Brown Tumor of Lumbar Spine in Chronic Renal Failure: a Case Report

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Abstract- Brown tumors may occur secondary to hyperparathyroidism in patients with chronic renal failure (CKD). They are increasingly rare because hyperparathyroidism in now diagnosed and treated at an early stage. We report 67-year-old man who had been on hemodialysis for CRF for over 3 years, who presented with back pain over the thoracolumbar junction from 2 years ago and because of pain he could not stand or walk in the last 3 months before surgery. Ambulation was regained after surgical decompression and stabilization. In conclusion, when brown tumor arises in the spine, surgery may be needed to preserve neurologic function.

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Keywords: Brown tumor; Hyperparathyroidism; Spinal cord compression; Spinal tumor

Introduction

The pathogenesis of hyperparathyroidism in chronic kidney disease is incompletely understood (1). Resistance to the normal level of PTH is a major factor contributing to the development of hypocalcemia, which, in turn is a stimulus to parathyroid glands enlargement (1). Brown tumor, also called osteoclastoma, is a lytic bone tumor caused by hyperparathyroidism, being more common in primary than in secondary hyperparathyroidism (2). They are not neoplasms, but can grow considerably in size and compress vital structures, particularly in the mandible, maxilla, ribs, and pelvis (3). Incidence rates of 1.5 to 13% have been reported in patients with CRF (2).

Skeletal brown tumors are relatively uncommon, and brown tumors that involve the spine are considered very rare (4). We report a case of brown tumor of the lumbar spine extending from L2 to L4 causing severe mechanical back pain.

Case Report

A 67-year-old man with a 3-year history of hemodialysis was admitted through the clinic for severe mechanical back pain. His pain had started 2 years ago and had worsened rapidly in the last 3 months. He had a history of bilateral THA at about 1 year earlier. Physical examination revealed tenderness over the posterior aspect of upper and mid lumbar spine. No neurologic deficit was evident. Radiographs of the spine disclosed osteolytic lesion of the vertebral body of L3 (Figure 1). Ct scan of the spine showed an osteolytic lesion that extend from the posterior L3 vertebral body to its right pedicle and both laminae and spinous process of L3 & L4 (Figure 2). Findings were negative from imaging studies done to look for other tumors, including computed tomography (CT) of the abdomen and pelvis, and a radionuclide bone scan. The only laboratory test abnormality was serum parathyroid level (PTH) elevation to 1500 pg/ml. serum levels of alkaline phosphatase, calcium and phosphate, were normal.

A percutaneous CT-guided biopsy of L3 was performed. Pathologic findings were consistent with giant cell tumor or brown tumor. The clinical setting characterized by renal failure responsible for secondary hyperparathyroidism strongly supported a diagnosis of brown tumor. No histologic evidence of malignancy was found.

Surgical decompression was performed. Following L3 laminectomy, soft brownish tissue was easily cleaved from the spinal canal and vertebral column and debulking of that tissue was performed. The extensive osteolysis and involvement of the mobile vertebral segment required internal fixation by pedicle screws and rods from L1 to L5 (Figure 3). An autologous bone graft was implanted posterolaterally from L1 to L5.

Subsequently, subtotal parathyroidectomy was

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performed. Serum PTH values fell to 11, four days after surgery. High dose calcium and vitamin D supplementation were required to maintain the serum calcium levels within the normal range.

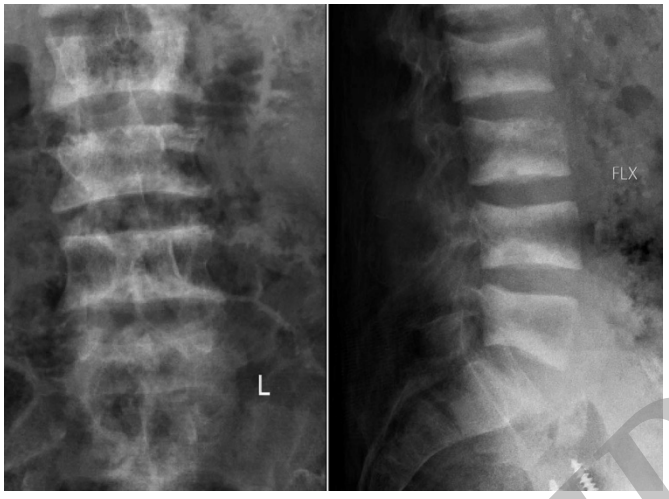

Figure 1. AP and Lat preoperative radiograph of spine show osteolytic lesion of the L3

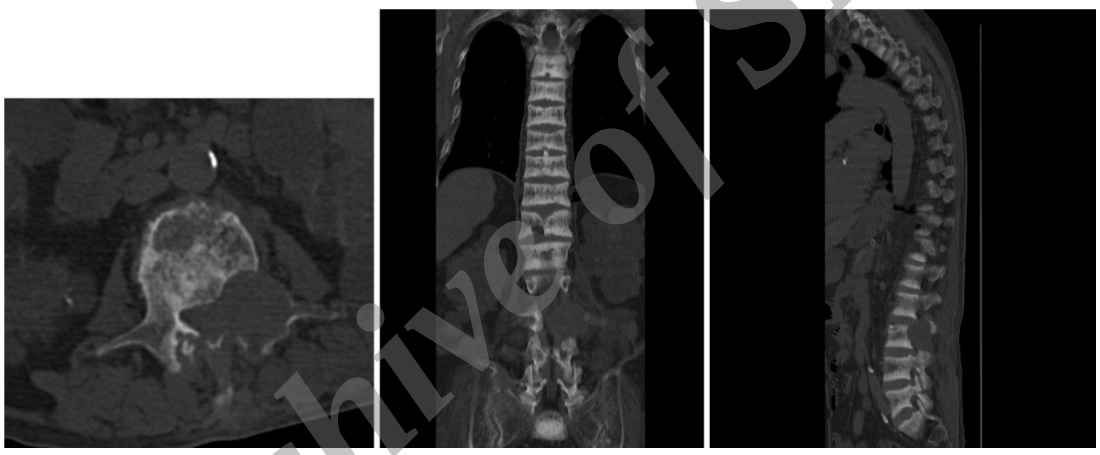

Figure 2. Preoperative axial, coronal and sagittal views of the spine show osteolytic lesion of the L3 and it's posterior elements

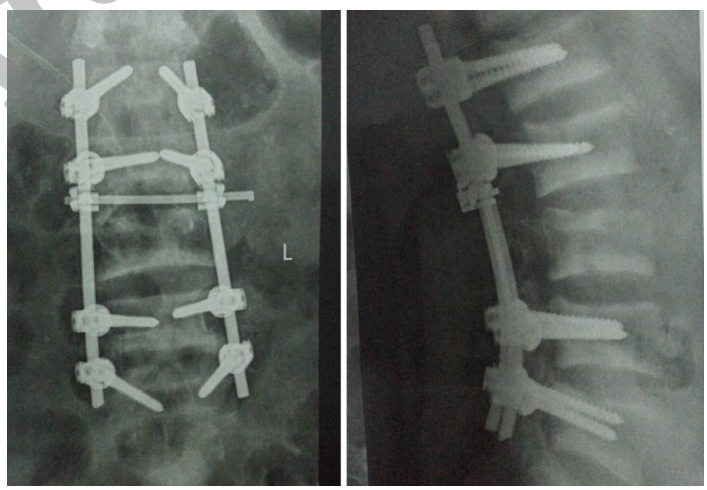

Figure 3. Post operative AP and Lat radiographs after internal fixation from L1 to L5, decompression and fusion

Discussion

Secondary hyperplasia of the parathyroids is a well-recognised complication of renal failure resulting in a spectrum of bone disorders described as renal osteodystrophy; these include osteomalacia, osteitis fibrosa and osteosclerosis (5).

Historically, brown tumors were seen more commonly in primary hyperparathyroidism (HPT) but in the last three decades the course of renal failure has been modified by the advent of renal dialysis and transplantation. The prevalence of brown tumor in these patients has been estimated as 1.5% in chronic renal failure and 1.7% in transplant recipients (5).

Brown tumors can have almost any appearance, from a purely lytic lesion to a sclerotic process. Generally when the patient's hyperparathyroidism is treated, the brown tumor undergoes sclerosis and will eventually disappear (6). If a brown tumor is going to be considered in the differential diagnosis, additional radiographic findings of HPT should be seen. Subperiosteal bone resorption is pathognomic for HPT and should be searched for in the phalanges particularly in the radial aspect of the middle phalanges, distal clavicles (resorption), medial aspect of the proximal tibial and sacroiliac joints (6). Computed tomography shows an osteolytic tumor of uniform tissue density replacing the cancellous bone of the vertebral body and neural arch. The cortex may be spared. Magnetic resonance imaging confirms that the mass is composed of tissue and provides an accurate evaluation of local spread (2).

The definite diagnosis depends on the examination of a tissue specimen. Cortical and trabecular bone are lost and replaced by loose connective tissue (7). Microscopically, excessive resorptive activity, is manifested by the presence of increased numbers of osteoclastic and accompanying erosion of bone surfaces. The marrow space contains increased amounts of loose fibrovascular tissue. Hemosiderin deposits are present, reflecting episodes of hemorrhage resulting from fractures of the weakened bone (7). In some instances, collections of osteoclasts, reactive giant cells, and hemorrhagic debris from a distinct mass, termed a brown tumor of HPT. Cystic changes are common in such lesions (hence the name osteitis fibrosa cystic), and they can be confused with primary bone neoplasms (7).

Brown tumor has a more favorable prognosis as compared to other lesions that have similar clinical and radiographic findings, such as metastatic lesions and giant cell tumors (8).

In a patient with multiple giant cell tumors, the most likely diagnosis is HPT, followed by Paget's disease and more rarely, true neoplastic GCT. The diagnosis of multiple neoplastic GCT should never be made in the absence of serial chemistries for calcium, phosphorous and PTH (9).

The spine is an uncommon site of brown tumor development (2). To our knowledge, only 10 other cases related to secondary hyperparathyroidism have been reported (Table 1). Five of the 10 patients presented with acute neurologic compromise that resolved fully after decompressive surgery.

Table 1. The 10 reported cases of spinal brown tumor

Author	Year	Age	Gender	Spinal segment	Hemodilysis	Symptoms	Treatment
Ericcon *et al.*(2)	1978	47	F	Cervical	No	Paresis, Pain	Resection,PT
Bohlman *e al*.(2)	1986	69	F	Thoracic	NO	Incipient paraplegia	GC
Pumar *et al*.(2)	1990	24	F	Thoracic	NO	Incipient paraplegia	Resection
Barlow(2)	1993	31	F	Cervical	Yes	Pain,neuralgia	Minerva,PT
Finemann(2)	1999	37	F	Thoracic	Yes,10ys	Incipient paraplegia	Resection,PT
Masutani *et al*.(2)	2001	39	F	Thoracic	Yes,11ys	Paraplegia	Resection,PT
Azria *et al.*(2)	2000	40	F	Thoracic	Yes	Pain	PT
Vandenbusche(2)	2004	34	F	Thoracic	Yes,2ys	Cord compression	Resection,fusion,PT
Kava *et al.*(10)	2007	72	M	Thoracic	Yes	Cord compression	Resection,Fusion
Jackson *et al.*(11)	2007	29	F	Cervicothoracic	NO	Radicular pain	Resection,Fusion

PT: Parathyroidectomy, GC: Glucocorticoid

Definitive treatment requires total or subtotal parathyroidectomy, which is usually followed by complete clearing of the lesions with remineralization of the vertebra. The tumor tissue within the spinal canal does not undergo remineralization, probably because it is not subjected to mechanical stress. After decompressive surgery, internal fixation may be required if the spine is unstable or impending fracture is anticipated (2).

Although uncommon, a brown tumor should be considered first in patients with renal failure and the vertebral osteolysis, particularly those on long-term hemodialysis. This benign tumor resolves after parathyroidectomy but can require emergent

decompressive surgery when it involves the spine.

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