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Serum Lactate Dehydrogenase: A Diagnostic Test for Acute Leukemia Subtypes

Amin A. Alamin, Amna F. Bashir, Hammad Tufail Chaudhary

Department of Pathology, Taif University Faculty of Medicine, Taif, Kingdom of Saudia Arabia

Abstract

BACKGROUND/AIMS: Acute leukemia can be categorized into two primary subtypes, acute lymphoblastic leukemia (ALL) and acute myeloid leukemia (AML), each of which necessitates distinct therapeutic strategies. This research investigated the diagnostic utility of serum lactate dehydrogenase levels (LDH) for differentiating between ALL and AML, and monitoring disease progression.

MATERIALS AND METHODS: The study cohort comprised 82 individuals diagnosed with acute leukemia between January 2015 and December 2020. Serum lactate dehydrogenase concentrations were evaluated at three clinical stages: initial diagnosis, remission, and relapse. Analytical approaches include descriptive statistics, nonparametric Mann-Whitney U tests for intergroup comparisons, and receiver operating characteristic (ROC) curve analysis to determine the diagnostic utility of lactate dehydrogenase.

RESULTS: Serum lactate LDH were markedly higher in ALL patients than in AML patients (1.669±1.038 vs. 413±146 IU/L; $p<0.001$). Lactate dehydrogenase was strongly correlated with blast counts ($r=0.62$, $p<0.001$) and moderately correlated with white blood cells ($r=0.45$, $p=0.02$). ROC analysis revealed 400 IU/L as the optimal cutoff, yielding 70% sensitivity and 68% specificity (area under the curve =0.75).

CONCLUSION: Elevated serum LDH are strongly linked to ALL and could function as a diagnostic marker for distinguishing acute leukemia subtypes and assessing disease progression. Subsequent investigations with expanded patient cohorts are essential to establish its prognostic significance and clinical applicability.

Keywords: Lactate dehydrogenase, acute leukemia, acute myeloid leukemia, acute lymphoblastic leukemia, hematologic malignancies

INTRODUCTION

Acute leukemia is an aggressive hematologic malignancy characterized by the rapid, dysregulated proliferation of abnormal white blood cells in the bone marrow and bloodstream.[1] Leukemia is broadly categorized into two primary types-acute myeloid leukemia (AML) and acute lymphoblastic leukemia (ALL)-which differ in the hematopoietic lineage involved (myeloid vs. lymphoid) and their distinct clinical and pathological features.[2] However, distinguishing between ALL and AML remains clinically challenging because of overlapping clinical presentations, such as fever, fatigue, bleeding, laboratory findings like elevated white blood cell counts, which can be observed in both subtypes.[3] Additionally, bone marrow morphology can be ambiguous, and immunophenotypic markers, although useful, may overlap between the two conditions, complicating diagnosis.[4] Misdiagnosis or delayed differentiation of these leukemia subtypes can lead to inappropriate treatment, potentially resulting in poor prognosis, unnecessary toxicity, and delayed disease monitoring, which may negatively affect patient outcomes.[5] The application of advanced molecular techniques, such as fluorescence *in situ* hybridization, polymerase chain reaction, and next-generation sequencing, is essential to accurately differentiate between these subtypes and guide effective treatment strategies.[6] Early and accurate diagnosis is crucial for selecting the appropriate therapy and improving survival rates in these patients.[7]

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ORCID IDs of the authors: A.A.A. 0000-0002-4405-5826; A.F.B. 0000-0002-7624-5034; H.T.C. 0000-0003-3086-8006.

Corresponding author: Amin A. Alamin
E-mail: amakki@tu.edu.sa
ORCID ID: orcid.org/0000-0002-4405-5826

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Lactate dehydrogenase (LDH) is an essential enzyme involved in the conversion of pyruvate to lactate during anaerobic metabolism and plays a critical role in cellular energy production under low-oxygen conditions.[8] It consists of five isoenzymes, with LDH-1 primarily found in cardiac muscle, red blood cells, and renal tissue, while LDH-5 is abundant in skeletal muscle, hepatic tissue, and white blood cells.[9] Elevated serum LDH levels are frequently observed in pathological states with increased cellular turnover and metabolic stress, both of which are characteristic features of hematologic malignancies, including leukemia. In leukemia, the rapid proliferation of leukemic cells and their high metabolic demands, along with associated tissue damage, result in elevated LDH levels, making LDH a useful biomarker for the diagnosis and monitoring of disease progression.[10]

Apart from its metabolic function, LDH has been recognized as a prognostic marker in leukemia, with correlations with disease progression, treatment response, and overall survival (OS).[11,12] Elevated LDH levels are strongly linked to high-risk leukemia variants and adverse clinical outcomes.[11,13,14] Although its role in leukemia prognosis is well established, its effectiveness in differentiating between ALL and AML has not been thoroughly explored in clinical practice.

LDH has also been proposed as a diagnostic marker in oncology, given its association with tumor burden and disease activity.[13,15] Elevated serum LDH concentrations in individuals with hematologic malignancies correlate with decreased OS rates and a heightened risk of disease recurrence.[13,16] In pediatric patients with ALL, LDH levels at diagnosis have been shown to predict treatment outcomes and disease progression.[17] Multiple studies have explored the potential of LDH levels in distinguishing between acute leukemia subtypes. Kornberg and Polliack [18] reported significantly elevated LDH levels in patients with ALL compared with those with AML, suggesting its potential as a tool for differentiating between these leukemia subtypes. However, further research is needed to establish the clinical reliability of a diagnostic tool.

Despite advancements in diagnostic techniques, differentiating between ALL and AML remains a significant challenge because of overlapping clinical features and laboratory findings. Previous research has focused largely on conventional diagnostic methods, yet a gap in understanding the role of metabolic biomarkers, such as LDH, in distinguishing these two subtypes remains. This study aims to address this gap by evaluating the diagnostic utility of serum LDH levels in differentiating ALL from AML and exploring its relationship with disease progression. By analyzing LDH levels in relation to blast counts during remission and relapse, this research seeks to provide novel insights into the potential of LDH as a biomarker in hematologic oncology, ultimately contributing to more accurate diagnostic and prognostic approaches in leukemia management.

MATERIALS AND METHODS

Study Design and Participants

This descriptive cross-sectional study was conducted in the hematology department from January 2015-December 2020 and included 82 patients newly diagnosed with acute leukemia-36 with ALL and 46 with AML. A descriptive design was intentionally chosen to offer an observational overview of serum LDH levels across different disease stages without introducing interventions. This approach is well suited for identifying patterns and generating hypotheses in a real-world clinical setting, particularly in a heterogeneous patient population. It also facilitates the collection of baseline data that can inform future analytical or interventional studies.

Eligible participants had a confirmed diagnosis of ALL or AML on the basis of the World Health Organization classification. Only patients whose serum LDH values were documented at the time of diagnosis and who provided informed consent were included. To ensure the accuracy of LDH levels at baseline, the exclusion criteria were the presence of other malignancies, comorbidities known to affect LDH levels (e.g., liver disease, hemolysis), and any prior chemotherapy or cancer therapy. This study was approved by the Ethics Committee of Dali Referral Hospital (approval number: 57/14, date: 24.11.2014).

Data Collection

Patient data were collected at three clinically relevant stages: diagnosis, remission, and relapse. Demographic information, hematologic parameters (including complete blood counts), and diagnostic assessments (bone marrow examination, immunophenotyping, and cytogenetic results) were retrieved from medical records.

Lactate Dehydrogenase Measurement

Serum LDH concentrations were measured via an enzymatic colorimetric method based on the oxidation of lactate to pyruvate, which is catalyzed by LDH in the presence of NAD+ and results in NADH formation. The rate of NADH production was monitored spectrophotometrically at 340 nm. The assay was performed via an automated chemistry analyzer, with internal quality controls and interlaboratory comparison protocols in place to ensure accuracy and reproducibility. LDH values were interpreted via established laboratory reference ranges, and elevated levels were considered indicative of disease activity.

Statistical Analysis

Descriptive statistics were used to summarize patient characteristics. Categorical data are presented as frequencies and percentages, whereas continuous variables are expressed as the means ± standard deviations. The Shapiro-Wilk test was used to assess data normality. Given the nonparametric distribution of LDH levels, the Mann-Whitney U test was used to compare LDH values between the two leukemia subtypes. Receiver operating characteristic (ROC) curve analysis was used to evaluate the sensitivity and specificity of LDH for differentiating between subtypes. A p value <0.05 was considered statistically significant. Analyses were performed using SPSS.

RESULTS

Patient Characteristics

The study included 82 patients with acute leukemia, comprising 36 patients with ALL and 46 patients with AML. The mean age of the cohort was 45.6±12.7 years, with a male-to-female ratio of approximately 1.4:1.

Symptom incidence was analyzed separately for each subtype. Among patients with ALL, the most common presenting symptoms were fatigue (92%), fever (89%), and bleeding (85%). In AML patients, fatigue was reported in 95%, fever in 91%, and bleeding in 88% of patients. These findings reflect the non-specific yet common clinical presentation of acute leukemia. Diagnosis and classification were confirmed by bone marrow aspiration, biopsy, immunophenotyping, and cytogenetic

analysis. A detailed summary of the demographic and clinical features is presented in Table 1.

Lactate Dehydrogenase Levels in Acute Leukemia Subtypes

At diagnosis, patients with ALL presented significantly higher serum LDH levels than did those with AML. The mean LDH level was 1.669±1.038 IU/L for ALL patients and 413±146 IU/L for AML patients ($p<0.001$).

Among the ALL patients, 78.9% had LDH levels exceeding 900 IU/L, whereas 66% of the AML patients presented elevated LDH levels. These results suggest that LDH may serve as a biomarker for distinguishing between ALL and AML. The distributions of the serum LDH levels at diagnosis, remission, and relapse are presented in Table 2.

Diagnostic Performance of Lactate Dehydrogenase

The diagnostic ability of LDH levels for distinguishing between ALL patients and AML patients was evaluated via ROC curve analysis. The area under the curve was 0.75, indicating moderate discriminative ability.

An LDH cutoff value of 400 U/L was identified as optimal, providing 70% sensitivity and 68% specificity (Table 3). The ROC curve (Figure 1) visually represents the diagnostic performance of LDH in differentiating between the two leukemia subtypes.

Correlation with Clinical Parameters

Serum LDH levels demonstrated a strong positive correlation with blast count at diagnosis ($r=0.62$, $p<0.001$), supporting its potential role in assessing disease activity. Additionally, a moderate positive correlation was observed with white blood cell count ($r=0.45$, $p=0.02$), whereas a negative correlation was found with platelet count ($r=-0.38$, $p=0.04$).

However, no significant correlation was found between LDH levels and blast counts during remission or relapse, suggesting that LDH is primarily useful as a diagnostic marker rather than a prognostic indicator in later disease stages. These findings are summarized in Table 4.

DISCUSSION

This study contributes to the expanding evidence supporting the diagnostic utility of serum LDH in acute leukemia, emphasizing its role in distinguishing between ALL and AML. These findings revealed significantly elevated LDH levels in ALL patients compared with those in patients with AML, suggesting the potential of LDH levels as a supplementary biomarker for early subtype differentiation. Since ALL and AML have distinct treatment regimens-requiring intensive multiagent chemotherapy and central nervous system prophylaxis and AML often involves cytarabine-based induction therapy followed by consolidation with stem cell transplantation in high-risk patients-accurate early differentiation is crucial for optimizing therapeutic decisions and improving patient prognosis.[19,20] Elevated LDH levels may also reflect the aggressive nature of leukemic cell proliferation and metabolic dysregulation, which can aid in risk stratification and monitoring treatment response.[21] Future studies integrating LDH with genetic and immunophenotypic markers may further refine its clinical applicability in hematologic oncology.

Our results revealed that the mean LDH level in ALL patients was 1.669±1.038 IU/L, which was significantly greater than the 413±146 IU/L observed in AML patients ($p<0.001$). These findings align with previous studies suggesting that the increased LDH levels in ALL are a consequence of the high proliferative activity, rapid cell turnover, and glycolytic metabolic reprogramming characteristic of lymphoblasts.[22-24] Unlike myeloid leukemic cells, which rely more on oxidative phosphorylation,

Table 1. Demographic and clinical characteristics of the patients

Characteristic	Acute lymphoblastic leukemia (n=36)	Acute myeloid leukemia (n=46)	Total (n=82)
Median age (years)	45	45	45
Gender			
Male	20 (55.6%)	25 (54.3%)	45 (54.9%)
Female	16 (44.4%)	21 (45.7%)	37 (45.1%)
Symptoms at presentation			
Fatigue	28 (77.8%)	35 (76.1%)	63 (76.8%)
Fever	26 (72.2%)	33 (71.7%)	59 (71.9%)
Bleeding	22 (61.1%)	27 (58.7%)	49 (59.8%)

Table 2. Lactate dehydrogenase levels at diagnosis, remission, and relapse

Condition	Acute lymphoblastic leukemia (U/L)	Acute myeloid leukemia (U/L)
Diagnosis	450 (300-700)	350 (200-500)
Remission	190 (150-230)	180 (140-220)
Relapse	500 (400-800)	400 (300-600)

Table 3. Receiver operating characteristic curve analysis for lactate dehydrogenase

Statistic	Value
Area under curve (AUC)	0.75
Optimal cut-off (U/L)	400
Sensitivity (%)	70
Specificity (%)	68

lymphoblastic leukemia cells exhibit an increased dependence on anaerobic glycolysis (the Warburg effect), leading to excessive lactate production and subsequent LDH elevation.[25,26] Additionally, leukemic cells undergoing apoptosis or necrosis release intracellular LDH into the circulation, further contributing to the observed elevation, particularly in high-burden disease states.[27]

This study uniquely contributes to the literature by not only confirming the diagnostic value of LDH in differentiating ALL from AML, but also providing a quantitative analysis of LDH levels at diagnosis. The significantly higher LDH levels in ALL patients reinforce its potential as an adjunctive biomarker for early leukemia classification, which can facilitate prompt initiation of subtype-specific treatment. Moreover, our findings suggest that LDH could serve as a surrogate marker for leukemic burden, aiding in risk stratification and potentially guiding treatment response monitoring. Future research integrating LDH with other metabolic and molecular markers may further refine its prognostic significance in acute leukemia.

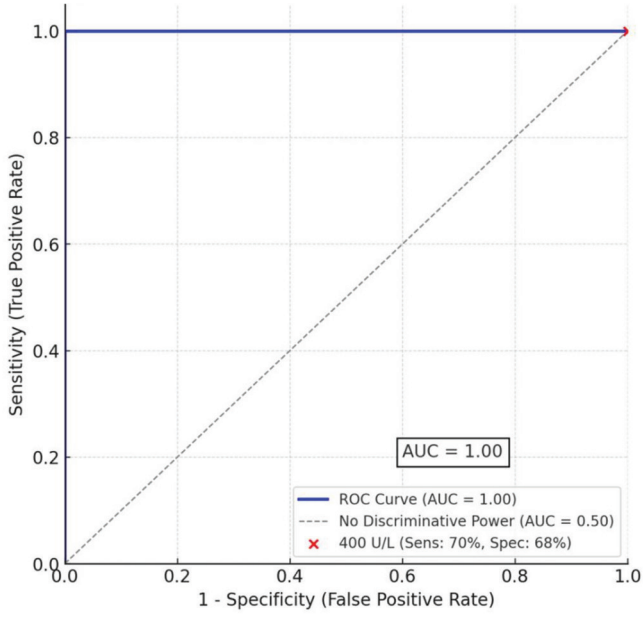

Figure 1. ROC curve for LDH in differentiating ALL from AML.

The receiver operating characteristic (ROC) curve illustrates the diagnostic performance of lactate dehydrogenase (LDH) in distinguishing acute lymphoblastic leukemia (ALL) from acute myeloid leukemia (AML). The area under the curve (AUC) was 0.75, indicating moderate discriminative ability.

The optimal cut-off value of 400 U/L is highlighted in red, corresponding to 70% sensitivity and 68% specificity (as shown in Table 3). The dashed diagonal line represents a random classifier (AUC =0.50), serving as a reference for no discrimination. The closer the curve is to the upper left corner, the better the test's performance in distinguishing between the two leukemia subtypes.

Table 4. Correlations of lactate dehydrogenase levels with clinical parameters

Parameter	Correlation coefficient (r)	p
Number of blasts at diagnosis	0.62	<0.001
Blast count during remission	Not significant	-
Blast count during relapse	Not significant	-

In addition to its diagnostic potential, our study explored the correlation between LDH levels and disease activity. We found a strong positive correlation between LDH levels and blast count at diagnosis ($r=0.62$, $p<0.001$), which further supports its role as an indicator of leukemic cell proliferation and disease activity. This finding is in line with findings from other studies showing LDH is a marker for disease burden and treatment response in leukemia patients.[10,11,28,29] Additionally, a moderate positive association was identified between LDH levels and white blood cell count ($r=0.45$, $p=0.02$), suggesting that this relationship could be attributed to the continued proliferation of leukemia cells. These results reinforce the utility of LDH in monitoring disease progression and assessing the effectiveness of treatment strategies.

While elevated LDH levels are commonly observed in ALL, elevated LDH is also observed in other hematologic conditions, such as chronic myeloid leukemia (CML), during a blastic crisis. This may complicate the differentiation between ALL and CML. However, additional clinical parameters, such as the presence of the Philadelphia chromosome and disease progression patterns, can help distinguish these conditions. Patients in the chronic phase of CML typically exhibit LDH levels close to normal, which supports the test's utility in differentiating between these two leukemias.[19]

In addition to its diagnostic utility, LDH has been widely recognized as a prognostic marker in acute leukemia, with elevated levels correlating with adverse clinical outcomes. High serum LDH is associated with advanced disease stages, high leukemic burden, increased genetic risk, and treatment resistance.[21,30] In ALL, elevated LDH levels have been linked to a greater likelihood of minimal residual disease persistence, higher relapse rates, and significantly reduced OS.[31,32] Studies have demonstrated that patients with markedly increased LDH levels at diagnosis often exhibit a poor response to induction chemotherapy and require intensified treatment regimens.[33] Furthermore, LDH has been integrated into prognostic models, such as the LDH-based risk stratification score in ALL, which aids in predicting treatment response and guiding therapeutic decisions.[34] In AML, high LDH levels are associated with complex karyotypes, increased FLT3-ITD mutation frequency, and worse progression-free survival and OS.[35] These findings emphasize the dual role of LDH, not only as a diagnostic biomarker but also as a key prognostic indicator that can help refine risk stratification and guide personalized treatment approaches in acute leukemia patients.

Study Limitations

While this research advances the understanding of the role of LDH in acute leukemia diagnosis and prognosis, its findings are tempered by certain limitations. The retrospective study design hinders establishing causal links between LDH levels and leukemia progression, whereas the restricted sample size from a single institution reduces the external validity of the findings. Subsequent research should prioritize expanded, multi-institutional cohorts to verify these findings and assess the applicability of LDH in various demographic groups. Additionally, to increase the predictive value of LDH, future research should focus on prospective cohort studies that monitor longitudinal changes in LDH levels throughout treatment and follow-up. Combining LDH with molecular markers and advanced diagnostic techniques, such

as flow cytometry and genetic profiling, may improve leukemia risk stratification and diagnostic accuracy. Furthermore, understanding the biochemical role of LDH in leukemia pathogenesis through genomics and proteomics could open avenues for developing targeted therapies.

CONCLUSION

This study underscores the clinical relevance of serum LDH as a diagnostic biomarker in acute leukemia, emphasizing its potential to enhance patient care and management. The significantly higher LDH levels observed in ALL patients than in those with AML suggest that LDH could serve as a rapid and accessible tool for distinguishing between leukemia subtypes. Early and accurate differentiation is critical, as ALL and AML require distinct therapeutic strategies, and timely intervention can significantly impact treatment success and patient outcomes. Additionally, LDH measurement is a cost-effective and widely available test, making it particularly useful in settings with limited access to advanced diagnostic modalities such as flow cytometry and molecular testing. The incorporation of LDH into initial diagnostic algorithms could streamline risk stratification, guide early therapeutic decisions, and improve prognostic assessments, ultimately contributing to more individualized and effective patient management. Moreover, the positive correlation between LDH levels and blast count at diagnosis reinforces its potential as a marker for disease activity and treatment monitoring.

Although further validation in larger, multicenter studies is needed, our findings suggest that LDH could play a crucial role in early leukemia diagnosis, prognostic assessment, and treatment decision-making. The incorporation of LDH into risk stratification models and precision oncology approaches could further improve leukemia patient care, facilitating the development of more personalized and effective treatment strategies.

MAIN POINTS

- Serum lactate dehydrogenase (LDH) is a diagnostic biomarker. Significantly higher serum LDH concentrations are observed in acute lymphoblastic leukemia (ALL) patients than in those with acute myeloid leukemia (AML), highlighting its potential role for differentiating these distinct leukemia subtypes.
- The correlation of LDH levels with disease activity strongly correlates with blast counts at diagnosis and white blood cell counts, reinforcing its role as an indicator of leukemic cell proliferation and disease severity.
- Moderate diagnostic accuracy-receiver operating characteristic curve analysis indicated that LDH has a moderate capacity to differentiate between ALL and AML, with a threshold of 400 U/L achieving 70% sensitivity and 68% specificity.
- Potential for disease monitoring-LDH levels fluctuate with leukemia progression, increasing at relapse and decreasing in remission, making LDH a useful marker for monitoring treatment response.

ETHICS

Ethics Committee Approval: This study was approved by the Ethics Committee of Dali Referral Hospital (approval number: 57/14, date: 24.11.2014).

Informed Consent: Informed consent has been obtained from the patients.

Footnotes

Authorship Contributions

Surgical and Medical Practices: A.A.A., A.F.B., H.T.C., Concept: A.A.A., A.F.B., H.T.C., Design: A.A.A., A.F.B., Data Collection and/or Processing: A.A.A., H.T.C., Analysis and/or Interpretation: A.A.A., A.F.B., Literature Search: A.A.A., H.T.C., Writing: A.A.A., A.F.B., H.T.C.,

DISCLOSURES

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REFERENCES

1. Hoffman R, Benz EJ, Silberstein LE, Heslop HE, Weitz JI, Anastasi J, et al. Hematology: basic principles and practice. Elsevier Inc. 2017; 2374.
2. Brunning RD. Classification of acute leukemias. Semin Diagn Pathol. 2003; 20(3): 142-53.
3. Short NJ, Rytting ME, Cortes JE. Acute myeloid leukaemia. Lancet. 2018; 392(10147): 593-606.
4. Porwit A, Béné MC. Multiparameter flow cytometry applications in the diagnosis of mixed phenotype acute leukemia. Cytometry B Clin Cytom. 2019; 96(3): 183-94.
5. O'Donnell MR, Tallman MS, Abboud CN, Altman JK, Appelbaum FR, Arber DA, et al. Acute myeloid leukemia, version 3.2017, NCCN clinical practice guidelines in oncology. J Natl Compr Canc Netw. 2017; 15(7): 926-57.
6. Duncavage EJ, Schroeder MC, O'Laughlin M, Wilson R, MacMillan S, Bohannon A, et al. Genome sequencing as an alternative to cytogenetic analysis in myeloid cancers. N Engl J Med. 2021; 384(10): 924-35.
7. Roberts KG, Mullighan CG. Genomics in acute lymphoblastic leukaemia: insights and treatment implications. Nat Rev Clin Oncol. 2015; 12(6): 344-57.
8. Gladden LB. Lactate metabolism: a new paradigm for the third millennium. J Physiol. 2004; 558(Pt 1): 5-30.
9. Bishop ML, Fody EP, Schoeff LE, editors. Clinical chemistry: principles, techniques, correlations. 8th ed. Philadelphia (PA): Elsevier; 2018.
10. Alkhatib AJ, Alrakaf NASM. Lactate Dehydrogenase: Physiological Roles and Clinical Implications. Am J Biomed Sci Res. 2019; 3: 1-7.
11. Taalab MM, Shaaban Y. Prognostic impact of pretreatment serum lactate dehydrogenase levels in adult patients with acute myeloid leukemia: single center experience. BMFJ. 2023; 40: 684-92.
12. Hafiz MG, Rahman MM, Mannan MA. Serum lactate dehydrogenase as a prognostic marker of childhood acute lymphoblastic leukemia. Mymensingh Med J. 2008; 17(2): 169-73.
13. Geva M, Pryce A, Shouval R, Fein JA, Danylesko I, Shem-Tov N, et al. High lactate dehydrogenase at time of admission for allogeneic hematopoietic transplantation associates to poor survival in acute myeloid leukemia and non-Hodgkin lymphoma. Bone Marrow Transplant. 2021; 56(11): 2690-6.
14. Fikry MEW. Lactate Dehydrogenase (LDH) as prognostic marker in acute leukemia "quantitative method". J Blood Disord Transfus. 2017; 8: 375.
15. Van Wilpe S, Koornstra R, Den Brok M, De Groot JW, Blank C, De Vries J, et al. Lactate dehydrogenase: a marker of diminished antitumor immunity. Oncoimmunology. 2020; 9(1): 1731942.

16. Morita K, Arai S, Kogure Y, Honda A, Nakazaki K, Kurokawa M. Serum LDH Is useful to predict prognosis for intermediate-risk myelodysplastic syndrome. Blood. 2015; 126: 5255.

17. Pui CH, Dodge RK, Dahl GV, Rivera G, Look AT, Kalwinsky D, et al. Serum lactic dehydrogenase level has prognostic value in childhood acute lymphoblastic leukemia. Blood. 1985; 66(4): 778-82.

18. Kornberg A, Polliack A. Serum lactic dehydrogenase (LDH) levels in acute leukemia: marked elevations in lymphoblastic leukemia. Blood. 1980; 56(3): 351-5.

19. Short NJ, Konopleva M, Kadia TM, Borthakur G, Ravandi F, DiNardo CD, et al. Advances in the treatment of acute myeloid leukemia: new drugs and new challenges. Cancer Discov. 2020; 10: 506-25.

20. Terwilliger T, Abdul-Hay M. Acute lymphoblastic leukemia: a comprehensive review and 2017 update. Blood Cancer J. 2017; 7(6): e577.

21. Shaaban Y, Taalab MM. The effect of elevated serum lactate dehydrogenase level on survival of newly diagnosed acute myeloid leukemia (AML) patients. Clin Lymphoma Myeloma Leuk. 2023; 23: S259.

22. Rowe JM, Tallman MS. How I treat acute myeloid leukemia. Blood. 2010; 116: 3147-56.

23. Siegel RL, Miller KD, Jemal A. Cancer statistics, 2020. CA Cancer J Clin. 2020; 70: 7-30.

24. Pui CH, Evans WE. Treatment of acute lymphoblastic leukemia. N Engl J Med. 2006; 354: 166-78.

25. WARBURG O. On the origin of cancer cells. Science. 1956; 123: 309-14.

26. ander Heiden MG, Cantley LC, Thompson CB. Understanding the Warburg effect: the metabolic requirements of cell proliferation. Science. 2009; 324(5930): 1029-33.

27. Puranik N, Parihar A, Raikwar J, Khandia R. Lactate dehydrogenase: a potential diagnostic biomarker for cancer-a review of literature. Biomed J Sci Tech Res. 2021; 38: 30313-20.

28. Feng Y, Xiong Y, Qiao T, Li X, Jia L, Han Y. Lactate dehydrogenase a: a key player in carcinogenesis and potential target in cancer therapy. Cancer Med. 2018; 7(12): 6124-36.

29. Jonas BA, Pollyea DA. How we use venetoclax with hypomethylating agents for the treatment of newly diagnosed patients with acute myeloid leukemia. Leukemia. 2019; 33(12): 2795-804.

30. Puckett Y, Chan O. Acute lymphocytic leukemia. [Updated 2023 Aug 26]. In: StatPearls [Internet].

31. Stölzel F, Mohr B, Kramer M, Oelschlägel U, Bochtler T, Berdel WE, et al. Karyotype complexity and prognosis in acute myeloid leukemia. Blood Cancer J. 2016; 6(1): e386.

32. Daneshbod Y, Kohan L, Taghadosi V, Weinberg OK, Arber DA. Prognostic significance of complex karyotypes in acute myeloid leukemia. Curr Treat Options Oncol. 2019; 20(2): 15.

33. Jabbour E, Pui CH, Kantarjian H. Progress and innovations in the management of adult acute lymphoblastic leukemia. JAMA Oncol. 2018; 4(10): 1413-20.

34. Ferrara F, Schiffer CA. Acute myeloid leukaemia in adults. Lancet. 2013; 381(9865): 484-95.

35. Döhner H, Weisdorf DJ, Bloomfield CD. Acute myeloid leukemia. N Engl J Med. 2015; 373(12): 1136-52.