Misdiagnosis of angioimmunoblastic T-cell lymphoma: A case report

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Abstract. Angioimmunoblastic T-cell lymphoma (AITL) is a specific subtype of peripheral T-cell lymphoma that is challenging to diagnose due to the lack of specific pathological characteristics. This report describes the case of a 56-year-old man with Hodgkin lymphoma in whom the gene rearrangement results were positive for TCRβDB+Jβ1/2. Pathological and immunochemical examinations revealed a diagnosis of lymphoma that was a composite of AITL and focal classical Hodgkin lymphoma. Unfortunately, he died soon after the correct diagnosis was made. This case shows that a combination of immunohistochemistry and gene rearrangement analysis can increase the diagnostic accuracy for AITL. A review of the literature on the misdiagnosis of AITL indicates that this disease progresses rapidly with a high mortality rate. Our experience, in this case, highlights the need for early diagnosis.

Introduction

Angioimmunoblastic T-cell lymphoma (AITL) is a subtype of peripheral T-cell lymphoma (PTCL) that accounts for 1-2% of all cases of non-Hodgkin lymphoma (HL) and 15-20% of cases of PTCL, and has a poor prognosis (1). The median age at diagnosis is ~65 years, and the primary clinical manifestations are fever, weight loss, urticaria, papules, red nodules, and skin lesions (2). Approximately 20-50% of patients with AITL have prodromal symptoms, and their skin manifestations can range from urticarial lesions to nodular tumors (3). Diagnosis is often delayed or masked due to the atypical biochemical and autoimmune manifestations, and it is common for the disease to have reached an advanced stage by the time an accurate diagnosis is made (4). As a result of the abnormal proliferative activity of B-cells (5), AITL is often accompanied by autoimmune disorders, such as hemolytic anemia and hypergammaglobulinemia. Epstein-Barr virus (EBV) has been found to play an important role in the pathogenesis of AITL (6). EBV can stimulate the activation of helper T-cells, leading to the development of tumors. Diagnosis of AITL is based on histopathological examination but remains challenging given the lack of specific pathological characteristics. This report suggests that a combination of immunohistochemistry and gene rearrangement can increase diagnostic accuracy. CD10 and CXCL13 are specifically expressed in AITL and can be used as characteristic markers for diagnostic purposes (7). Clonal rearrangement of the IgH gene and TCRγ may also be of significance in terms of the diagnosis (8).

Case report

The patient was a 56-year-old man who was initially diagnosed with HL for which he received doxorubicin hydrochloride liposome, bleomycin, vindesine, and dacarbazine (ABVD), and doxorubicin hydrochloride liposome, vindesine, and dacarbazine (AVD) chemotherapy in April 2020. However, a decrease in his CD21 levels and disruption of the follicular dendritic cell (FDC) network (CD20+; PAX-5+; CD3+; Ki-67+: 20-30%; CD10-; BCL-6+; MUM-1+; PD-1+; CXCL-13-; CD30+; CD15-) was also detected. Molecular detection showed monoclonal rearrangement of TCRβDB+Jβ1/2 and oligoclonal rearrangement of Vβ + Jβ2. A review of all details concerning unsatisfactory treatment led to a diagnosis of compound lymphoma consisting of AITL and focal classical HL (CHL).

In March 2020, the patient was admitted to Hebei General Hospital (Shijiazhuang, China) after a 4-month history of skin redness and itching. Physical examination revealed extensive redness, swelling, and rough skin on the head, neck, and limbs. There were multiple enlarged lymph nodes in the right armpit and on both sides of the groin. The largest node was ~4 cm in diameter with a smooth surface and was non-tender and mobile. The laboratory findings are presented in Table I. A proliferative disease of the lymphatic system was suspected

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Abbreviations: AITL, angioimmunoblastic T-cell lymphoma; CHL, classical Hodgkin lymphoma; EBV, Epstein-Barr virus; HL, Hodgkin lymphoma; PTCL, peripheral T-cell lymphoma; RS, Reed-Sternberg; TFH, T follicular helper

Key words: lymphoma, gene rearrangement, immunohistochemistry, pathology, diagnosis

initially; a punch biopsy was performed, a histological analysis of which documented CHL, lymphocyte-rich type, with molecular detection of EBER positivity. Flow cytometry revealed that 96.1% of the nuclear cells in the bone marrow were CD3+ lymphocytes, with some showing positive expression for CD3 and CD5, some expressing TCRrd, and a small number expressing CD8 (Fig. 1). Radiological imaging detected lymph node infiltration in multiple organs. After various examinations, the patient was diagnosed as having lymphocyte-predominant HL. The patient completed chemotherapy that consisted of ABVD (doxorubicin hydrochloride liposome 40 mg IV, bleomycin 10 mg/m^2 IV, vindesine 4 mg IV, and dacarbazine 375 mg/m^2 IV on days 1 and 15), followed by AVD as a second course.

After two courses of chemotherapy, a CT showed that the patient's lymph nodes were smaller than before (Fig. 2). However, the patient appeared to have worsening pruritus. Immunohistochemical examination of the lymph node specimen revealed CD20+, PAX-5+, CD3+, Ki-67+ (20-30%), CD10-, BCL-6+, MUM-1+, PD-1+, CXCL-13-, CD30+, and CD15-; (Fig. 3). T-cell clone analysis was performed using the BIOMED-2 polymerase chain reaction protocol. On molecular examination, TCRβDB+Jβ1/2 showed monoclonal rearrangement and Vβ+Jβ2 showed oligoclonal rearrangement with PTPRD gene mutation. The pathological diagnosis was non-Hodgkin peripheral (mature) T-cell lymphoma, prone to AITL. Thus, the patient was diagnosed with lymphoma, which was a composite of AITL and HL (International Prognostic Index, 3; Prognostic Index for T-cell lymphoma, 2). Given this diagnosis, the patient was scheduled for etoposide, prednisone, vincristine, cyclophosphamide, and doxorubicin (EDOCH). However, the patient died of a lung infection before starting further treatment (Fig. 4).

Table I. Results of laboratory findings on admission.

Laboratory indicator	Normal range	Result
CBC		
WBC, x10^9/l	3.5-9.5	15.00
LY, x10^9/l	1.1-3.2	5.12
HGB, g/l	115-150	151
PLTx10^9/l	125-350	292
β2-microglobulin, μg/ml	0.9-2.7	4.288
Biochemistry		
TP, g/l	65-85	53.5
GLO , g/l	20-40	22.3
LDH, U/l	120-250	323.9
HBDH, U/l	72-182	222.4
TG, mmol/l	0.1-1.7	1.84
Coombs test		
Direct Coombs test		Negative
Indirect Coombs test		Negative
Tumor Marker		
CA 125, U/ml	0-35	125.100
VEGF, pg/ml	0-142	178.4

CBC, complete blood count; WBC, white blood cell; LY, lymphocyte cell; HGB, hemoglobin; PLT, plate; TP, total protein; GLO, globulins; LDH, lactate dehydrogenase; HBDH, GHB dehydrogenase; TG, triglyceride; VEGF, vascular endothelial growth factor.

Discussion

Although this patient had obvious skin symptoms, the pathological results and immunohistochemical analysis n his initial admission were consistent with a diagnosis of CHL. However, the patient's pruritic symptoms worsened after ABVD chemotherapy. Therefore, a lymph node pathology examination was repeated. Finally, the patient was diagnosed as having a composite of lymphocyte-rich type HL and AITL.

AITL is a specific subtype of PTCL that originates from T follicular helper (TFH) cells and is often accompanied by fever, night sweats, weight loss, lymphadenopathy, skin rash, and other clinical manifestations (9). Skin involvement is one of the most common extranodal manifestations of the disease (10). However, the heterogeneous presentation of AITL means that most cases are not diagnosed until weeks or months after the onset of symptoms (11).

Although the diagnosis of AITL relies on lymph node biopsy, certain patients may not be diagnosed until after 2-3 lymph node biopsies (12). The pathological features include destruction of lymph node structure, tumor cells primarily medium in size, lightly stained or transparent cytoplasm, generally with round or oval nuclei, and atypical cells. Large cells of varying numbers are scattered in the background of inflammatory cells, including eosinophils, lymphocytes, and plasma cells. The TFH phenotype is positive for CD3, CD4, and CD10 (4). CD5 and CD7 expression are largely absent (13). CD30 is found in 20% of patients (14). Cytoplasmic CXCL13 is expressed almost uniformly and is specific for AITL (15). TFH expression of PD-1, ICOS, BCL-6, and CD200 can be distinguished from that in benign lymphoproliferative diseases and other subtypes of PTCL. In ~60% of patients, TCR gene rearrangements are observed, as seen in TCβ, TCD, and TCG, whereas some have an IgH gene rearrangement (16). In recent years, with advances in the field of genomics, patients with AITL have been found to have a high number of TET2, RHOA, IDH2, and DNMT3A mutations (especially in TET2), which are associated with a poor prognosis (17). However, there is still no systematic method for the identification of AITL, and its diagnosis remains difficult.

There is a strong correlation between AITL and EBV infection. EBV-infected B-cells can transmit EBV protein signals on their surface to T-cells via major histocompatibility complex II molecules when TFH cells interact with B-cells, which upregulate the expression of CD ligands, provide antigen and costimulatory signals for activation of T-cells, promote the secretion of the chemokine CXCL13 (6), and lead to activation of B-cells. Laforga *et al* (18) hypothesized that EBV promotes the proliferation of B-cells in AITL. They suggested that as a result of the effects of EBV, CD8-positive T-cells become immunosuppressed, leading to immune evasion of EBV-positive B-cells. EBV-infected B-cells exhibit abnormal proliferation and may be polyclonal, oligoclonal, or monoclonal. If the immunoglobulin structure is disrupted, there are three possible outcomes for

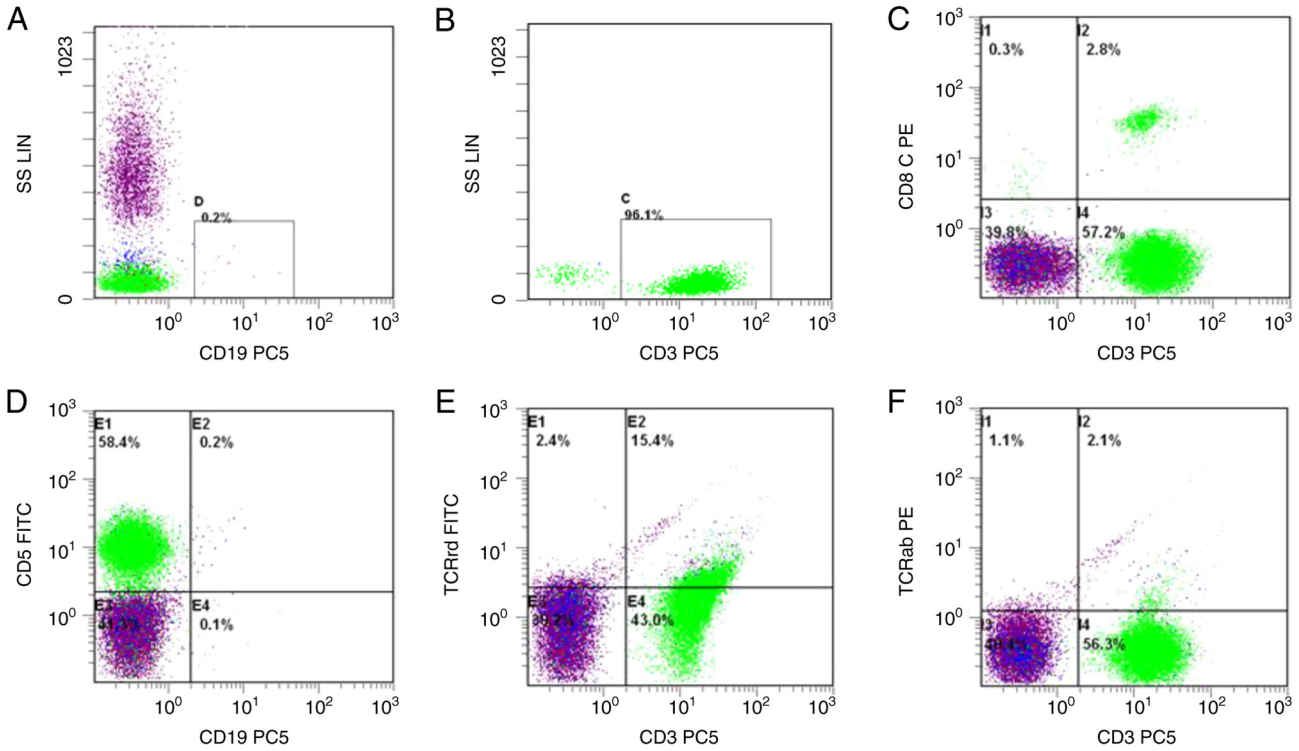

Figure 1. Flow cytometry results for bone marrow. (A) CD19+ lymphocytes (group D) account for 0.2% of nuclear cells. (B) CD3+ lymphocytes (group C) account for 96.1% of all nuclear cells. (C-F) Positive expression of CD3 and CD5, partial expression of TCRrd, and limited expression of CD8.

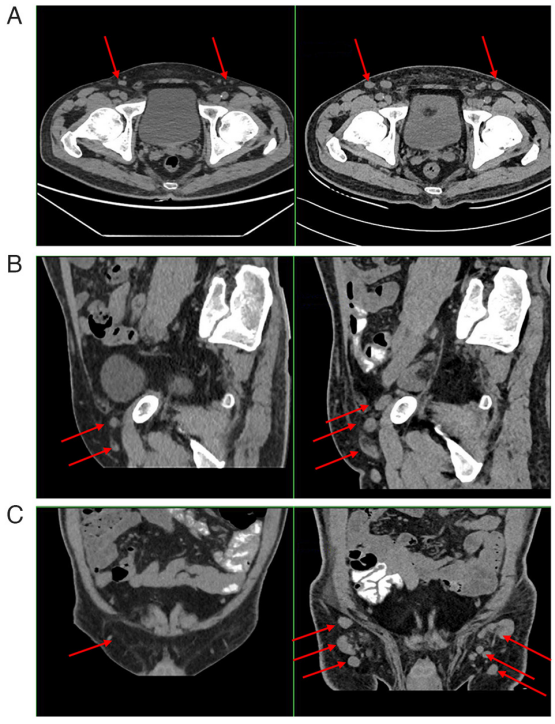

Figure 2. CT images of and enlarged lymph node. (A) Cross section, (B) sagittal plane, and (C) coronal plane images. Red arrows indicate lymph nodes. The images before chemotherapy are shown on the left and the images following chemotherapy are shown on the right.

B-cells: Proliferation resembling that of Reed-Sternberg (RS) cells; proliferation resembling that of CHL; or the development of CHL.

The 2008 edition of the WHO Classification of Tumors of Hematopoietic and Lymphoid Tissues mentions RS-like cells being seen in early AITL for the first time (19). Destruction of lymph node structure and infiltration of plasma cells, tissue cells, and other inflammatory cells is observed in both AITL and CHL, and both diseases are associated with EBV infection (20), and most immune phenotypes do not have specificity, so diagnosis cannot be determined solely based on immune phenotypes. The immunophenotype of CHL includes CD15+, CD30+, PAX-5 weakly positive, CD3-, CD20-(mainly), CD45-, and CD79a-, which combined with RS cells, assist in the diagnosis (21,22). CHL and AITL are two types of lymphoma with very different prognoses. While CHL has a good prognosis with a 5-year overall survival rate of >80% (23), AITL has a poor prognosis. A retrospective analysis found the complete response rate to be 25% and the median overall survival to be only 14.9 months after CHOP chemotherapy in elderly patients with AITL (24). At present, ABVD is the first-line treatment for CHL, and most patients benefit from it. However, AITL progresses rapidly with a high mortality rate. Therefore, clinical phenotype, pathological morphology, immunohistochemistry, and gene rearrangement studies are important for early and correct diagnoses of AITL.

AITL is frequently misdiagnosed given its nonspecific clinical and histologic findings. A summary of a review of the literature on the misdiagnosis of AITL is shown in Table II. It was found that AITL can not only be misdiagnosed as another hematological disease but also as a disease involving another system and that more than one lymph node biopsy is required for a definitive diagnosis.

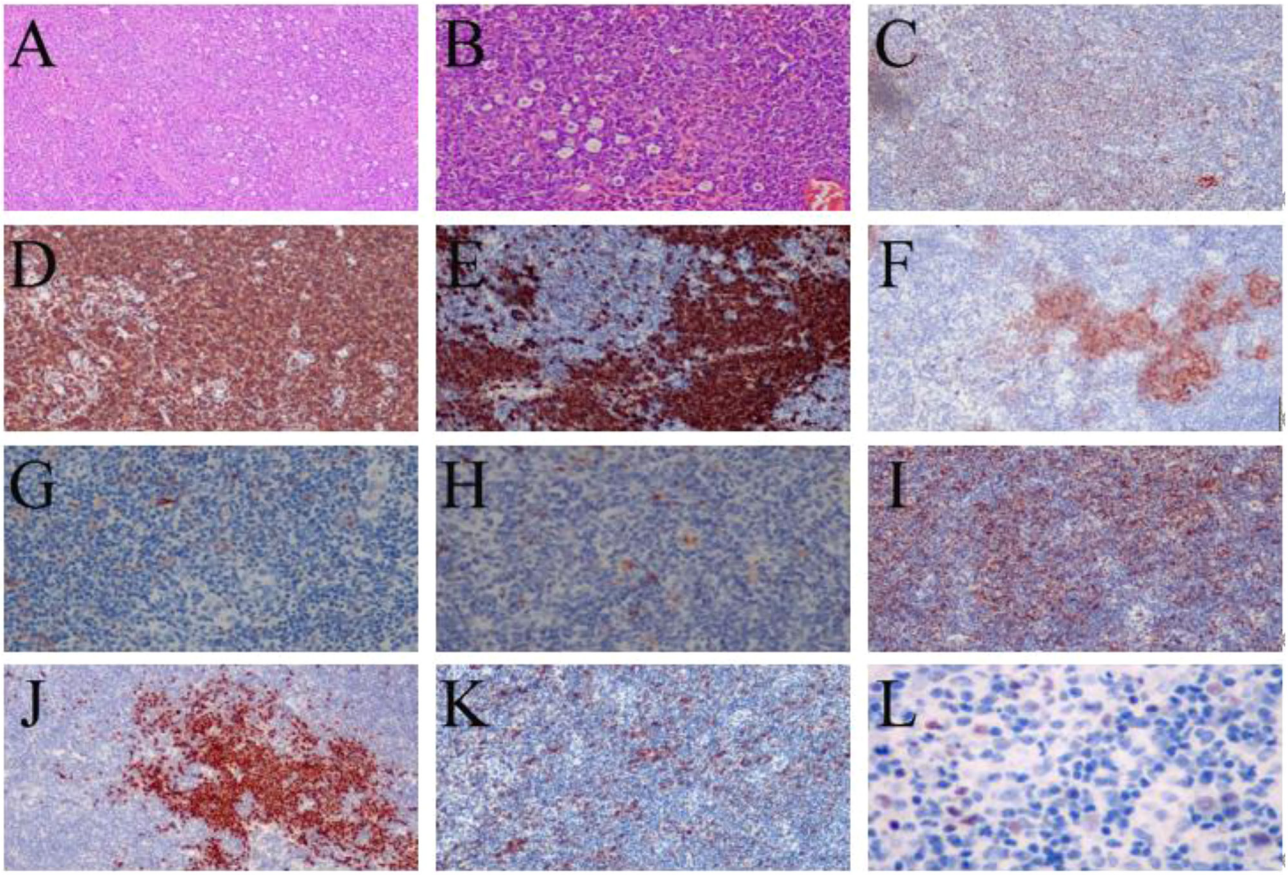

Figure 3. Immunostaining of the right inguinal lymph node for cytokines. (A) HE staining x40 magnification; (B) HE staining, x100 magnification; (C) BCL-6 negative staining, x100 magnification. (D) CD3 positive staining, x100 magnification. (E) CD20 positive staining, 100x magnification. (F) CD21 staining negative, x100 magnification. (G) CXCL13 negative staining, x100 magnification. (H) Ki-67 positive staining, x100 magnification. (I) MUM-1 positive staining, x100 magnification. (J) PAX-5 positive staining, x100 magnification. (K) PD-1 positive staining, x100 magnification. (L) EBER (molecular diagnosis) positive staining. Based on (A and B), the structure of the lymph node was partially destroyed, with scattered atypical large cells, rich cytoplasm, large nuclei, with some cells possessing some double nuclei and larger nucleoli. There were T-cell lymphomas in multiple lymph nodes, and certain lymph nodes also contained focal classical Hodgkin's lymphoma. HE, hematoxylin-eosin.

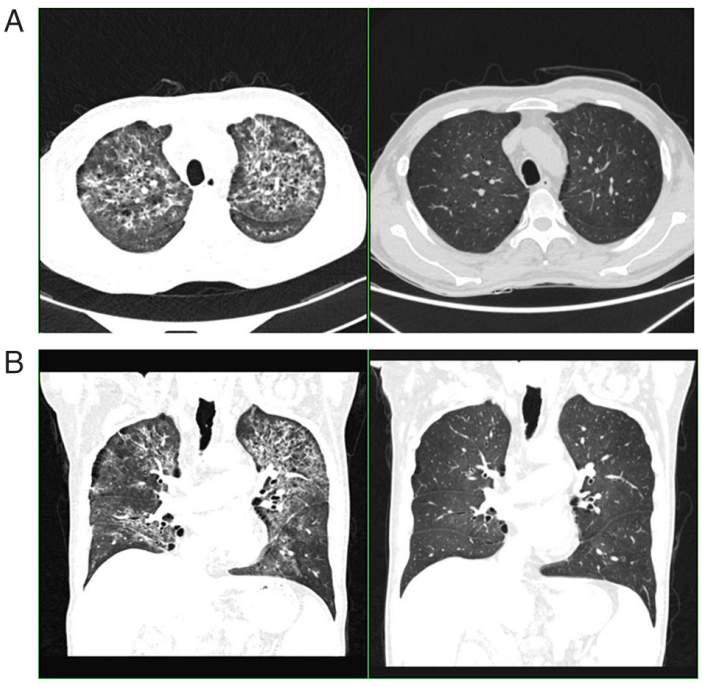

Figure 4. CT images of pulmonary infection. (A) Cross section, and (B) coronal planes. The images after two courses of chemotherapy are shown on the right. The patient had a severe pulmonary infection and extensive exudation.

Table II. Literature review of misdiagnosis of AITL.

First author/s, year	Age, years	Sex	Misdiagnosis	Method leading to misdiagnosis	Method of diagnosis	(Ref.)
van den Akker and Chen, 2021	62	M	Reactive lymphadenopathy	FNA	Excisional biopsy	(25)
Ellis *et al*, 2018	72	F	DLBCL	LN biopsy	Reexamination, IHC	(26)
Keefe *et al*, 2022	65	M	DRESS syndrome	clinical signs and symptoms, PET-CT	LN biopsy	(27)
Trimech *et al*, 2021	62	M	Richter syndrome	PET-CT, BM uptake	LN biopsy	(28)
Papadi *et al*, 2012	55	F	SPBIP	LN biopsy and BM aspirate	LN biopsy, clonal TCR gene rearrangement	(29)
	70	F	SPBIP	Clinical and morphologic features	Flow cytometry and gene rearrangement	
Smithberger *et al*, 2010	79	F	Inflammatory dermatosis	Skin biopsy	LN biopsy	(30)
Ahsanuddin *et al*, 2011	76, 46, 60	F, F, F	Plasma cell leukemia	Smear morphology and manual differential of peripheral blood	LN biopsy and BM biopsy	(31)
Han *et al*, 2019	70	M	Drug fever and allergic purpura, septicemia	Medication history, surgery history	LN biopsy	(32)
Kaffenberger *et al*, 2015	59, 68	M, M	MALT	Skin biopsy	LN biopsy	(33)
Szablewski *et al*, 2019	41, 60, 67	M, M, M	CHL, B cell lymphoma	Skin biopsy	A second review of the skin biopsy, LN biopsy	(34)
Suárez, 2016	77	F	Marginal-zone B-cell-lymphoma	Skin biopsy	Skin biopsy, LN biopsy	(35)
Laforga, 2010	58	M	HL	Autoimmune phenomena	Touch imprints of LN biopsy	(18)

M, male; F, female; LN, lymph node; FNA, fine-needle aspiration; DLBCL, diffuse large B-cell lymphoma; LN, lymph node; IHC, immunohistochemical staining; DRESS syndrome, drug reaction with eosinophilia and systemic symptoms; PET-CT, positron emission tomography-computed tomography; BM, bone marrow; SPBIP, systemic polyclonal B-immunoblastic proliferation; MALT, marginal zone lymphoma; CHL, classical Hodgkin lymphoma; ATLL, adult T-cell leukemia/lymphoma.

In conclusion, AITL is a specific subtype of peripheral T-cell lymphoma that is challenging to diagnose. Moreover, treatment is often delayed by misdiagnosis. The present case underscores the importance of early and accurate diagnosis of AITL and the potential for a poor prognosis. More than one pathological examination should be performed to reduce the risk of misdiagnosis. Clinical phenotype, lymph node biopsies, immunohistochemistry, and gene rearrangement analyses should all be considered for early and accurate diagnosis and appropriate treatment.

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Availability of data and materials

The datasets used and/or analyzed during the current research are available from the corresponding author on reasonable request.

Authors' contributions

YL conceived and designed the study. XG collected the data and wrote the manuscript. LK treated the patient and contributed to draft and revise the manuscript. JL advised on patient treatment and participated in revising the manuscript. YL, XG, LK and JL confirm the authenticity of all the raw data. All authors have read and approved the final manuscript.

Ethics approval and consent to participate

Not applicable.

Patient consent for publication

This report was published with the written consent of the patient's relatives.

Competing interests

The authors declare that they have no competing interests.

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