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Case Report

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Primary spinal extramedullary diffuse large B–cell lymphoma presenting with initial spinal cord compression: a case report

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ABSTRACT

Extranodal lymphomas, by definition, can involve any organ or tissue. Brain parenchyma, spinal cord, eyes, cranial nerves, and meninx are extranodal regions that show involvement at much lower rates. It is quite rare for lymphoma patients to present to the hospital with symptoms and findings associated with spinal cord compression as the initial presentation. This condition can lead to irreversible autonomic dysfunction, and motor and sensory loss. Here, we present a rare primary spinal intradural extramedullary diffuse large B–cell lymphoma (DLBCL) case who presented with acute neurological symptoms and no findings of cerebral involvement or involvement at any other site. Magnetic resonance imaging (MRI) was performed for the spinal region. Surgical procedure was performed for the detected mass. Immunohistochemical staining of the excised mass was performed. In the surgical operation, the mass showed partial invasion of the vertebral bone and the surrounding muscle. Considering the rapid progression of the neurological deficit, surgical resection was performed primarily for spinal decompression. Subsequently, 6 courses of rituximab, cyclophosphamide, doxorubicin, vincristine, and prednisolone (R–CHOP) chemotherapy regimen were given for the residual tissue, and radiotherapy was administered to the involved region. Timely and effective treatment achieved a complete recovery of motor and sensory function. The patient remains in complete remission without clinical or radiological relapse under follow-up after nearly 4 years. Although it is a rare condition, primary spinal intradural extramedullary DLBCL disease should be suspected in patients who present with acute neurological symptoms and have no signs of cerebral involvement or involvement elsewhere, and necessary investigations should be performed by paying attention to this.

Keywords: Epidural, diffuse large B–cell lymphoma, lymphoma, spinal cord compression, spinal non–Hodgkin lymphoma

INTRODUCTION

Primary central nervous system (CNS) lymphomas comprise 4% of all brain tumors and 4-6% of extranodal lymphomas. The extranodal disease typically manifests as intermediate and high-grade non–Hodgkin lymphoma (NHL).[1] Extranodal lymphomas define the involvement of any organ or tissue, while the brain parenchyma, spinal cord, eyes, cranial nerves, and meninx are extranodal regions that show involvement much less commonly. Approximately 90-95% of primary CNS lymphomas are large B–cell lymphomas (DLBCL) and the risk is higher in patients with inherited immunodeficiency syndromes, HIV/AIDS, and in organ transplant recipients.[2,3] Spinal cord lymphoma is the least prevalent form of CNS lymphoma and is associated with a poor prognosis if untreated. It can involve the lower cervical or upper thoracic regions and manifests symptoms associated with the involved region.[4] Magnetic resonance imaging (MRI) examination can demonstrate an expansile mass lesion. The differential diagnosis includes glioma, metastatic tumor, toxoplasma infection, sarcoidosis, abscess, and progressive multifocal leukoencephalopathy. A tissue biopsy is essential for a definitive diagnosis.

As the initial presentation, it is quite unusual for lymphoma patients to present to the hospital with symptoms and findings associated with spinal cord compression. However, spinal cord compression, which can lead to irreversible autonomic dysfunction, and motor and sensory loss, occurs in 0.1-6.5% of NHL patients and can be the presenting problem.[5] Cases of primary spinal DLBCL are quite rare, with only a few reported cases and short case series.[6] Here, we present a rare primary isolated spinal intradural extramedullary DLBCL case who presented with acute neurological symptoms and did not show cerebral involvement or involvement at any other site.

CASE

41–year–old male patient presented to our hospital with thoracic back pain and progressive complaints of weakness, numbness, and difficulty in ambulation in bilateral lower extremities. His history included hypertension controlled by medical treatment. His history was unremarkable for recent diseases and medication and narcotic drug use. Neurological examination revealed 3/5 strength in bilateral legs and a sensory deficit up to the level of the umbilicus. Bilateral lower extremities showed loss of sensation and reduced motor function, with no fecal or urinary incontinence. On other systemic examinations, there were no hepatomegaly, splenomegaly, lymphadenopathy, or palpable masses. On spinal MRI examination, a well–circumscribed intradural extramedullary mass with a craniocaudal extension of 6 cm and an AP diameter of 1 cm that was isointense to the spinal cord on T1–weighted sequences and slightly hyperintense on T2–weighted series, and showed diffuse homogenous contrast enhancement after intravenous contrast agent injection was determined between the vertebral levels T6 and T8. Also, the narrowing of the spinal cord secondary to compression by the lesion was visualized (**Figure 1 A, B**).

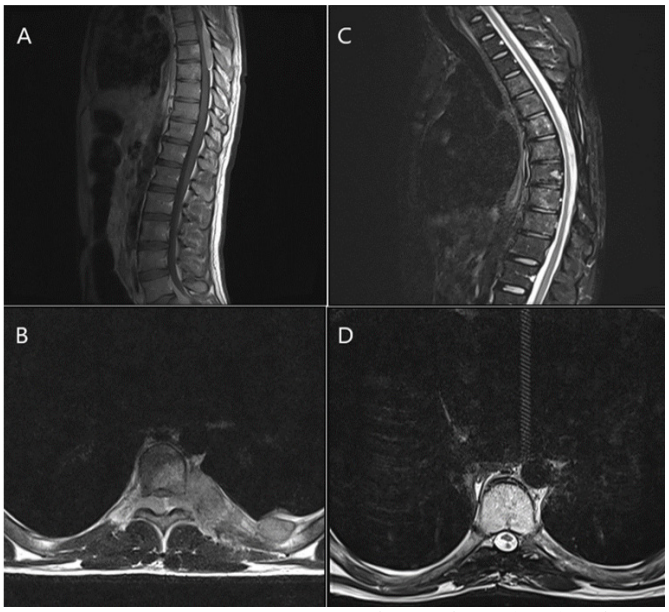

Figure 1. Magnetic resonance images at the initial visit (**A, B**) and post–treatment visit (**C, D**). Sagittal (**A**) plane at T6–8 and axial (**B**) planes at T7 show abnormal lesion located intradural extramedullary space of the thoracic spinal canal. Sagittal (**C**) and axial (**D**) planes had been shown complete disappearance of post–treatment with chemotherapy

Considering the rapid progression of the neurological deficit, resection of the dural mass and spinal decompression were planned. In the surgical operation, the mass showed partial invasion of the vertebral bone and the surrounding muscle. The mass invading the dura was resected and laminectomy was performed at T6–T9. The mass extended to the right and left neural foramina and this section could not be completely resected as it was not anatomically suitable. On histopathological examination of the mass, there was diffuse malignant infiltration by large atypical lymphoid cells with prominent nucleoli and a coarse chromatin structure. On immunohistochemical examination, neoplastic cells showed; CD20 (+, diffuse), CD3 (–), MPO (–), Tdt (–), CD1a (–), S100 (–), ALK (–), CD68 (–), CK (–), actin (–), vimentin (–) staining, and the Ki67 proliferation index was 70% (**Figure 2**). The pathology department reported the mass to be consistent with a diffuse large B cell lymphoma (centroblasts type). Cervical–thoracic–abdominopelvic computed tomography (CT) was performed to determine the extent of the disease, and no masses, organomegaly, or enlarged lymph nodes were detected. Bone marrow aspiration and biopsy did not show bone marrow involvement. Other detailed laboratory examinations did not reveal any pathological results.

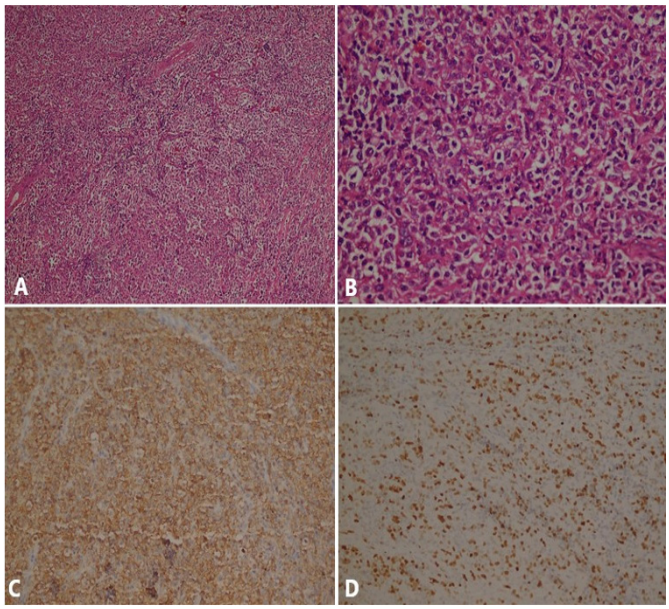

Figure 2. A. Tissue shows diffuse infiltration (Hematoxylin–eosin ×100). **B.** Atypical lymphoid cells with narrow cytoplasm, vesicular nuclei, and occasional prominent nucleoli (Hematoxylin–eosin ×400). **C.** On immunohistochemical examination, diffuse staining with CD20 in neoplastic lymphoid cells (×200). **D.** Ki67 proliferation was evaluated as high (70%) (×200)

After diagnosis, the patient was planned to undergo chemotherapy and radiotherapy. The patient received chemotherapy consisting of rituximab cyclophosphamide, doxorubicin, vincristine, and prednisone (R–CHOP) and was administered with six cycles. After chemotherapy, radiotherapy was given at a total dose of 40 Gy as 2 Gy per fraction. The strength of the bilateral lower extremity muscle groups showed daily improvement and the patient was able to walk normally with two courses of chemotherapy, after approximately six weeks. The neurological examination performed at the end of the patient's treatment revealed no sensory or motor deficits. Follow-up spinal MRI showed no evidence of any mass. The patient remains in remission without clinical or radiological relapse under follow-up after nearly 3 years.

DISCUSSION

A substantial portion of spinal compression cases is linked to metastatic tumors (lung carcinoma, prostate carcinoma, multiple myeloma, etc.), with a rate as large as 85%. The annual incidence of spinal cord compression among patients who showed cancer–related death was 3.4%, and approximately 15% of these cases were of Hodgkin's lymphoma and non–Hodgkin lymphoma.[5]

Compression associated with primary NHL is a condition of infrequent occurrence in clinical practice. Patients in

this group typically receive a diagnosis by presenting with neurological deficits arising from the local effect of the mass, or, although rarely, with back pain. Primary spinal lymphomas may not be clinically and radiologically distinct from other spinal lesion. Spinal intramedullary lymphomas are not associated with pathognomonic radiological findings, however, MRI can provide accurate and reliable data for the correct localization of the mass and post–treatment follows–up.[7] Accordingly, our case presented progressive neurological symptoms that resulted from the mass effect. Before the diagnosis, the lesion was detected using MRI, and post–treatment follow–up also utilized MRI for the evaluation of the response and the monitoring of relapse.

Spinal DLBCL is an aggressive form of lymphoma that is linked to rapid progression and mortality if untreated. The importance of early diagnosis is evident from the high morbidity and mortality rates, particularly in older patients. In spinal DLBCLs, aggressive surgical intervention is usually not recommended as the cases are both chemo– and radiosensitive, and as it can lead to a postoperative delay in initiation of chemotherapy and risk of neurological deficits.[8] The optimal treatment of DLBCL with spinal compression is reported to be controversial. Treatments usually include chemotherapy, radiotherapy, surgery, or a combination of these methods. Chemotherapy regimens based on rituximab continue to represent the standard of care in the management of DLBCL. Among these, R-CHOP remains the most widely employed, while polatuzumab vedotin, rituximab, cyclophosphamide, doxorubicin, prednisone (Pola-R-CHP) and rituximab, etoposide, cyclophosphamide, doxorubicin, vincristine, prednisone (R-EPOCH) are also utilized as alternative frontline therapeutic options.[9-12] In these patients, intrathecal therapy is not routinely planned; however, in cases considered high-risk, two cycles of systemic high-dose methotrexate are recommended.[3] Radiotherapy is effective in all CNS lymphomas including DLBCL; however, it must not be employed as the first–line treatment due to neurotoxicity and not offering a survival advantage.[13]

On the other hand, since NHLs are highly chemosensitive and radiosensitive tumors, certain clinicians state that non–surgical treatments involving chemotherapy and radiotherapy can achieve rapid improvement of spinal cord and nerve root compression.[14] However, decompression surgery can be considered a primary option for the improvement of neurological symptoms.[15] Accordingly, our case primarily underwent surgical decompression for both diagnostic and treatment purposes, as there were neurological deficits induced by spinal compression. Subsequently, for the residual mass, first chemotherapy, and then radiotherapy was administered as a combined treatment. As a result of this treatment approach, the patient has now remained relapse–free under follow–up for nearly 3 years.

CONCLUSION

As a result, the differential diagnosis of patients who present with a spinal mass should be made carefully. It must be considered that, although rarely, DLBCLs can present as massive disease-causing spinal compression, and that clinically significant improvement can be achieved by timely and effective treatment. In patients who present with spinal compression, early decompression, particularly using surgery, is of great importance. Considering that spinal DLBCL is a malignant disease, appropriate treatment approaches play a vital role in achieving neurological recovery, longer survival times, and better life quality.

ETHICAL DECLARATIONS

Informed Consent

Written informed consent was obtained from the patient included in this report. Signed consent forms are retained by the authors and are available upon request.

Peer Review Process

This report underwent external peer review.

Conflict of Interest

The authors declare no conflicts of interest.

Financial Disclosure

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Author Contributions

Concept/Design: ÖC, ÖE, CD; Data Collection and/or Processing: ÖC, AD, MA; Analysis and/or Interpretation: ÖC, ÖE, İA, CD; Literature Search: ÖC, AD, MA; Writing the Manuscript: ÖC, ÖE; Critical Review: CD, AD, İA. All authors read and approved the final manuscript.

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