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CASE REPORT

Asian variant intravascular large B-cell lymphoma with highly suspected central nervous system involvement: A case report

Yong-Pyo Lee, Seung-Myoung Son, Jihyun Kwon

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Abstract

BACKGROUND

Intravascular large B-cell lymphoma (IVLBCL) is a rare subtype of extranodal lymphoma. In particular, the Asian variant of IVLBCL is characterized by hemophagocytic lymphohistiocytosis along with bone marrow involvement. However, central nervous system (CNS) involvement is uncommon in this variant compared to the Western variant. Here, we report a case of typical Asian variant IVLBCL with highly suspected CNS involvement and discuss the nature of the disease and its genetic aberration.

CASE SUMMARY

A 67-year-old female patient complained of gradually worsening cognitive impairment. While hospitalized, she developed a high fever and showed marked bicytopenia. Intracranial imaging revealed a suspected leptomeningeal disease. Although no malignant cells were found in the cerebrospinal fluid (CSF), the protein and lactate dehydrogenase levels in CSF were increased. Bone marrow examination revealed an increased number of hemophagocytic histiocytes, and ^{18}F-fluorodeoxyglucose (FDG) positron emission tomography with computerized tomography scan revealed increased FDG uptake in both adrenal glands, the liver, and the right ethmoid sinus. A tissue biopsy showed atypical large lymphoid cells with prominent nucleoli in the vessels, and the tumor cells were positive for CD20, BCL2, BCL6, and IRF4/MUM1. In addition, targeted sequencing identified *MYD88*, *TET2*, and *PIM1* mutations. Consequently, we diagnosed the patient with the Asian variant of IVLBCL with highly suspected CNS involvement.

CONCLUSION

Suspicion of IVLBCL and immediate diagnosis lead to timely treatment. Moreover, careful CNS examination at diagnosis is recommended.

Key Words: Intravascular large B-cell lymphoma; Asian variant; Hemophagocytic lymphohistiocytosis; Central nervous system involvement; Genetic alteration; Case report

Core Tip: Intravascular large B-cell lymphoma (IVLBCL) is a rare but clinically aggressive lymphoproliferative disease. Given its aggressive nature, immediate diagnosis of IVLBCL and timely treatment are critical for better clinical outcomes. As central nervous system (CNS) involvement adversely affects prognosis if IVLBCL, active CNS examination is required at diagnosis. In addition, along with conventional pathology, targeted sequencing contributes to diagnosis and provides a basis for use of targeted agents. Here, we report a case of Asian variant IVLBCL with highly suspected CNS involvement. We first describe the clinical course of disease and then discuss the genetic aberrations found in the patient.

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INTRODUCTION

Intravascular large B-cell lymphoma (IVLBCL), characterized by growth of lymphoma cells within the lumen of blood vessels, is a rare type of lymphoid malignancy[1]. According to the World Health Organization classification, IVLBCL is divided into classic, hemophagocytic syndrome–associated, and isolated cutaneous variants. In addition, classification into Asian and Western variants according to the clinical manifestation and geographic distribution is also practiced[2,3]. The Asian variant of IVLBCL predominantly involves the liver, spleen, and bone marrow and often accompanies hemophagocytic lymphohistiocytosis (HLH), while the Western variant frequently affects the skin and central nervous system (CNS)[3]. Aside from its aggressive nature, IVLBCL patients usually present non-specific symptoms only, which can delay accurate diagnosis and ultimately lead to dismal clinical outcomes. In addition, due to the alterations of various molecules and chemokines that regulate the interaction between lymphoma cells and vascular endothelial cells[1,4], extravascular invasion is unusual, and overt lymphadenopathy is rare compared to other types of lymphoma. Thus, although there has been research on the ^{18}F-fluorodeoxyglucose (FDG) positron emission tomography with computed tomography (PET-CT) features of IVLBCL[5], the definitive role of PET-CT imaging in the diagnosis and staging of IVLBCL remains ambiguous[6]. Here, we report a characteristic Asian variant IVLBCL patient with HLH and highly suspected CNS involvement. We describe the patient's clinical features and the course of disease and discuss observed genetic aberrations.

CASE PRESENTATION

Chief complaints

A 67-year-old female patient visited the department of neurology for deteriorating cognitive function.

History of present illness

The patient was able to walk with a cane and take care of herself. However, upon presentation, her cognitive function had deteriorated, she could not recognize her neighbors, and she had difficulty walking unassisted.

History of past illness

She had been diagnosed with cerebellar ataxia a few years prior and was on the medications such as cilostazol and atorvastatin.

Personal and family history

She had no personal or family history.

Physical examination

At the time of examination, she had a mild fever of 37.8°C, but her other vital signs were stable, and she reported no symptoms other than deteriorated cognitive function. There was no sensory deficit, and motor power was intact, although her coordination was poor.

Table 1 Patient characteristics and laboratory findings at diagnosis

Sex/age (yr)	ECOG	Disease involvement sites	Ann Arbor stage	IPI	Cell of origin	WBC (10^3/μL)	Hb (g/dL)	Plt (10^3/μL)	LDH (IU/L)	CRP (mg/dL)	Ferritin (ng/mL)
F/67	4	Right ethmoid sinus, liver, spleen, bilateral adrenal glands	IV	5	Non-GCB	5.35	8.5	77	891	5.26	835

CRP: C-reactive protein; ECOG: Eastern Cooperative Oncology Group performance status; GCB: Germinal center B-cell; Hb: Hemoglobin; IPI: International Prognostic Index; LDH: Lactate dehydrogenase; Plt: Platelet; WBC: White blood cell.

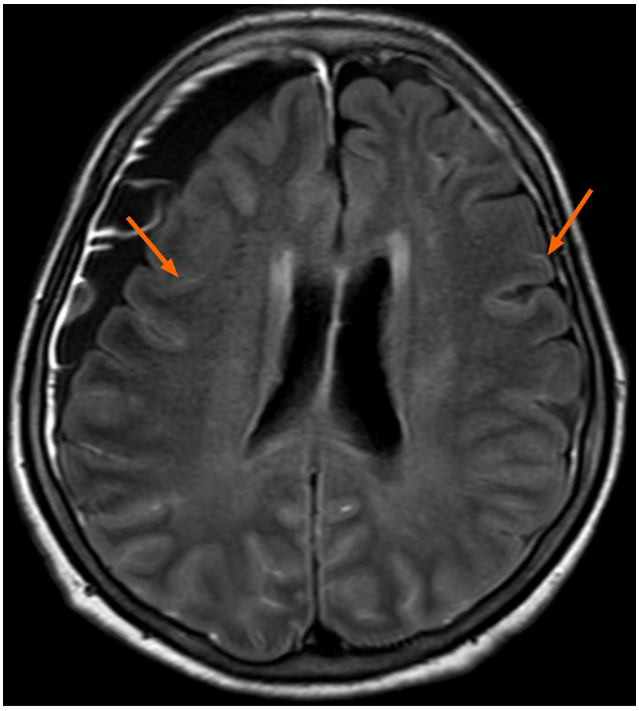

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Figure 1 Axial T2-FLAIR brain magnetic resonance imaging reveals pachymeningeal enhancement in both frontoparietal convexities.

Laboratory examinations

Laboratory testing confirmed bicytopenia (hemoglobin, 8.5 g/dL; platelet count, 77 × 10^3/μL) and elevated C-reactive protein (5.26 mg/dL) and lactate dehydrogenase (LDH) (891 IU/L) levels (Table 1). She was confirmed to have a urinary tract infection caused by *Escherichia coli*.

Imaging examinations

Following brain magnetic resonance imaging (MRI), a focal diffusion restrictive lesion in the left parietal lobe and chronic subdural hemorrhage in the right frontal convexity were observed. In addition, pachymeningeal enhancement of the bilateral frontoparietal convexities was noted, suggesting leptomeningeal disease (Figure 1).

Three consecutive lumbar punctures were performed, and a consistent increase in protein and LDH levels in cerebrospinal fluid (CSF) was observed. No malignant cells were observed and the CSF pressure was within the normal range (7.5 cmH_2O). Abdominal CT showed bilateral enlargement of adrenal glands along with hepatomegaly and splenomegaly. Meanwhile, her cognitive function and the results of blood tests were worsening (*e.g.*, exacerbation of cytopenia, elevation of ferritin and triglyceride levels), and, despite improved urinalysis findings after antibiotic therapy, she developed a high fever up to 39°C. Thus, the patient was referred to a hematologist who performed immediate bone marrow exam. Increased numbers of hemophagocytic histiocytes were found without malignant cells, suggesting secondary HLH. PET-CT was performed to identify the underlying disease. An increased FDG uptake was observed in both adrenal glands, the liver, and the right ethmoid sinus (Figure 2), and a biopsy of the right ethmoid sinus was ordered. Histological examination revealed atypical large lymphoid cells with prominent nucleoli in the vessels (Figure 3A and B). Immunohistochemical analysis showed that the tumor cells were positive for CD20, BCL2, BCL6, and IRF4/MUM1 (Figure 3C-F) but negative for CD3 and CD10. In addition, *MYD88*, *TET2*, and *PIM1* mutations were identified by targeted sequencing using tissues (Table 2).

Table 2 Variants found in next-generation sequencing

Tier 1 variants				Tier 2 variants				Tier 3 variants			
Gene	DNA	Protein	VAF	Gene	DNA	Protein	VAF	Gene	DNA	Protein	VAF
MYD88	c.755T>C	p.Leu252Pro	54.8%	*PIM1*	c.237G>C	p.Glu79Asp	30.77%	*NOTCH1*	c.6283C>T	p.Arg2095Cys	37.7%
								ETV6	c.1123G>A	p.Gly375Arg	14.4%
TET2	c.3280A>T	p.Lys1094Ter	16.0%	*BTG2*	c.97C>T	p.Gln33Ter	19.6%	*HIST1H1E*	c.367G>A	p.Ala123Thr	15.6%
								TBL1XR1	c.848G>A	p.Ser283Asn	16.5%

DNA: Deoxyribonucleic acid; VAF: Variant allele frequency.

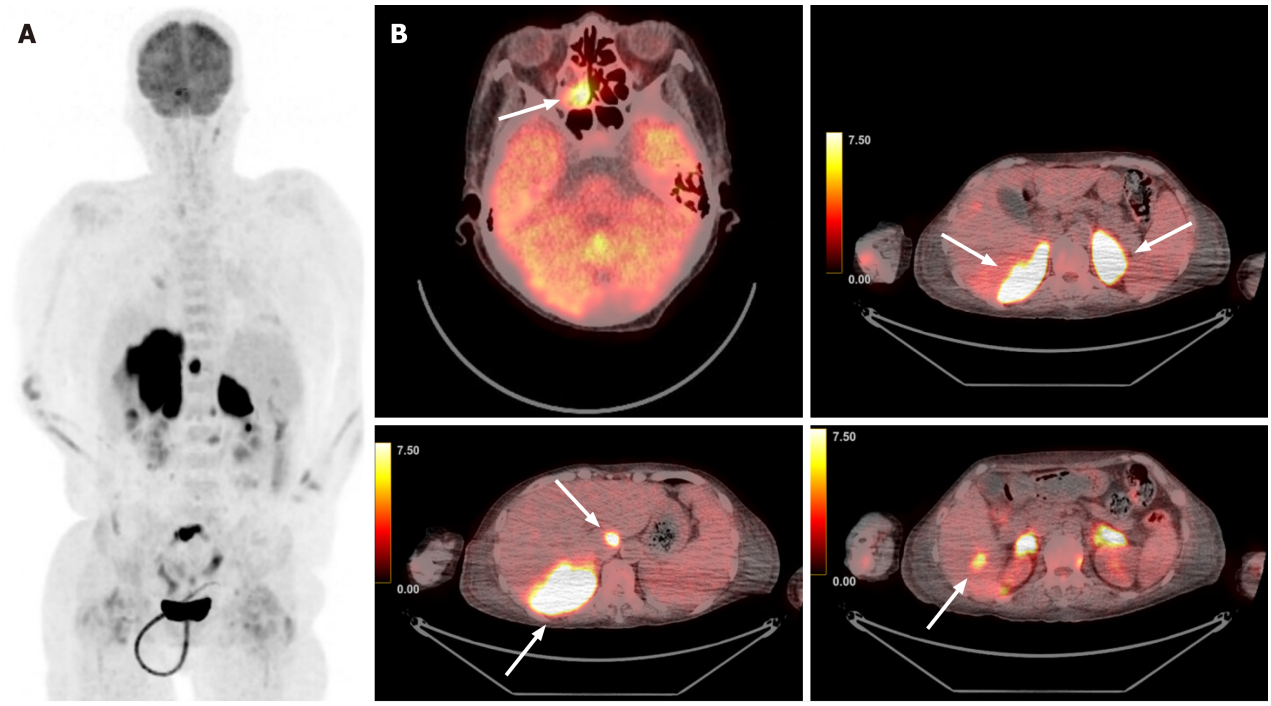

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Figure 2 An ^{18}F-fluorodeoxyglucose (FDG) positron emission tomography with computed tomography scan shows high FDG uptake in the right ethmoid sinus, liver, and both adrenal glands.

FINAL DIAGNOSIS

The Asian variant of IVLBCL was diagnosed. In addition, although there was no cytological confirmation, the IVLBCL was considered to be accompanied by CNS involvement based on the findings of brain MRI and CSF analysis as well as her clinical manifestation.

TREATMENT

Intravenous methylprednisolone administration at a dose of 1 mg/kg was started immediately after the biopsy, and, following the final diagnosis, immunochemotherapy including rituximab and CNS-directed therapy with methotrexate (MTX) was considered. However, due to her poor performance status and economic issues, she decided to receive only steroid therapy and best supportive care.

OUTCOME AND FOLLOW-UP

The patient deteriorated and passed away two weeks after her diagnosis.

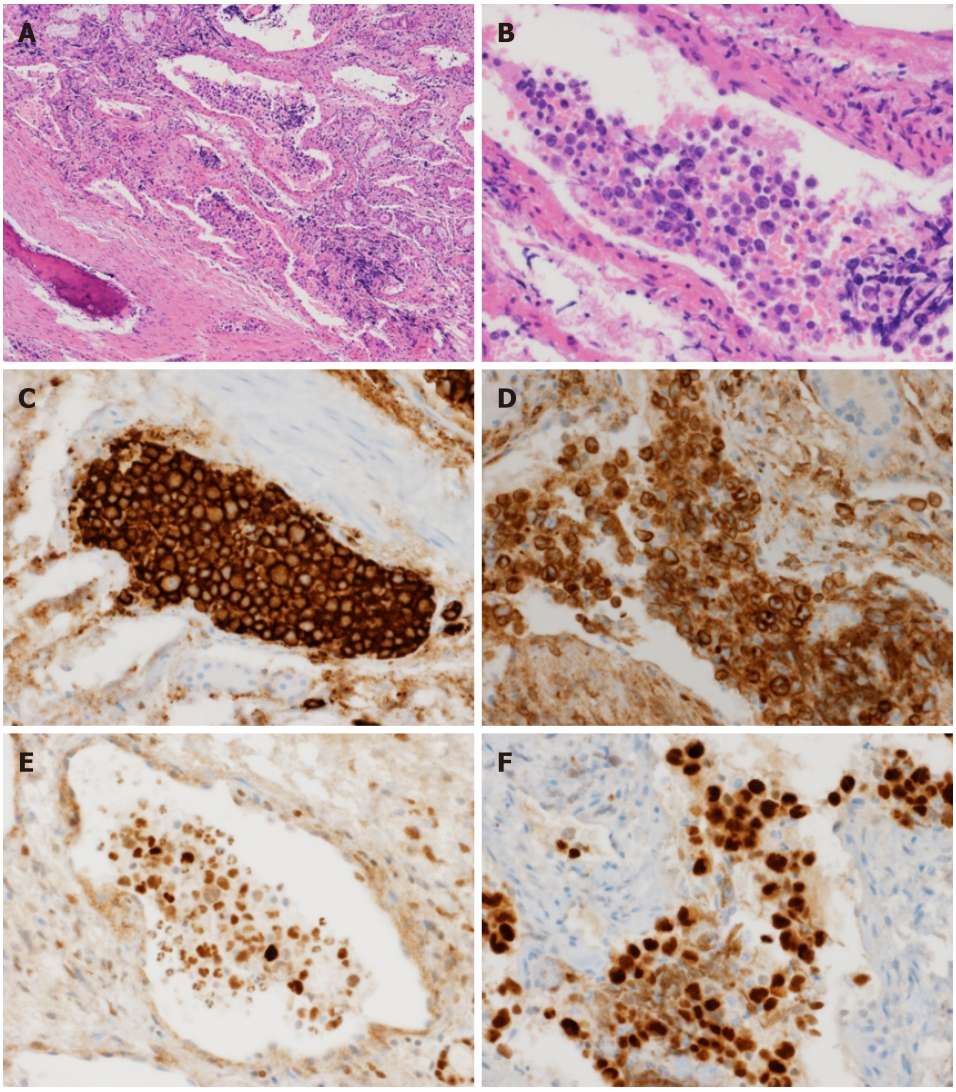

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Figure 3 Haematoxylin and eosin stained section of the biopsy specimen demonstrates atypical large lymphoid cells with prominent nucleoli in the blood vessel. A: Haematoxylin and eosin (H&E) (×100); B: H&E (×400); C: Immunohistochemical staining: CD20 (×400); D: BCL2 (×400); E: BCL6 (×400); F: IRF4/MUM1 (×400).

DISCUSSION

Despite a quantum leap of cancer diagnostic technology, the diagnosis of IVLBCL remains challenging due to the ambiguous signs and symptoms that do not precisely reflect the characteristics of the disease. In particular, approximately 20%-30% of Asian variant IVLBCL cases have CNS involvement at diagnosis[3,7], which is associated with poor prognosis[8]. However, since malignant lymphocytes are rarely found in CSF and there are no previously described pathognomonic findings on MRI[9], auxiliary diagnostic tools may often be required. Recently introduced less-invasive diagnostic methods using peripheral blood or CSF, such as liquid biopsy[10], or mutation detection using circulating tumor DNA[11,12] can play a complementary role in diagnosing IVLBCL. Therefore, when diagnosing IVLBCL, a multidisciplinary approach and an integrated diagnostic process are needed to analyze each symptom according to involved organ, including active CNS examination.

Malignant lymphoma derived from T-cells or natural killer cells is one of the leading causes of HLH in adults[13,14], but B-cell origin lymphoproliferative disease can also provoke HLH[14]. Indeed, the Asian variant of IVLBCL is commonly accompanied by HLH[2,3,6]. Therefore, in adult patients suspected of secondary HLH, systemic evaluation and biopsy based on PET-CT scan should be performed promptly. However, as opposed to the nodal diffuse large B-cell lymphoma (DLBCL), IVLBCL mainly involves extranodal sites and shows various levels (usually mild to moderate) of FDG uptake in PET-CT[5], and in general, the selection of PET-CT-based biopsy lesions may be difficult under these circumstances. Nonetheless, in diagnosing IVLBCL, when infectious or inflammatory diseases are excluded, such findings may help to select the appropriate biopsy site[5]. Therefore, despite some limitations, PET-CT may play an important role in the diagnosis of IVLBCL.

The absence of a prospective study due to the rarity of the disease hindered the establishment of standard of care for IVLBCL. Thus, IVLBCL has been managed by adapting the treatment strategy of DLBCL, where several immunophenotype overlaps[9]. Anthracycline-based chemotherapy with rituximab presented favorable clinical outcomes in both East and West[15,16]. In addition, CNS-directed therapy is an essential part of IVLBCL management. Considering that malignant cells can penetrate the CNS parenchyma through blood vessels[17], intrathecal MTX therapy alone for CNS-involving disease would not be sufficient. Recently, several reports have introduced high-dose IV MTX-based treatment based on the primary CNS lymphoma (PCNSL) treatment strategy[17,18], and this approach seems reasonable given the poor CNS penetration of systemic immunochemotherapy. In several reports of genetic alterations in IVLBCL, mutations in *MYD88* and *CD79B* were frequently detected[19,20], usually in primary testicular DLBCL and PCNSL, where extranodal site involvement is common[21]. With the advances in understanding of the disease and its biology, novel therapeutic approaches to IVLBCL are continuously being attempted. As Bruton's tyrosine kinase inhibitors block the nuclear factor kappa B pathway, patients with B-cell lymphomas harboring *MYD88* and/or *CD79B* mutations are expected to show better treatment responses[22]. Indeed, an interim analysis of a phase II study for treatment-naïve IVLBCL patients using zanubrutinib was recently reported, demonstrating promising efficacy[23]. As such, future studies to improve clinical outcomes with precision treatment for the disease in addition to conventional treatment are required.

CONCLUSION

In conclusion, given the aggressive nature of IVLBCL, suspicion of the disease and subsequent immediate and accurate diagnosis lead to timely treatment, which results in better clinical outcomes. In addition, considering its poor prognosis, careful examination of CNS involvement at diagnosis is recommended. Even if CNS invasion of IVLBCL is not confirmed, active CNS-directed therapy is required when highly suspected.

FOOTNOTES

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