ORIGINAL RESEARCH

Evaluation of total Serum Creatine Phosphokinase and Serum Lactate Dehydrogenase levels in patients with Oral Leukoplakia & Oral Cancer

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ABSTRACT

Background: The diagnosis of malignancies and premalignant lesions has been aided by the use of numerous tumour markers and enzyme studies. The present study evaluated total serum creatine phosphokinase and serum lactate dehydrogenase levels in patients with oral cancer and oral leukoplakia. **Materials & Methods:** Group I comprised of Oral leukoplakia patients, group II had oral cancer patients and healthy controls were kept in group III. 5 mL of venous blood was collected and used for estimation of serum CK and LDH levels using Siemens autoanalyzer. **Results:** Group I had 22 males and 10 females, group II had 20 males and 12 females and group III had 16 males and 16 females. The mean LDH level in group I was 290.2 IU/L, in group II was 318.4 IU/L and in group III was 145.2 IU/L. The difference was significant (P< 0.05). The mean CPK in group I was 78.2 IU/L, in group II was 70.8 IU/L and in group III was 106.4 IU/L. The difference was significant (P< 0.05). **Conclusion:** Serum LDH and CK measurement is a simple, non-invasive process that may be employed in patients with OSCC and oral cancer as a biochemical marker.

Key words: LDH, tumour markers, Oral Squamous cell carcinoma

INTRODUCTION

All cancers that start in the oral tissues are included under the umbrella term of "oral cancer," which is also a significant indicator of poor health and disease around the world. A significant degree of morbidity and death (about 50%) are features of the condition. 9.8% of India's anticipated 644,600 accidental cancer cases are head and neck cancer.[1] A multistage process leads to oral squamous cell carcinoma (OSCC), which develops from normal cells to dysplastic cells (precancerous lesions).[2]

The development of oral squamous cell carcinoma (OSCC) occurs in stages, starting with normal cells and progressing to dysplastic cells (precancerous lesions) and then squamous cell carcinoma. The majority of precancerous lesions in the oral cavity, or oral leukoplakia, or OL, account for 85% of all such lesions. An increase in glycolytic activity and a switch from aerobic to anaerobic glycolysis are linked to the development of cancer.[3]

Prior until now, the diagnosis of malignancies and premalignant lesions has been aided by the use of numerous tumour markers and enzyme studies.[4] Among all the physiological fluids, blood has emerged as the preferred medium for the investigation of biochemical indicators. Certain cancers, including big gut malignancies, hemangiopericytoma, and adenocarcinoma, have been discovered to have high levels of markers such carcinoembryonic antigen (CEA), lactate dehydrogenase (LDH), and phosphohexose isomerase (PHI).[5] It has been proposed that the enzyme creatine phosphokinase (CK) serves as a marker for a variety of malignancies, including liver, colon, and lung carcinomas.[6] The present study evaluated total serum creatine phosphokinase and serum lactate dehydrogenase levels in patients with oral cancer and oral leukoplakia.

MATERIALS & METHODS

The present study comprised of 64 patients of oral leukoplakia and oral cancer of both genders. All patients gave their written consent for the participation in the study.

Data such as name, age, gender etc. was recorded. Group I comprised of Oral leukoplakia patients, group II had oral cancer patients and healthy controls were kept in group III. 5 mL of venous blood was collected from the antecubital vein and was then centrifuged at 3000 rpm for 10 minutes to separate the serum. The biochemical estimation of serum CK and LDH levels using Siemens autoanalyzer was carried out using serum. Data thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Groups	Group I (32)	Group II (32)	Group III (32)
Status	Oral leukoplakia	OSCC	Control
M:F	22:10	20:12	16:16

Table I shows that group I had 22 males and 10 females, group II had 20 males and 12 females and group III had 16 males and 16 females.

Table II Estimation of LDH in all groups

Groups	LDH (IU/L)	P value
Group I	290.2	0.01
Group II	318.4	
Group III	145.2	

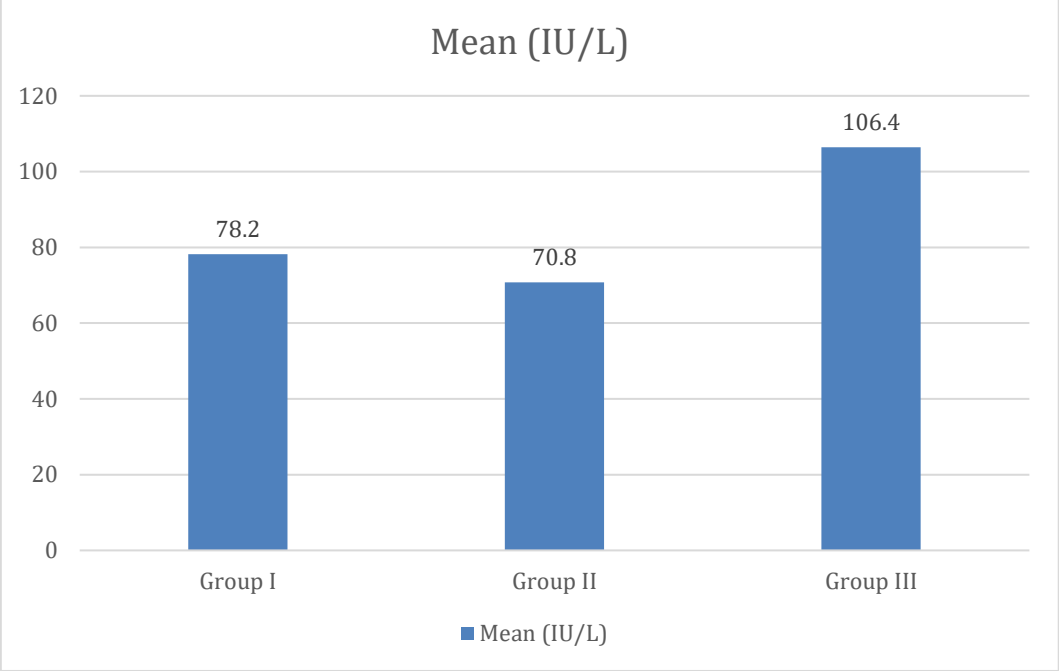
Table II shows that mean LDH level in group I was 290.2 IU/L, in group II was 318.4 IU/L and in group III was 145.2 IU/L. The difference was significant (P< 0.05).

Table III Estimation of serum creatine phosphokinase in all groups

Groups	CPK (IU/L)	P value
Group I	78.2	0.01
Group II	70.8	
Group III	106.4	

Table III, graph I shows that mean CPK in group I was 78.2 IU/L, in group II was 70.8 IU/L and in group III was 106.4 IU/L. The difference was significant (P< 0.05).

Graph I Estimation of serum creatine phosphokinase



DISCUSSION

A white lesion, a red lesion, a mixed red/white lesion, a lump, an ulcer with fissuring or elevated edges, pain or numbness, a loose tooth, an extraction socket that hasn't healed, induration, the fixing of a lesion to deeper tissues, enlarged lymph nodes, dysphagia, and weight loss are examples of classic signs.[7,8] With the increase in the glycolytic activity the concomitant increase in lactate dehydrogenase (LDH) enzyme may be reflected in certain tissues.[9,10] The present study evaluated total serum creatine phosphokinase and

serum lactate dehydrogenase levels in patients with oral cancer and oral leukoplakia.

We found that group I had 22 males and 10 females, group II had 20 males and 12 females and group III had 16 males and 16 females. Clinically and histopathologically determined oral leukoplakia and oral malignancies were included by Bhayya H et al[11], with 20 patients in each category. Twenty healthy patients without lesions, tobacco-related habits, or any systemic disorders made up the control group. When compared to the control group, it was discovered that total serum CK levels were statistically considerably lower in both oral leukoplakia and oral cancer. When compared to the control group, total serum LDH levels were statistically substantially higher in both oral leukoplakia and oral cancer.

We mean LDH level in group I was 290.2 IU/L, in group II was 318.4 IU/L and in group III was 145.2 IU/L. Patra S et al.[12], found that with the development of the malignancy, CK activity in the muscle gradually diminished until it was finally undetectable during the final stage of dedifferentiation. Lactate dehydrogenase (LD) levels in saliva were found to change in relation to oral leukoplakia (OL) and oral cancer (OC), according to Shetty SR et al.[13] The study included 75 patients—25 with oral leukoplakia, 25 with oral cancer, and 25 healthy controls (HC) who reported to the department of oral medicine and radiology. In all three study groups (OL, OC, and HC), the mean salivary lactate dehydrogenase levels were higher in males than in females. In the control group, oral leukoplakia group, and oral cancer group, the salivary lactate dehydrogenase levels were 79.50 4.67 IU/L, 136.46 3.36 IU/L, and 148.77 4.83 IU/L, respectively. There was a significant difference in the mean salivary levels of the above groups.

We mean CPK in group I was 78.2 IU/L, in group II was 70.8 IU/L and in group III was 106.4 IU/L. Swei H. Tsung[14] measured total creatine kinase (CK; EC 2.7.3.2) activity and isoenzyme pattern in normal and neoplastic tissues. Found that CK activity was detected in all the examined tissue and concluded that total CK activity was very low in most tumour tissues. Joshi et al[15] studied clinically diagnosed 30 cases each of OL and 30 healthy individuals of comparable age served as control. Unstimulated whole saliva was aseptically collected and was processed immediately for LDH isoenzymes measurement by agarose gel electrophoresis. Biopsy specimen obtained was processed and stained by hematoxylin and eosin. Sections of OL and OSCC cases were scrutinized histopathologically and appropriately graded for epithelial dysplasia and differentiation of carcinoma respectively. The present salivary analysis for LDH isoenzyme reveals an overall increased salivary LDH isoenzyme level in OL and OSCC cases and a significant correlation between levels of salivary LDH isoenzymes and histopathologic grades of dysplasia in OL and OSCC. Salivary analysis of LDH will definitely provide the clinician and/or the patient himself with an efficient, non- invasive and friendly new tool for diagnosis and monitoring of oral precancer and cancer.

The limitation the study is small sample size.

CONCLUSION

Authors found that serum LDH and CK measurement is a simple, non-invasive process that may be employed in patients with OSCC and oral cancer as a biochemical marker.

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