other pathological conditions.

Among modern diagnostic imaging modalities, CT scan and MR images seem to be useful for distinguishing this type of VO from other pathological conditions.^{2,13-16} Van Lom KJ *et al*¹⁶ reported that the prevertebral soft tissue extension in VO seen on CT usually completely surrounded the spine anteriorly in contrast to neoplasm characterized by no or only partial paravertebral soft tissue extension, and also neoplasms are more likely to involve the posterior elements than infection. But there was no such obvious soft tissue involvement of the posterior elements in our case. On the other hand, the characteristic finding in MR images was that the highest signal intensity of the infectious lesions on T2-weighted image was equal to or higher than that of the cerebrospinal fluid (CSF), in contrast to the less intense malignant lesions.¹³ In our case, this finding was a diagnostic clue except for histological findings of the surgical specimen. Although not available in our case, gadolinium-enhanced MR images seem to be valuable for differentiation, showing peripheral enhancement of the abscess or exudative fluid with lack of uptake centrally.¹⁵ There is another pathological condition to distinguish from the MR images of this type of PVO. It is post-traumatic avascular necrosis of the vertebral body, of which the highest signal intensity on the T2 weighted image in the extended position also shows an equal or higher signal intensity of the CSF, and its gadolinium-enhanced MR image shows marginal enhancement of the necrotic lesion changed to vacuum cleft.

In this case, a non-infectious pathologic process was suspected, but it proved to be infection. Not infrequently, the opposite situation occurs. This case is an excellent illustration of why we should always culture what we biopsy, and biopsy what we culture.

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