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T-Cell-Rich Large B-Cell Lymphoma: Biology and Prognosis

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ABSTRACT

Background: T-cell/histiocyte-rich large B-cell lymphoma (T/HRBCL, THRLBCL) is an uncommon morphological variant of diffuse large B-cell lymphoma (DLBCL) characterized by a distinctive tumor microenvironment and variable clinical behavior.

Objective: to analyze the clinicopathologic feature of T/HRBCL at a single tertiary center and compare presentation, treatment, and survival with a contemporaneous cohort of conventional DLBCL.

Methods: this retrospective cohort study was conducted at the Oncology Center, Mansoura University between January 2015 and December 2022. Thirty-six patients with T/HRBCL were included and compared with 203 randomly selected, unmatched DLBCL controls. Demographic, laboratory, and pathologic data, treatments, and outcomes were extracted. Responses were assessed by Lugano criteria, and OS and PFS were estimated using Kaplan–Meier methods with log-rank testing.

Results: Patients with T/HRBCL were younger, predominantly male, and more frequently presented with B-symptoms, elevated LDH, and advanced disease compared with conventional DLBCL. Most received R-CHOP, achieving a complete remission rate of 59.4%, slightly lower than DLBCL (67.3%) but not statistically significant. Salvage regimens, particularly GDP (gemcitabine, dexamethasone and cisplatin), produced encouraging results with a CR rate of 62.5%. Despite initial responses, survival was inferior in T/HRBCL, with median PFS (progression-free survival) and OS (overall survival) significantly shorter than in DLBCL (14 vs. 40 months for PFS, $p = 0.001$; 67 months vs. not reached for OS, $p < 0.001$). Male sex was an independent adverse prognostic factor for OS.

Conclusion: T/HRBCL presented with distinct clinicopathologic features and earlier progression compared with conventional DLBCL despite frequent use of R-CHOP. The data support careful early risk-stratification and exploration of biology-driven therapeutic strategies.

Keywords: THRLBCL, T-cell/histiocyte-rich large B-cell lymphoma, DLBCL, Prognosis, R-CHOP.

INTRODUCTION

Diffuse large B-cell lymphoma (DLBCL) is recognized as the most prevalent lymphoid malignancy observed in the adult population, constituting a significant proportion, nearly 40%, of all non-Hodgkin lymphoma cases. Within this broad spectrum of B-cell malignancies, T-cell/histiocyte-rich large B-cell lymphoma (THRLBCL) represents an uncommon morphological subtype, accounting for only 1% to 3% of cases [(1)].

Despite its low incidence, the accurate identification of this variant is critically important due to its distinct histopathological presentation and unique clinical features, which differentiate it from the more common forms of DLBCL. Histologically, THRLBCL is defined by a sparse distribution of large neoplastic B cells, typically constituting fewer than 10% of total cellularity, dispersed within an abundant reactive background composed predominantly of T cells and numerous histiocytes. Despite the high T-cell-to-tumor-cell ratio, the malignant B cells employ immune evasion strategies that neutralize the host's cytotoxic response. This process is mediated in part through cytokine signaling pathways and by the expression of programmed death ligand 1 (PD-L1) on both tumor and stromal cells, which engage the PD-1 receptor on T cells, inducing functional exhaustion and immune tolerance [(2)]. The reactive T cells are characterized predominantly by a CD8+ TIA-1+ granzyme B− cytotoxic phenotype, indicative of an active yet ultimately ineffective immune response. Immunophenotypically, the malignant B cells resemble germinal center B cells, expressing pan-B-cell markers such as CD20 and CD79a, as well as BCL-6, but with minimal or absent CD10 expression, a feature useful for differential diagnosis [(3)].

The distinct morphological pattern exhibited by T-cell/histocyte-rich large B-cell lymphoma (THRLBCL) presents a significant diagnostic challenge for pathologists. This unique histological presentation, characterized by a sparse infiltrate of malignant B cells within a dense reactive microenvironment, frequently results in the misclassification of the entity as other lymphoid neoplasms, including peripheral T-cell lymphoma or nodular lymphocyte-predominant Hodgkin lymphoma [(1, 5)].

This potential for misdiagnosis underscores the need for careful immunophenotypic analysis. Clinically,

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THRLBCL is acknowledged as an aggressive pathological entity. The majority of affected patients are diagnosed at an advanced clinical stage, and this advanced presentation is typically characterized by extensive nodal involvement, which is observed in more than 60% of cases [4]. This aggressive behavior and widespread dissemination upon initial diagnosis contribute to a challenging clinical course and often a guarded prognosis.

Therapeutically, THRLBCL is generally managed with rituximab-based immunochemotherapy, most commonly R-CHOP (rituximab, cyclophosphamide, doxorubicin, vincristine and prednisone) [4, 5].

Historically, prognosis was poor, with reported overall survival rates of less than 50% at three years in the pre-rituximab era [6].

The incorporation of rituximab significantly improved outcomes across DLBCL subtypes, but the efficacy of R-CHOP in THRLBCL remains a subject of debate. Several studies indicate that a proportion of patients continue to experience inferior responses and outcomes compared with conventional DLBCL [5].

Given the aggressive clinical and biological profile of THRLBCL, alternative treatment strategies have been explored. Retrospective analyses suggest that intensified regimens, such as dose-adjusted R-EPOCH or sequential R-CHOP followed by R-ICE (ifosfamide, carboplatin and etoposide), may achieve superior results compared with R-CHOP alone [7]. These findings support the hypothesis that the unique pathobiology of THRLBCL may necessitate more potent therapeutic approaches to overcome intrinsic resistance mechanisms and improve long-term outcomes.

AIM OF THE STUDY

This study aims to provide a comprehensive analysis of THRLBCL outcomes in the modern treatment era. We compare the efficacy of different rituximab-based immunochemotherapy regimens to identify an optimal treatment strategy. The findings provide crucial evidence for guiding clinical practice and highlight the necessity of considering alternative, more aggressive therapeutic approaches for newly diagnosed patients with this challenging lymphoma subtype.

PATIENTS AND METHODS

Study design and setting: A retrospective cohort study was conducted at the Oncology Center, Mansoura University (OCMU), Egypt, covering the period from January 2015 to December 2022. The institutional review board approved the study.

Patients: All patients with a histopathological diagnosis of T/HRBCL confirmed by expert hematopathologists were included (n=36). A comparison group of 203 randomly selected patients with conventional DLBCL was used as a control cohort.

Inclusion criteria: Histologically confirmed T/HRBCL or DLBCL. Complete clinical, laboratory, and follow-up data. Minimum follow-up of six months.

Exclusion criteria: Relapsed/refractory cases referred after prior treatment. Incomplete medical records.

Data collection: Clinical data included demographics, performance status, B-symptoms, and disease stage. Laboratory parameters included CBC, LDH, liver function (including bilirubin), and viral serology (HCV). Staging was based on Ann Arbor classification; prognostic risk was assessed by International Prognostic Index (IPI).

Treatment and response assessment: Treatment regimens were extracted from records. R-CHOP was the standard first-line therapy; dose-modified regimens were used for frail patients. Salvage therapies included GDP, DHAP, and ICE. Response was evaluated using Lugano criteria.

Ethical consideration: The research protocol received approval from Institutional Research Board (IRB) of Mansoura University, as well as administrative clearance from the respective healthcare facilities where the study was conducted. Owing to its retrospective nature, informed consent was obtained, nevertheless, strict confidentiality was maintained, and patient anonymity was safeguarded against unauthorized disclosure. The study adhered to the ethical standards of Declaration of Helsinki and complied with all relevant clinical research guidelines.

Statistical analysis

Data were tabulated and analyzed using SPSS software version 26.0 (IBM Corp., Armonk, NY, USA). Quantitative variables were compared using the independent t-test for normally distributed data and the Mann–Whitney U test for non-parametric distributions. Categorical variables were analyzed with the Chi-square test. A p-value ≤ 0.05 was considered statistically significant. Survival outcomes, including overall survival (OS) and progression-free survival (PFS), were estimated using the Kaplan–Meier method and compared with the log-rank test. Univariate and multivariate Cox proportional hazards models were applied to determine independent prognostic factors for survival.

RESULTS

A total of 620 patients with DLBCL were identified. Of these, 240 were initially randomly selected

for the control arm. However, due to missing records, 37 patients were excluded, resulting in a final control group of 203 patients. Additionally, all 36 available patients with (T/HRBCL) were included in the analysis, representing approximately 5.55% of the total cases. At the end of the follow-up, with a median of 36 months, 18 out of 36 patients in the T/HRBCL group had died (50%), while the remaining 18 patients (50%) were either alive or lost to follow-up. In the DLBCL group, 34 out of 203 patients had died (16.7%), with 169 patients (83.3%) either alive or lost to follow-up (Figure 1).

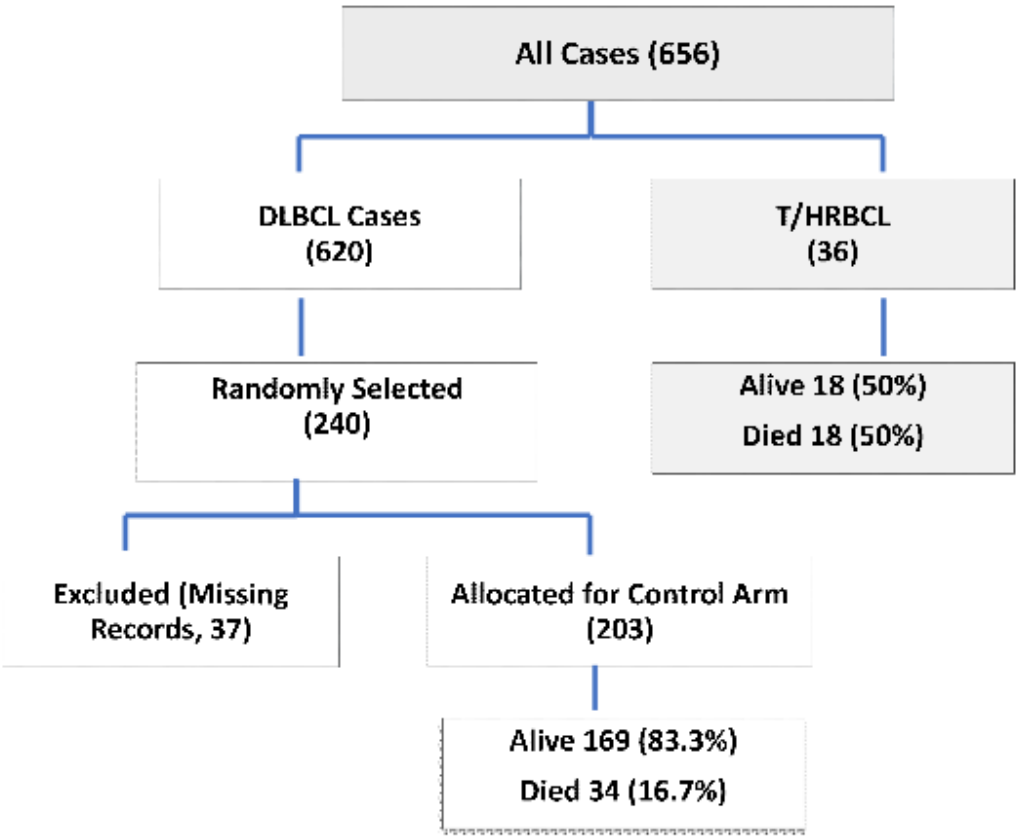

Figure 1: Flow diagram of patient selection and allocation to study groups

Table 1 present the baseline characteristics of the T/HRBCL population. The median age of the patients was 39.5 years (range 21-66 years). The incidence was higher in males, accounting for 69.4% of cases. HCV antibodies were positive in 33% of the cases. B symptoms were present in 58% of the patients at the time of initial presentation. Regarding baseline laboratory investigations, more than half of the patients had anemia at presentation, while bone marrow infiltration was observed in only 5.6% of cases. Elevated LDH levels were noted in 89% of the patients. The majority of THRBCL patients presented with advanced-stage disease, with only 19.5% having stage I or II disease. Furthermore, a significant proportion (36%) were classified as high or intermediate-high risk based on the IPI score.

Table (1): Baseline characteristics of the T/HRBCL population

		N (36)	%
Sex	**Female**	11	30.6%
	Male	25	69.4%
Age Category (year)	**≤40**	19	52.8%
	>40-≤60	15	41.7%
	>60	2	5.6%
DM		3	8.3%
Hypertension		3	8.3%
HCV		12	33.3%
B-Symptoms		21	58.3%
WBCs Count	**Leucopenia**	8	22.2%
	Leukocytosis	5	13.9%
Anemia		20	55.6%
Thrombocytopenia		7	19.4%
Elevated serum bilirubin		8	22.2%
Elevated LDH		32	88.9%
Stage	**I**	1	2.8%
	II	6	16.7%
	III	21	58.3%
	IV	8	22.2%
BM infiltration		2	5.6%
Extranodal		4	11.1%
Bulky Disease		4	11.1%
IPI score	**Low**	5	13.9%
	Low-Intermediate	18	50.0%
	High-Intermediate	11	30.6%
	High	2	5.6%

Table (2) show the most patients (89%) received R-CHOP as first-line treatment, with 55.4% achieving CR (complete remission). Four patients received R-miniCHOP or COP as first-line treatment due to compromised cardiac or liver function, or frailty, with 50% (2 patients) achieving CR. For patients who relapsed or failed to achieve remission with initial therapy, salvage chemotherapy was administered. Among the 13 patients who received the DHAP protocol as salvage chemotherapy, the CR rate was 46%. Of the 8 patients treated with the GDP regimen, 62.5% (5 patients) achieved CR. Additionally, 1 patient received ICE as salvage therapy and achieved CR.

Table (2): Response to treatment

Regimen		N (%)	CR rate, N (%)
First line Chemotherapy	**R-CHOP**	32 (88.9%)	19 (%59.4)
	R-MiniCHOP/COP	4 (11.1%)	2 (50%)
Salvage Chemotherapy	**GDP**	8 (36.4%)	5 (62.5%)
	DHAP	13 (59.1%)	6 (46.2%)
	ICE	1 (4.5%)	1 (100%)

* In patients who received R-mini-CHOP/COP CRR was 50%.

Table 3 present a comparative analysis of the characteristics between the DLBCL and T/HRBCL groups. The T/HRBCL cohort demonstrated a significantly higher incidence among males and in patients under 60 years of age, with p-values of 0.038 and 0.001 respectively. Additionally, the T/HRBCL group exhibited a significantly higher prevalence of B symptoms, hyperbilirubinemia, and elevated LDH levels. Conversely, the DLBCL group had a significantly higher IPI score ($p = 0.002$). No significant differences were observed between the groups for the remaining characteristics outlined in the table.

Table (3): Comparison of the characteristics between the 2 arms

		DLBCL (203) N (%)	T/HRBCL (36) N (%)	p
Sex	**F**	100 (49.3%)	11 (30.6%)	0.038
	M	103 (50.7%)	25 (69.4%)	
Age Category (year)	**≤60**	137 (67.5%)	34 (94.4%)	0.001
	>60	66 (32.5%)	2 (5.6%)	
DM		36 (17.7%)	3 (8.3%)	0.16
Hypertension		36 (17.7%)	3 (8.3%)	0.16
HCV		79 (38.9%)	12 (33.3%)	0.52
Stage	**I-II**	29 (14.3%)	7 (19.4%)	0.42
	III-IV	174 (85.7%)	29 (80.6%)	
Anemia		118 (58.1%)	20 (55.6%)	0.77
Thrombocytopenia		38 (18.7%)	7 (19.4%)	0.91
Hyperbilirubinemia		16 (7.9%)	8 (22.2%)	0.008
B-Symptoms		46 (22.7%)	21 (58.3%)	0.001
Elevated LDH		139 (68.5%)	32 (88.9%)	0.012
BM infiltration		24 (11.8%)	2 (5.6%)	0.26
IPI Score	**Low** Low-Intermediate/	74 (36.5%)	23 (63.9%)	0.002
	High-Intermediate/High	129 (63.5%)	13 (36.1%)	
First-Line	**R-CHOP**	167 (82.3%)	32 (88.9%)	
	R-MiniCHOP	31 (15.3%)	4 (11.1%)	
	R-COP	5 (2.5%)	0 (0.0%)	

CR (complete remission) rate was evaluated for both the T/HRBCL and DLBCL groups, focusing exclusively on patients who received R-CHOP therapy. In the T/HRBCL group, the CR rate was 59.4%, while in the DLBCL group, it was slightly higher at 67.3%. Despite the numerical difference favoring the DLBCL group, this variation did not reach statistical significance (p = 0.38). For a visual representation of these findings, refer to Figure 2.

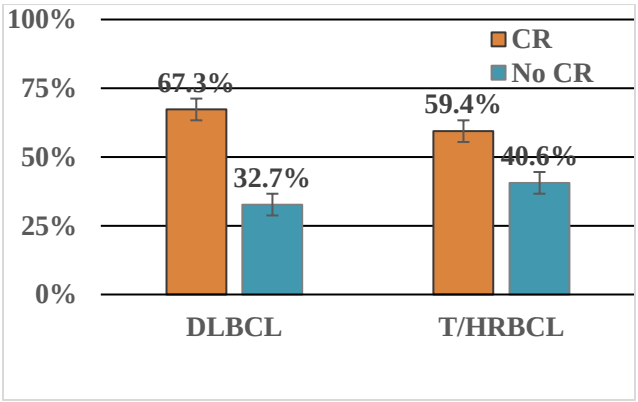

Figure 2: Comparison of Complete Remission Rates Between DLBCL and T/HRBCL.

Figure 3 show Kaplan-Meier Curves for OS and PFS in the T/HRBCL Variant. The median OS was 67 months, while the median PFS was 14 months (95% CI: 7-21 months). The substantial difference between OS and PFS is mainly due to disease progression within the first 24 months, particularly during the initial 12 months. Beyond this period, the OS and PFS curves become parallel, indicating similar rates of progression and survival thereafter.

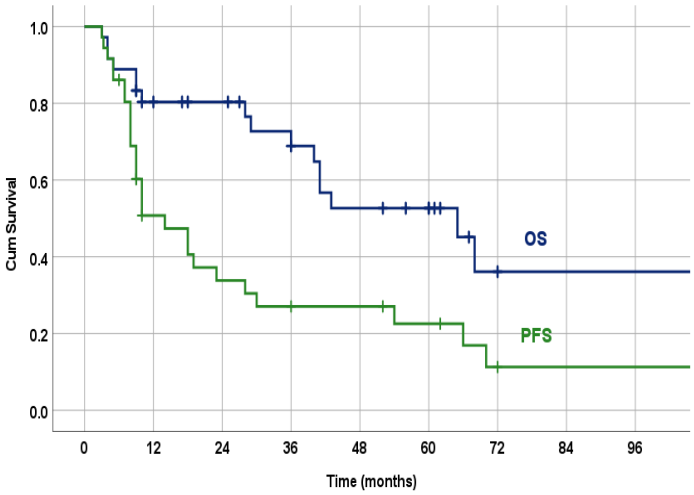

Figure 3: Kaplan-Meier Curves for OS and PFS in T/HRBCL Variant

The median PFS in the DLBCL group was 40 months (95% CI: not reached), significantly longer than the 14 months observed in the T/HRBCL group (95% CI: 7-21 months; p = 0.001), as shown in Figure 4.

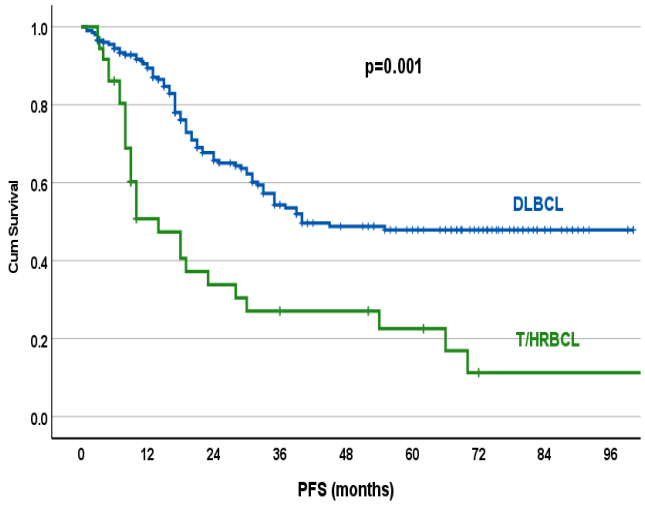

Figure 4: Kaplan-Meier Curves Comparing PFS between DLBCL and T/HRBCL.

The median OS for T/HRBCL arm was 67 months (95% CI, 37-92 months) compared to not reached in DLBCL. This difference was statistically significant (p < 0.001) Figure 5.

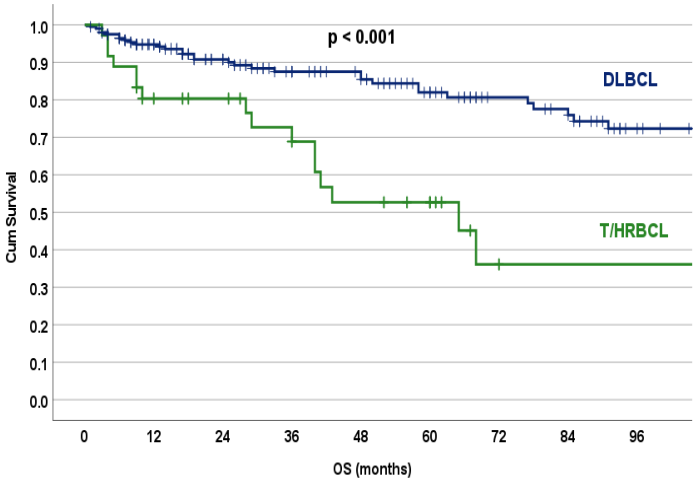

Figure 5: Kaplan-Meier Curve Comparing Overall Survival Between DLBCL and T/HRBCL.

Table 4 show Cox univariate analysis of OS revealed that only male sex was a significant poor prognostic factor. Other variables, including age over 60 years, HCV positivity, advanced stage, anemia, elevated LDH levels, presence of B symptoms, and high IPI score, did not show prognostic significance for OS.

Table (4): Univariate Analysis of Prognostic Factors of OS

	HR	95.0% CI	p.
Male sex	9.580	1.72 – 53.1	0.01
Age >60	5.636	0.40 - 9.37	0.20
HCV positive	1.585	0.44 - 5.5	0.47
Stage III and IV	0.279	0.05 - 1.46	0.13
Anemia	1.737	0.36 - 8.40	0.49
B-Symptoms	2.458	0.74 - 8.14	0.14
Elevated serum LDH	1.579	0.17 – 14.2	0.68
IPI score (high intermediate/ high)	2.474	0.48 - 12.5	0.27

DISCUSSION

T/HRBCL constitutes a biologically and clinically distinct variant of DLBCL, with disease characteristics that exert a profound influence on prognosis. In our cohort, affected patients were significantly younger, predominantly males, and more frequently exhibited B-symptoms, elevated LDH levels, and advanced-stage disease at diagnosis when compared with conventional DLBCL. These findings underscore the aggressive clinical behavior of T/HRBCL and corroborate previous reports describing its presentation as more symptomatic and advanced [8].

Granting the majority of patients in this series received R-CHOP, the CR rate was inferior to that typically achieved in DLBCL, and both progression-free survival and overall survival were significantly shorter. These observations indicate that standard immunochemotherapy, while capable of inducing remission, does not confer durable disease control in T/HRBCL. Similar conclusions were drawn by **Ollila *et al.*** [4] who reported inferior long-term outcomes in T/HRBCL despite rituximab introduction. By contrast, **Robin *et al.*** [5] suggested that intensified therapeutic regimens may partially improve outcomes in select patients, although evidence remains limited.

In the present study, the median age at diagnosis was 39.5 years with a marked male predominance (69.4%), in contrast to **Wei et al.** [9] who described a higher median age of onset, typically between 49 and 57 years. Furthermore, HCV seropositivity was identified in one-third of cases, which consistent with **Couronné *et al.*** [10] who reported associations between chronic viral infection and NHL pathogenesis. To our knowledge, this represents the first report to suggest a potential link between HCV infection and the development of T/HRBCL.

The aggressive nature of the disease was further reflected by the high prevalence of B-symptoms (58.3%), advanced disease stage (III–IV in 80.5%), and elevated

LDH levels (88.9%). These proportions were significantly higher than those observed in conventional DLBCL (22.7% and 68.5% respectively; p = 0.001 and 0.012). These findings parallel earlier comparative studies conducted between 2010 and 2015, which similarly demonstrated higher rates of B-symptoms (44% vs. 28%) and advanced disease (81% vs. 56%) in T/HRBCL compared with DLBCL-NOS [4].

Therapeutically, R-CHOP achieved a CR rate of 59.4%, modestly lower than the 67.3% reported in DLBCL, although the difference was not statistically significant. This suggests that R-CHOP retains efficacy in T/HRBCL despite the aggressive clinical features. Importantly, patients who did not achieve remission or who relapsed responded favorably to salvage regimens, with GDP demonstrating a CR rate of 62.5%, indicating potential utility as a second-line option.

The limited durability of remission in T/HRBCL is likely attributable to its distinctive molecular and microenvironmental features. Genomic analyses by **Schuhmacher *et al.*** [11] identified recurrent alterations in genes regulating immune responses, transcriptional programs, and somatic hypermutation, supporting intrinsic mechanisms of tumor persistence under immune pressure. Complementary spatial profiling studies demonstrated upregulation of the PD-1/PD-L1 axis and signatures of immune exhaustion, indicating that malignant B cells evade immune surveillance despite abundant reactive T-cell infiltrates [12]. Collectively, these findings provide a mechanistic explanation for the suboptimal clinical outcomes observed.

The inferior survival outcomes in this cohort further substantiate the recognition of T/HRBCL as a biologically high-risk lymphoma. Notably, the association of male sex with worse prognosis mirrors prior reports, suggesting potential sex-linked biological or immunological factors that warrant further exploration [13].

In light of these challenges, novel therapeutic strategies are urgently required. While CD19-directed CAR, T-cell therapy has demonstrated transformative efficacy in relapsed/refractory DLBCL, registry-level data reported by **Pophali *et al.*** [14] indicate only modest disease control in T/HRBCL. Similarly, institutional experiences from **Nair *et al.*** [15] highlighted limited durable responses, reinforcing the impact of the profoundly immune-evasive microenvironment. Given the prominent PD-1/PD-L1 axis dysregulation in T/HRBCL, immune checkpoint inhibition represents a biologically rational approach. Reviews by **Xie *et al.*** [16] have emphasized the therapeutic relevance of PD-1/PD-L1 inhibitors in lymphomas driven by immune exhaustion. Early clinical experiences combining CAR T-cell therapy with checkpoint blockade, or employing PD-1 inhibitors in relapsed T/HRBCL, suggest potential benefit, although robust prospective data remain lacking [17].

Taken together, the available evidence highlights the necessity of a multipronged therapeutic strategy for T/HRBCL. Approaches integrating immune checkpoint blockade, epigenetic modulation, and CAR T-cell optimization may be required to achieve durable disease control. Our findings, together with the broader literature, strongly support the notion that T/HRBCL should not be regarded as a mere morphological variant of DLBCL but rather as a distinct clinicopathological entity with unique biological features and therapeutic vulnerabilities.

CONCLUSION

T/HRBCL represents an aggressive variant of large B-cell lymphoma with distinct biological and clinical features. Although the overall prognosis was poorer than that of DLBCL, first-line treatment with R-CHOP remains effective in a significant proportion of patients. However, the high rate of early disease progression highlights the need for close monitoring and potentially more aggressive or novel therapeutic strategies to improve outcomes in this population.

LIMITATIONS

Our study is limited by its retrospective nature, relatively small sample size, and single-center design, which may introduce selection bias and limit generalizability. The lack of prospective molecular or immune profiling in all patients constrains the ability to correlate specific biomarkers with outcomes. In addition, the DLBCL comparator cohort was unmatched, which may introduce confounding. Finally, in some patients salvage therapies were heterogeneous, making it difficult to isolate the effect of specific regimens.

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