CASE REPORT

Anterior horn lesion of spinal cord as the initial manifestation of multiple myeloma: A case report

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Abstract: Multiple myeloma (MM) is a malignant proliferation of abnormal plasma cell tumour that often violates multiple body systems. Its clinical manifestations vary greatly, thus leading to a high rate of misdiagnosis. Herein, we report a clinical case of non-compressive spinal cord lesion, which manifested early signs of multiple myeloma. Retrospective analyses of MM pathogenesis, clinical manifestation, and diagnosis were based on the clinical features and diagnostic criteria of MM that have been published in the literature. This study was initiated to improve current awareness of MM and to reduce the rate of misdiagnosis.

Keywords: multiple myeloma; anterior horn lesion of spinal cord; misdiagnosis


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Introduction

A 70-year-old male patient was admitted to our department after complaining of upper left limb weakness that persisted for two months, with noticeable development of limb muscle atrophy in the two weeks prior to hospitalization. Initially, the patient experienced an onset of upper left limb weakness and numbness with no apparent cause, having difficulty lifting his left limb and picking up small objects. He was later diagnosed with cerebral infarction and treated at a local hospital but did not see improvement in his condition. Subsequently, the patient suffered from thenar atrophy on his left hand with general weakness in his lower limbs and required great effort to go up a flight of stairs.

Cervical magnetic resonance imaging (MRI) scan showed cervical degenerations and intervertebral disc herniation at C3-4, C4-5, and C5-6. Moreover, the scan also revealed dural sac and left nerve root compression. He was then diagnosed with cervical spondylodiscitis and was treated in a local hospital but showed no signs of improvement. Subsequently, the patient was referred to our hospital for further diagnosis. Admission examination revealed that the muscle strength of the patient’s upper left limb was given a muscle strength rating of 4, while that of other limbs was given a rating of 5[1]. The tendon of his upper left limb showed hyporeflexia whereas other limbs were normal. His left thenar muscle, along with his limb muscles, was showing atrophy; however, further physical examination revealed no other abnormalities.

The results of laboratory tests performed upon the patient’s admission are as follows: leukocytes count was $11.4 \times 10^9/L$, erythrocytes count was $2.67 \times 10^{12}/L$, hemoglobin was 86 g/L, total protein was 131.5 g/L, serum creatinine was 113 μmol/L, and serum calcium was within the normal range. A blood image showed an increase in the total amount of leukocytes and the rouleaux formation of erythrocytes, but the thrombocytes were seemingly normal. Hematology consultation was as follows – immunofixation electrophoresis (IFE): IgG (+), K (+); serum protein electrophoresis (SPEP): 23.9% albumin, 5.8% alpha, 63.2% gamma, M-protein positive; immunoglobulin (A, G, M) + serum chain determination:
92 g/L IgG, 0.2 g/L IgA, 0.86 g/L IgM; immunoglobulin light-chain K: 147 g/L; determination of urinary light-chain: 4460.0 μg/L of immunoglobulin light-chain K; bone marrow aspiration: plasma cells >30%.

The aforementioned results indicated a clear and definite diagnosis of multiple myeloma (MM, IgG type), and the patient was immediately transferred to the Department of Hematology for a bortezomib- and dexamethasone (BD)-based chemotherapy. After a course of treatment, the patient experienced a lessening of limb weakness and exhibited remission of muscle atrophy. While the patient underwent his therapy, we tracked his electromyography (EMG) readings and found denervation and fasciculation potentials in his limb muscles. The insertion potentials were extended with the appearance of giant potentials at a synchronization rate of 90%, in addition to an extended latency period for the F-wave upper limb nerve inspection, which led to low extraction rate. Pathological changes of the anterior horn cells of spinal cord were found and no other obvious abnormalities were detected based on the chest computed tomography (CT) scan, as well as posterior pelvis, skull, femur, and lateral plain scanning.

Discussion

MM is an abnormal proliferation of plasma cells that causes malignant neoplasm, consisting of up to 10% of haematological malignancies[2] and it mainly affects the elderly population[3]. It has relatively non-specific onset symptoms as it shows a variety of clinical manifestations but lacks specificity. The vast majority of MM patients have (but are not restricted to) invasive myeloma cells and bone marrow, and the condition usually presents itself clinically in the form of osteolytic lesion or osteoporosis, hypercalcemia, and anemia[4].

Myeloma cells can also break the cortical bone via direct invasion or blood-borne metastasis into soft tissues and the neural system, causing extramedullary disease and a variety of neurological symptoms[5]. A large proportion of newly diagnosed patients shows extramedullary invasion as the main clinical manifestation[6]. Therefore, MM is easily misdiagnosed owing to the lack of disease-specific clinical manifestation. It has been reported that the rate of domestic MM misdiagnosis in China is 56.44%. The most common misdiagnoses are osteoarthritis and other common neurological diseases at 32.2% and 2.97%, respectively[7].

In our patient, the disease manifested itself as a non-compressive spinal cord lesion caused by limb muscle atrophy, accompanied by anemia, infections, and renal dysfunction, which is extremely rare in a clinical setting.

The neural pathogenesis of MM and its clinical manifestations are as follows:

(1) Spinal cord lesions that can be categorized as either compressive or non-compressive in nature. The incidence of spinal cord compression is about 20%, occurring in the thoracic spine and extending mostly into the spinal canal in secondary MM, which causes compression around the dura and is often accompanied by vertebral collapse[6]. Common initial clinical manifestations include lower back pain, numbness of the limbs and trunk, paresthesia, paraplegia, and sphincter disorders. X-ray results usually show osteoporosis and compression fractures with occasional soap bubble-like expansion or paraspinal shadows.

Meanwhile, the occurrence of non-compressive spinal cord lesion is extremely rare, appearing as non-specific spinal cord lesion and the degeneration of anterior horn of spinal cord. If the lesion invades the spinal vascular structure, it might potentially cause myelomalacia. In our patient, his electromyography analysis showed anterior horn lesion of spinal cord; after undergoing BD chemotherapy, his symptoms were relieved and this suggested a non-compressive spinal cord lesion of MM.

(2) Myeloma-like meningitis is caused by the spread of plasma cells in pia mater, commonly seen in the IgA- and IgM-type of MM, and it is usually accompanied by meningeal irritation symptoms such as headache, nausea, and vomiting. Plasma cells can be detected in the blood of up to 45% of patients while 90% of patients have immature plasma cells in their cerebrospinal fluid, indicating a poor prognosis[8].

(3) Peripheral neuropathy may cause plasma cells to increase abnormally, leading to the binding of M-protein to the myelin of peripheral nerve, particularly myelin-associated glycoprotein[9]. M-protein can be deposited in between myelin sheath gaps and causes secondary demyelination[10]. This results in acute or chronic progressive distal sensory-based movement disorders, which are associated with muscle atrophy and tendon reflexes and is commonly known as dyskinesia. The manifestation of POEMS (polyneuropathy, organomegaly, endocrinopathy, M-protein spike, and skin manifestations) syndrome has been described in association with it[11].

(4) Cranial nerve lesion is mostly caused by the nerve compression of plasmacytoma that is formed in the brain of MM patients[12]. Its clinical manifestations are mostly hemiplegia, partial body sensation, hemianopia, aphasia, and other focal symptoms.

Prevention for misdiagnosis

MM has a variety of clinical manifestations and lacks specificity, which often results in a high rate of misdiag-
nosis in the first instance. It is important for clinicians, especially those with non-hematological background, to increase their overall awareness on MM in order to improve the diagnosis rate of MM during a patient’s first visit[13]. For neurologists, a detailed review of the patient’s medical history and a thorough physical examination would be necessary when attending to elderly patients with nerve lesion of unknown reason, along with the following conditions: (1) unexplained bone pain, osteoporosis, and pathological fracture; (2) unexplained anemia, hemorrhage, an increasing erythrocyte sedimentation rate, and hypercalcemia; (3) unexplained edema, proteinuria, and kidney lesion; and (4) unexplained abnormalities of liver function and immunoglobulin. In addition, a blood film, hematuria immunoglobulin electrophoresis, skeletal x-ray, and bone marrow cytology examinations must also be used to assist diagnosis.

**Conflict of interest**

The authors declare no potential conflict of interest with respect to the research, authorship, and/or publication of this article.

**References**