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Case report

Diffuse large B-cell lymphoma with contemporary involvement of central and peripheral nervous system: A case report and literature review

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ABSTRACT

Introduction: Simultaneous involvement of the peripheral nervous system (PNS) and central nervous system (CNS) during the same period in diffuse large B-cell lymphoma (DLBCL) is rarely documented. In this particular case, the diagnosis of diffuse large B-cell lymphoma was pathologically confirmed, with invasion into the basal ganglia, diencephalon, and several peripheral nerves. The initial clinical manifestations were dyspnoea and hyperventilation.

Case presentation: The patient presented to the hospital with fatigue, dyspnoea, and limb pain for over 7 months, accompanied by progressive breathlessness and unconsciousness in the last 6 days. Initial treatment with glucocorticoids for Guillain-Barre syndrome (GBS) proved ineffective in controlling the severe shortness of breath and hyperventilation, necessitating the use of ventilator-assisted ventilation. 18-Fluorodeoxyglucose positron emission tomography/computed tomography (^{18}FDG PET/CT) showed that the basal ganglia, brainstem, and multiple peripheral nerves were thickened and metabolically active. There were atypical cells in the cerebrospinal fluid; the pathology indicated invasive B-cell lymphoma, demonstrating a propensity toward diffuse large B-cell lymphoma (DLBCL). After receiving chemotherapy, the patient regained consciousness and was successfully weaned off ventilator assistance but died of severe pneumonia.

Discussion: The early clinical manifestations of DLBCL lack specificity, and multifocal DLBCL complicates the diagnostic process. When a single primary disease cannot explain multiple symptoms, the possibility of DLBCL should be considered, and nervous system invasion should be considered when nervous system symptoms are present. Once nervous system involvement occurs in DLBCL, whether the central or peripheral nervous system, it indicates a poor prognosis.

1. Introduction

Lymphoma is one of the most common malignancies in China, with Non-Hodgkin's Lymphoma (NHL) ranking 10th in incidence and mortality among males [1]. Involvement of the central nervous system (CNS) only occurs in 5–25% of NHL cases, correlating with

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a poor prognosis and short-term survival [2]. The most common pathologic type of NHL is diffuse large B-cell lymphoma (DLBCL), which exhibits a higher incidence of CNS involvement in patients with immune dysfunction [2,3]. DLBCL can originate in the lymph nodes, gastrointestinal tract, CNS, or anywhere in the body; however, simultaneous involvement of the peripheral nervous system (PNS) and CNS is rare [4,5]. We report a case of DLBCL involving both the PNS and CNS, initially misdiagnosed as Guillain-Barré syndrome (GBS). We also present a literature review aiming to guide accurate diagnosis of DLBCL involving both the CNS and PNS, prevent misdiagnosis, and improve selection of treatment.

2. Case presentation

A 73-year-old male presented to our emergency department on December 10, 2021 with a history limb pain and fatigue lasting more than 7 months, as well as progressive breathlessness and unconsciousness over the last 6 days. He was initially admitted to the first hospital on May 4, 2021 due to left leg and right arm pain and weakness. After a lumbar magnetic resonance imaging (MRI) examination, the patient was diagnosed with lumbar spinal stenosis and sciatic nerve damage. Symptoms improved after he received anti-inflammatory, pain relief, nutritional nerve, and physical therapy, and he was discharged on May 17, 2021. Soon after discharge, on July 15, 2021, the symptoms recurred, necessitating readmission to the hospital. The relevant parameters improved upon examination. Cerebrospinal fluid (CSF) examination revealed a white blood cell count of 800,000/L, red blood cell count of 0–3/HP, total protein level of 619 mg/L, and high anti-sulfatide IgG titres. Electromyography (EMG) showed bilateral median nerve and left sciatic nerve dysfunction. Electroencephalography (EEG) showed mild abnormalities and the results of the flash visual evoked potential test were negative. The patient was diagnosed with autoimmune peripheral neuropathy and received 40 mg methylprednisolone daily for neurotrophic therapy. As symptoms improved, the patient was discharged on August 2, 2021.

However, these symptoms recurred and gradually worsened, necessitating admission to a second hospital on November 10, 2021. Arterial blood gas analysis with room air revealed hyperventilation (pH 7.504, $PaCO_2$ 21.2 mmHg, AB 16.6 mmol/L, SB 20.5 mmol/L). CSF analysis revealed a total protein concentration of 444 mg/L. MRI revealed bilateral mesencephalon atrophy, leukoaraiosis, and mild cerebral atrophy and EMG showed peripheral nerve damage. The patient was diagnosed with GBS at the hospital and received methylprednisolone pulse therapy (1 g/day for 3 days, 0.5 g/day for 3 days, 0.12 g/day for 3 days) and subsequent maintenance therapy (60 mg/day). His limb weakness improved; however, he gradually developed delirium, slurred speech, difficulty in swallowing, shortness of breath, fever, night sweats, and weight loss of approximately 15 kg in 7 months. The patient was transferred to our hospital due to worsening shortness of breath, severe respiratory alkalosis, hypocapnia ($PaCO_2$ 11.9 mmHg), and unconsciousness. He was in a state of lethargy at admission, with 20–30 deep breaths per minute, articulation disorder, limbs muscle atrophy, diminished tendon reflexes, and a negative pathological reflex, but without lymphadenectasis.

Clinical manifestations of hyperventilation and respiratory alkalosis were alleviated through sedatives and muscle relaxants combined with mechanical ventilation. Anti-GM1, -GD1a, -GD1b, and -sulfatide antibody levels in the CSF and serum were normal.

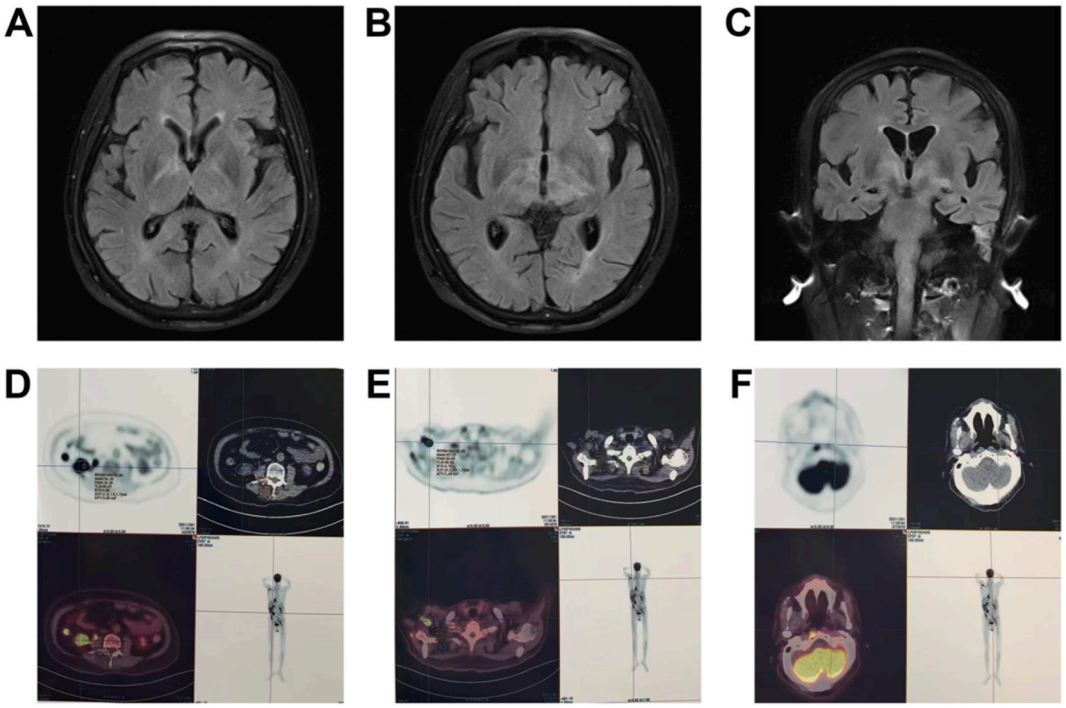

Fig. 1. Changes of head MRI (A–C) and systemic ^{18}FDG-PET (D–F) in this patient.

CSF examination revealed albuminocytological dissociation; white blood cell count, 500,000/L; total protein, 1038.1 mg/L; glucose 5.4 mmol/L, and Pandy's reaction was weakly positive. Repeated EMG showed peripheral nerve damage, abnormal F-wave, a suspicious conduction block in the left ulnar nerve, and spontaneous potential in the left anterior tibial tract at rest.

Brain MRI revealed abnormal signals in both periventricular areas (Fig. 1A–C). Examination with 18-Fluorodeoxyglucose positron emission tomography (^{18}FDG-PET) revealed diffusely increased FDG uptake in the bilateral basal ganglia, brainstem, enlargement and thickening of the right axillary nerve and left sciatic nerve, FDG-avid masses next to the right side and bottom of the right ventricle and the psoas major, and a linear FDG-avid lesion in the lateral right adrenal gland, suggestive of lymphoma (Fig. 1D–F).

Two bone marrow biopsies showed no lymphomatous infiltration; however, CSF cytology revealed an elevated lymphocyte count featuring atypical lymphocytes with distinct characteristics such as varying cell sizes, enlarged cell bodies, surface protuberances, variable nuclear shapes, and protruding nuclei, all of which are strongly indicative of lymphoma (Fig. 2 G - J). A biopsy of the right psoas major muscle mass guided by interventional radiology revealed aggressive B-cell lymphoma prone to DLBCL, a non-germinal centre B cell immunophenotype (Fig. 3 K - N). Immunohistochemistry revealed positive markers; CD20 (+), CD79a (+), CXCL13 (+), Bcl-6 (+), MUM1 (+), and CD21 (+); the percentage of Ki-67-positive cells was 95%. In situ hybridization results were negative for Epstein-Barr virus -encoded RNA (EBER) .

After confirming the pathological diagnosis, the patient was transferred to an oncology centre hospital, where he was administered oral orelabrutinib (150 mg/day), temozolomide (150 mg/day), and intravenous dexamethasone (10 mg every 12 h). Subsequently, hyperventilation and unconsciousness were alleviated, his overall condition improved, and he was successfully weaned from mechanical ventilation and recovered consciousness. However, he died of severe pneumonia a month later on February 2, 2022.

3. Discussion

Lymphoma is a lymphoid malignancy, with almost 90% of cases being NHL. According to the latest GLOBOCAN data, NHL was responsible for 544,000 new cases and 260,000 deaths worldwid In 2020 [6]. And DLBCL is the most common lymphoid malignancy in adults [7]. Secondary central nervous system lymphoma (SCNSL) refers to the involvement of the CNS in patients with aggressive lymphoma, whereas neurolymphomatosis (NL) refers to isolated peripheral nervous system infiltration. CNS involvement is uncommon in patients with DLBCL compared to other high-grade lymphomas, such as Burkitt's lymphoma and lymphoblastic lymphoma [8,9]. In addition, the incidence of CNS involvement has been further reduced (<5%) since the introduction of rituximab-containing regimens as the first-line therapy [10]. Meanwhite NL is also a rare clinical disease, although its precise incidence is unknown, it is estimated to occur in approximately 0.2% of all NHL patients, based on a report by Choi et al. [11]. NL with SCNSL is uncommon and has been the subject of very few case reports worldwide. Here, we report a rare case of NL in a patient with SCNSL.

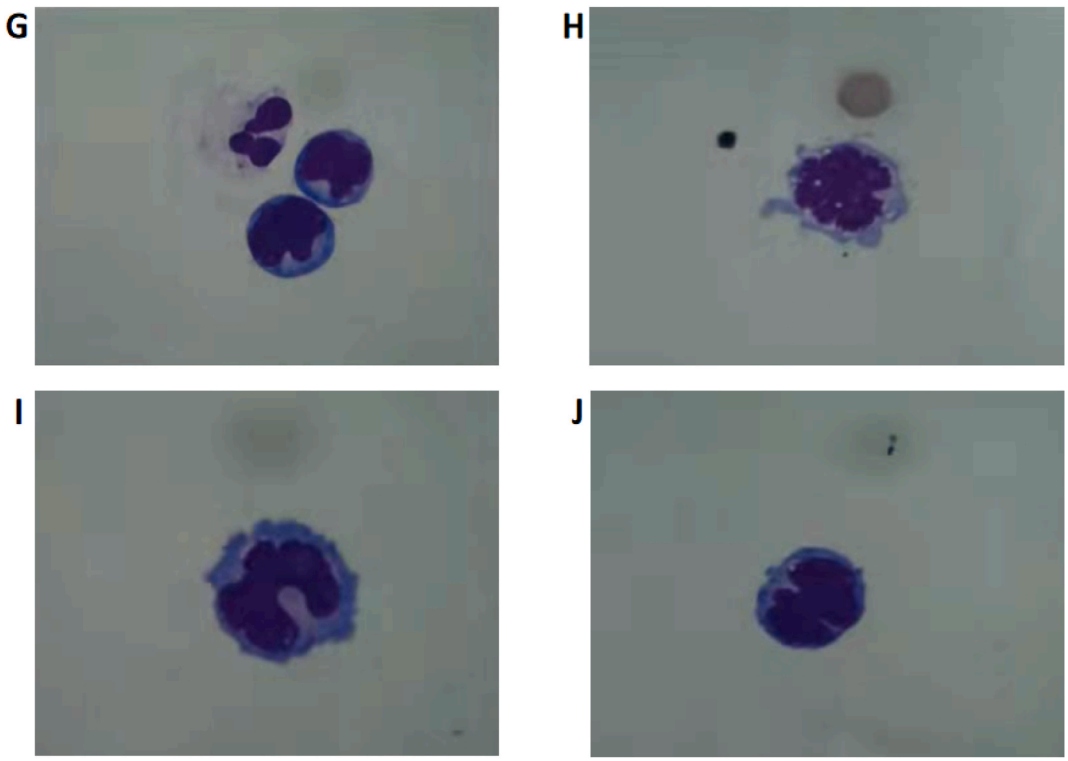

Fig. 2. Anomalous lymphocytes (G–J) were found in the cerebrospinal fluid of this patient.

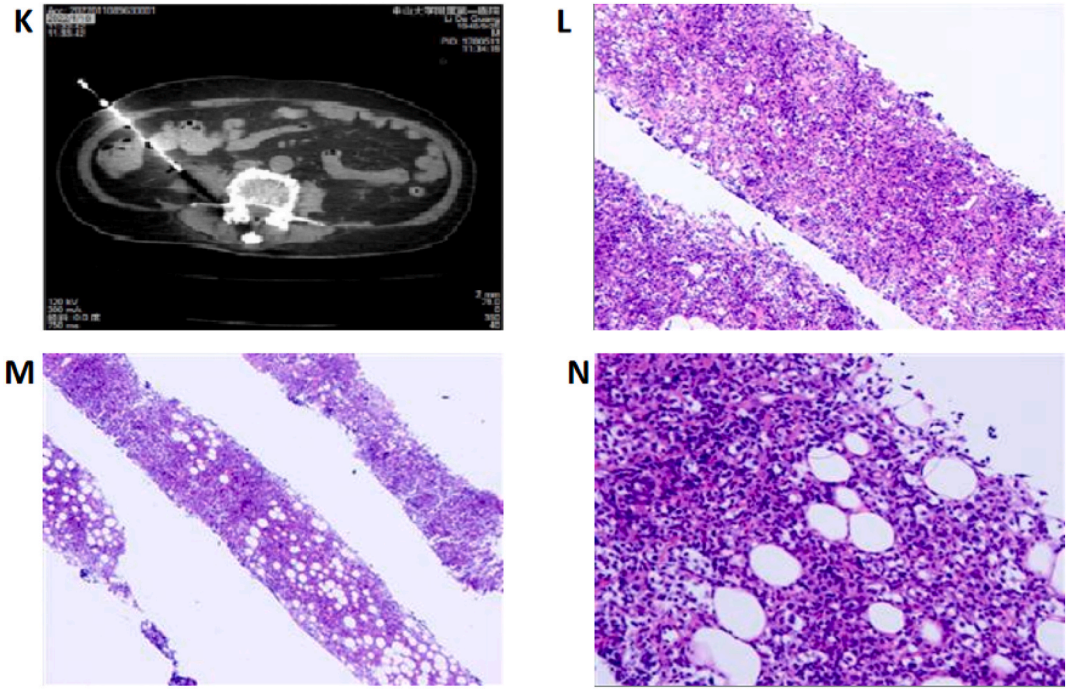

Fig. 3. The patient was diagnosed with aggressive B-cell lymphoma by biopsy of the left psoas major (K–N).

DLBCL can occur anywhere in the body and has a variety of manifestations depending on its location. Most symptoms are nonspecific, particularly in the early stages. Approximately 30–40% of patients present with only general symptoms, such as B-cell symptoms (fever, weight loss, and night sweats) and signs, including painless lymphadenectasis, splenomegaly, and regional masses [12]. Symptoms of CNS involvement also depend on the infiltration location, and patients may suffer from multiple cranial nerve damage, chronic headache, personality changes, altered mental status, and seizures [13,14]. According to previous reports, although SCNSL involvement can be leptomeningeal, parenchymal, or both, the parenchyma is rarely involved [15]. However, in a study by Malikova et al., most patients had parenchymal disease with multiple lesions, which might be related to the poor blood-brain barrier (BBB) penetration of rituximab [16]. Several studies in the rituximab era revealed that the median time from lymphoma diagnosis to CNS involvement in patients with DLBCL was approximately 9 months. Of these cases, 80% occurred after first-line therapy, mainly during the initial relapse period, while the remainder occurred during first-line therapy, with 50–60% of patients showing isolated brain parenchyma involvement [17–20]. The most common symptoms included painful peripheral neuropathy (76%), sensory or motor dysfunction (mixed motor-sensory neuropathy), and cranial neuropathy [21–23]. The initial symptoms in the case presented herein were characteristic of PNS dysfunction and included pain and weakness in the limbs with no lymphadenopathy, followed by a gradual presentation of B cell symptoms. However, due to the nonspecific symptoms and relief post-steroid treatment, lymphoma was overlooked in the first two hospitals. The patient was later transferred to our hospital with severe shortness of breath, hyperventilation, and progressive unconsciousness. SCNSL manifesting as central neurogenic hyperventilation (CNH) is rare and is typically observed in comatose patients. There are fewer than 30 case reports of conscious patients with CNH in the literature, mostly involving primary central nervous system lymphoma (PCNSL) [24,25].

The diagnosis of DLBCL requires pathological biopsy and immunohistochemistry. Stereotactic biopsies are considered the standard procedure for PCNSL diagnosis but may vary for SCNSL diagnosis. SCNSL is diagnosed when patients with DLBCL present with typical symptoms of CNS infiltration, imaging abnormalities, and pathological evidence [26]. Although brain MRI changes are alleviated after exposure to corticosteroids, several studies recommend it as the preferred imaging modality for diagnosing SCNSL [26–28]. ^{18}FDG-PET/CT is currently the most sensitive and specific imaging technique for the detection of lymphoma and plays an increasingly important role in the diagnosis of NL [29]. CSF cytology and flow cytometry can also confirm the diagnosis of SCNSL. Biopsy of the involved neurological structures is the gold standard for the diagnosis of NL. However, it only has a sensitivity of 88%, with limitations due to the difficulty in obtaining biopsy samples from the target nerves and some negative findings, as well as the potential complications of neurological damage and sequelae. Notably, only half of the patients in previous reports underwent nerve biopsies [21,30]. For patients presenting with characteristic manifestations of primary tumours in the clinic, chemotherapy can be started empirically without a biopsy [31]. Our patient was diagnosed with non-GCB DLBCL based on right lumbar biopsy and immunohistochemistry results. ^{18}FDG-PET/CT indicated lymphoma infiltration of the right axillary nerve and left sciatic nerve, which, combined with the clinical symptoms, suggested a diagnosis of NL. Furthermore, brain MRI, ^{18}FDG-PET/CT, and CSF findings revealed intracranial involvement, which can be diagnosed as SCNSL.

A study involving 1085 cases of DLBCL revealed a 5-year overall survival (OS) of 62.5%, and a 5-year progression-free survival of

54.2% [12]. DLBCL with CNS involvement may indicate a poor prognosis, and a previous study showed that the median OS of NL cases was 10 months [21]. Another study showed that the median OS of patients with SCNSL was only 3.9 months, and the 2-year OS rate was only 20% [20]. Therefore, patients with lymphoma with a high risk of CNS invasion require greater attention, and timely and appropriate clinical management.

Previous studies revealed that the cerebral parenchyma is the main site of CNS infiltration lesions in patients with lymphoma, as drugs in the subarachnoid space can barely cross the BBB to achieve adequate concentrations in the CNS. Consequently, guidelines from the National Comprehensive Cancer Network (NCCN) and European Society for Medical Oncology (ESMO) have not yet defined the optimal protocol for CNS prophylaxis [32]. Drug delivery via intrathecal (IT) injection is the most common method of CNS prophylaxis in patients with lymphoma. Moreover, CNS prophylactic management recommended in the NCCN guidelines includes four to eight doses of IT or Ara-C (or both), or a high dose of intravenous methotrexate [33]. According to the guidelines of the British Committee for Standard in Haematology on the prevention of SCNSL, CNS prevention therapy should be administered to patients with high-grade NHL and extranodal localization (e.g., testicular, breast), raised serum lactate dehydrogenase (LDH), or more than one extranodal localization [34]. The patient in this report had a high risk of CNS involvement, presenting with stage IV lymphoma manifestations and multiple extranodal lesions, and was eventually diagnosed with NL and SCNSL at our hospital. After the diagnosis was confirmed, the patient was transferred to an oncology centre, where he underwent orelabrutinib, temozolomide, and corticosteroid therapy. Although he regained consciousness and his symptoms of discomfort were temporarily relieved, he eventually died of severe pneumonia; his OS was lower than the median for SCNSL and NL reported in previous studies, suggesting a worse prognosis for the combination of SCNSL and NL. However, further clinical studies are required for its validation.

4. Conclusion

Over the course of the disease, DLBCL can present with various non-specific early-stage symptoms and multiple lesions, complicating the diagnosis and potentially resulting in misdiagnosis. Therefore, a DLBCL diagnosis should be considered when a single primary disease cannot explain the clinical presentation, and clinicians must be more aware of potential CNS infiltration when patients exhibit neurological symptoms. Once DLBCL progresses to CNS infiltration, clinical outcomes are poor, and prompt diagnosis and appropriate treatment are required to improve the prognosis.

Ethical statement

This study was approved by the Ethics Committee of The First Affiliated Hospital of Sun Yat-sen University (approval number: [2022] 446), Patients were consented by an informed consent process that was reviewed by the Ethics Committee of The First Affiliated Hospital of Sun Yat-sen University and certify that the study was performed in accordance with the ethical standards as laid down in the 1964 Declaration of Helsinki.

Informed consent statement

We declare that informed consent was obtained from patient's families prior to inclusion, and they consented to the publishing of all images, clinical data, and other data included in the manuscript.

Data availability statement

The authors declare that all medical records in the report are true and objective, and we can provide original medical records if necessary.

CRediT authorship contribution statement

Chuwen Tang: Writing – original draft, Validation, Methodology, Investigation, Formal analysis, Data curation. **Peng Jiang:** Writing – original draft, Investigation, Formal analysis, Data curation. **Jinhui Tang:** Visualization, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Jinli Liao:** Supervision, Resources, Methodology, Formal analysis, Data curation, Conceptualization. **Qingli Zeng:** Writing – review & editing, Visualization, Resources, Methodology, Formal analysis, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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