Foot-drop due to involvement of lumbosacral plexus in diffuse large B-cell lymphoma

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ABSTRACT

Extranodal nerve system involvement is seen in approximately 5% of the patients with non-Hodgkin's lymphomas. It causes as primary or secondary infiltration of brain, leptomeninges, or peripheral nerves and the signs of spinal cord compression. Here we present a 67-year-old woman with advanced stage diffuse large B-cell lymphoma who developed foot-drop due to lumbosacral plexus involvement.

Key Words: Diffuse large B-cell lymphoma, Lumbosacral plexus, Foot-drop.

ÖZET

Difüz büyük B-hücreli lenfomada lumbosakral pleksus tutulumuna bağlı düşük ayak


Anahtar Kelimeler: Difüz büyük B-hücreli lenfoma, Lumbosakral pleksus, Düşük ayak.
INTRODUCTION

Diffuse large B-cell lymphoma (DLBCL), which is the most common lymphoma worldwide, is responsible for about one-third of non-Hodgkin’s lymphomas (NHL)\(^1,2\). Thirty to 40% of the patients with DLBCL have extranodal involvement, with gastrointestinal tract and Waldeyer ring being the most commonly involved sites\(^1-4\). Involvement of nervous system as an extranodal involvement is seen in approximately 5% of the patients with NHL. It usually takes the form of infiltration in the brain, leptomeningeal or peripheral nerves and compression of the spinal cord\(^5-7\). The compression of the spinal cord is rare in NHL and most commonly occurs in thoracic vertebrae\(^6,8-10\).

Here we present a case with DLBCL who developed foot-drop due to lumbosacral plexus infiltration.

A CASE REPORT

A 67-year-old woman presented with fatigue, cramps worsening with exercise on her entire right leg and weakness for three months. Her medical history, revealed cataract operation. Her father, brother and sister died from liver cirrhosis, NHL and brain tumor, respectively. On her physical examination, blood pressure was 100/70 mmHg, heart rate 78 beats per minute, respiratory rate 16 per minute, and body temperature 36.7°C and conjunctivae were pale. Lymphadenopathy (diameter 2 x 2 cm) on the right axillary area, 4 cm splenomegaly and 6 cm hepatomegaly were detected. On the neurological examination, flexion, adduction and eversion of the hip and extension of the knee were restricted and muscle strength was 4/5. There were sensory losses in the genital area, lateral, anterior and medial hip regions and medial side of the lower legs. The reflexes of cremaster and patellar on right side were diminished. On the follow-up, Achilles reflex on right side was hypoactive and urinary incontinence occurred. The hematological parameters were hemoglobin 9.5 g/dL, hematocrit 27.8%, white blood cell count 2500 cell/mm\(^3\) and platelet count 19.1000/mm\(^3\). Peripheral smear was normal. Erythrocyte sedimentation rate was 63 mm/hour, lactate dehydrogenase 935 IU/L, ferritin 718 ng/mL and D-dimer 1289 mg/mL. Protein electrophoresis, immune globulin values, vitamin B\(_{12}\) levels, and thyroid function tests were in normal limits. PPD, HBsAg, anti-hepatitis C virus (HCV), anti-human immunodeficiency virus (HIV), and cytomegalovirus (CMV)-IgM tests were negative. The aspiration and biopsy of bone marrow revealed minimal dysplastic changes in the erythroid cells and lymphoma infiltration. Chest X-ray, electrocardiography, and venous Doppler ultrasonography of lower extremities were normal. On thoracic tomography, there were multiple lymphadenopathies, infiltration areas of widespread reticular view in the parenchyma and pleural effusion on the right side. Abdominal tomography showed hepatosplenomegaly, multiple conglomerated lymphadenopathies (diameter of 5 x 4 cm) surrounding the aorta and inferior vena cava in the retroperitoneal area and mass lesion in the right adductor lodge. CD20 (+) DLBCL was found in the biopsy specimen of the right axillary lymphadenopathy. On the electromyography (EMG), mild degeneration in the muscles innervated by L2, S\(_2\) and S\(_3\) on the right side and severe degeneration in the muscles innervated by L\(_5\) (peroneal neuropathy due to involvement of sacral plexus) were detected. Malignant soft tissue infiltration involving the lumbosacral plexus and obturator area on the right side of the pelvis and the paravertebral area, lumbar spondylosis, and disc protrusions of L\(_1-2\), L\(_2-3\), L\(_4-5\) and L\(_5-S\(_1\) intervertebral discs were observed on magnetic resonance imaging (MRI) of the lumbar plexus (Figure 1). Cranial MRI was normal.

According to Ann-Arbor classification, Stage IVA DLBCL with 5 IPI (International Prognostic Index) was diagnosed. Six regimens of R-CHOP (rituximab and cyclophosphamide, adriamycin, oncovin and prednisolone) were given. Foot-drop and gait difficulty partially regressed. The
patient then received radiotherapy (RT) of 45 Gy on para-aortic region including the right sacroiliac area. After RT and chemotherapy, while thoracic tomography was normal, increase in soft tissue was observed in the retroperitoneal area surrounding the aorta and inferior vena cava. MRI of the lumbosacral plexus revealed a mass of soft tissue extending from the mid-line distally along the paracaval side of the anterior aspect of the spinal aorta and to the piriformis and obturator internus muscles by surrounding the internal iliac vein. Increase in soft tissue as observed on the tomography and MRI, was considered as fibrosis secondary to the RT (Figure 2). Positron emission tomography (PET) supported this appearance (Figure 3). On the follow-up, bone marrow involvement disappeared. On the EMG, there were neurological motor unit potential changes in the muscles innervated by L2-5 and S1 on the right side. The patient has been followed in remission for six months.

**DISCUSSION**

After the examinations of lymph node and bone marrow, the patient was diagnosed as advanced stage DLBCL. Additionally, she had polyradiculopathy and foot-drop. In the patients with lymphoma, peripheral neuropathy may appear due to direct peripheral nerve invasion, spinal cord compression, monoclonal gammapathy, anti-neoplastic drugs, and autoimmunity. Guillain-Barré syndrome, hypothyroidism, megaloblastic anemia, HIV and CMV infections should also be considered in the differential diagnosis of peripheral neuropathy. We excluded the other causes of neuropathy in our patient because there were negative viral markers, normal thyroid function and normal serum level of vitamin B12. Moreover, spinal disc compression was considered as the findings related to peroneal neuropathy on the EMG and mass lesions on MRI of the lumbosacral area. Hematological and neurological findings disappeared following R-CHOP chemotherapy and RT. These lesions detected radiologically were considered to be related to fibrosis.
Lymphomas cause spinal cord compressions in 6.8 to 10% of the patients. Spinal cord, dura mater, meninx or vertebra may be infiltrated. The infiltration of the lumbosacral region is rare. In polyradiculopathies due to infiltrations of the lumbosacral region, back pain, weakness, sensory loss, paresthesia and such findings as urinary frequency and constipation related to autonomic dysfunction as well as spasticity, ataxia, increased or decreased deep tendon reflexes, pathological reflexes or cauda equina syndrome may develop[7-10]. CT, MRI and PET are the most useful diagnostic tools in the diagnosis of lumbosacral infiltration[7,9,14].

Hollender et al.[15] reported the involvement of central nervous system in 170 (6.6%) of 2561 patients with NHL. While 85% of these patients had advanced stage, 140 patients were secondary NHL. The overall median survival was 2.6 months and seven of the patients had spinal cord infiltration.

Sciatic nerve infiltration of NHL may be rarely seen[6,16]. In these patients, paresthesia, numbness, weakness and pain in the lower extremity occurred, but no foot-drop. According to the working classification, these patients were in intermediate or high grade NHL. Cauda equina syndrome due to spinal cord involvement may result in paraplegia in NHL. These patients were reported as advanced stage DLBCL. Their median survival was short[17,18]. Patients with DLBCL with progressive neuropathy leading to spinal cord compression and sensory and motor losses have been reported. Although triceps palsy occurred in a patient related to brachial plexus compression, foot-drop was not seen in these patients[9,19].

In conclusion, foot-drop may develop due to lumbosacral cord compression as an extranodal involvement in the patients with advanced stage DLBCL. Thus, lymphoma should be considered in the patients with peripheral neuropathy and spinal cord compression.

REFERENCES


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