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COGNITIVE DERANGEMENT IN SMOKELESS TOBACCO USERS.

Physiology

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ABSTRACT

Background: Tobacco use is a public health concern worldwide. Nicotine (highly addictive psychoactive drug), which is a main constituent of smokeless tobacco exerts multiple local and systemic effects. Chronic exposure from SLT has also been reported to cause neuro-muscular disease. Tobacco chewing may cause cognitive decline. It is associated with deficiency in executive function, cognitive flexibility, intellectual abilities learning and memory processing. However we don't have much literature available, so we planned to assess cognitive function in SLT users using MoCA test.
Objective: To assess cognitive function MoCA (Monteral Cognitive Assessment) tests in chronic tobacco chewers and to see its effect by increasing number of pouch year on cognition.
Method: The study was conducted in Department of Physiology, PGIMS, Rohtak. A total of 80 subjects in the age group of 25-50 years were included, and divided into two groups. Group I included 40 chronic tobacco chewers with history of minimum 2 pack years and group II were controls who didn't use tobacco in any form. Subjects with a history of any drug abuse, alcoholism, diabetes, hypertension, COPD or any neurological disease were excluded from the study. Further tobacco chewers were subdivided into 3 groups A, B and C based on pouch year. The tobacco chewers and the controls were subjected to MoCA test.
Results: The mean age of chewer was 31.52±6.5 years and control 30.5±4.9 years. The mean MoCA score was significantly decreased ($p<0.05$) in SLT users. Group C (tobacco chewer 10-15 pouch year) shows positive correlation with Group B and Group A indicating decrease in cognitive function with increasing number of pouch year.
Conclusion: Smokeless tobacco has deleterious effect on cognition. The deleterious effect on cognition increases with increasing number of pouch years in tobacco chewers.

KEYWORDS

Cognitive function, tobacco chewer

INTRODUCTION

An increasing awareness of the ill effects of smoking, has led people to contemplate whether chewing of tobacco is a safer alternative. Chewing tobacco is as bad or probably a lot worse. While in developed nations, use of cigarettes, cigars and pipes is more rampant; tobacco chewing forms a large component of nicotine addiction primarily in South East Asia.[1]

Multiple systemic effects of Smokeless tobacco chewing are due to the absorption of significant amount of nicotine. Nicotine also exerts a sympathomimetic effect on the cardiovascular system. Chronic exposure from SLT has been reported to cause neuromuscular disease manifested as a loss of muscle bulk, strength and endurance.[1] Nicotine present in all tobacco products, is well absorbed from mucosal surfaces, respiratory tract and skin resulting in local effects like irritation of oral mucosa, gum recession, exposure of neck of the tooth, and abrasion of the enamel surface. Chronic inflammation initiates epithelial dysplasia of buccal mucosa, eventually resulting in leukoplakia and many other premalignant lesions like lichen planus, lichenoid lesions, erythroplakia etc.[2]

However its effect on cognition is still in question.

Hence, in our study we have used MoCA(Montral Cognitive Assessment) test to assess cognitive functions in chronic tobacco chewers .

MATERIAL AND METHOD

Study design

The study was conducted in the Department of Physiology, PGIMS, Rohtak. A total of 80 male volunteers of age group 25-50 years were included in the study. The subjects were selected from the labourers (and their relatives) coming to PGIMS, Rohtak. Informed consent was obtained from all the participants and the study protocol was approved by Institutional Ethical Committee.

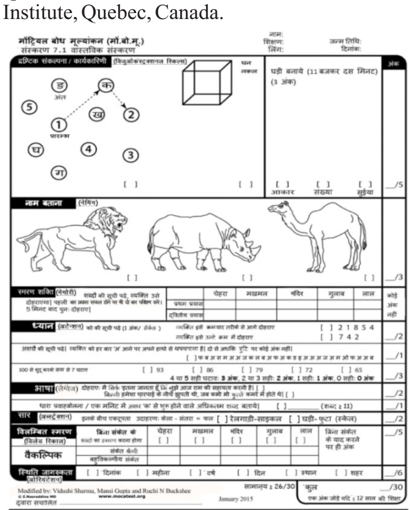
The subjects were selected with minimum literacy level upto primary classes and similar socioeconomic background. Subjects with a history of any chronic illness like diabetes mellitus, hypertension, COPD, liver disease or any drug abuse were excluded from the study. We included the tobacco chewers having chewing history of minimum 2 pouch years. Pouch year were calculated considering (i) total years chewed, and (ii) daily consumption.[3]

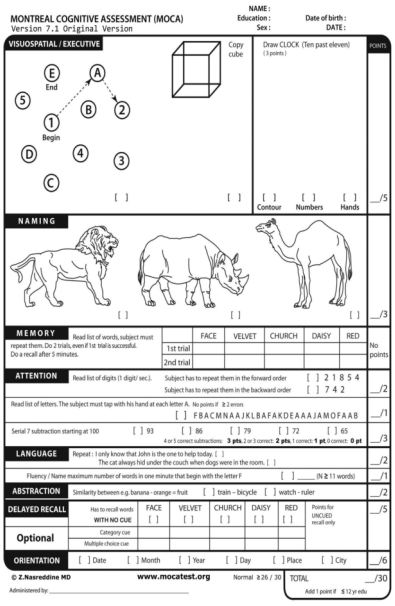
Further sub grouping were done on the basis of number of pouch years in chewers or group I- Group A(< 5 pouch years), Group B(6-10 pouch years) and Group C(> 10 pouch years).

Assessment of cognitive function

Montreal Cognitive Assessment (MoCA)

To assess cognitive function, Montreal Cognitive Assessment (MoCA) was used. The MoCA test is a one-page, 30-point test administered in approximately 10-15 minutes. The MoCA assesses various cognitive domains. A score below 26 points indicates mild cognitive impairment. The proforma for the test is attached both in English and Hindi. The permission to use this test has been obtained from MoCA Clinic and Institute, Quebec, Canada.





Statistical analysis

Data was collected and analyzed using SPSS v. 20, and is presented as mean ± standard deviations. To assess the significance of the differences between chewers and controls, the student t- test was applied. The strength of correlations between variables was assessed using Pearson's correlation coefficient and its statistical significance with t distribution test. A p- value of <0.05 was considered the threshold for statistical significance.

RESULTS

Table 1: Comparison of different Anthropometric parameters among chronic chewers and controls

Variable	Chewers	Controls
Age (years)	31.52±6.5	30.5±4.9
Height (cm)	164.33±52	165.07±5.95
Weight (kg)	62.2±7.3	63.47±8.80
BMI (kg/m^2)	22.74±1.96	23.31±3.23

Fig 1 shows comparison of anthropometric measures among chewers and controls.

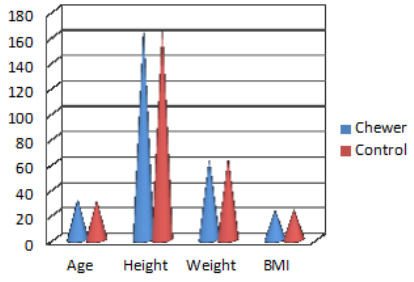


Table 2

Parameters	Group 1 (chewers) Mean ±SD	Group 2 (control) Mean ±SD	P value
VISUOSPATIAL	3.82±1.15	4.12±1.04	0.2
ATTENTION	5.35±0.80	5.72±0.55	0.011*
LANGUAGE	1.075±0.94	1.25±86	0.42
ABSTRACT	1.12±0.51	1.42±0.59	0.02
DELAYED RECALL	4.05±0.81	4.57±0.54	0.001*
ORIENTATION	5.95±0.31	6±0	0.32

*p<0.05= Significant
**p<0.001=Highly significant

Table 3: Comparison of MoCA Score in chronic tobacco chewers and controls.

Parameters	Chewer	Control	P value
MoCA cumulative score	24.45±2.5	26.15±1.83	0.0008*

*p<0.05= Significant
**p<0.001=Highly significant

The mean MoCA score in chewer is 24.45±2.5 and that for control is 26.15±1.83. The mean MoCA score is significantly reduced in chewers (p<0.05).

Fig: 2 shows comparison of MoCA score in chewers and controls

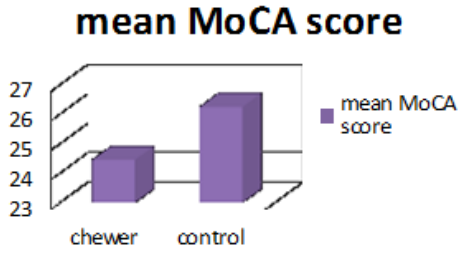


Table 4: MoCA score among subdivisions of tobacco chewers on the basis of pack years

	Group A(<5 pouch years)	Group B(6-10 Pouch years)	Group C(11-15 pouch years)	P value
MoCA score	27.07±1.03	24.53±0.96	21.92±2.09	0.000*

Table 5: Post hoc test

GROUP	P value
Group AVs Group B	0.000*
Group B Vs Group C	0.000*
Group C Vs Group A	0.000*

Table 6: Correlation between Group A, Group B and Group C

Group	Correlation co-efficient
A and B	0.86
B and C	0.20

Table 7: Prevalence of Mild cognitive impairment (MCI)

	Chewers	Controls
MoCA score	22/40= 55%	13/40=32.5%

DISCUSSION

In India, khaini or tobacco lime mixtures (12%) is the most commonly used smokeless tobacco products, followed by gutkha, a mixture of tobacco, lime and areca nut mixture (8%), betel quid with tobacco (6%) and applying tobacco as dentifrice (5%). Smokeless tobacco (SLT) products such as snuff and chewing tobacco contains high level of nicotine, which reaches to the lungs through the skin and mucosal lining of the mouth and nose. Nicotine reaches peak levels in the bloodstream and brain rapidly depending upon which form it is consumed.[4] Gaede et al (1941) found 0.6gm of tobacco contained 15 mg of nicotine, of which 33% was absorbed in ½ an Hour after chewing; 50% at 2Hours; 60% at 4Hours and 90% at 8Hours.[5] In addition to nicotine, smokeless tobacco also contains nitrosamine, sodium, glucose, glycyrrhizinic acid and grit.[2]

The deleterious effects of chronic tobacco chewing on human cardiac [6] and pulmonary function [7], peripheral vascular systems [6] and its carcinogenic properties [8] have already been emphasised in literature. Chronic tobacco chewing is associated with alteration in blood lipid profile, resulting in atherogenic risk factor primarily low HDL-C. Nicotine, present in the tobacco, causes lipolysis and increases free fatty acids content leading to overproduction of VLDL-C; Total triglycerides; LDL-C and Lowered HDL-C.[9] Devendra et al and Mukherjee et al observed that chewing tobacco users have significantly higher level of Fasting blood sugar than in control group. They proposed that nicotine present in the tobacco may hamper the maintenance of blood glucose level possibly by lowering insulin sensitivity and the adiponectin which is involved in the maintenance of blood glucose levels.[10, 11-13] Lowering of adiponectin has been linked to the possibility of developing type 2 diabetes.[14] Smokeless tobacco use is also considered a potential cause of sodium retention and so it has a poor blood pressure control because of its nicotine, sodium, and licorice content. In some experimental trials it is found that SLT causes significant elevation in systolic as well as diastolic blood pressure. It may elevate blood pressure up to 90 minutes after use.[15] Chronic exposure from SLT has been reported to cause neuromuscular disease manifested as a loss of muscle bulk, strength and endurance.[8,16] Recently, Gupta et al studied the effects of tobacco chewing on

cardiovascular and autonomic functions and found imbalance of autonomic activity in tobacco chewers.

In the paucity of literature about the cognitive function in smokeless tobacco users, we have evaluated cognitive function using Montreal cognitive assessment (MoCA) as a clinical test in chronic tobacco chewers. In the present study, we observed significant decrease ($p<0.05$) in the mean MoCA score indicating mild cognitive impairment in tobacco chewers. On assessing the different cognitive domains there was significant reduction in memory and abstract reasoning, which is often reduced in cases of dementia. Jose Contreras Vidal stated that tobacco use on the job can reduce an individual capability to learn new visuomotor mapping and adapt his or her performance to new visual feedback. His research was supported by Smokeless Tobacco Research Council.[17] He observed that chewing tobacco or consuming nicotine in any form makes it harder for people to perform complex tasks involving hand and eye coordination. Tobacco chewers proved slower learning and adjusting to changing visual patterns on a computer screen. They also exhibited slower, jerkier movements and were less accurate overall than non tobacco users.[18]

We may attribute our findings of cognitive derangement in tobacco chewers to Smokeless tobacco product which induces oxidative stress resulting in imbalance between formation of reactive oxygen species and antioxidants. Antioxidant rich foods such as green-leafy vegetables and fruits reduce the oxidative stress caused by tobacco. Usually it is seen that the subjects, who are chronic tobacco chewers are economically poor and also because of lack of antioxidant rich diet, makes them more vulnerable to tobacco-induced oxidative stress.[19]

Smokeless tobacco may also increases the risk of dementia through cardiovascular disease related- mechanisms.[20] As discussed earlier, smokeless tobacco causes altered lipid profile, which can cause atherosclerosis, leading to narrowing of vessels and deprive brain cells for proper perfusion, nutrient supply and by- product exchange forming a dependent risk factor for cognitive impairment and dementia.[20]

CONCLUSION

Smokeless tobacco has deleterious effect on cognition. MoCA is an efficient tool to assess cognitive function subclinically. The deleterious effect on cognition increases with increasing number of pouch years in tobacco chewers. It is of great importance that the masses can be educated even to quit SLT.

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