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RESEARCH ARTICLE

LEVEL OF SERUM CREATINE PHOSPHOKINASE IN ORAL ERYTHRO LEUKOPLAKIA- A BIOCHEMICAL STUDY

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ABSTRACT

Context: Oral cancer develop from a potentially malignant disorders.
Early diagnosis of these are necessary in order to prevent them from developing into malignant ones which can reduce the mortality and morbidity and improve the quality of life of a person .The different diagnostic procedures available now are histopathology, cytology, histochemistry and Enzyme assays. However histopathology has many limitations which can be overlooked by an enzyme essay technique.
Keeping this in mind a study was planned to evaluate the association between the serum CPK and oral Erytro Leukoplakia.
Aims: Aims and Objectives of the study-
1. To assess the level of CPK in erythro leukoplakic patients and normal subjects .
2. To compare the result

Study design Out patients visiting the department of Oral Medicine and Radiology, Coorg Institute of Dental Sciences, Virajpet in the age range of 30-50 years old males. With smoking habit for more than 5 yrs. 20 subjects were selected and the lesion was subjected to histopathological examination and blood samples were collected and sent for biochemical analysis.

Statistical analysis used: student t test

Result: Mean CPK value of the control group was 96.6 and that of cases was 102.25 . Statistical analysis was done using student t test and it was not statistically significant (p=0.441). Correlation between age and CPK value was done and the correlation(r) is -0.083 but it was not significant statistically (p=0.611).

Conclusions: From this study it can be concluded that a definite correlation between CPK enzyme and oral erythro leukoplakia lesions cannot be obtained as it is dependent on different factors like the severity of lesion, grades of dysplasia and invasiveness of lesion.

INTRODUCTION

Moderisation and fast food style habits has increased the prevalence of different diseases one among being head and neck cancer. The statics of cancer is estimated to be 22 million out of which 10 million cases are diagnosed every year. In a country between the races the cancer can show variations. Generally the lung cancer is the most common and breast cancer in females. With regard to head and neck cancer also shows a similar sex predilection. Males are attacked more than females with respect to oral cavity in particular[1].

The most common site for oral cancer is from lining buccal mucosa. The overall percentage being 5.5%. of all malignancies. The concept of precancer was given 1972 in WHO workshop[2]. But this concept of precancer was redefined in a workshop held in UK 2005 to potentially malignant disorders. These can transform into malignant lesions, by an another concept of Field cancerization given by Slaughter was explained[3].

All these lesions show some amount of histopathological changes which are consistent to oral squamous cell carcinomas. The severity of histopathological changes depend upon the cancerous cell invasion. The severity cannot be judged clinically by routine examination hence an early detection of these lesions are necessary which well documented.[4, 5]

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The different techniques and their significance is also well documented.[6-11] Bodily fluids are the most easily available eg. saliva and serum. Lab investigation using these do not need a complicated invasive procedure as is required in routine biopsy and also the contraindication for biopsy are also overlooked The importance of biological biomarkers and their significance in relation with dyspalsia is also documented.[12] Hence taking a muscle enzyme CPK as a biological marker a study was planned. CPK had shown its significance in different studies done by different authors.[5]

METHODS AND MATERIAL

Out patients visiting the department of Oral Medicine and Radiology, Coorg Institute of Dental Sciences Virajpet in the age range of 30-50 years old males. With smoking habit for more than 5 yrs. 20 subjects were selected and the lesion was subjected to histopathological examination and blood samples were collected and sent for biochemical analysis. All the demographic details were recorded along with dental status of patient. 20 Patients were selected after through clinical examination. A detailed case history was taken along with description of the lesion on the buccal mucosa. Only classical case of oral leukoplakia was included. The lesion was subjected to toluidine blue staning and thereafter biopsy and histopathological examination.

Inclusion criteria – All male patients of age 30-50 yrs having a smoking habit for more than 5 years were selected and patient willing for the study and treatment thereafter.

The Exclusion criteria were physically and mentally disabled and Patient with history systemic illness and patients not willing for biopsy.

Result - Mean CPK value of the control group was found to be 96.6 with a standard deviation of 27.82 and a standard error of 6.22. (Table 1, figure 1). Mean CPK of the cases was 102.25 with a standard deviation of 16.73 and a standard error of 3.74. (Table 1, figure 1). When the mean CPK of the cases are compared with the controls using student t test it was not statistically significant (p=0.441) (table 1, figure 1). Correlation between age and CPK value the correlation(r) was -0.083 which means a probable negative correlation and the P value was 0.611 which showed that it was not significant statistically.(figure 2 ,3).

Table 1 Mean and standard deviation

Group	Number	Mean	Standard deviation
Control	20	96.6	27.827
Case	20	102.3	16.739

Students t test ,P value =0.441 ,Not significant

Table 2 Correlation between Age and CPK value

Variable	Mean	Standard deviation
Age	39.8	5.331
CPK value	99.43	22.846

Correlation = -0.083 , p=0.611 not significant

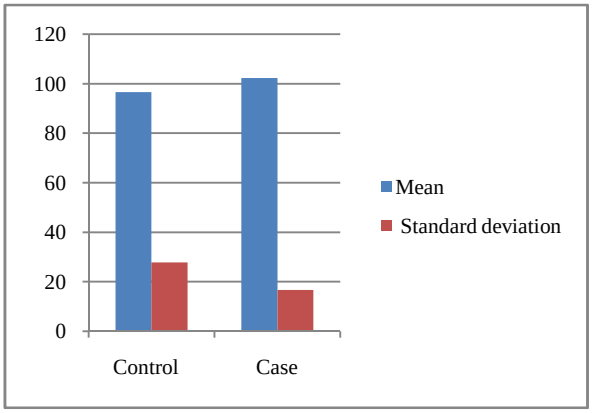

Figure 1 Mean CPK value in Case and Control

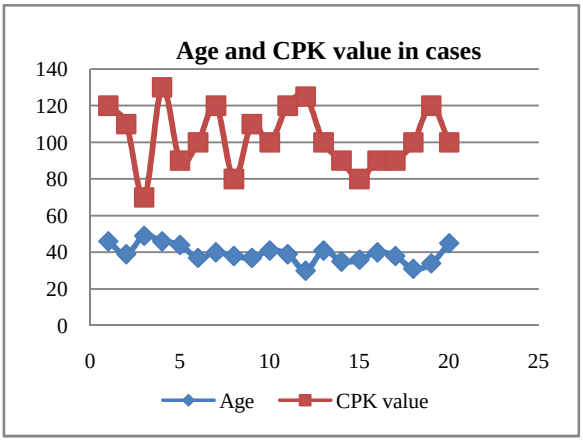

Figure 2 Age and CPK value in case.

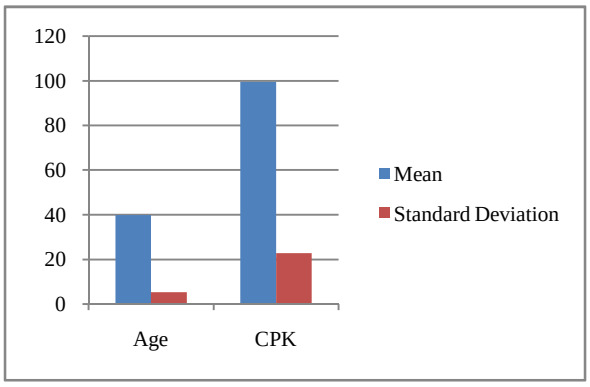

Figure 3 Mean of age and CPK in cases.

DISCUSSION

Oral Cancer like other cancers possess a great threat to life. It can leave a person debilitated for the remaining period of life and affecting a person's quality of life. The annual incidence of oral cancer is well documented. The etiological factors are different deleterious oral habits. The prevalence of potentially malignant disorders from these habits are well documented .[9] There are different studies done related to each of these lesions showing the habitual significance. A study done by Shyam *et al*, Surendran *et al* and Prasad *et al* showed the significance of smoking. They also mentioned the prevalence of theses lesions in their study along with other studies [13-15] When the

cells undergoes potentially malignant transformation then dysplastic features can be identified as described by Dost *et al* in their study[4] As degree of dysplastic changes denote the severity of the lesion for malignant transformation there are different methods for evaluating it other than the usual biopsy.[9,10]

Our study outlines an alternative method to the usual biopsy-histopathology procedure where in biopsy can have many contraindications. Our study makes use of muscle enzyme in the context that these lesions cause dysplastic in their later stage which involve the muscle layer damage.

Spoorthy *et al* has done a study on leukoplakia patients taking enzyme CPK.[16] Our study was centered to find out the influence of CPK on oral erythroplakia patients .Similar to this other studies were also carried out using micronutrients to find out their influence.[10] They have found out a positive correlation. The key factor could be that, the probable alteration in CPK value of serum is centred on damage to the muscle layer in the involved buccal mucosa in these lesions .Similar studies were done on hamsters to find out the influence of CPK on muscle damage in prostate , lung ,GIT cancers but got varying result showing an influence of this enzyme.[16] The influence of the CPK in muscle damage can be related to the fact that enzyme CPK is a constituent in muscle fibre and is released during a muscle damage which was stated in our previous study in Oral Submucous Fibrosis. However the exact cause for release of this enzyme is not clearly understood. The cause for muscle damage can be due to the overuse, trauma and local irritation due to carcinogens, all which can cause damage to fibrils leading to release of the enzyme.[17]

The overuse can be due to many deleterious habits like, pan chewing, betel nut chewing, areca nut chewing and smoking. These habits can cause damage to the oral mucosa by forming hyperkeratosis initially, and followed by invasion of deep layers on persisitent usage. The advanced clinical presentation would be like non healing ulcers for long time.

In our study we could not find a staticall significance between the CPK and oral erythrolekoplakia. The correlation between the age did not show much significance. The probable reason for the insignificance can be due to the damage done to muscle in oral erythroplakia could be minimal even though the lesion is active when compared to leukoplakia and OSMf.[16,17] This can be attributed to the different pathophysiology of Oral erythroplakia against OSMF where in there is muscle fibre fibrosis. Our study showed the correlation as 0.083 between age and CPK value which is negative. This shows that even though as the age increases there is no significant change in enzyme release in muscle when underlying mucosa and muscle fibres are involved in dysplastic changes. However in our previous study in OSMF there is considerable muscle damage but not in oral leukoplakia.[17] The statical insignificance of our study can be attributed to the altered pathophysiology of oral erytroleukoplakia whetrein this lesion does not show any fibrosis. In our study all the participants had lesion which showed mild dysplastic changes but this alone cannot be taken as marker for grading.[18] Other factor for statiscal insignificance can be due to small sample group . However with similar less study subjects the significance of CPK is shown in study done by Spoorthy *et al*[16]. This can be substantiated for the different cytokines released during the chronic irritation to the oral epithelium which in turn reflects its value in the serum, irrespective of the severity of muscle damage produced. However when CPK alone is taken the amount of muscle damage induced is of great importence. At this juncture it can be said that the amount of muscle damage caused in our many be sufficient for release of CPK.

The value is not constant but gets reduced as the muscle recovers from the injury .This type of sudden muscle damage is more seen in cardiac muscle, after a myocardial infarction.[17] Whatsoever when considereing enzyme CPK solely in cases of oral leukoplakia irrespective of grade of dysplasia the P value is not significant. This could be due mild dyspalsia. So much severe grades of dyspalsia or a carcinoma in situ might show an alteration in serum CPK value. With these factors it can be said that the study of oral leukoplakia lesions with respect t to CPK enzyme cannot be regarded as authenticated one until and unless a large volume of subjects with higher grade of dyspalsia is studied.

CONCLUSION

From this study it can be concluded that a definite correlation between CPK enzyme and oral leukoplakia lesions cannot be obtained as theit is dependent on different factors like the severity of lesion, grades of dysplasia and invasiveness of lesion etc. Furthermore a study with more samples might give some significance of CPK with oral leukoplakia lesions. However considering the few limitation in our study were the lesions size, grading of dyspalsia, duration, gender relation, individual physical activity which can be overcomed in a large multicentre trials.

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