ORIGINAL ARTICLE

Serum LDH Concentration Level in Hodgkin's and Non-Hodgkin's Lymphomas

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ABSTRACT

Background: Mostly, the lymphomas that are reported globally are either Hodgkin's Lymphoma or Non-Hodgkin Lymphoma. LDH is an enzyme involved in glycolysis and is considered a biomarker of cellular activity and tumor dynamics.

Objective: The study primarily aims to evaluate LDH concentrations in the blood of patients with Hodgkin and Non-Hodgkin Lymphomas. The secondary objective is to determine the association between LDH levels and disease stage, and tumor proliferative index (Ki-67).

Methods: This is a cross-sectional study conducted at Jinnah Postgraduate Medical Center in Karachi from April 2025 to September 2025. Serum LDH was evaluated in all patients with Hodgkin and Non-Hodgkin Lymphoma. Patients with infections, concurrent malignancies, and missing data were excluded from the study. Demographics, LDH levels, Ann Arbor Stage, and tumor proliferative index (Ki-67) were recorded. A non-parametric method and Spearman's correlation were employed in SPSS.

Results: Among 200 equally divided patients of HL and NHL, LDH levels are higher in the NHL group (median 350 U/L) as compared to the HL group (median 220 U/L), consistent with the non-normal distribution. With progressing disease stage, LDH levels elevated steadily in the NHL group ($p<0.001$). A significant positive relationship has been found between LDH and Ki-67 proliferation index ($r=0.52$, $p<0.001$).

Conclusion: LDH appears to be a cost-effective and practical biomarker for identifying tumor burden, biological aggressiveness, and tumor proliferation. Its higher levels in the NHL indicate its significance for risk identification in resource-limited settings.

Keywords: *Lymphoma, Hodgkin lymphoma, non-Hodgkin lymphoma, lactate dehydrogenase, Ki-67 antigen.*

INTRODUCTION

Lymphomas are a diverse group of malignancies arising from lymphoid tissues, broadly divided into two principal categories: Hodgkin lymphoma and non-Hodgkin lymphoma. Hodgkin and Non-Hodgkin lymphomas account for a considerable proportion of hematological cancers worldwide [1]. According to GLOBOCAN 2022, the incidence rate of Hodgkin Lymphoma (HL) was 37.7% across Asia, while the incidence rate of Non-Hodgkin Lymphoma (NHL) was 42.5% in Asia [2, 3]. Treatment decisions and prognosis are largely dependent on the extent and biological behavior of the disease. Over the past few decades, serum Lactate dehydrogenase (LDH) has emerged as an economical and practical biomarker in oncology for evaluating tumor burden in Hodgkin and Non-Hodgkin Lymphoma [4]. This enzyme tends to rise in circulation with cell destruction and increasing tumor burden, indicating that LDH correlates with advanced-stage malignancy and less favorable outcomes [5, 6].

The presence of Reed-Sternberg cells characterizes Hodgkin lymphoma. It is classified by the World Health Organization (WHO) into two main subtypes: classical HL (comprising nodular sclerosis, mixed cellularity, lymphocyte-rich, and lymphocyte-depleted variants) and nodular lymphocyte-predominant HL. Disease extent is described using the Ann Arbor staging system (Stages I-IV), where Stage I denotes involvement of a single lymph node region and Stage IV indicates disseminated extranodal disease.

Non-Hodgkin Lymphoma consists of B-cell and T-cell types. The B-cell types include follicular lymphoma, marginal zone lymphoma, diffuse large B-cell lymphoma (DLBCL), and Burkitt lymphoma. In contrast, T-cell lymphomas are peripheral T-cell lymphoma and anaplastic large cell lymphoma. NHL is staged according to the Ann Arbor System, with stage III-IV as the advanced disease stage. The biological heterogeneity among these subtypes and stages reflects LDH variation, making LDH a potential biomarker for assessing the lymphoma spectrum.

LDH has been identified as the independent predictor of poor prognosis in cases of non-Hodgkin's lymphoma [7, 8]. It is used as a risk assessment tool, such as the NCCN-IPI score, in which elevated levels indicate extranodal spread, bulky disease, and increased tumor proliferation [9,10]. In Hodgkin Lymphoma, LDH elevation is less associated with disease prognosis and is more relevant for advanced disease stages. The

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association between LDH and lymphomas can be explained by the Warburg effect, in which tumor cells shift to aerobic glycolysis, increasing LDH production and subsequently raising LDH levels in circulation [11-13].

The main objective of this study is to determine serum LDH concentrations in patients with HL and NHL. Secondary objectives are to identify associations between LDH and disease stage, histological subtypes, and proliferative index (Ki-67). By defining these correlations, LDH may serve as an economical yet practical biomarker to assist physicians in evaluating the disease stage and tumor burden.

METHODOLOGY

A cross-sectional study was carried out at the Department of Oncology, Jinnah Postgraduate Medical Center, Karachi, from April 2025 to September 2025. The study was performed with permission of the Institutional Review Board (IRB) of Jinnah Postgraduate Medical Center, Karachi (Reference No. F.2-81/2025-GENL/306/JPMC). All histologically confirmed, treatment-naïve patients with Hodgkin and Non-Hodgkin Lymphoma are eligible for enrollment in the study. Exclusion criteria included active infections, concurrent malignancies, and incomplete laboratory records. Written consent was obtained from all patients.

A sample size of 75 patients per group was calculated, using the mean serum LDH levels (384.2 ± 264.9) and non-LDH (289.5 ± 121.7) [14], with a 95% confidence interval and 80% power. Sample size calculation was performed using the online OpenEpi tool, using a two-sample mean size calculation. The computed sample size was increased to 100 to increase the study's power. Thus, 100 patients per group were enrolled in this study.

Serum LDH was measured at baseline, before initiation of any antineoplastic therapy, using a kinetic colorimetric assay on the Roche Cobas c501 automated analyzer (Roche Diagnostics, Mannheim, Germany). The institutional laboratory reference range for serum LDH is 120-240 U/L. Values above this range were defined as elevated. Ki-67 proliferation index was retrieved from diagnostic biopsy immunohistochemistry reports where available; cases in which Ki-67 data were absent from the pathology record were excluded from the Ki-67 correlation analysis (n=28; 14% of total cohort). Histological subtype distribution among HL patients was as follows: nodular sclerosis (n=62, 62%), mixed cellularity (n=28, 28%), lymphocyte-rich (n=7, 7%), and lymphocyte-depleted (n=3, 3%). Among NHL patients, the most frequent subtypes were DLBCL (n=55, 55%), follicular lymphoma (n=18, 18%), peripheral T-cell lymphoma (n=12, 12%), marginal zone lymphoma (n=9, 9%), and other subtypes (n=6, 6%).

Data were collected from current medical records and hospital documentation. After obtaining consent, we extracted relevant details from clinical files, histopathology, and laboratory reports. Data was collected through a standardized data form. It consists of information for each subject: age and sex; lymphoma subtype, classified according to WHO guidelines; Ann Arbor stage; serum LDH, measured in U/L; and, where possible, the Ki-67 percentage from diagnostic biopsy samples. Using the same form for everyone minimized variation in how data were recorded.

All statistical work was carried out using SPSS (version appropriate for the study period). We first assessed the normality of continuous variables using the Shapiro-Wilk test. Normally distributed data were summarized as means with standard deviations, while non-normally distributed data were summarized as medians with interquartile ranges. For two-group comparisons, we applied the independent t-test or the Mann-Whitney U test, depending on the distribution. When comparing three or more groups, one-way ANOVA or Kruskal-Wallis testing was used. Categorical variables underwent chi-square or Fisher's exact testing as appropriate. To explore relationships between LDH and continuous measures like Ki-67, we calculated Spearman's correlation coefficients. A p-value below 0.05 (two-tailed) marked statistical significance across all analyses.

RESULTS

Compared to HL patients, NHL patients had a significantly higher mean age (47.0 *vs.* 35.5 years, p<0.001). In both groups, a male predominance was noted, but this was not significant (p=0.309). Compared to HL patients, NHL patients were more likely to present with advanced-stage disease (Stage III-IV) (71% *vs.* 52%) (p=0.006). Furthermore, the NHL group had more comorbidities (32%) than the HL group (18%) (p=0.022) (**Table 1**).

Table 1: Baseline characteristics of studied lymphoma patients (n=200).

Variables	Hodgkin Lymphoma	Non-Hodgkin Lymphomas	Total	p-value
Age (years), mean ± SD	35.5 ± 12.1	47.0 ± 15.3	41.0 ± 14.6	*<0.001
Gender				
Male	58 (58)	65 (65)	123 (61.5)	0.309
Female	42 (42)	35 (35)	77 (38.5)	
Clinical stage, n (%)				
Stage I-II	48(48)	29 (29)	77 (38.5)	*0.006
Stage III-IV	52 (52)	71 (71)	123 (61.5)	
Comorbidities, n (%)	18 (18)	32 (32)	50 (25)	*0.022

*Significant at p<0.05

Patients with non-Hodgkin's lymphoma had significantly greater serum LDH levels than those with Hodgkin's lymphoma. Table **2** indicates that the median LDH for HL was 220 U/L (range: 124-695 U/L), and for NHL, 350 U/L (range: 101-3539 U/L). The p-value was statistically significant (p<0.001).

A comparison of serum LDH between the two groups revealed a clear and statistically significant difference. Given that LDH data were non-normally distributed (Shapiro-Wilk test, p<0.05), the primary comparison was performed using the Mann-Whitney U test. The median LDH for NHL patients (350 U/L, IQR: 210-680 U/L) was significantly higher than for HL patients (220 U/L, IQR: 155-320 U/L; p<0.001). For descriptive purposes, mean values were also recorded: NHL 612 U/L (SD 201.7) *versus* HL 245 U/L (SD 98); the large SD in the NHL group reflects the marked skewness and heterogeneity of LDH values in this cohort. A supplementary independent t-test similarly confirmed this difference (t = -6.45, p < 0.001), though non-parametric results should be considered primary. Because LDH data did not follow a normal distribution—as confirmed by the Shapiro-Wilk test—we also ran a non-parametric Mann-Whitney U test as a safeguard. That analysis returned the same conclusion (p < 0.001), reinforcing confidence in the finding.

Table 2: Comparison of serum LDH levels between Hodgkin lymphoma and non-Hodgkin lymphomas (n=200).

Group	Mean LDH (U/L)	SD	Median	Min-Max	p-value (Independent t-test)	Mean Difference
Hodgkin Lymphoma	245	98.4	220	124-695	p < 0.001	-367 U/L
Non-Hodgkin Lymphoma	612	201.7	350	101-3539		

Patients with stage I disease demonstrated no significant difference in LDH levels between HL and NHL (p=0.290). Serum LDH was higher in patients having stage II (p=0.003), III (p<0.001), and IV (p<0.001) (**Table 3**).

Table 3: Comparison of serum LDH Levels between HL and NHL stratified on clinical stages (n=200).

Clinical Stage	Hodgkin Lymphoma	Non-Hodgkin Lymphomas	p-value
I	190.6 ± 55.8	210.2 ± 72.3	0.290
II	215.3 ± 78.7	295.4 ± 98.7	*0.003
III	260.9 ± 92.5	480.7 ± 170.2	*<0.001
IV	340.2 ± 108.6	890.6 ± 356.8	*<0.001

Data is expressed as mean ± SD,*Significant at p<0.05

Fig. (**1**) displays a scatter plot showing the correlation between the Ki-67 proliferation index and serum LDH levels. Spearman's rank correlation revealed a positive correlation between serum LDH and the Ki-67 proliferative index (r=0.52, p<0.001), indicating that lymphomas with a high Ki-67 proliferative index exhibit higher LDH levels.

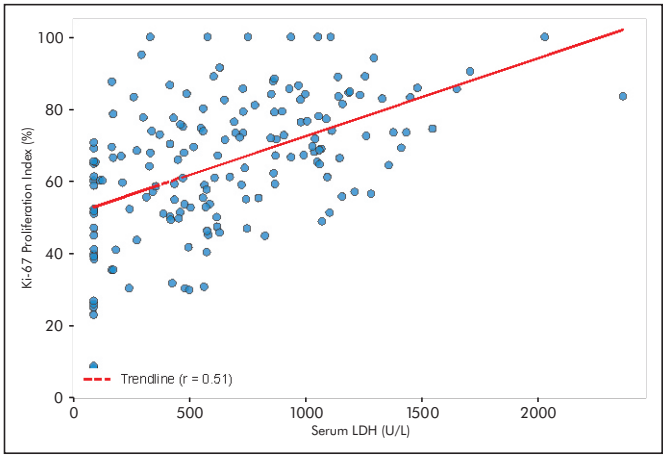

Fig. (1): A scatter plot showing the association between LDH levels and Ki-67 index across all the cases (Hodgkin lymphoma and non-Hodgkin lymphomas combined; n=172).

DISCUSSION

Our study clearly demonstrated the differences in serum LDH levels between Hodgkin and Non-Hodgkin's Lymphoma. Patients with NHL have significantly higher LDH concentrations than those with HL. While LDH's biological relevance is well-established in the Western world, this study contributes data from the Pakistani population, where patient demographics and lymphoma subtypes differ from those in the West, and where advanced diagnostic facilities are limited. The concurrent assessment of LDH and Ki-67 immunohistochemistry across both lymphoma types in a single cohort adds value in resource-limited settings. This assessment is aligned with the work by Gaikwad *et al.*, who reported similar findings [4]. Bakirtas *et al.* observed that pre-treatment LDH predicted the disease stage in diffuse large B-cell lymphoma [15]. The Laboratory Prognostic Index, which incorporates LDH with hemoglobin and β2-microglobulin, increased the significance of LDH in aggressive NHL subtypes [16].

Elevated LDH levels in the blood indicate an advanced disease stage, a finding more pronounced in NHL than in HL, where values increased fourfold between stages I and IV, aligning with our study. Liu and colleagues found that elevated serum LDH levels are a predictor of overall survival among 402 patients with advanced DLBCL [17]. These observations align with the established NCCN-IPI score, which assigns additional weight to LDH [18]. Similarly, Rabinovich and coworkers reported that LDH measured at CAR-T cell infusion predicted overall survival in patients with DLBCL [7].

A key finding was a positive relationship between LDH and Ki-67, offering a predictor for clinical patterns. Tumor cells shift towards aerobic glycolysis, which increases LDH production and its release into circulation; this process is known as the Warburg effect. Vastrad and

associates reported that Ki-67 exceeding 20% correlated strongly with elevated LDH and predicted worse progression-free survival in marginal zone lymphoma [19]. Liu and colleagues similarly identified both markers as independent variables in their DLBCL nomogram [17]. At the same time, Li and associates demonstrated that pre-treatment levels predicted CAR-T immunotherapy outcomes with considerable accuracy [20]. Early evidence suggests that LDH captures elements beyond tumor mass; in Hodgkin's lymphoma, Sincan and colleagues found that LDH correlated with interleukin-18 and β2-microglobulin, reflecting the immune activation mentioned [21].

The diversity of LDH values in our NHL group—from normal to multi-fold elevation—reflects the biological heterogeneity of this category. Indolent subtypes show minimal LDH variations while aggressive variants demonstrate marked elevations, consistent with LDH as a continuous correlate of disease stage. Future studies incorporating multivariable analysis adjusting for stage, age, histological subtype, and other potential confounders would strengthen causal inferences. The cross-sectional design of the present study precludes concluding survival, treatment response, or outcomes; the observed associations should be interpreted accordingly. We acknowledge limitations: the single-center cross-sectional design limits generalizability, and non-malignant conditions can affect LDH levels despite the exclusion criteria [22]. Longitudinal studies tracking LDH during treatment could clarify its prognostic value, particularly with novel therapies [7]. In resource-constrained settings, LDH is particularly relevant—a pediatric HL study from South Africa demonstrated that LDH, combined with ferritin and white cell counts, predicted chemosensitivity with considerable accuracy, potentially identifying patients who may safely forgo radiotherapy when PET-CT is unavailable [23]. These findings indicate that routine assessment of LDH concentrations may support risk stratification in resource-limited settings with limited access to advanced diagnostic facilities. However, prospective longitudinal studies are needed for validation before clinical recommendations can be made.

CONCLUSION

In conclusion, this study found that LDH concentrations differ between HL and NHL, with higher levels seen in the NHL group. The data further indicated that with aggressive histotypes and advanced disease stage, LDH levels are raised progressively. An important correlation is observed between LDH and Ki-67 (tumor proliferative index), indicating a direct relationship between elevated LDH and the proliferative activity of tumor cells.

According to the findings of this study, LDH appears to be an economical and readily available biomarker for identifying tumor burden, disease stage, and proliferative activity in HL and NHL. Because it is a cross-sectional study, there are certain limitations, such as the absence of survival rates and treatment outcomes, which warrant caution when extending these findings to direct treatment-guiding recommendations. Prospective longitudinal studies are required to determine the use of LDH levels in decision-making strategies. However, in resource-limited settings where diagnostic modalities are unavailable, determining LDH concentrations may contribute to meaningful risk assessment along with established clinical guidelines.

ETHICS APPROVAL

This study was approved by the Institutional Review Board (IRB) of Jinnah Postgraduate Medical Center, Karachi (Reference No. F.2-81/2025-GENL/306/JPMC). All procedures performed in studies involving human participants were following the ethical standards of the institutional and/ or national research committee and the Helsinki Declaration.

CONSENT FOR PUBLICATION

Written informed consent was obtained from all participants before enrolment in the study.

AVAILABILITY OF DATA

The datasets used and analyzed during the current study are available from the corresponding author upon reasonable request.

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None.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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AUTHORS' CONTRIBUTION

PM: study conception and design, data collection, manuscript drafting. GH: study design, statistical analysis, critical review, and revision of manuscript. SB and MA: data collection and manuscript drafting. All authors read and approved the final manuscript.

GENERATIVE AI AND AI-ASSISTED TECHNOLOGIES IN THE WRITING PROCESS

AI-assisted language editing tools were used solely for grammar and language refinement. All scientific content, interpretation, and conclusions are the sole responsibility of the authors.

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