ORIGINAL ARTICLE

PROGNOSTIC IMPACT OF LACTATE DEHYDROGENASE IN LOW AND HIGH-RISK MYELODYSPLASTIC SYNDROMES: A SINGLE CENTER EXPERIENCE FROM PAKISTAN

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Background: The higher level of serum lactate dehydrogenase (LDH) is linked to a worse prognosis in myelodysplastic syndromes. Therefore, the present study was planned to investigate the prognostic utility of baseline lactate dehydrogenase (LDH) in predicting survival of low and high-risk myelodysplastic syndrome (MDS) patients. **Methods:** This cross-sectional study was conducted at National Institute of Blood Diseases and Bone Marrow Transplantation (NIBD-BMT), PECHS campus, Karachi, Pakistan from January 2022 to January 2024. A total of 44 newly diagnosed MDS patients were included. The Complete Blood counts (CBC) were analyzed by using Sysmex XN-1000 (Sysmex Corporation, Kobe, Japan). The IPSS was calculated for risk stratification. Serum LDH levels were done by using Cobas c311 (Roche Diagnostics, Germany). Baseline LDH <220IU/l was considered normal. All parameters were analyzed by using SPSS version 23. **Results:** In a total of 44 de novo MDS patients, 29 (65.9%) were male. The median age was 54 ranging 7–87 years. Among the patients, 32 (72.7%) had LDH ≥220IU/l. No significant differences were found between LDH levels and International Prognostic Scoring System (IPSS) risk stratified groups (p=0.311). Significant association of LDH levels was found with cytogenetic risk category (p=0.011). The median survival time for individuals with LDH ≥220 IU/l was 18 months (95% CI: 8.86–27.14), compared to 19 months (95% CI: 10.97–27.03) for LDH < 220IU/l (p=0.296). **Conclusion:** The present study did not identify significant association between LDH levels and MDS classification, risk stratification, or survival outcomes. Our findings underscore the importance of further research to elucidate the role of LDH in MDS prognosis.

Keywords: Myelodysplastic syndrome; Cytogenetics; International Prognostic Scoring System; Lactate dehydrogenase

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INTRODUCTION

Myelodysplastic neoplasms (MDS) are clonal hematopoietic neoplasms that have predisposition to bone marrow failure or acute myeloid leukemia (AML) with a combination of unexplained prolonged cytopenias and morphologic dysplasia.[1] In most cases, the blood count aberration in MDS is chronic in nature (usually lasting 4 months or even more) and cannot be completely attributed to any medication, toxin, or coexisting disease.[1] The blast cells in bone marrow, number and complexity of cytopenias, and cytogenetic abnormalities all affect the prognosis in MDS.[2] The growth factors, lenalidomide, and transfusions are used to treat people with lower risk myelodysplastic syndromes, particularly for anemia. However allogeneic stem cell transplantation and hypomethylating drugs are used to treat individuals who are at high risk.[2,3]

As MDS is a heterogeneous disease, the different scoring systems have stratified MDS patients according to the prognosis.[4,5] The number of peripheral cytopenias, the cytogenetic pattern, and the bone marrow blasts are evaluated to calculate the IPSS and the survival of MDS patients have traditionally been predicted using the revised IPSS-R.[5] The clinical, analytical, and cytogenetic changes are the basis of IPSS score, which has received extensive validation.[6,7] The impact of somatic mutation on the prognosis of MDS patients has been examined in numerous studies.[8–11] Recently, a risk stratification score for MDS called IPSS-M has been evaluated which has improved the outcome prediction for both leukemia free survival (LFS) and overall survival (OS) by incorporating molecular data to clinical and analytical parameters.[12] Although several studies have supported the IPSS's overall usefulness, it is still challenging to assess survival and the emergence of AML in specific MDS patients.[13]

In order to increase the predictive potential of the IPSS, it appears useful to evaluate other prognostic factors that may be taken into consideration and the

blood level of lactate dehydrogenase (LDH) activity is one of these factors. Several investigations have demonstrated that LDH is correlated to poor prognosis in MDS.[2,14-17] Seven German and Austrian organizations that manage MDS registries have formed a working group to validate and improve prognostication in MDS. The MDS patients identified at these facilities was gathered and then centrally examined using the working group's database.[13] The analysis of LDH in the prognosis of MDS had also been studied in Asian population. Studies from China, India and South Korea have reported significant correlation of LDH with prognosis of MDS.[18,19]

In Pakistan, clinico-hematological characteristics of MDS patients have been studied in detailed and only one study has evaluated the molecular profile of MDS.[20–24] There is none of the national study in Pakistan which has assessed the prognostic utility of LDH in the disease. The LDH is an easily performed, cost effective parameter and previous studies have indicated its utility to predict disease progression. Therefore, the current study was done to evaluate the utility of LDH as prognostic marker and whether the incorporation of LDH at baseline assessment in low and high risk MDS would impart any additional utility to predict the survival.

MATERIAL AND METHODS

This cross-sectional study was conducted at NIBD, PECHS campus, Karachi, Pakistan from January 2022 to January 2024. Approval from Institutional Ethical Committee (IRB approval #: NIBD/IRB-241/11-2022) was acquired. Informed and written consent was taken from all the patients. Inclusion criteria were newly diagnosed MDS patients of either gender or age, while secondary or therapy related MDS were excluded. Categorization of MDS and its types was based on 2016 Revision to the World Health Organization Classification of Myelodysplastic Syndromes classification.[25] At the time of diagnosis, all demographics, clinical and laboratory variables including blood cell counts, bone marrow blast, LDH, WHO type, and cytogenetic findings were evaluated. The Complete blood counts (CBC) were assessed by Sysmex XN-1000 (Sysmex Corporation, Kobe, Japan). The analysis of cytogenetic was performed by using the International System for Cytogenetic Nomenclature Criteria.[26] The serum LDH levels were analyzed by Cobas c311 (Roche Diagnostics, Germany). LDH <220 IU/l were taken as normal while those ≥220 IU/l being taken as high threshold value. The survival was also assessed and the time from MDS diagnosis to AML evolution was evaluated.

Data was analyzed by using SPSS version 23.0. The association of all categorical variables was analyzed by chi-square or fisher exact test. The Kruskal–Wallis test variance analysis for nonparametric data was used to find the significance of differences of baseline LDH levels in MDS disease sub groups and for IPSS risk groups. Mann-Whitney U was applied to find difference between the groups. To determine the probability of overall survival Kaplan Meier method was employed. Significance difference in survival between the patient's groups was calculated by Log rank test. Differences were considered significant with $p<0.05$.

RESULTS

In a total of 44 de novo MDS patients, 29 (65.9%) were males and 15 (34.1%) females. The median age was 54 (ranging between 07–87 years). Most patients of MDS were classified as MDS-EB2, MDS-EB1, MDS-SLD, and MDS-MLD, 11 (25.0%), 10 (22.7%), 7(15.9%), and 6 (13.6%), respectively. Table 01 depicts the descriptive characteristics of all MDS patients along with their classification. The most frequent disease complication observed was febrile neutropenia in 20 (45.5%) followed by gastrointestinal bleeding in 11 (25.0%), urosepsis 10 (22.7%), and pneumonia in 10 (22.7%) patients respectively. The frequency distribution of complications with respect to LDH levels during the course of the study is shown in a figure-1.

Among the patients, 32 (72.7%) had LDH ≥220 IU/l, while 12 (27.3%) had <220 IU/l. No significant association was observed between LDH levels and the classification types of MDS (p=0.207). Our findings also revealed no significant correlation between LDH levels and overall survival (p=0.311) or the incidence of AML transformation (p=0.653). Significant association of LDH levels was found with cytogenetics risk category (p=0.011), and cytogenetics findings (p=0.007). An insignificant difference was observed in LDH levels between IPSS risk groups (p=0.311). Table-2 shows the details about the association of baseline LDH levels with respect to characteristics and outcome of MDS patients.

After comparing different risk groups, it was found that the low-risk group had higher median LDH levels 259 (ranging 229–328 IU/I) than higher-risk group with median levels of 253 (ranging 193–299 IU/I). The Mann-Whitney U test revealed no statistically significant difference in LDH levels between the IPSS risk groups (p=0.446). Kruskal-Wallis test performed to analyze the relationship between MDS WHO classification 2016 groups and LDH levels found no statistically significant association (p=0.814). The median duration of follow up was 12 months (ranging between 1–24 months). The median survival time for individuals with LDH ≥220IU/l was 18 months (95% CI: 8.86-27.14 months), compared to 19 months (95% CI: 10.97–27.03 months) with LDH <220 IU/l. The log rank test did not indicate any significant association (p=0.296), as shown in figure-02. Twenty-two MDS patients who were identified as IPSS high risk, had median survival time of 14 months (95% CI: 5.01–22.99 months), while 22 patients who were low risk, had median survival of 23

months (95% CI: 21.04–24.96). The log-rank test did not demonstrate any significant association (p=0.114), as shown in figure-03. Over the course of the study, 07 patients were transformed into AML. Six of them had LDH levels >220 IU/l. Out of the 06 cases, 02 patients were classified as low risk as per IPSS, while 04 were categorized as high risk.

Table-1: Descriptive characteristics of MDS patients with respect to MDS classification (n=44)

WHO category	N	Age (IQR)	Hb (g/dl)	ANC (*10^9/L)	Platelet counts (*10^9/L)	BM blast (%)
Hypoplastic MDS	04	47 (27-77)	8.6 (7.1-10.2)	1.32 (0.2-2.3)	77 (25-110)	03(2.2-3.7)
MDS-EB1	10	61 (39-70)	7.7 (6.6-8.9)	1.17 (0.5-2.6)	40 (15-76)	07(01-09)
MDS-EB2	11	53 (32-60)	8.9 (8-9.4)	1.72 (0.8-5.2)	48 (32-80)	11(07-15)
MDS-MLD	06	5 1(40-64)	6.2 (5.1-8.9)	0.77 (0.3-2.5)	94 (34-168)	04(02-02)
MDS-SLD	07	58 (52-70)	6.2 (5.3-8.8)	1 (0.5-5.0)	118 (35-246)	03(02-04)
MDS-U	01	_	_	_	_	_
MDS based on defining cytogenetics abnormality (Del7q)	01	_	_	_	_	_
MDS Del5q	01	_	_	_	_	_
MDS-RS-SLD	01	_	_	_	_	_
MDS/MPN	02	_	_	_	_	_
Total MDS patients	44	54 (36-65)	8.2 (6.7-9.1)	1 (0.6-2.7)	47 (27-120)	04 (02-08)

Table-2: Association of baseline LDH levels with respect to characteristics and outcome in MDS patients

Characteristics and outcomes	Total (%)	LDH levels		p-value
	n=44 (%)	≥220 IU/l n=32 (%)	<220 IU/l n=12 (%)	
Gender				0.555
Male	29 (65.9%)	20 (62.5%)	09 (75%)	
Female	15 (34.1%)	12 (37.5%)	03 (25%)	
MDS Classification				0.207
MDS-EB2	11 (25.0%)	09 (28.1%)	02 (16.7%)	
MDS-EB1	10 (22.7%)	05 (15.6%)	05 (41.7%)	
MDS-SLD	07 (15.9%)	05 (15.6%)	02 (16.7%)	
MDS-MLD	06 (13.6%)	06 (18.8%)	-	
Hypoplastic MDS	04 (9.1%)	03 (9.4%)	01 (8.3%)	
MDS/MPN	02 (4.5%)	02 (6.3%)	-	
MDS-RS-SLD	01 (2.3%)	01 (3.1%)	-	
MDS Del5q	01 (2.3%)	-	01 (8.3%)	
MDS based on defining cytogenetics abnormality (Del7q)	01 (2.3%)	01 (3.1%)	-	
MDS-U	01 (2.3%)	-	01 (8.3%)	
Cytogenetics				0.007
Normal	26 (59.1%)	23 (71.9%)	03 (25%)	
Abnormal	18 (40.9%)	09 (28.1%)	09 (75%)	
Cytogenetics Risk Category				0.011
Good	30 (68.2%)	25 (78.1%)	05 (41.7%)	
Very good	01 (2.3%)	-	01 (8.3%)	
Intermediate	06 (13.6%)	02 (6.3%)	04 (33.3%)	
Poor	01 (2.3%)	-	01 (8.3%)	
Very poor	06 (13.6%)	05 (15.6%)	01 (8.3%)	
IPSS Category				0.311
High	22 (50.0%)	14 (43.8%)	08 (66.7%)	
Low	22 (50.0%)	18 (56.3%)	04 (33.3%)	
Transfusion frequency				0.164
≤2 months	27 (61.4%)	10 (31.3%)	07 (58.3%)	
>2 months	17 (38.6%)	22 (68.8%)	05 (41.7%)	
Admission frequency				0.412
≤2 months	25 (56.8%)	16 (50.0%)	09 (75%)	
>2 months	18 (40.9%)	15 (46.9%)	03 (25%)	
Never	01 (2.3%)	01 (3.1%)	-	
Survival Status				0.311
Death	22(50.0%)	18 (56.3%)	04 (33.3%)	
Alive	22(50.0%)	14(43.8%)	08(66.7%)	
Transformed to AML				0.653
No	37(84.1%)	26(81.3%)	11(91.7%)	
Yes	07(15.9%)	06 (18.8%)	01 (8.3%)	

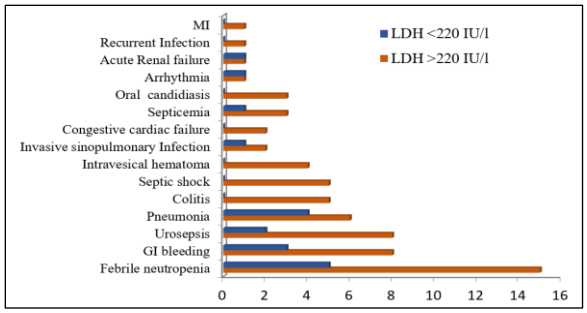

Figure-1: Frequency distribution of complications in mds patients with respect to LDH levels

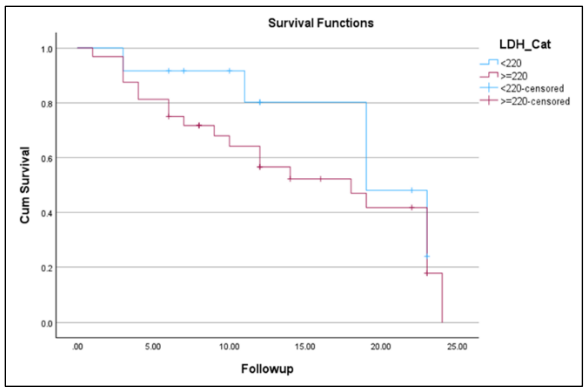

Figure-2: Kaplan-Meier survival curve illustrating the association between the survival time and LDH levels in MDS patients

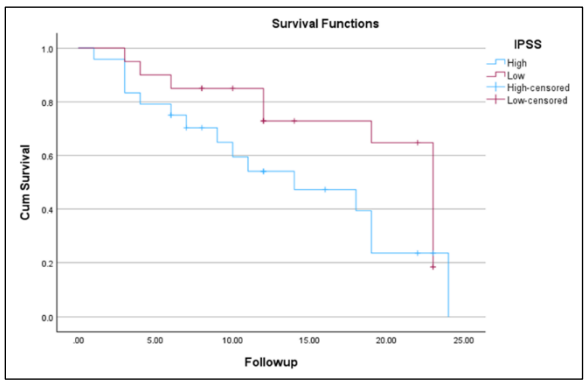

Figure-3: Kaplan-Meier survival curve demonstrating the relationship between survival probabilities and risk status

DISCUSSION

Metabolic alterations in the cancer cells involve high glucose uptake and abnormal activity of lactate dehydrogenase (LDH), which mediates glucose conversion to lactic acid. Elevated serum LDH levels are commonly observed in cancer patients and are associated with poor clinical outcomes and treatment resistance.[27] Consequently, LDH determination has become a standard adjunctive tool in cancer diagnosis and treatment monitoring.[28] The significance of serum LDH levels in MDS patients has been an area of considerable interest, reflecting its potential prognostic value and its association with disease progression and survival outcomes.[15]

The current study aimed to explore the prognostic relevance of LDH levels in MDS patients. In this study involving 44 MDS patients, the majority of patients (72.7%) exhibited LDH levels ≥220 IU/l. Although the median survival time for patients with LDH <220 IU/l was slightly higher compared to those with LDH >220 IU/l (19 months vs 14 months), the survival analysis did not reveal any significant association (p=0.296). These results are in contrast with the prior studies.[13,19,29]

Wimazal *et al*. from Austria observed that upon diagnosis among 221 patients with MDS, the median LDH levels were 206 U/l (ranging from 101 to 2600 U/l).[29] Patients exhibiting elevated LDH levels (>240 U/l) experienced a median survival period of 26.8 months, significantly shorter than those with normal LDH levels (44.6 months; p<0.05). Additionally, they observed a notable increased in LDH levels preceding disease progression, suggesting its potential as an independent prognostic variable.[10] Another study by Malayath revealed that among the 39 patients initially presenting with elevated LDH levels (≥500 IU/L.), 51.3% patients passed away during the follow-up period compared to 14.1% with initially low LDH levels (<500 IU/L).[3] Zgabg *et al*. from China observed that the median LDH level at diagnosis was 214 U/L, ranging from 102 to 865 U/L and notably, patients with elevated LDH levels (>240 U/L) exhibited a substantially shorter median survival time of 25.6 months compared to those with normal LDH levels (56.8 months), demonstrating a statistically significant association (p<0.05).[30] Another study demonstrated an association between elevated serum LDH levels and reduced median survival in patients with MDS.[13] Specifically, patients with LDH levels equal to or exceeding 300 U/l exhibiting significantly shorter median survival of 10.3 months compared to those with LDH levels below 300 U/l (p<0.01), which is in contrast to the present findings as we did not find any significant association.[15] Rana *et al*. in a recent local study emphasized that serum LDH levels can be utilized to distinguish between megaloblastic anemia from other anemia, especially MDS before proceeding for bone marrow analysis.[31]

In our study, patients of MDS were classified as MDS-EB2, MDS-EB1, MDS-SLD, and MDS-MLD, 25.0%, 22.7%, 15.9%, and 13.6%, respectively. The regional data shows difference in distribution of MDS classification as demonstrated by a study conducted by Abraham *et al*, where MDS-MLD was the most common MDS classification noted in 62.5%

patients while MDS-EB2, and MDS-EB1 were found in 12.5%, 10.4% MDS patients, respectively.[15] Another study done by Gupta *et al* had MDS-MLD, MDS-EB2, and MDS-EB1 as the most common MDS sub type seen in 42.0%, 22.0%, and 21.3% patients respectively.[32] These findings reveal difference in pattern of distribution of MDS classification among patients from Pakistan which further necessitates future research exploring various aspects of MDS. The discrepancies between our results and those of previous investigations could be attributed to several factors such as variations in patient population; including demographics, disease severity, and treatment modalities contributing to variations in LDH levels and their prognostic implications. Additionally, variations in study methodologies, such as sample size, follow-up duration, and analytical techniques for LDH measurement could also influence the observed outcomes. Moreover, the multifactorial nature of MDS, characterized by heterogeneous disease biology and variable clinical trajectories, underscores the complexity of interpreting LDH levels as a singular prognostic marker.

CONCLUSION

While the current study did not identify significant associations between LDH levels and MDS classification, risk stratification, or survival outcomes, the findings underscore the importance of further research to elucidate the role of LDH in MDS prognosis. Integrating data from future perspective and multicenter studies, considering diverse patient cohorts and incorporating comprehensive prognostic models, may provide deeper insights into the clinical significance of LDH in MDS management which could potentially contribute to personalized treatment strategies.

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The authors declare that they have no conflict of interests.

CONTRIBUTIONS OF AUTHORS

NA: conceptualization of the study, did study design and contributed to manuscript writing, editing, critically reviewed and approval of final manuscript. LM contributed in data collection and statistical analysis.NF contributed to statistical analysis. VK and AA contributed in manuscript writing. All authors reviewed the final manuscript draft.

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