EURASIAN JOURNAL OF EMERGENCY MEDICINE
Original Article
DOI: 10.4274/eajem.galenos.2025.80008
Eurasian J Emerg Med.

Does Lactate Dehydrogenase Act as an Early Warning System Predicting Mortality in Trauma Patients?

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Abstract

Aim: Trauma is one of the most prevalent causes of death and disability in middle age, and early diagnosis and treatment are critical in minimizing mortality and morbidity. Lactate dehydrogenase (LDH) is an indicator of inflammation in many diseases. We aim to present this study, in which we measure the power of LDH to show us mortality in the blood sample taken at the first examination in trauma patients, in the light of the literature.

Materials and Methods: Trauma patients of emergency medicine department between November 2020 and November 2021, were analyzed. Trauma mechanism, consultations, mortality-morbidity, and scoring were evaluated retrospectively.

Results: The glasgow coma scale (GCS), injury severity score (ISS), and new injury severity score (NISS) values of the patients were 14.58±1.76 (95% confidence interval: 14.43-14.73), 14 (0-76), and 18 (0-101) respectively. The data for leukocytes, hemoglobin, platelets, glucose, aspartate aminotransferase (AST), and LDH were significantly different between the control and patient groups. The comparison of leukocyte, hemoglobin, glucose, creatinine, AST, and LDH data between the survivors and the deceased in the patient group revealed a statistically significant difference. ROC analysis was then applied to evaluate these markers in the non-surviving patients. ISS, NISS, GCS, alanine aminotransferase, AST and LDH were found to be significant.

Conclusion: LDH elevation, which is studied between routine procedures, may be beneficial for physicians working in the periphery both in patient referral and in making an early operation decision. Thus, we believe that mortality and morbidity will decrease with the use of LDH, a cost-effective marker.

Keywords: Trauma, LDH, radiology, mortality, emergency service

Introduction

In addition to being the leading cause of death in patients under 45 years of age, trauma is associated with complications and late death (1). Among these complications, multiple organ dysfunction syndrome (MODS) is the most common and causes a significant increase in mortality (2,3). Therefore, initiating effective treatment early in trauma patients requires identifying patients at high risk of developing MODS. This approach is at the forefront of reducing victimization resulting from trauma.

In recent years, several studies have investigated trauma-related mortality and its predictors in emergency settings in Türkiye. A study comparing falls from height and traffic accidents, reported that trauma mechanism significantly influences mortality rates, underlining the need for mechanism-based prognostic tools (4).

Another study focusing on pediatric trauma emphasized the importance of early physiological markers in identifying high-risk patients in the emergency department (5). Additionally, mortality predictors in traffic accidents were explored in a study

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Cite this article as: Çanakçı SE, Tunalı Türkdoğan F, Türkmen F, Türkdoğan KA, Kapçı M, Küçükgül F. Does lactate dehydrogenase act as an early warning system predicting mortality in trauma patients? Eurasian J Emerg Med. [Epub Ahead of Print].

Received: 04.06.2025
Accepted: 26.08.2025
Epub: 13.11.2025

that highlighted the potential role of simple parameters for early prognostication (6). In light of these national findings, our study aims to evaluate whether lactate dehydrogenase (LDH)—a low-cost and routinely available biomarker—could serve as an early predictor of trauma severity and mortality in adult patients.

Serum LDH is a low-cost test that is included in the regular procedures requested during a hospital admission evaluation. As a result, it could be an appropriate marker for physicians to use in determining the prognosis of trauma patients without incurring additional costs or requiring additional time or blood samples. LDH is a cytoplasmic enzyme that is found in every major organ, including the brain, lung, liver, and heart (7). Serum LDH levels are thought to be an indicator of inflammation and the extent of tissue damage. (8-13). The object of this retrospective study was to examine the association between mortality in trauma patients and serum LDH concentration.

Materials and Methods

Patients and Methods

Five hundred fourteen cases of trauma patients, who were admitted to the emergency medicine clinic between November 2020 and November 2021, were analyzed. Trauma mechanism, consultations, mortality-morbidity and scoring were evaluated retrospectively. As the control group, 130 people who underwent non-traumatic routine control were included in the study. Approval was obtained from the University of Health Sciences Türkiye, Bakırköy Dr. Sadi Konuk Training and Research Hospital Clinical Research Ethics Committee (decision number: 2022-16-01; date: 15.08.2022).

Inclusion and Exclusion Criteria

In this retrospective study, patients whose laboratory values were assessed later and whose glasgow coma scale (GCS), injury severity score (ISS), and new injury severity score (NISS) values were recorded at admission to the emergency department were included.

Patients with trauma or malignancy (liver, lung, and hematological malignancy) who were using anticoagulant drugs were removed from the study.

Patients who came for non-traumatic routine control were included as the control group, while patients with malignancies (liver, lung, and hematological malignancies) and those using anticoagulant drugs were left out of the study.

Statistical Analysis

The normal distribution analysis was performed by considering the data in the study: Five parameters [Skewness-Kurtosis, standard deviation (Std)/mean, Q-Q plots, histogram, and Shapiro-Wilk test]. Normally distributed parameters with sufficient data were shown as mean ± Std, and the independent samples t-test was applied for pairwise comparisons. The median (minimum-maximum) Mann-Whitney U test was used for groups that did not have enough data or did not have a normal distribution. The chi-square test was applied to analyze the frequency of categorical data. The ROC curve was developed by examining the sensitivity and specificity of diagnostic tests. A multiple logistic regression analysis was performed with risk factors for estimating mortality. In the study, a significance level of $\alpha=0.05$ was used, and a p-value less than α was considered significant.

Results

In this study, there were 514 individuals in the patient group, and the mean age was 39.88±13.45 [95% confidence interval (CI): 37.85-41.91]. Of the patients, 161 were female, and 353 were male. The GCS, ISS, and NISS values of the patients were 14.58±1.76 (95% CI: 14.43-14.73), 14 (0-76), and 18 (0-101), respectively.

Leukocyte, hemoglobin, platelet, glucose, aspartate aminotransferase (AST), and LDH data revealed statistically significant differences between the control and patient groups. Creatinine and alanine aminotransferase (ALT), data did not reveal any statistically significant distinctions between the control and patient groups ($p>0.05$). While patient group leukocyte data increased by 20% relative to control group data, patient group hemoglobin data decreased. The patient group had greater levels of glucose, blood urea nitrogen, AST, and LDH than the control group (Table 1).

The mortality frequency observed in those who underwent surgery (2), was statistically significantly different from the expected mortality frequency (8.5) ($p<0.001$). There is no substantial difference between expected and observed mortality rates for other causes of trauma (Table 2).

The observed frequency of death (2) of patients who received neurosurgery consultation was statistically different from the expected frequency of death (6) ($p=0.007$). The observed death frequency of patients who received pediatric surgical consultation (3) was different from the observed death frequency in another group, and this difference was statistically significant ($p=0.022$). No statistically significant differences were found between the expected and observed death frequencies of patients who received other consultations ($p>0.05$).

There was a statistically significant difference in the comparison of leukocyte, hemoglobin, glucose, creatinine, AST, and LDH data between the survivors and the deceased in the patient group. The p-values were 0.006, 0.027, 0.015, 0.031, $p<0.001$, and $p<0.001$, respectively (Table 3).

Table 1. Comparison of control and patient group data.			
	Control (n=130)	Patient (n=514)	p value
Leukocyte	10190 (4650-30950)	12030 (1175-53580)	<0.001
Hemoglobin	13.25±1.89	12.68±2.21	0.007
Platelet	253.50 (66-491)	245 (16-862)	0.034
Glucose	103 (63-436)	117 (33-678)	<0.001
Blood urea nitrogen	27 (6-148)	31 (3-470)	<0.001
Creatinin	0.73 (0.41-3.47)	0.76 (0.33-3.89)	0.127
Alanine aminotransferase	18 (6-266)	20 (6.00-1396)	0.254
Aspartate aminotransferase	24 (13-371)	29 (8-1058)	0.001
Lactate dehydrogenase	252 (43-870)	270 (105-3166)	0.016
Data are presented as mean ± SD or median (minimum-maximum)			

Table 2. Comparison of trauma mechanisms patients who survived and those who died			
	Survival n=503	Died n=11	p value
Surgery			
No	108 (21.5)	9 (81.8)	**<0.001**
Yes	395 (21.5)	2 (18.2)	
Gunshot wound			
No	488 (97)	11 (100)	1.000
Yes	15 (3)	0 (0)	
Stab wound			
No	448 (89.1)	10 (90.9)	1.000
Yes	55 (10.9)	1 (9.1)	
Falls			
No	342 (68)	10 (90.9)	0.186
Yes	161 (32)	1 (9.1)	
Traffic Accident			
No	343 (68.2)	5 (45.5)	0.188
Yes	160 (31.8)	6 (54.5)	
Falling from Height			
No	436 (86.7)	9 (81.8)	0.649
Yes	67 (13.3)	2 (18.2)	
Judicial Case			
No	275 (54.7)	9 (81.8)	0.122
Yes	228 (45.3)	2 (18.2)	
Data are presented as n (%).			

The leukocyte, glucose, AST, ALT and LDH frequencies, categorized as normal and abnormal according to clinical cut-off values, were statistically significant. The p-value was respectively: p=0.011, p=0.021, p=0.002, p<0.001, and p<0.001.

Table 3. Comparison of blood values between patients who survived and those who died			
	Survival	Died	p value
Leukocyte	11870 (1175-40650)	17280 (7930-53580)	0.006
Hemoglobin	13 (3-18)	9.20 (6.30-14.90)	0.027
Platelet	245 (16-862)	222 (105-437)	0.312
Glucose	117 (33-421)	158 (73-678)	0.015
BUN	31 (3-470)	24 (14-73)	0.134
Creatinin	0.75 (0.33-3.20)	0.85 (0.63-3.89)	0.031
Alanine aminotransferase	20 (6-1143)	169 (11-1396)	0.002
Aspartate aminotransferase	29 (8-675)	241 (16-1058)	<0.001
Lactate dehydrogenase	267 (105-1900)	1036 (232-3166)	<0.001

The sensitivity and specificity values of risk factors are shown in Table 4. The ROC curve of ALT, AST and LDH is shown in Figure 1, and ISS, NISS and GCS are shown in Figure 2.

ROC analysis was then applied to evaluate these markers in the non-surviving patients. ISS [area under the curve (AUC): 0.843, 95% CI: 0.709-0.977, p<0.001), NISS (AUC: 0.874, 95% CI: 0.750-0.998, p<0.001) GCS (AUC: 0.937, 95% CI: 0.834-1.000, p<0.001), ALT (AUC: 0.773, 95% CI: 0.587-0.960), AST (AUC: 0.808, 95% CI: 0.636-0.980, p<0.001) and LDH (AUC: 0.896, 95% CI: 0.782-1.000) were found to be significant (Table 4).

To this end, we analyzed LDH and GCS characteristics in binary logistic regression models. LDH was revealed to be the most important parameter in defining the mortality rate of patients in a multiple binary logistic regression analysis. (Table 5). The predictive power of the model was measured using Nagelkerke's R^2 coefficient. In terms of longevity, this model is 99.49% accurate and 45.46% accurate in predicting death (Table 3). There is a 59.1% variance explained in the probability of death according to the Nagelkerke's R^2 value (Table 5).

Discussion

In this study, we found that leukocytes, hemoglobin, platelets, glucose, AST, and LDH serum values increased with trauma and were closely related to survival among patients. In the ROC analysis performed between these blood values, GCS and LDH reached the best sensitivity and specificity. This analysis also included trauma scoring systems such as ISS, NISS, and GCS. LDH was found to be more effective in determining mortality in the binary regression analysis performed among these groups.

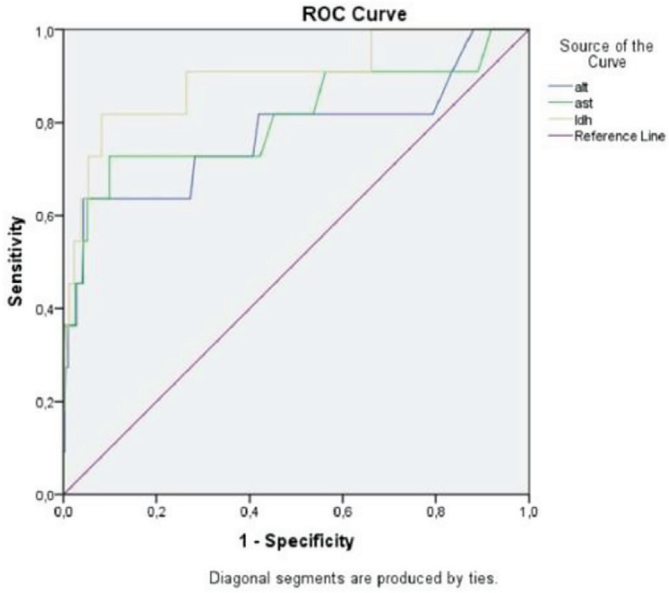

Figure 1. Chart showing ROC analysis of ALT, AST, and LDH

ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, LDH: Lactate dehydrogenase

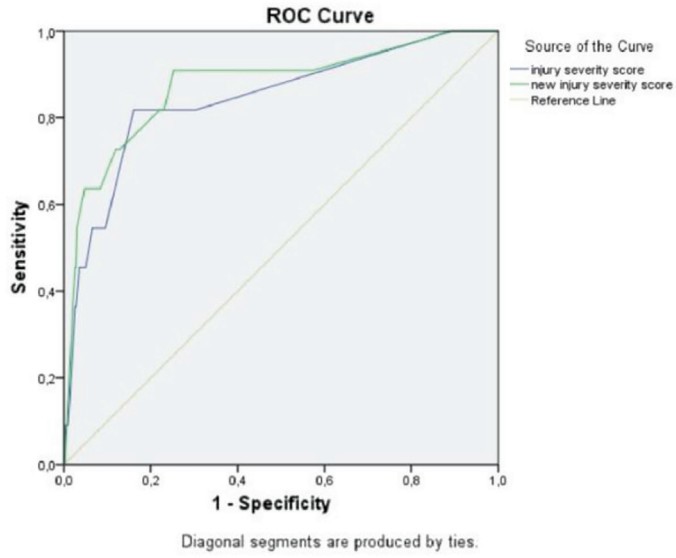

Figure 2. Chart showing ROC analysis of ISS and NISS

ISS: Injury severity score, NISS: New injury severity score

Table 4. ROC curves of risk factors

Risk factor	AUC (95% CI)	Cut-off value	p value	Sensitivity	Specificity
Injury severity score	0.843 (0.709-0.977)	23.5	<0.001	0.818	0.839
New injury severity score	0.874(0.750-0.998)	24.5	<0.001	0.909	0.748
Glasgow coma scale	0.937 (0.834-1.000)	14.5	<0.001	0.909	0.911
Alanine aminotransferase	0.773 (0.587-0.960)	144.5	0.002	0.636	0.957
Aspartate aminotransferase	0.808 (0.636-0.980)	109.5	<0.001	0.727	0.901
Lactate dehydrogenase	0.896 (0.782-1.000)	621	<0.001	0.818	0.919

AUC: Area under the curve, CI: Confidence interval, ROC: Receiver operating characteristic

Table 5. Multiple logistic regression analysis of risk factors on mortality

	B	SE	Wald	DF	Sig.	Exp (B)	95% CI for exp (B)	
							Lower	Upper
LDH	0.003	0.001	12.04	1	0.001	1.003	1.001	1.005
GCS	-0.436	0.098	19.734	1	0	0.647	0.534	0.784
Constant	-0.36	1.213	0.001	1	0.976	0.965		
			Model summary					
Step	-2 Log likehood	Cox and Snell R-square	Nagelkerke's square	Step				
1	44.395	0.131	0.591	1				
	Observed		Predicted					
			Mortality					
	Mortality		No	Yes				
		No	99.49%	0.51%				
		Yes	54.54%	45.46%				

LDH: Lactate dehydrogenase, GCS: Glasgow coma scale, SE: Standard error, B: Regression coefficient, DF: Degrees of freedom, CI: Confidence interval

LDH accelerates the process 14 times, by catalyzing the coordinated interconversion of pyruvate to lactate and nicotinamide adenine dinucleotide (NADH) to NAD+ (14,15).

As a result, the increasing lactate level will be closely correlated with the rise in LDH levels. The transfer of a hydride ion from NADH to pyruvate, at carbon C2 of pyruvate, furthers the chemical process. The initial stage in the molecular process is the binding of NADH to enzymes. This binding involves a large number of residues in the active site. When NADH binds, it helps lactate bind by interacting with the LDH residues. LDH-NAD+ lactate and LDH-NADH-pyruvate are two tertiary complexes that are created when a hydride moves quickly in both directions simultaneously (16). In our study, we think elevated LDH is the underlying cause of higher LDH levels and mortality in patients compared to the control group.

LDH is considered a marker of tissue damage following trauma. In cases of acute trauma, LDH levels generally increase within the first 48 hours, and this elevation can be used as an important parameter in evaluating patient prognosis. Specifically, the increase in LDH levels within 48 hours after admission to the intensive care unit demonstrates high sensitivity and specificity in predicting mortality (17). In chronic trauma processes, however, LDH levels require longer-term monitoring. For example, in patients who have suffered an acute ischemic stroke, elevated LDH levels at hospital admission serve as an independent indicator of prognosis and long-term mortality risk (18). These findings highlight the potential of LDH as a prognostic biomarker in both acute and chronic trauma processes.

In our study, we aimed to investigate the potential role of serum LDH levels measured at the initial admission of trauma patients in assessing the risk of mortality and morbidity. In this context, we believe that the use of LDH as a prognostic marker in both acute and chronic trauma processes may contribute to clinical practice.

The relationship between trauma, inflammation, and tissue injury constitutes a cornerstone of clinical practice. LDH has emerged as a significant biomarker in the evaluation of these processes. LDH is an enzyme present in various tissues and organs, released into the bloodstream as a result of cellular damage. Therefore, its serum levels are considered an indicator of inflammation and tissue injury (19). The association between LDH and inflammation has been highlighted in several studies. Particularly in severe inflammatory conditions such as sepsis, elevated serum LDH levels can be used to determine patient prognosis. For instance, one study reported that high LDH levels in septic patients were associated with poor prognosis (20). Among the systemic effects of trauma are not only inflammation and tissue damage but also organ dysfunction. LDH may aid in the evaluation of these systemic effects. For example, one study found that elevated LDH levels were associated with organ dysfunction (21). In conclusion, LDH may be used as an important biomarker in the assessment of inflammation, tissue damage, and the systemic effects of trauma. Its role in these processes can contribute to clinical practice and assist in determining patient prognosis.

Both the activity of LDH and the concentration of global actin in the blood were observed to rise after trauma, as was previously reported by Hazeldine et al. (14) In cases with a fatal outcome, we discovered that LDH was considerably greater than that in the control group. LDH activity in serum samples was also compared between patients with isolated traumatic brain injury (TBI) and healthy controls, and it was observed that patients with TBI had greater LDH levels.

Since the serum concentration of LDH isoenzymes reflects tissue-specific pathological conditions, the quantification of LDH is of clinical interest (22). Their studies found that it was high in pericardial and peritoneal fluids (23), metastatic melanoma and fast-growing cancers (24,25), and this was associated with apparent inflammation. Hazeldine et al. (14) determined in their study that trauma may be the initiation of inflammation in the body. Therefore, we attribute the cause of the high LDH in the trauma patients in our study, especially in the deceased patients, to the inflammation that developed as a result of the trauma.

Study Limitations

This study has several limitations. Its retrospective and single-center design limits the generalizability of the findings. The relatively small sample size may have reduced statistical power. LDH was measured only at admission, without evaluating dynamic changes over time. This single time-point measurement restricts the ability to capture temporal fluctuations that could provide a more accurate prognostic assessment, as serial measurements are known to yield more valid and reliable results when evaluating prognostic biomarkers. Additionally, comparisons with other established biomarkers (e.g., C-reaktif protein, lactate, procalcitonin) were not performed. The heterogeneity in trauma types and exclusion of critically ill patients referred to tertiary centers may have influenced mortality outcomes. Moreover, detailed data regarding the type and extent of injury (e.g., crushing, penetrating, or burn trauma) were not consistently available due to the retrospective nature of the study, limiting our ability to assess their specific impact on LDH levels. Therefore, future prospective, multicenter studies with serial LDH measurements and comprehensive trauma classification are warranted to better elucidate LDH's prognostic value in trauma patients.

Conclusion

In our study, which determined that surgical intervention reduces mortality in appropriate patients, we found that LDH elevation—measured upon initial admission—was associated with trauma severity and mortality. Given its routine availability and low-cost, LDH may serve as an accessible early indicator in trauma triage, especially in peripheral or resource-limited settings. However, considering the limitations of single-time-point measurement, we propose that this study should be regarded as a preliminary investigation suggesting a potential link between LDH and trauma outcomes. Future prospective studies incorporating serial LDH measurements are necessary to determine whether LDH can reliably predict mortality and morbidity in trauma patients.

Ethics

Ethics Committee Approval: Approval was obtained from the University of Health Sciences Türkiye, Bakırköy Dr. Sadi Konuk and Research Hospital Clinical Research Ethics Committee (decision number: 2022-16-01; date: 15.08.2022).

Informed Consent: This retrospective study.

Footnotes

Author Contributions

Surgical and Medical Practices: F.T., M.K., Concept: S.E.Ç., F.T.T., M.K., Design: S.E.Ç., K.A.T., Data Collection or Processing: F.T.T., F.T., Analysis or Interpretation: S.E.Ç., K.A.T., M.K., Literature Search: S.E.Ç., F.T.T., Writing: S.E.Ç., K.A.T., M.K., F.K.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

References

1. Trunkey DD. Trauma. Accidental and intentional injuries account for more years of life lost in the U.S. than cancer and heart disease. Among the prescribed remedies are improved preventive efforts, speedier surgery and further research. Sci Am. 1983:249:28-35.
2. Reed D, Kemmerly SA. Infection control and prevention: a review of hospital-acquired infections and the economic implications. Ochsner J. 2009;9:27-31.
3. Jin J, Qian H, Wu G, Bao N, Song Y. Neutrophil-derived long noncoding RNA IL-7R predicts development of multiple organ dysfunction syndrome in patients with trauma. Eur J Trauma Emerg Surg. 2022;48:1545-53.
4. Gür A, Çelik BK, Çakmak F. Evaluation of mortality in height-experiencing falls in patients with height and traffic accidents. Eurasian J Emerg Med. 2024;23:196-202.
5. Yiğit E, Turtay MG, Çolak C. Examination of pediatric trauma patients admitted to the emergency department. Eurasian J Emerg Med. 2024;23:40-8.
6. Tortum F, Bayramoğlu A. Can we predict mortality in traffic accidents in emergency department? Eurasian J Crit Care. 2019;1:1-6.
7. Podlasek SJ, McPherson RA, Threatte GA. Characterization of apparent lactate dehydrogenase isoenzyme 6: a lactateindependent dehydrogenase. Clin Chem. 1984;30:266-70.
8. Turner CL, Budohoski K, Smith C, Hutchinson PJ, Kirkpatrick PJ, Murray GD. Elevated baseline C-reactive protein as a predictor of outcome after aneurysmal subarachnoid hemorrhage: data from the simvastatin in aneurysmal subarachnoid hemorrhage (STASH) trial. Neurosurgery. 2015;77:786-92.
9. Srinivasan A, Aggarwal A, Gaudihalli S, Mohanty M, Dhandapani M, Singh H, et al. Impact of early leukocytosis and elevated high-sensitivity C-reactive protein on delayed cerebral ischemia and neurologic outcome after subarachnoid hemorrhage. World Neurosurg. 2016;90:91-5.
10. Maiuri F, Gallicchio B, Donati P, Carandente M. The blood leukocyte count and its prognostic significance in subarachnoid hemorrhage. J Neurosurg Sci. 1987;31:45-8.
11. Weir B, Disney L, Grace M, Roberts P. Daily trends in white blood cell count and temperature after subarachnoid hemorrhage from aneurysm. Neurosurgery. 1989;25:161-5.
12. Zhang Y, Li L, Jia L, Li T, Di Y, Wang P, et al. Neutrophil counts as promising marker for predicting in-hospital mortality in aneurysmal subarachnoid hemorrhage. Stroke. 2021;52:3266-75.
13. Rao CJ, Shukla PK, Mohanty S, Reddy YJ. Predictive value of serum lactate dehydrogenase in head injury. J Neurol Neurosurg Psychiatry. 1978;41:948-53.
14. Hazeldine J, Dinsdale RJ, Naumann DN, Acharjee A, Bishop JRB, Lord JM, et al. Traumatic injury is associated with reduced deoxyribonuclease activity and dysregulation of the actin scavenging system. Burns Trauma. 2021;9:001.
15. Farhana A, Lappin SL. Biochemistry, Lactate Dehydrogenase. In: StatPearls. Available from: https://www.ncbi.nlm.nih.gov/books/NBK557536
16. Shi Y, Pinto BM. Human lactate dehydrogenase a inhibitors: a molecular dynamics investigation. PLoS One. 2014;9:86365.
17. Yan W, Bläsius F, Wahl T, Hildebrand F, Balmayor ER, Greven J, et al. Lactate dehydrogenase can be used for differential diagnosis to identify patients with severe polytrauma with or without chest injury-a retrospective study. PLOS ONE. 2024;19:0308228.
18. Feng J, Liu R, Chen X. Association between lactate dehydrogenase and 28-day all-cause mortality in patients with non-traumatic intracerebral hemorrhage: a retrospective analysisof the MIMIC-IV database. Biomol Biomed. 2025;25:663-71.
19. Kristjansson RP, Oddsson A, Helgason H, Sveinbjornsson G, Arnadottir GA, Jensson BO, et al. Common and rare variants associating with serum levels of creatine kinase and lactate dehydrogenase. Nature Communications. 2016;7:10572.
20. Thompson JJ, McGovern J, Roxburgh CSD, Edwards J, Dolan RD, McMillan DC. The relationship between LDH and GLIM criteria for cancer cachexia: systematic review and meta-analysis. Crit Rev Oncol Hematol. 2024;199:104378.
21. Bello S, Lasierra AB, López-Vergara L, de Diego C, Torralba L, de Gopegui PR, et al. IL-6 and cfDNA monitoring throughout COVID-19 hospitalization are accurate markers of its outcomes. Respir Res. 2023;24:125.
22. 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;7:6124-36.
23. Mercer RM, Corcoran JP, Porcel JM, Rahman NM, Psallidas I. Interpreting pleural fluid results. Clin Med (Lond). 2019;19:213-17.
24. Jurisic V, Radenkovic S, Konjevic G. The actual role of LDH as tumor marker, biochemical and clinical aspects. Adv Exp Med Biol. 2015;867:115-24.
25. Mishra D, Banerjee D. Lactate dehydrogenases as metabolic links between tumor and stroma in the tumor microenvironment. Cancers (Basel). 2019;11:750.