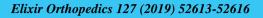




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ABSTRACT

We report the case of spinal cord compression from pathological fractures of the vertebral bodies with Gammapathy monoclonal in conjunction with multiple myeloma in a 52-year-old man who had recently been examined for paraplegia after two weeks of full pain. In our kwnoledge we thing that is a rare condition and must mainly been reported as a case presentation in the literature.

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Keywords

Multiple Myeloma, Spinal Cord Compression, Pathologic Fracture.

Introduction

Spinal cord compression (SCC) from spinal metastasis is a common complication in cancer and if left untreated, permanent paraplegia or quadriplegia will occur (1). The incidence is probably increasing owing to continual advancements in the treatment of cancer that have led to prolongation of life and a greater probability of secondary involvement of the spinal cord. The first detailed description of the disease was made in 1884 by the English physician Solly (2), Timely diagnosis is crucial in preventing permanent neurologic damage. Once SCC is suspected, diagnostic imaging of the spine should be obtained to confirm diagnosis. The treatment decision should be made based on multiple factors, including tumor histology, retropulsion of bony fragments, performance status of the patient and status of extraspinal systemic disease (3).

Multiple myeloma is malignant multicentric hemato poietic neoplasm characterized by errant proliferation of a single clone of plasma cells within the bone marrow (4)This leads to accumulation of the malignant plasma cells in bone marrow, with subsequent marrow compromise and destruction of bone. While this disease is typically multicentric, it most commonly affects the anterior column of the spine, resulting in vertebral body collapse and cord compression in 5% of cases (5).

Reports on osteocondensis of multiple pathologic fracture of bone as a cause of cord compression are rare. It most commonly affects the spine, with symptoms occurring when the disease spreadsthrough the vertebral body cortex and invades the surrounding tissues. This results in vertebral body collapse and subsequent spinal cord compression (6).

We present an unusual case of spinal cord compression as a result of osteocondasis lesion in pathologic fractures on multiple myeloma with sudden onset paraplegia resulting from a osteocondesis lesion.

Diagnosis

The most common presenting symptoms of MM are fatigue and bone pain. Anemia occurs in approximately 75% of patients and contributes to fatigue. Osteolytic skeletal lesions can be detected in approximately 80% of patients.

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Other common findings at presentation include hypercalcemia(15%), and elevated serum creatinine $\geq 2 \text{ mg/dL}$ (20%) (7).

A monoclonal (M) protein in the serum or urine is a cardinal feature of MM, but is seen in only 82% of patients by serum protein electrophoresis (7). The sensitivity increases to 93% when serum immunofixation is added, and to 97% with the addition of either the serum free light chain (FLC) assay or a 24 hour urine studies (8). Thus if MM is suspected, the recommended screening strategy is to order serum protein electrophoresis, serum immunofixation, and either a serum FLC assay or a 24 hour urine protein electrophoresis with immunofixation. The M protein type is IgG in approximately 50%, IgA in 20%, immunoglobulin light chain only in 20%, IgD in 2%, and IgM in 0.5% (7). About 2–3% of MM has no detectable M protein, and is referred to as non-secretory MM (9).

The baseline diagnostic work up required for the diagnosis of MM includes complete blood count, serum calcium, serum creatinine, serum and urine protein electro phoresis with immunofixation, serum FLC assay, and bone marrow examination. In addition, low dose whole body computed tomography (CT), or fluoro-deoxyglucose (FDG) positron emission tomography/CT (PET/CT), or at minimum, plain radiographs of the entire skeleton are required to detect osteolytic bone lesions

Traitment

There is no curative treatment but many drugs can help to prolong survival by 5 to 6 years after the diagnosis (4). Successful treatment and management of spinal myeloma disease requires a multidisciplinary approach that addresses the following: treatment of the myeloma with systemic chemotherapy; adequate control of pain; relief of spinal cord or cauda equina compression and maintenance of spinal stability. To reduce the risk of further spinal bone destruction or permanent deformity/neurological dysfunction and the impact on patient quality of life, it is imperative these measures are addressed in a systematic manner

Although and define the role for surgery in the spinal involvement of myeloma from the perspective of spine

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surgeon. The mechanical status of the spine and neurological status are two important factors to be assessed. As most surgeons recognize, these local factors are one of the many components used to determine management of the patient with spinal lesions caused by a myeloma.

Case Report

A 52 year old man had recently been examined to our facility with a two weeks history of lower back pain that was initially mild then increased in severity. This was followed by a sudden onset of flaccid paraplegia. Magnetic resonance imaging (MRI) of thoracolumbar spine showed : In osseous structures and bone marrow: a partial fracture along the upper haif of L3 vertebral body anterior aspect; Undisplaced fracture along the D12, L1, L2 3 vertebral body posterior 1/3rd; Inter discal herniation with sclerosis of the end plate of the vertebral body along D12-L1, L1-L2, L2-L3, L3-L4, L4-L5; Dorsal expation of the D11 vertebral body with spinal canal stenosis noted of about 34-36%; Dorsal expation of the D3 vertebral body with spinal canal stenosis noted of about 22-24%

Intervertebral discs and Motion segments: L1-L2, L2-L3, L3-L4: Middle global disc protrusion noted indending the tecal sac with mild compression over the bilateral exiting nerve roots. Bilateral facet osteoarthropathy is noted (Fig.1)

Laboratory investigations revealed the following: - Blood examination **revealed** anemia (Hb-10 g/dL), Myelocytes (02%), HyperAcid urique: 19.3 mg/dL, Total serum protein 9.89 mg/dL; Hypoalbumin 44%, hyperAlpha2 :13.4%, hypoBeta1:4%, HyperGamma: 30.3%; Hypercalcemia: 14.84 mg/dL; HyperAzote uréique: 45.5mg/dL; Urea–11.0 mmol/L Creatinine: 3.26 mg/dL

Protein Electrophoresis showed a decreased Albumin Concentration and Increased Alpha2 and M Concentration in the Gamma Globulin Protein Fraction M Peak in the Gamma Globulin Protein Fraction Compatible with Monoclonal Gammapathy The immunotypage of proteins showed a reduction of IgG of the heavy chain in the gamma fraction and a reduction in the Kappa Legere chain in the gamma fraction compatible with a Gammapaty monoclonale IgG-Kappa. A monoclonal (M)

The test for Bence–Jones proteinuria was positive. A bone marrow biopsy revealed a plasmocytosis at 30% thus confirming the diagnosis of multiple myeloma

The patient was treated with dexamethasone 40mg/ weekly repeated every 4 weeks associeted with Thalidomide 200 mg oral day 1-28.and and Zoledronic acid once-monthly: 4 mg intravenously over 15 minutes. The indication for surgery was neurological deficit due to spinal cord compression posterior. A decompression and stabilization was performed. Better therapeutic regimens including autologous stem cells transplants were considered, but financial and technical constraints did not allow us envisage other protocols apart from the Thalidomide/ dexamethasone association. Four weeks later, the patient presented with severe anaemia accompanied with the renal insufficiency and died a day later a blood transfusion

Discussion

MM is a hematological multicentric disorder. It comprises 1% of malignant tumors and 10-15% of hematopoietic neoplasms (10). However, as MM is a bone-marrow based neoplastic proliferation of plasma cells that secrete a monoclonal immunoglobulin, skeleton related events are not uncommon. Among these skeleton events, the spine is one of the most commonly involved sites and pathological fractures of the spinal column are the most common spinal involvement of MM. Spinal cord compression is reported to develop in 11-24% of patients with MM (11) and in metastatic prostate cancer is not rare occurring in 1 to 12% of patients (12).

Most cord-compression lesions occur due to a pathological fracture of the involved vertebral body or extension of a vertebral body myeloma lesion.



Figure 1. Magnetic resonance imaging (MRI) of thoracolumbar spine showed : In osseous structures and bone marrow : a partial fracture along the upper haif of L3 vertebral body anterior aspect ; Undisplaced fracture along the D12, L1, L2 3 vertebral body posterior 1/3rd ; Inter discal herniation with sclerosis of the end plate of the vertebral body along D12-L1, L1-L2, L2-L3, L3-L4, L4-L5.

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We present a rare case of spinal cord compression from a pathological fractures of the vertebral body combined with Gammapathy monoclonal in conjunction with multiple myeloma who had recently been examined for paraplegia after two weeks of full pain. The presentation of the disease in this case is atypical. Rather than the bone pains which are the most common presenting features of myeloma in about 58% of cases (4), the patient presented with spinal cord compression due to multifocal collapse of the vertebrae. This type of presentation was found in only 0.5% of 646 cases of myeloma recruited by Cheema and collaborators over a period of 17 years (13).

S. Vincent Rajkumar and all described a case of the spinal cord compression from a pathological fractures of the vertebral body without epidural myeloma combined (14)

MRI is the best diagnostic modality for detecting spinal cord compression, epidural masses and marrow involvement of MM.

Magnetic resonance imaging (MRI) of thoracolumbar spine showed in this patient:

1. In osseous structures and bone marrow:

- a partial fracture along the upper haif of L3 vertebral body anterior aspect.

-Undisplaced fracture along the D12, L1, L2 3 vertebral body posterior 1/3rd

-Inter discal herniation with sclerosis of the end plate of the vertebral body along D12-L1, L1-L2, L2-L3, L3-L4, L4-L5.

-Dorsal expation of the D11 vertebral body with spinal canal stenosis noted of about 34-36%

--Dorsal expation of the D3 vertebral body with spinal canal stenosis noted of about 22-24%

2. Intervertebral discs and Motion segments:

L1-L2, L2-L3, L3-L4 : Middle global disc protrusion noted indending the tecal sac with mild compression over the bilateral exiting nerve roots. Bilateral facet osteoarthropathy is noted

Protein Electrophoresis showed a decreased Albumin Concentration and Increased Alpha2 and M Concentration in the Gamma Globulin Protein Fraction M Peak in the Gamma Globulin Protein Fraction Compatible with Gammapathy monoclonal

The immunotypage of proteins showed a reduction of IgG of the heavy chain in the gamma fraction with a fall in the Kappa Legere chain in the gamma fraction compatible with IgG-Kappa Gammapathy monoclonal. The results of most laboratory tests were similar to those published in the literature for multiple myeloma. The anaemia was normochromic and normocytic and there was no thrombopaenia, which is found in only 10 to 15% of cases (4). The erythrocyte sedimentation rate was very high in accordance with the abnormal gammaglobulin hypersecretion (15), though the severe anaemia could equally be responsible. The serum calcium was very high and the kidney function test altered. There was a hyperproteinaemia with a gammaglobulin spike on serum protein electrophoresis. The bone marrow biopsy showed a plasmocytosis greater than 10% and a plain radiograph of the skull revealed lytic lesions. The test for Bence-Jones proteins was positive in our patient protein in the serum or urine is a cardinal feature of MM, but is seen in only 82% of patients by serum protein electrophoresis (16). The sensitivity increases to 93% when serum immunofixation is added, The M protein type is IgG in approximately 50%, IgA in 20%, immunoglobulin light chain only in 20%, IgD in 2%, and IgM in 0.5% (16). About 2-3% of MM has no detectable M protein, and is referred to as non-secretory MM.

The complete blood count showed; hypercalcemia, renal insufficiency, anemia.

The therapeutic strategy for the spinal cord compression from a pathological fractures of the vertebral body combined with multiple myeloma as one of devastating complications of multiple myeloma is still under debate. By review of cases with different clinical courses and related literatures, the authors tried to suggest therapeutic strategy for optimal clinical results. Close evaluation for mechanical stability and neurologic status, and multidiscipline approach are important factors leading successful results for this systemic disease.

The European Society of Medical Oncology (ESMO) and the Italian Society of Haematology (ISH) recommend autologous stem cell transplantation, after an initial induction treatment with vincristine, doxorubicin and dexamethasone as the ideal treatment in young patients (17). However, because it is not accessible in our context, we considered our patient as not a candidate for transplantation and treated her with the association of from the Thalidomide/dexamethasone.

Patients with a chemosensitive malignancy, chemo therapy are an attractive option because it can also treat tumor deposits elsewhere in the body. Targeting the myeloma cells, anti-myeloma treatments can disrupt the interaction with the bone microenvironment, thus inhibiting the osteoclastogenic effect. (18). Surgery.To date, surgical management of myeloma spinal disease has been similar to the management of spinal metastases in solid cancers. (19).

Direct decompressive surgical resection treatment of spinal cord compression caused by metastatic cancer, Lancet reported that patients with metastatic cancer(excluding myeloma) who presented with spinal cord compression and were treated with surgical decompression followed by radiotherapy had significantly improved outcomes compared to patients treated with radiotherapy alone (20).

Surgery for spinal metastases has consisted of simple decompressive laminectomy Results obtained in retrospective case series, however, have shown that this treatment provides little benefit to the patient. With the advent of better patientrelated selection practices, in conjunction with new surgical techniques and improved postoperative care, the ability of surgical therapy to play an important and beneficial role in the multidisciplinary care of cancer patients with spinal disease has improved significantly. Controversy remains, however, with respect to the relative merits of surgery, radiotherapy, chemotherapy, or a combination of these treatments. (21)

For some patients with spinal metastasis and spinal cord compression, newer surgical techniques are better than laminectomy or radiotherapy alone in relieving pain and restoring function. While radiotherapy remains the standard for spinal metastases due to myeloma, lymphoma, and many types of adenocarcinoma, proper surgical treatment can significantly improve function and outcome in selected patients (22).

We could not identify the exact cause of death in our patient. Taking into consideration the association of severe anaemia, kidnay insuffisancy is the most probable hypothesis. **Conclusion**

The multiple fractures without trauma of the spine the, we must suspect MM and The spinal cord compression in multiple myeloma is a neurosurgical emergency and the Efforts must be concentrated on his prevention. The diagnosis must be confirmed by MRI associeted with serum protein electrophoresis and serum immunofixation. The surgery decompression in the management of metastatic spinal cord compression is expanding and can consist of a combination of surgery, radiation treatment, and chemo therapy. Treatment modalities are not mutually exclusive and must be individualized for patients evaluated in a multidisciplinary setting.

Competing interests

The authors declare that they have no competing interests **Authors' contributions**

All the authors have read and approved the final version of the manuscript.

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