Olgu Sunumu

Spinal Cord Compression as the First Presentation of Disseminated Non-Hodgkin's Lymphoma: Case Report

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✔ Spinal cord compression as the first presentation of non-Hodgkin's lymphoma is a rare entity. In this paper, we presented a patient with disseminated non-Hodgkin's lymphoma initially presenting with progressive severe weakness and paresthesia of his legs and walking difficulty with a 5-month history of back pain. Imaging findings of vertebral non-Hodgkin's lymphoma were also described with its associated pancreatic, renal and bilateral adrenal gland involvements.

Key words: Non-Hodgkin's lymphoma, spinal cord compression, magnetic resonance imaging, vertebral involvement

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İlk Bulgu Olarak Spinal Kord Kompresyonu Olan Dissemine Non-Hodgkin Lenfoma Olgusu: Olgu Sunumu

✔ Non-Hodgkin lenfomanın ilk bulgu olarak spinal kord basısı yapması nadir bir durumdur. Bu çalışmada 5 aydır sırt ağrısı yakınmaları olan ve progressif, ileri derecede bacaklarında güçsüzlüğü ve parestezileri olan, yaygın non-Hodgkin lenfomalı bir hasta sunulmuştur. Vertebral non-Hodgkin lenfomanın görüntüleme bulguları pankreatik, renal ve bilateral adrenal bez tutulumlarıyla birlikte sunulmuştur.

Anahtar kelimeler: Non-Hodgkin lenfoma, spinal kord kompresyonu, manyetik rezonans görüntüleme, vertebral tutulum

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Lymphoma is a systemic disease and spinal involvement by lymphoma is not uncommon [(1)]. However lymphoma presenting initially with local symptoms and signs of spinal cord compression is a rare entity with an estimated incidence of less than 5 percent [(2)]. Early diagnosis and treatment of this disease is crucial to prevent partial or total neurological impairments [(3)]. Diagnosis is made primarily with a lymph node biopsy [(3)]. Magnetic resonance imaging (MRI) of the spine has proved to be exceptionally useful in the evaluation of spinal lymphoma and for ruling out other causes of cord compression [(1,3)]. In this report, we presented a case of disseminated non-Hodgkin's lymphoma (NHL) involving spine, liver, spleen, pancreas, stomach, lymph nodes and bilateral adrenal glands. Presenting symptom was back pain, followed by signs and symptoms of spinal cord compression. We also wanted to present MR findings of vertebral lymphoma.

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CASE REPORT

A 22-year-old male was admitted to our hospital emergency department with progressive severe weakness, and paresthesia of his legs, and walking difficulty developed 5 days before his admittance. He also mentioned about 5-month history of severe back pain. He had sought for medical care 4 months ago with the complaint of back pain and was treated with the diagnosis of low back pain (LBP) in the previous first-level referral hospital without any relief, and no neurological deficits were mentioned in his previous medical records. At that time, the plain radiograms of lumbar vertebrae were reported as normal. No further imaging studies were done then. The pain had gradually become intense and he also complained about neck and arm pain bilaterally which were not relieved by strict bed rest and analgesic drugs during the last 4 weeks. He did not give a history of urinary incontinence, headache, fever, chills, dysuria, hematuria, or abdominal pain. He mentioned about weight loss of 5 kg within the last 3 months.. Neurological examination assess ed muscular strengths in the right (proximally 4+/5, and distally 5/5), and left (4/5, and 5/5, respectively) legs. Babinski sign was negative. There was no clonus bilaterally. Examination of the arms was unremarkable. Laboratory results at the time of admission were as follows; WBC, 6.6K/uL; RBC, 3.65 M/uL; hgb, 8.8 g/dL; htc, 27.6 %; PLT, 376; prothrombin time (PT), 17.1 sec; PT %, 65 %; total protein, 6.4 g/dL; albumin, 2.6 g/dL; total bilirubin, 6.0 g/dL; AST, 56 U/L; ALT, 31 U/L; LDH, 487 U/L; alkaline phosphatase, 110 IU/L; creatinin (Cr), 0.9 mg/dL; Na,138 mmol/L; K, 4.5 mmol/L; Cl, 99 mmol/L; Ca, 10.0 mg/dL; blood urea nitrogen (BUN), 18 mg/dL; amylase, 72 U/L; CEA, 1.24 ng/ml; AFP, 0.649 IU/mL.

MRI scans of the cervical, dorsal and lumbar spine were requested. Sagittal and axial images were obtained using Fast Spin Echo (FSE) techniques with a 1.5-Tesla system. MRI showed diffuse involvement of cervical, thoracic, and lumbar vertebrae. There was involvement of the paravertebral soft tissue at levels of the T3, T11-T12, and L4-L5. There were C5, T3, T9, T11, T12, L4, L5 pathologic compression fractures with retropulsion of the posterior vertebral bodies of T9, and T11-T12 depressing ventral spinal cord (Fig. 1, Fig. 2). Intermediate- and high signal intensities relative to skeletal muscle on T1-weighted images, and high-signal intensities on T2-weighted images were observed. Some lesions had high signal intensities relative to

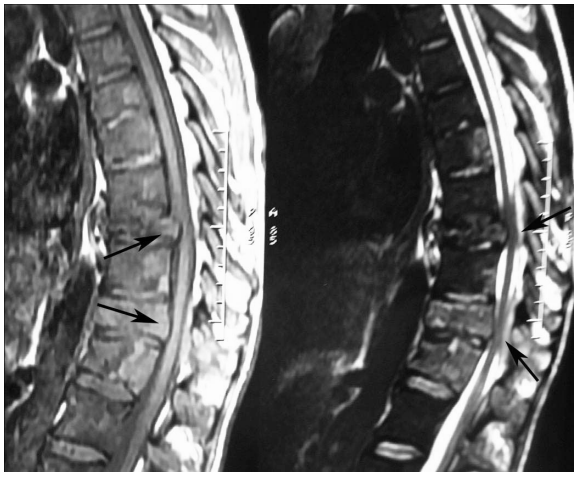

Figure 1. Sagittal T1-weighted (left) and T2-weighted (right) fast spin-echo MR images of thoracic vertebrae show compression of the cord (black arrows).

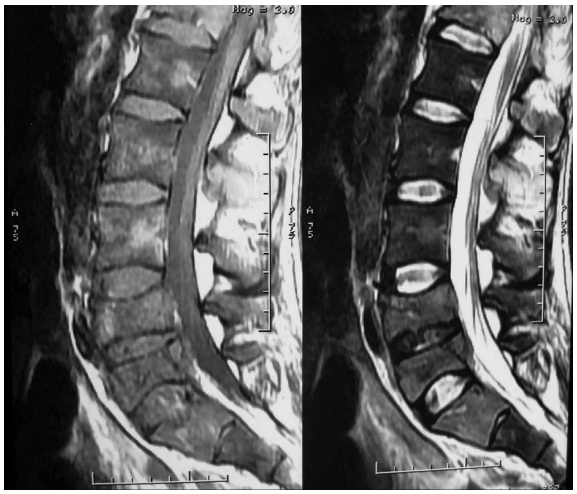

Figure 2. Sagittal MR images demonstrate multiple lesions with intermediate- and high signal intensty on T1-weighted images (left), high-signal intensity on T2-weighted images (right) in some lumbar vertebrae.

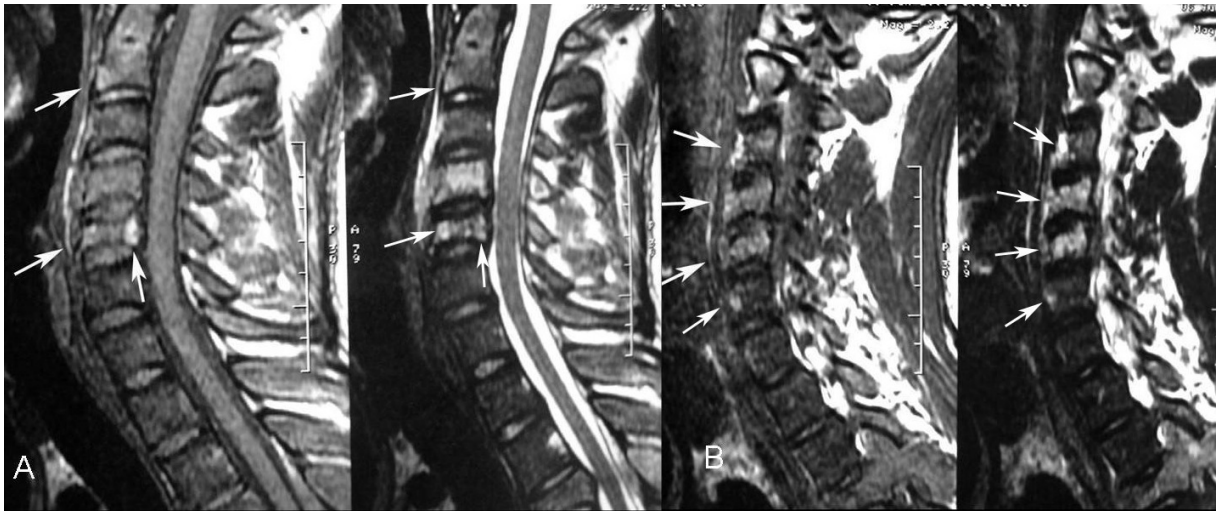

Figure 3. A-B: At sagittal MR images multiple lesions with intermediate- and high signal intensty on T1-weighted images (left), high-signal intensity on T2-weighted images (right) were seen in many cervical vertebrae (white arrows).

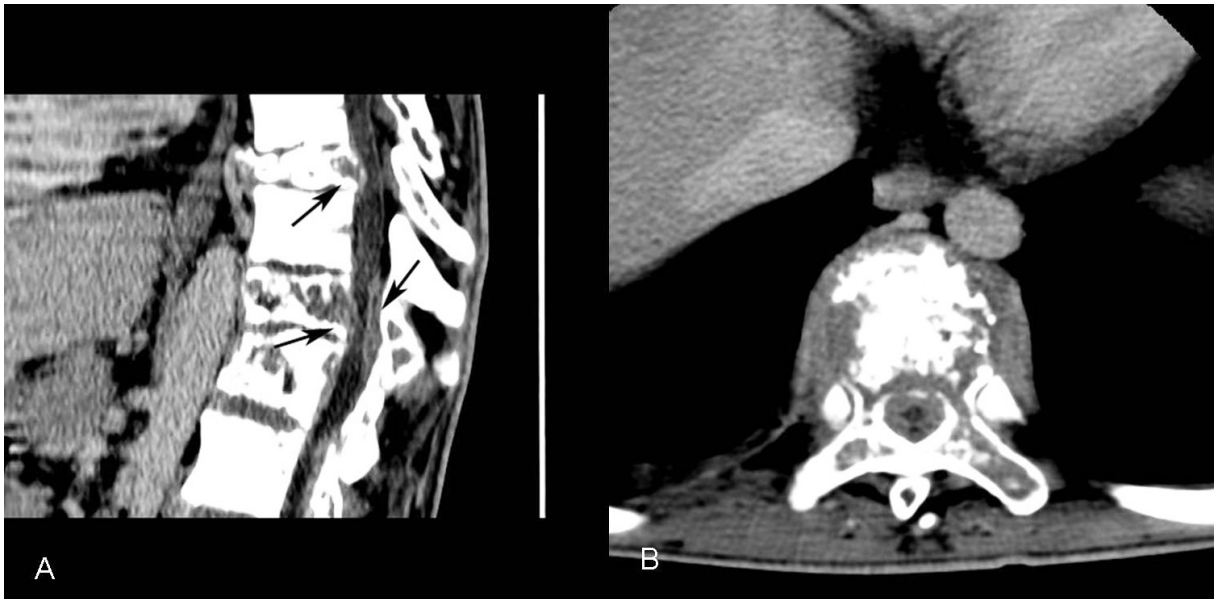

Figure 4. Contrast-enhanced CT scan of spine with sagittal (A) and axial (B) reformatted (soft window) images through thoracic vertebrea show vertebral involvement and compression of the cord (arrows).

skeletal muscles on T1-weighted images and high signal intensities relative to spinal cord on T2-weighted images (Fig. 3). Multi-slice CT examination with reformatted images was also performed to further delineate the osseous lesions which also demonstrated spinal cord compression with paravertebral soft tissue (Fig. 4). Abdominal ultrasonographic (US) examination was performed for the detection of the primary lesions which might give rise to metastatic vertebral lesions. Hepatosplenomegaly, diffuse enlargement of pancreas with minimal heterogeneity, mild nephromegaly and multiple renal round hypoechoic solid lesions were detected. There were several small-sized lymph nodes around the head of pancreas. Thorax and abdominal CT scans were performed both to disclose extent of the disease and also examine the lesions in detail. Contrast-enhanced multi-slice CT scan confirmed hepatosplenomegaly, mild

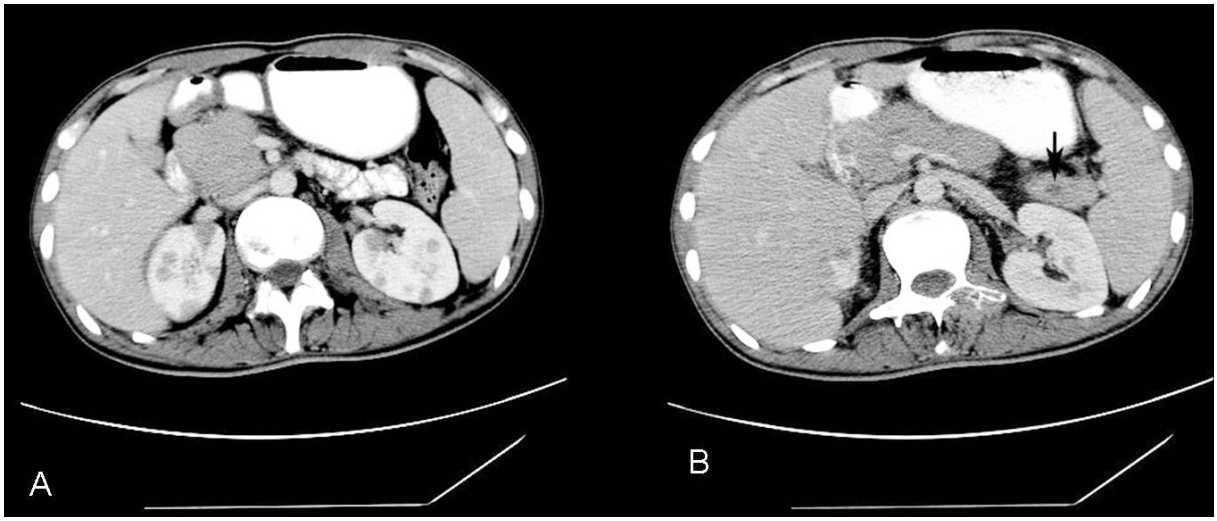

Figure 5. Contrast-enhanced CT scan reveals diffuse enlargement of pancreas with hypodens area within the tail (black arrow). Note bilateral multiple renal masses with homogenous attenuation, smooth borders and low contrast enhancement.

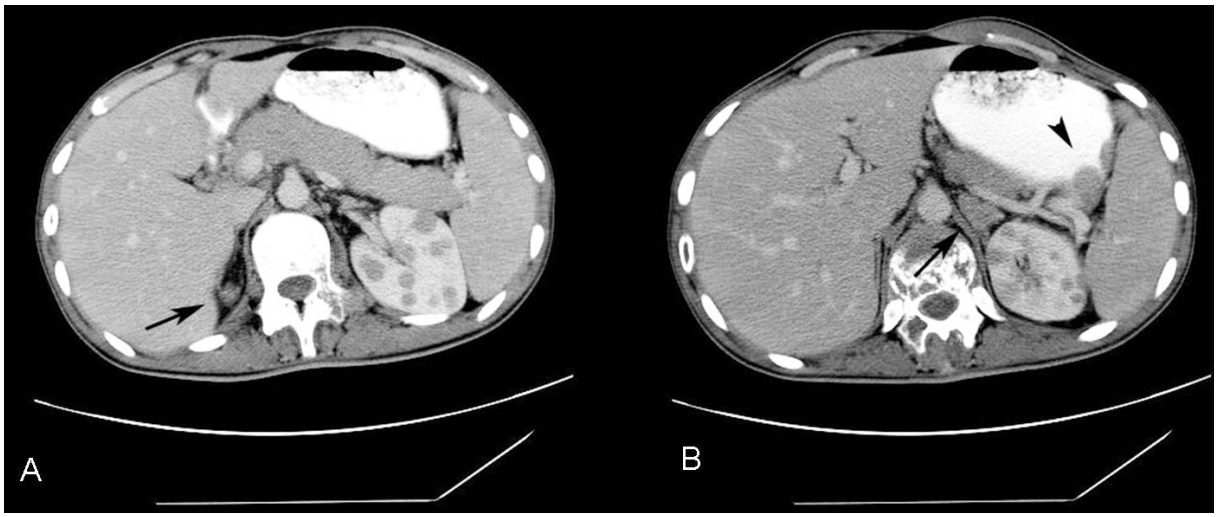

Figure 6. Contrast-enhanced CT scan adrenal glands demonstrates is small-low density lesions in the adrenal glands bilaterally (black arrows). There are also polypoid lesions in the stomach (arrowhead).

nephromegaly and multiple hypodense solid lesions within the kidneys. There was mild diffuse enlargement of pancreas with hypodense area within the tail of the pancreas (Fig. 5). In addition, hypodense solid lesions in the adrenal glands and gastric polypoid lesions were detected on abdominal CT (Fig. 6). On CT and US examinations. lymphadenopathy in the paraaortic regions and dilatation of the bile duct were not observed. Thorax CT examination revealed multiple mediastinal and hilar lymphadenopathies. CT scans also showed multiple osteolytic bone lesions with or without cortical destruction in the ribs, vertebrae, scapula, and right sacroiliac joint. Soft tissue masses were also observed associated with some bone lesions. Whole body bone scan using technetium-99m methylene diphosphonate (99mTc MDP) demonstrated abnormal intense uptake located in the cranium, ribs, bilateral humerus, cervical-thoracic-lumbar vertebrae, and femurs bilaterally. Histopathological examination of the biopsy specimen and special marker studies demonstrated NHL of a diffuse large B-cell type. The patient was treated

with both radiotheraphy and systemic CHOP (cyclophosphamide, adriamycin, vincristine, and prednisone) chemotheraphy.

DISCUSSION

Lymphomas can arise from lymphoreticular tissue anywhere in the body and spread through hematogenous dissemination to distant sites [4]. Skeletal involvement occurs in 5-16 % of the cases with non-Hodgkin's lymphoma. It can be of primary (stage I) in less than 1 % of the cases, or more commonly, secondary (stage IV) type in the context of disseminated disease [5]. Lymphoma commonly involves paravertebral lymph nodes and then invades adjacent vertebrae and extends through the intervertebral foramina to the epidural space. Sometimes lymphoma infiltrates only vertebral bone marrow or extradural space [1]. It often involves several vertebral levels and locations predominantly in the upper- and middle thoracic and middle-and lower lumbar segments. In our case, the patient had metastatic spinal lymphomas in multiple locations including cervical, thoracic and lumbar spinal segments and vertebrae predominantly with or without involvement of paravertebral mass.

Typical imaging appearance for non-Hodgkin's lymphomatous infiltration of bone is that of an osteolytic lesion with a permeative "moth-eaten" pattern of bone destruction in the metaphyseal (or less commonly epiphyseal or diaphyseal) region of a long bone. Lesions with both an osteolytic and an osteosclerotic component, or purely osteosclerotic lesions may be also encountered [5]. Conventional radiography is not sufficient for the demonstration of bone involvement [1]. CT can disclose compression fracture or sclerosis of the vertebra, but early bone marrow infiltration may be skipped [1].

On MRI the vertebral lesion usually exhibits decreased signal intensity relative to bone marrow on T1-weighted images like other types of metastases. Sometimes it demonstrates a diffusely hypointense signal in some diseased vertebral bodies as well as superimposed focal areas of lower signal intensity resulting from focal nodules of lymphoma. On T2-weighted images, lymphoma has a slightly higher signal intensity relative to bone marrow or inhomogeneously iso- and hyperintense because of the uneven infiltration of lymphoma [1]. Paravertebral lesions are hypo- or isointense relative to the surrounding muscle tissue on T1-weighted images. Epidural extension from adjacent vertebrae and paravertebral lesions are common growth patterns in spinal lymphoma. They can demonstrate mostly equal or slightly lower signal intensities relative to the cord on T1-weighted spin echo images [1]. In our patient we observed higher signal intensities for some vertebral lesions on T1- and T2-weighted MR images. This finding has been also documented as primary lymphomatous involvement of the bone by other investigators [6,7]. In our patient, MRI examination was also useful to demonstrate location and distribution of the lesions, paravertebral soft tissue masses, compression fractures and retropulsion of the posterior vertebral body into the spinal canal, the degree of compression on the spinal cord and the multiplicity of these lesions.

Primary pancreatic lymphoma is rarely seen, and fewer than 2 % of extranodal NHLs arise within the pancreas. Secondary involvement of the pancreas in patients with diffuse disease is more common representing about 30 % of the patients with NHL [8]. Secondary involvement of the pancreas represents a direct extension from contiguous lymphoid tissue or via lymphogenous spread [8]. The most common findings are abdominal pain and weight loss [8].

CT is the imaging technique most frequently used for the detection and characterization of pancreatic lymphomas. Two different morphologic patterns of pancreatic involvement are

encountered such as a localized, well-defined tumoral form, and a diffuse infiltrating form with enlargement of most of the pancreatic gland [8,9]. A bulky homogeneous tumoral mass without marked dilatation of Wirsung's duct, associated with patency of the peripancreatic vessels, lack of calcification or necrosis, and lymph node involvement below the level of the renal veins have been described suggesting the diagnosis of pancreatic lymphoma rather than adenocarcinoma [8]. In our case, CT examination demonstrated diffuse enlargement of pancreas with a small hypodense area within the tail. US examination also revealed ill-defined heterogeneous hypoechoic areas especially within the head of the pancreas.

Renal lymphomas are most often seen in conjunction with multisystemic, disseminated lymphomas [9,10]. Lymphoma typically involves the kidney in one of several recognizable patterns. These patterns include single or multiple masses, invasion from contiguous retroperitoneal disease, perirenal disease, and diffuse renal infiltration [10]. In our case US and CT examinations manifested multiple renal mass lesions bilaterally.

NHL results in radiologically visualized adrenal involvement in 4 % of the cases. The incidence of primary adrenal lymphoma is quite rare. There is usually other retroperitoneal involvement. Lesions are usually bilateral and resemble metastases [11]. Our case also had bilateral lymphomatous adrenal involvement.

The presenting complaint of our patient was back pain. Back pain is a common symptom with numerous etiologic factors. Tumor metastasis to the spinal cord can result in diverse symptoms, depending on the level of spinal compression Tumoral compressions of the spinal cord result in neurological symptoms and medical emergencies. Kombogiorgas et al. reported a case of non-Hodgkin lymphoma with a history of rapidly progressive tetraparesis as a result of spinal cord compression [12]. Some patients might have only back pain which they or their clinicians might not attribute importance, as is in our case. Every clinician should remember that malignancy is one of the reasons of back pain, even if in youngsters.

CONCLUSION

An interesting case of disseminated lymphoma , presenting initially with spinal cord compression which led to delayed diagnosis was reported in this study. Early diagnosis and treatment may prevent further damage to the nervous system.

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