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ORIGINAL ARTICLE

Clinicopathological Characteristics and Prognostic Factors in Angioimmunoblastic T-cell Lymphoma: a Retrospective Analysis

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SUMMARY

Background: **Our study aimed to analyze the molecular and clinical characteristics of angioimmunoblastic T-cell lymphoma (AITL), identify prognostic factors, and evaluate their implications for patient outcomes.**
Methods: **This retrospective study analyzed 33 patients diagnosed with AITL between 2012 and 2022 at our center. Clinical data, laboratory parameters, pathological findings, and treatment outcomes were evaluated. Survival analyses were performed using the Kaplan-Meier method, and prognostic factors were identified through univariate Cox regression analysis.**
Results: **The median age was 64.1 ± 7.2 years, with male predominance (63.6%). Most patients presented with advanced disease (69.7% stage IV). Immunophenotypic analysis confirmed high expression of follicular helper T-cell markers, including PD-1 (96.9%), CXCL13 (83.3%), and BCL-6 (96.6%). EBV was detected in 72.7% of specimens by EBER-ISH and 90% by EBV-DNA PCR. Univariate analysis identified lower hemoglobin, decreased platelet counts, low albumin levels, and elevated β2-microglobulin as significant negative prognostic factors for progression-free survival (PFS). For overall survival (OS), low albumin levels (HR 0.8, 95% CI 0.69 - 0.92, p = 0.002) and elevated β2-microglobulin (HR 1.32, 95% CI 1.03 - 1.69, p = 0.029) were significant predictors of inferior outcomes. Interestingly, PD-1 positivity was associated with significantly better PFS (HR 0.03, 95% CI 0 - 0.52, p = 0.016).**
Conclusions: **This study highlights the aggressive nature of AITL and identifies several readily accessible laboratory parameters as important prognostic factors. The protective effect of PD-1 positivity on survival outcomes warrants further investigation. While CHOP/CHOPE remains the standard treatment, the addition of novel targeted therapies shows promise for improving patient outcomes in this challenging lymphoma subtype.**
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INTRODUCTION

Angioimmunoblastic T-cell lymphoma (AITL) is a rare and aggressive peripheral T-cell lymphoma (PTCL) that accounts for approximately 15 - 20% of all PTCL cases worldwide [1,2]. It represents one of the most common subtypes within the PTCL classification and is characterized by a distinct clinical presentation and molecular pathogenesis [3]. AITL typically affects elderly individ-

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uals and presents with generalized lymphadenopathy, hepatosplenomegaly, B symptoms (fever, night sweats, weight loss), and various immunological abnormalities [4,5].

The pathogenesis of angioimmunoblastic T-cell lymphoma represents a complex network involving transformed T cells, the surrounding supportive microenvironment, and various alterations at the genetic and molecular levels [6]. Recent advances in molecular profiling have identified recurrent genetic mutations in AITL, including those affecting epigenetic regulators (TET2, DNMT3A, IDH2), T-cell receptor signaling pathway components (RHOA, VAV1), and other key cellular processes [7-9]. Additionally, the frequent association with Epstein-Barr virus (EBV) infection suggests a potential role for viral factors in disease progression [10, 11]. Histologically, AITL lymph nodes exhibit architectural disruption, expanded follicular dendritic cells, and proliferating high endothelial venules. The malignant cells express both T-cell antigens and specific TFH markers (BCL6, CD10, CXCL13, ICOS, PD-1). Gene expression studies confirm these neoplastic cells share molecular features with normal TFH cells, establishing their cellular origin [12].

Despite advancements in our understanding of AITL biology, the clinical management remains challenging, with limited treatment options and poor overall survival rates [13]. Standard chemotherapy regimens like CHOP (cyclophosphamide, doxorubicin, vincristine, prednisone) or CHOPE (CHOP plus etoposide) show modest efficacy [14,15], while novel approaches targeting PD-1, CD30, and other molecular markers are emerging as potential therapeutic strategies [16,17].

In this study, we aim to comprehensively analyze the molecular and clinical characteristics of AITL, identify prognostic factors, and evaluate their implications for patient outcomes. By integrating genomic, immunophenotypic, and clinical data, we hope to contribute to a more precise understanding of AITL pathogenesis and prognosis, ultimately guiding more effective treatment strategies.

MATERIALS AND METHODS

Patient selection and clinical data collection

We conducted a retrospective analysis of 33 patients diagnosed with angioimmunoblastic T-cell lymphoma between 2012 and 2022 in our center. Patients with pathologically confirmed AITL diagnosis according to WHO classification and available clinical data (baseline information, staging, treatment regimens, efficacy evaluation, and follow-up records) were included, while those with acute myocardial infarction or cerebral infarction within the past 6 months, uncontrolled hypertension or symptomatic arrhythmia, pregnancy or lactation, or concurrent second malignancy were excluded from the study. All patients provided written informed consent prior to participation.

Clinical data were collected from medical records, including age, gender, Ann Arbor stage, Eastern Cooperative Oncology Group (ECOG) performance status, International Prognostic Index (IPI) scores, and laboratory findings. Laboratory parameters collected included complete blood count, liver and kidney function tests, lactate dehydrogenase (LDH), C-reactive protein (CRP), β2-microglobulin (β2-MG), serum ferritin, and immunoglobulin G (IgG) levels. Additionally, EBV-DNA copy numbers were measured using quantitative PCR in peripheral blood samples.

Pathological analysis

Upon enrollment, pathological specimens including hematoxylin-eosin stained slides, immunohistochemistry preparations, and Epstein-Barr virus encoded RNA in situ hybridization (EBER-ISH) were meticulously reviewed by expert hemato-pathologists to confirm the diagnosis of AITL. Diagnostic criteria adhered to established pathological hallmarks: partial or complete effacement of lymph node architecture, prominent vascular proliferation with arborized high endothelial venules, extrafollicular follicular dendritic cell meshwork expansion, and infiltration by atypical CD4+ T cells expressing at least two T-follicular helper (TFH) markers (CD10, BCL6, PD-1, CXCL13). Additionally, the presence of large CD20+ B immunoblasts, with or without evidence of Epstein-Barr virus infection, was documented. TFH marker positivity was rigorously defined as expression in ≥ 20% of tumor cells. Patients with concurrent or secondary diffuse large B-cell lymphoma and those with nodal peripheral T-cell lymphoma exhibiting TFH phenotype (as classified in the 2016 WHO classification revision) were systematically excluded from the analysis to maintain diagnostic homogeneity. Polymerase chain reaction (PCR) was used to analyze T-cell receptor (TCR) and immunoglobulin heavy chain (IGH) gene rearrangements.

Treatment and response assessment

Patients received various treatment regimens, including CHOP/CHOPE (n = 22), azacitidine plus CHOP (Aza + CHOP, n = 7), anti-CD30 antibody plus CHOP (anti-CD30 + CHOP, n = 3), or anti-PD-1 antibody plus CHOP (anti-PD1 + CHOP, n = 1). Treatment response was assessed based on the Lugano classification criteria [18].

Statistical analysis

Progression-free survival (PFS) was defined as the duration from diagnosis to either disease progression, relapse, or death from any cause. Overall survival (OS) was calculated from the date of diagnosis to death from any cause or the last follow-up date. Survival analyses were performed using the Kaplan-Meier method, and differences between groups were compared using the log-rank test. Univariate analyses were conducted using Cox proportional hazards regression models to identify factors associated with survival outcomes. Statistical

Table 1. Clinical characteristic.

Characteristic	No (%)
Age, Median (range)	64.1 ± 7.2
< 60	9 (27.3)
≥ 60	24 (72.7)
Gender	
Female	12 (36.4)
Male	21 (63.6)
Ann Arbor stage	
III	10 (30.3)
IV	23 (69.7)
ECOG performance status	
≤ 1	26 (78.8)
> 1	7 (21.2)
Ann Arbor stage	
III	10 (30.3)
IV	23 (69.7)
IPI score	
≤ 2	22 (66.7)
> 3	11 (33.3)
Bone marrow infiltration	
No	20 (60.6)
Yes	13 (39.4)
EBV - DNA ≥ 500 copies/mL, n = 20	
No	2 (10)
Yes	18 (90)
WBC, x 10^9/L	5.5 (4.0, 6.8)
Monocytes percentage, %	9.0 (6.5, 11.6)
Hemoglobin, g/L	122.0 (108.0, 132.0)
Platelet, x 10^9/L	154.0 (135.0, 190.0)
LDH level, U/L	225.0 (187.5, 337.0)
Albumin level, g/L	37.9 (33.1, 41.2)
CRP level, mg/L	8.2 (4.2, 29.8)
β2-MG	3.7 (2.7, 4.6)
Serum ferritin, ng/mL	272.3 (186.6, 422.2)
IgG level	1,042.0 (870.5, 1,732.0)
Treatments	
CHOP/CHOPE	22 (66.7)
Aza + CHOP	7 (21.2)
anti-CD30 + CHOP	3 (9.1)
anti-PD1 + CHOP	1 (3.0)

WBC white blood cell count, CRP C-reactive protein, β2-MG β2-microglobulin, LDH lactic dehydrogenase, CHOP Cyclophosphamide + Hydroxydoxorubicin + Oncovin + Prednisone, CHOPE CHOP + Etoposide, AZA azacitidine.

significance was set at $p < 0.05$.

RESULTS

Clinical and demographic characteristics

This study included 33 patients diagnosed with AITL. The median age was 64.1 ± 7.2 years, with a male predominance (63.6%, n = 21). Most patients presented with advanced disease: 23 patients (69.7%) had Ann Arbor stage IV, and 10 patients (30.3%) had stage III disease. Good performance status (ECOG ≤ 1) was observed in 26 patients (78.8%). Eleven patients (33.3%) had an IPI score > 3, and 13 patients (39.4%) exhibited bone marrow infiltration (Table 1).

Laboratory findings revealed median values of WBC count at 5.5 x 10^9/L, hemoglobin at 122.0 g/L, and platelet count at 154.0 x 10^9/L. Elevated LDH (median 225.0 U/L), CRP (median 8.2 mg/L), and β2-MG (median 3.7) were common findings. Among 20 patients tested for EBV-DNA, 18 (90%) had levels ≥ 500 copies/mL, suggesting a high prevalence of EBV infection in this cohort.

Pathological Findings

The pathological analysis demonstrated a consistent immunophenotypic profile characteristic of AITL (Table 2). All examined cases showed positivity for CD4 (32/32, 100%), while CD3 expression was detected in most cases (27/31). CD10, a marker frequently associated with AITL, was positive in 27/33 cases. High expression of follicular helper T-cell (TFH) markers was observed, including PD-1 (31/32), CXCL13 (25/30), and BCL-6 (28/29). CD30 positivity was detected in 29/31 cases, and EBER-ISH was positive in 24/33 specimens, confirming the frequent association of AITL with EBV infection. A high proliferation index (Ki-67 ≥ 60%) was observed in 11/32 cases. TCR gene rearrangement was detected in 10/11 tested samples, while IGH rearrangement was found in only 1/6 cases.

Survival analysis and prognostic factors

The Kaplan-Meier survival analysis demonstrated the patterns of overall progression-free survival (PFS) and overall survival (OS) across the entire cohort (Figure 1A and 1B). When stratified by treatment methods, differential survival patterns emerged (Figure 1C and 1D), suggesting that treatment approach significantly impacts patient outcomes.

Univariate analysis identified several factors significantly associated with PFS (Table 3). Lower hemoglobin levels (HR 0.97, 95% CI 0.94 - 1.0, p = 0.029), lower platelet counts (HR 0.98, 95% CI 0.97 - 0.99, p = 0.003), decreased albumin levels (HR 0.82, 95% CI 0.72 - 0.94, p = 0.003), and elevated β2-MG (HR 1.25, 95% CI 1.06 - 1.46, p = 0.007) were significantly associated with inferior PFS. Interestingly, PD-1 positivity was associated with significantly better PFS (HR 0.03, 95% CI 0 - 0.52, p = 0.016).

Table 2. Pathological characteristics.

Pathological finding	Event (n)	%
CD3 positive	27/31	87.09
CD4 positive	32/32	100
CD8 positive	18/31	58.06
CD10 positive	27/33	81.8
CXCL13 positive	25/30	83.3
BCL-6 positive	28/29	96.5
PD-1 positive	31/32	96.8
Ki-67 index ≥ 60%	11/32	34.4
EBER- ISH positive	24/33	72.7
CD30 positive	29/31	93.5
TCR rearrangement	10/11	90.9
IGH rearrangement	1/6	16.7

Table 3. Univariate analysis for OS and PFS in advanced angioimmunoblastic T-cell Lymphoma.

Variables	RFS		OS	
	HR (95% CI)	p-value	HR (95% CI)	p-value
Gender, male	1.5 (0.47, 4.82)	0.487	0.98 (0.28, 3.38)	0.972
Age > 60 years	1.51 (0.41, 5.6)	0.534	1.25 (0.32, 4.89)	0.753
Ann Arbor stage III vs. IV	3.92 (0.87, 17.62)	0.075	6.11 (0.77, 48.27)	0.086
Bone marrow involvement	1.00 (1, 1.01)	0.724	2.8 (0.81, 9.63)	0.102
ECOG performance status ≥ 1	1.04 (0.33, 3.34)	0.942	0.9 (0.24, 3.38)	0.874
IPI scores > 3	2.68 (0.95, 7.51)	0.061	1.19 (0.37, 3.75)	0.773
WBC	0.95 (0.75, 1.21)	0.692	0.83 (0.62, 1.11)	0.206
Monocytes percentage	0.99 (0.91, 1.07)	0.769	1.01 (0.93, 1.1)	0.756
Hemoglobin	0.97 (0.94, 1)	0.029	0.98 (0.95, 1.01)	0.226
Platelet	0.98 (0.97, 0.99)	0.003	0.99 (0.98, 1)	0.12
Albumin	0.82 (0.72, 0.94)	0.003	0.8 (0.69, 0.92)	0.002
LDH	1.0 (1.0, 1.0)	0.746	1.0 (1.0, 1.0)	0.458
β2-MG	1.25 (1.06, 1.46)	0.007	1.32 (1.03, 1.69)	0.029
CRP	1.01 (0.99, 1.03)	0.238	1.0 (0.98, 1.03)	0.522
Serum ferritin	1.0 (1.0, 1.0)	0.824	1.0 (1.0, 1.0)	0.743
PD1 positive	0.03 (0, 0.52)	0.016	0.26 (0.03, 2.14)	0.21
Ki67 index > 60%	0.53 (0.15, 1.93)	0.335	0.73 (0.19, 2.76)	0.644
EBV-DNA ≥ 500 copies/mL	1.14 (0.13, 9.93)	0.908	0.82 (0.09, 7.46)	0.863
IgG level	1.0 (1.0, 1.0)	0.298	1.0 (1.0, 1.0)	0.1

For OS, lower albumin levels (HR 0.8, 95% CI 0.69 - 0.92, p = 0.002) and elevated β2-MG (HR 1.32, 95% CI 1.03 - 1.69, p = 0.029) emerged as significant prognostic factors. Although not reaching statistical significance, Ann Arbor stage III versus IV showed a trend toward impact on both PFS (HR 3.92, 95% CI 0.87 - 17.62, p = 0.075) and OS (HR 6.11, 95% CI 0.77 - 48.27, p = 0.086).

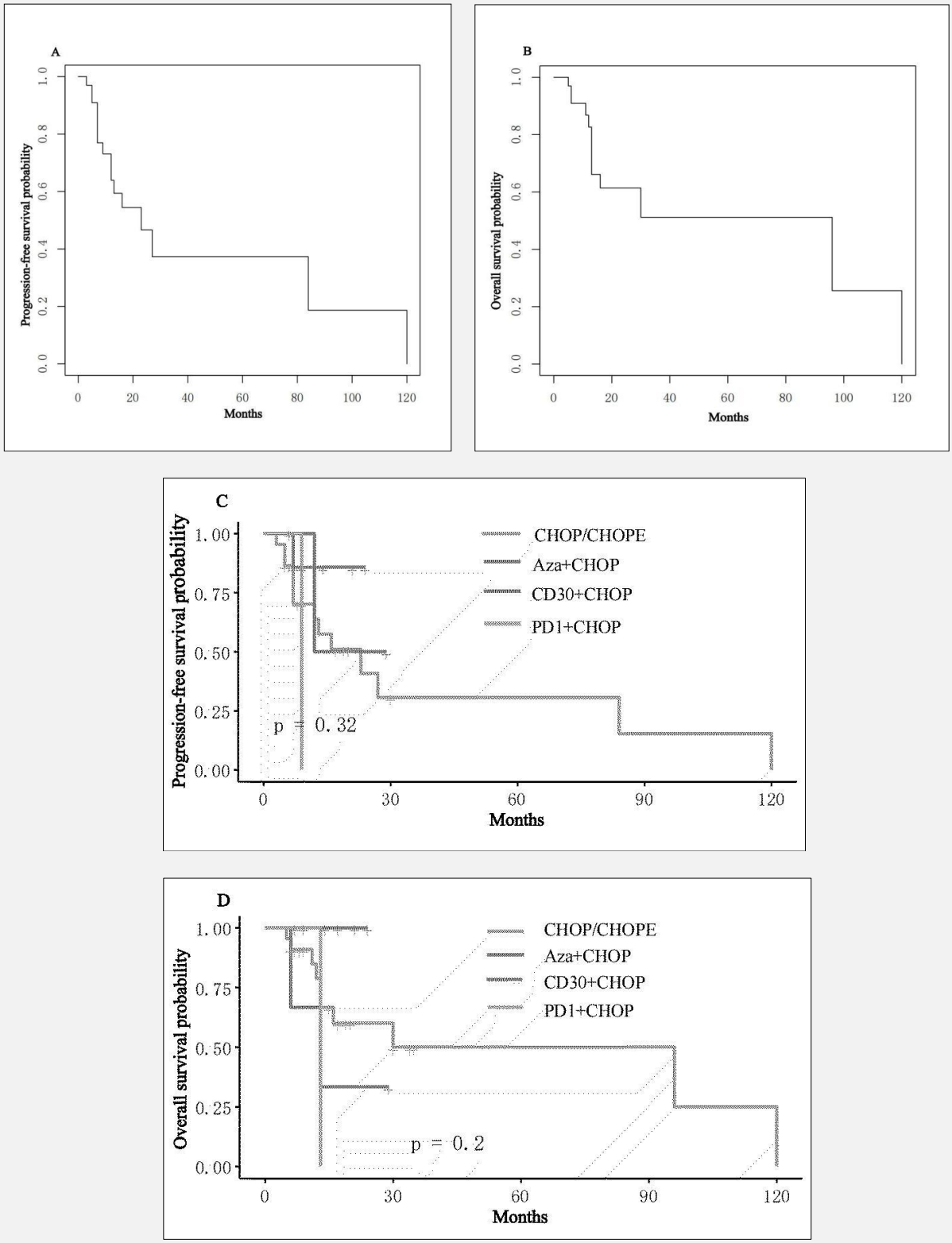

Figure 1. Survival of the patients with AITL, PFS (A) and OS (B).

Survival probabilities of the AITL cohort. Kaplan-Meier curves show PFS (C) and OS (D) in patients with AITL stratified by treatment methods.

Overall survival outcomes
Kaplan-Meier survival analysis was performed to evaluate the prognostic outcomes of patients with AITL. Figure 1A displays the progression-free survival (PFS) curve for the entire cohort, while Figure 1B illustrates the overall survival (OS) pattern. The median PFS and OS were nine months and thirteen months, respectively.

Treatment-stratified survival analysis
To assess the impact of different therapeutic approaches on patient outcomes, we stratified the cohort by treatment methods. Figure 1C shows the PFS curves for patients grouped by treatment protocols, while Figure 1D presents the corresponding OS curves. The analysis revealed differential survival patterns across treatment groups.

DISCUSSION

This comprehensive analysis of 33 patients with AITL provides valuable insights into the molecular and clinical characteristics of this aggressive lymphoma subtype. Our findings highlight several key aspects of AITL pathobiology and clinical management.
The demographic profile of our cohort aligns with the established understanding of AITL as a disease predominantly affecting elderly individuals, with a median age of 64.1 years and male predominance [4,19]. The high proportion of patients presenting with advanced-stage disease (69.7% with stage IV) underscores the aggressive nature of AITL and potentially reflects diagnostic delays common in this disease entity [15].
Our pathological findings confirm the characteristic immunophenotype of AITL, with high expression of TFH-related markers (PD-1, CXCL13, BCL-6) and frequent CD10 positive [20,21]. The high prevalence of EBV infection (72.7% by EBER-ISH and 90% by EBV-DNA PCR) corroborates the well-established association between AITL and EBV [22,23]. While the exact role of EBV in AITL pathogenesis remains debated, it is thought to contribute to the proliferation of B cells within the tumor microenvironment and potentially influence disease progression [24,25].
The survival analysis identified several prognostic biomarkers in AITL. Lower albumin levels and elevated β2-MG emerged as significant predictors of both inferior PFS and OS, suggesting that these readily accessible laboratory parameters could be incorporated into risk stratification models. The protective effect of PD-1 positivity on PFS in our study, which is consistent with Yuta Tsuyuki et al. from Japan, found that PD-L1 expression positively correlated with survival in 39 patients with CNS diffuse large B-cell lymphoma in their retrospective analysis [26]. The result may reflect the biological heterogeneity within AITL or the complex interplay between tumor cells and the immune microenvironment [27].
Our treatment stratification analysis suggests differential outcomes based on therapeutic approach. While CHOP/CHOPE remains the backbone of treatment, the addition of novel agents targeting specific molecular markers (CD30, PD-1) or epigenetic modulators (azacitidine) appears promising. Despite the limited cohort size, which precluded the establishment of statistically significant differences. These findings align with the emerging paradigm of precision medicine in lymphoma management, where therapy is increasingly tailored to the molecular profile of the disease [28,29].
We acknowledge several limitations in our study. The retrospective design and the relatively small sample size could potentially restrict the broader applicability of our results. Additionally, comprehensive molecular profiling including next-generation sequencing was not performed, which might have offered more profound understanding of the genetic landscape of AITL.
In conclusion, our study contributes to the growing body of knowledge on AITL by characterizing its clinical, pathological, and prognostic features. The identification of albumin, β2-MG, and PD-1 as potential prognostic biomarkers warrants further investigation in larger cohorts. Future studies should focus on integrating clinical, pathological, and molecular data to develop risk-adapted treatment strategies and explore novel therapeutic approaches targeting the unique biological features of AITL.

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No funding was received for this study.

Declaration of Interest:
The authors declare no conflicts of interest.

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