ORIGINAL RESEARCH

Assessment of Serum Lactate Dehydrogenase in Oral Potentially Malignant Disorders and Oral Squamous Cell Carcinoma

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Abstract

Introduction: Oral squamous cell carcinoma (OSCC) remains the most common cause of cancer-related deaths in man, with several risk factors such as cigarette smoking, HPV virus infection, and exposure to UV radiation. The frequent delay in diagnosis and the often-limited response of tumors to therapy serve as a major factor in failure of treatment of cancer. So, early detection and treatment is the key to improve outcomes. Serum Lactate dehydrogenase (LDH) in oral potentially malignant disorders (OPMDs) and OSCC serves as an important prognostic factor. LDH, usually intracellular, comes into extracellular space upon tissue damage and necrosis. High LDH levels have been widely reported in multiple cancer types and are associated with poor prognosis in solid tumors.

Aim & Objectives: 1. To compare and assess serum LDH in OPMDs and normal healthy individuals. 2. To compare and assess serum LDH in patients of OPMDs & OSCC.

Materials and Method: Study samples included total 150 subjects with histo-pathologically diagnosed cases of OSCC, OED, OLP, OSMF with size of 30 cases each, reported at Department of Oral Pathology & Microbiology. After obtaining consent from the patients, serum samples were collected & analysed for LDH levels. Serum samples from healthy individuals were taken as a control group. Collected data were statistically analyzed using SPSS 22. Results were expressed as mean ± standard deviation by independent t test. One-way ANOVA (p-value of ≤ 0.05) & Post-Hoc Tukey's correction (p-value of ≤ 0.005) were done for multiple group comparisons and for group-wise comparisons, respectively.

Result: Our findings demonstrated statistically significant ($p = 0.00027$), stepwise increase in serum LDH from the control group through OPMDs to frank malignancy, with OSCC patients exhibiting the highest LDH levels. All pair-wise group comparisons using post hoc Bonferroni tests also showed significant difference in serum LDH levels ($p < 0.005$).

Conclusion: LDH may be a useful, non-invasive biomarker in differentiating between benign, potentially malignant, and malignant oral disorders, though its diagnostic accuracy should always be interpreted in conjunction with clinical and histopathological findings.

Keywords: Serum, Lactate dehydrogenase, OSCC, OPMDs, ANOVA, OLP, OED, OSMF.

Introduction

Oral cancer (OC) stands as the primary cause of cancer-related deaths among men in India, with significantly high rates also documented across Europe, Australia, and New Zealand.[1] Research has established several key risk factors in the development of OC, including cigarette smoking, infection with human papillomavirus (HPV), and exposure to ultraviolet radiation.[2-6] One notable feature of oral cancers is their tendency to spread (metastasize) to nearby lymph nodes before reaching distant organs, and the extent of this lymph node involvement is a crucial determinant of survival rates in patients with oral squamous cell carcinoma (OSCC). Major challenges in improving survival rates for OSCC lie in the frequent delay in diagnosis and the often-limited response of tumors to treatment.

Consequently, early and precise detection remain the single most significant factor in determining the prognosis

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of OSCC. Over the years, researchers have investigated a wide range of tissue-based molecular markers to anticipate and recognize the progression from normal oral tissues to pre-cancerous changes and, ultimately, to OSCC.[7] Despite these advances, no perfect biomarker has yet been identified.

The evolution of molecular biology has also opened new avenues for detecting tumor markers in readily accessible biological fluids such as blood, saliva, and urine. Recent studies have suggested that certain serum markers—notably, squamous cell carcinoma antigen (SCC-Ag), ferritin, and carcinoembryonic antigen (CEA)—are markedly elevated in OSCC patients, pointing to their potential role in diagnosis.[8]

Tissue damage and cell death are common features in a variety of cancers, including OSCC. Identifying a biomarker that reflects tissue injury would facilitate detection and monitoring in OSCC. Lactate dehydrogenase (LDH), a ubiquitous intracellular enzyme, is released into the extracellular environment upon tissue damage and necrosis.[10] High LDH levels have been widely reported in multiple cancer types and are associated with poor prognosis in solid

tumors. Even minor tissue injury can lead to measurable increases in LDH levels.[11,12] LDH has been extensively studied, with elevated levels indicating a poor prognosis across various solid tumors. Studies by Suh SY et al. showed that serum LDH predicts survival in untreatable cancers.[13] While Zhou GQ et al. demonstrated its correlation with clinical staging in nasopharyngeal carcinoma.[14] Further, elevated LDH levels have been detected in patients with OPMDs and OSCC, separately.

Despite this, there's still a lack of research directly comparing and correlating serum LDH in OPMDs & OSCC. Therefore, this study was undertaken to evaluate and compare serum LDH among people with OPMDs, OSCC, and healthy controls. The aim was to clarify whether LDH could serve as an effective biomarker for identifying and monitoring OPMDs & OSCC patients.

Material & Methods:

Tissue Specimens:

The present study was prospective type of study. Due approval from Institutional Ethical Committee, with approval number MIDSR/IEC-204/837/265/2024 was taken &, the study subjects were recruited from the Department of Oral Pathology & Microbiology. Before conducting the study, the purpose, design and procedure were explained to the patient and written consent was obtained from each participant.Study sample included serum samples of diagnosed cases of OSCC, OED, OLP and OSMF with sample size of 30 cases each. Serum samples of healthy individuals were taken as a control group.

Total numbers of subjects selected were 150 and they are divided into five groups as:

- Group 1: 30 samples of Normal Healthy Individual (Control)
- Group 2: 30 cases of OLP
- Group 3: 30 cases of OSMF
- Group 4: 30 cases of OED (10 Mild + 10 Moderate + 10 Severe)
- Group 5: 30 cases of OSCC (20WDSCC + 6 MDSCC + 4 PDSCC)

Inclusion Criteria:

- All histo-pathologically diagnosed cases of OSCC, OED, OLP & OSMF without any history of prior treatment for the same lesion.
- Healthy individuals as control group without any oral habits.

Exclusion Criteria:

- Patient not willing for participation in the study.
- Patients with any H/O or undergoing treatment for malignancy, hepatitis, hypothyroidism, anemia (hemolytic or pernicious anemia), lung disease, liver disease, kidney disease, pancreatitis, muscle trauma, muscular dystrophy, arrhythmia, pulmonary infarction and stroke.
- Patients undergoing treatment for the diagnosed conditions of OSCC, OED, OLP & OSMF.
- Patients with H/O myocardial incidence within past 2 weeks.
- Patients with H/O consumption of aspirin, narcotics or alcohol.
- Patients with H/O recent anesthesia or surgical procedure.

Out of 30 cases of OSCC, 20 cases were WDSCC, 6 cases were MDSCC and 4 cases were of PDSCC. For OED 10 cases were having mild dysplasia, 10 were of moderate dysplasia and 10 cases were of sever dysplasia.

Method of Sample Collection & Biochemical analysis:

Serum sample collection from study subjects were done according to the method recommended by National Institute of Health (NIH- Bethesda, MD, USA, 2009). 5ml of venous blood were collected from anticubital vein with proper aseptic precautions. The blood sample was kept at room temperature for 30-60 minutes for clot formation & serum separation. Then, sedimented blood sample was centrifuged at 3000 rpm for 10-15 minutes. Supernatant serum was separated out with the help of a micro-pipette. Quantitative estimation of LDH was done using was a semi-automated biochemical analyzer & commercially available Ames Autopak LDH reagent kit, within 3hrs of sample collection.

Statistical Analysis:

Results were expressed as mean ± standard deviation by independent t test. One-way ANOVA was used for multiple group comparisons followed by Post-HocBonferroni's correction for group wise comparisons. For ANOVA, p-value of ≤ 0.05 was considered for statistical significance, while for Post-Hoc test, p-vale of ≤ 0.005 was considered for statistical significance.

Results:

Table 1 summarizes the gender-wise distribution of study subjects including the control group. Out of 150 participated study subjects, 86 (71.67%) were male and 64 (53.33%) were female subjects. The age distribution of the study subjects ranges from 2nd to 6th decade, with the mean age of 44.6 yrs.

In the present study, mean value of serum LDH level for control, OED, OLP, OSMF & OSCC were 198.80 ± 23.52, 369.53 ± 27.52, 225.56 ± 24.47, 299.66 ± 32.43 & 473.23 ± 30.91 respec-

tively. In all study subjects, serum LDH levels were elevated as compared to control, with maximum increase of serum LDH in OSCC patients followed by OED, OSMF and OLP patients (Graph 1). The increased levels of serum LDH was statistically significant with *p-value* of 0.00027 which was < 0.05 (Table 2).

Individual group comparison using Bonferroni *post hoc* tests showed statistically significant difference between control group and every study group with *p-value* for every group comparison < 0.05 (Table 3). Maximum difference for mean value of LDH was observed between OSCC & Control group (274.43) followed by OSCC & OLP group (247.67). While the least difference in LDH levels were observed between control & OLP group (26.77) (Table 3).

Discussion:

In this study, we evaluated serum lactate dehydrogenase (LDH) levels among control subjects (198.80 ± 23.52), patients with oral lichen planus (OLP) (225.56 ± 24.47), oral submucous fibrosis (OSMF) (299.66 ± 32.43), oral epithelial dysplasia (OED) (369.53 ± 27.52), and oral squamous cell carcinoma (OSCC) (473.23 ± 30.91). Our findings demonstrate a significant, stepwise increase in serum LDH from the control group through OPMDs to frank malignancy, with OSCC patients exhibiting the highest LDH levels followed by OED, OSMF and OLP patients. Both our mean differences and post hoc analysis indicate that LDH may help distinguish not only between healthy and diseased states but also among various OPMDs and OSCC. This gradient was statistically significant as revealed by ANOVA (p = 0.00027), and all pair-wise group comparisons using post hoc Bonferroni tests were highly significant (p < 0.005), with highest difference in normal and OSCC (274.43), followed by OLP v/s OSCC (247.67) and least in normal v/s OLP (26.77). These findings suggest that as the oral epithelial pathology advances from chronic inflammation (OLP, OSMF) to dysplasia (OED) and carcinoma (OSCC), there is a corresponding increase in serum LDH. This may reflect enhanced anaerobic glycolysis (Warburg effect), tissue necrosis, and metabolic reprogramming, and rapid cellular proliferation, characteristics of malignant transformation. Meanwhile, inflammatory conditions such as OLP and OSMF showed moderately increased LDH, possibly due to chronic tissue inflammation and necrosis.

These results are in concordance with several previous studies that have established the diagnostic and prognostic

Table 1: Distribution of Study Subjects

Study Groups	Male	Female	Total
Control	16	14	30
OLP	07	23	30
OSMF	24	06	30
OED	18	12	30
OSCC	21	09	30
Total	86(71.67%)	64(53.33%)	150

Control - Normal Healthy Individual, OLP– Oral Lichen Planus, OSMF – Oral Submucous Fibrosis, OED – Oral Epithelial Dysplasia, OSCC – Oral Squamous Cell Carcinoma.

Table 2: Comparison of mean LDH levels in Control Group, OPMD & OSCC (ANOVA)

Groups		Serum LDH (IU/L) Mean±SD
Control		198.80±23.52
OED		369.53±27.52
OLP		225.56±24.47
OSMF		299.66±32.43
OSCC		473.23±30.91
ANOVA	F value	476.10
	Fcrit	2.43
	p value	0.00027* <0.05 Significant

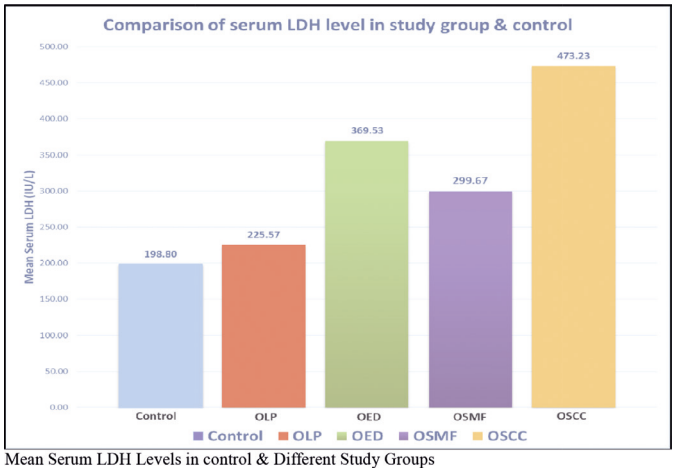
Mean difference is significant at thep-value≤0.05, *Significant.

Table 3: Multiple Comparisons of serum LDH by Post-hoc Tests (Bonferroni's Multiple Comparisons)

Comparison Groups	Mean difference	p-value (Significance)
Control vs. OSCC	274.43	0.0003977*
Control vs. OSMF	100.87	0.0005831*
Control vs. OLP	26.77	0.0006182*
Control vs. OED	170.73	0.0001657*
OED vs. OLP	143.97	0.0003207*
OED vs. OSMF	69.87	0.0001350*
OED vs. OSCC	103.70	0.0007279*
OLP vs. OSMF	74.10	0.0003239*
OLP vs. OSCC	247.67	0.0002762*
OSMF vs. OSCC	173.57	0.0005141*

Mean difference is significant at the p-value ≤0.005, *Significant

Graph 1:



Mean Serum LDH Levels in control & Different Study Groups

value of LDH in oral OPMDs and OSCC. For instance, study by Anitha et al.,(2022)[15] demonstrated statistically significant elevated levels of LDH in serum and saliva samples of OSCC patients when compared to healthy individuals, consistent with results reported by Joshi et al.,[16] Pereira T et al.,[17] Gholizadeh et al.,[18] Pandey et al. (2021),[19] Anthwal N et al. (2020),[20] and Kumar et.al.[21] suggesting increased cell turnover and tissue breakdown with progression. Bhuvneshwari et al.,(2022)[22] and Patel S et al.,(2019)[23] also found that salivary LDH was increased in patients with leukoplakia and OSCC, as compared to normal individuals. Similarly, Vishwanath et al. (2013)[24] documented rising LDH activity with increasing severity of dysplasia and malignancy in oral lesions, consistent with our observation of a gradational rise from OLP and OSMF to OED and OSCC, and also concluded that serum LDH could serve as a non-invasive biomarker for malignant transformation in oral lesions. On comparisons between the histopathological grades of OSCC group the level of LDH were found to increase from well differentiated to moderately differentiated to further increase in poorly differentiated patients, similar to those studied by Bhuvneshwari et al.[22] and Patel et al.,[23] who reported that increased serum LDH level had a positive correlation with the histological grading of OSCC.

There are some controversies for the clinical value of serum LDH in oral lesions. For instance, Joshi et al. (2011)[25] observed that while LDH was elevated in OSCC, its levels in OPMDs did not consistently differ from healthy subjects, indicating a limitation if used in isolation. Patel et al. reported no significant difference in serum LDH between OSCC, OPMD, and control groups.[23] Shah et al. (2023) measured both serum and salivary LDH across patients with habits, OPMDs, and OSCC, reporting that differences in serum LDH were non-significant (P>0.05, P>0.05), although salivary LDH levels did show significance. Rao et al. (2024)[26] compared serum and salivary LDH levels in OSCC and OPMD patients versus controls, reporting that serum LDH levels showed high variability and were affected by sample handling and analytical processes, leading to inconsistent results.[27] Such discrepancies may result from differences in selection criteria, sample sizes, or technical aspects of LDH assay. Some variability may also arise from underlying systemic conditions or lifestyle factors that can non-specifically elevate LDH.

Higher LDH reflects higher tissue damage and increased cellular turnover, and necrosis within the affected oral tissues, serving as a non-specific but valuable biomarker differentiating malignant from pre-malignant and healthy states, thereby serving as an adjunctive diagnostic tool. Positive correlation between serum LDH and clinical staging, as observed in previous studies along with our findings, suggests that monitoring serum LDH levels could aid clinicians in assessing tumor burden, aggressiveness, and progression in OPMDs & OSCC patients. Easily measured via minimally invasive blood sampling, SLDH offers practical advantages for routine screening, especially in high-risk populations, and can augment the predictive value of established tumor markers when used in combination. The findings of our study, strengthened by consistent trends across multiple comparative groups and robust statistical significance, underscore the potential of serum LDH as an adjunct marker in screening and monitoring OPMDs. Due to its association with poorer prognosis at higher levels, serum LDH can also inform risk stratification and help tailor follow-up and therapeutic strategies. Nonetheless, its integration into clinical protocols should be guided by further validation in larger studies to ensure specificity and reliability across diverse patient populations. When used in conjunction with other serum or salivary biomarkers (such as SCC-Ag, CEA, or specific salivary proteins), serum LDH may enhance the accuracy of diagnostic and prognostic models for OPMDs & OSCC. Its non-invasive nature and significant association with pathology makes it a feasible, cost-effective screening adjunct, particularly in resource-limited settings. Integrating serum LDH values into clinical workflows can guide the intensity of monitoring and intervention, paving the way for more personalized and timely management of OPMDs & OSCC.

Conclusion:

In conclusion, our findings align with the majority of literature suggesting elevated serum LDH in OPMDs and OSCC compared to healthy individuals. LDH may be a useful, non-invasive biomarker in differentiating between benign, potentially malignant, and malignant oral disorders, though its diagnostic accuracy should always be interpreted in conjunction with clinical and histopathological findings.

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