

INFLUENCE OF CHRONIC CIGARETTES SMOKING ON SERUM TESTOSTERONE AND PROLACTIN LEVELS AMONG SUDANESE SMOKERS

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ABSTRACT

Objective: to estimate testosterone and prolactin levels in chronic Sudanese smokers regardless of their fertility functions.

Materials and methods: the study was conducted at Omdurman area-Khatoum state-Sudan during the period from December 2012 to March 2013, it involved a control group of apparently healthy non smokers (N=50) matched for age with a test group of smokers (N=50). The range of both groups was 20-60 years old. Serum testosterone and prolactin levels were measured in Omdurman Military hospital by using ELISA full automated human analyzer (Human-Germany). Appropriate statistical tests were used to assess significant differences in the means of the studied concentrations between smokers and control group.

Results: The smokers showed lower levels of testosterone [M±SD = 3.0±1.9ng/ml] compared with control group [M±SD = 6.1±2.8ng/ml], this difference was considered statistically significant [P = 0.03 which is less than 0.05]. While there is no significant differences in prolactin levels in smokers [M±SD = 7.3± 2.0ng/ml] compared with control group [M±SD = 7.6±1.8ng/ml P = 0.83 which is higher than 0.05].

Conclusion: The study added further evidence for possible harmful consequence of smoking on lowering the testosterone levels which will affect on male mass muscles as much as male fertility functions in the future.

Key words: Cigarette, nicotine, respiratory, cardiovascular, testosterone, prolactin

INTRODUCTION

Smoking is the most common method of consuming tobacco (1), the resulting vapors are then inhaled and the active substances (nicotine)

absorbed through the alveoli in the lungs and trigger chemical reactions in nerve endings (2), Dopamine and epinephrine are released, which are often associated with pleasure (3).

The hazards of cigarette smoking are well recognized worldwide (4). The World Health Organization (WHO) estimates that tobacco caused 5.4 million deaths in 2004 about 100 million deaths over the course of the 20th century. Yet significant numbers of people continue to smoke in developing countries (5).

Cigarette smoking is a known risk factor for many diseases such as respiratory, cardiovascular, and neoplastic and also affects the male's fertility functions (6-9). Nicotine it acts as vasoconstrictor (10), which means it cause blood vessels to become narrower and reduces blood flow. This constriction is sometimes to blame for men's reduced sexual arousal. Furthermore, erectile dysfunction resulting from smoking can be difficult to treat when caused by damage to blood vessels (11;12).

Smoking has multiple effects on hormone secretion, some of which are associated with important clinical implications (13).

Nicotine from cigarette smoking increases circulating levels of cortisol, growth hormone and prolactin in male chronic smokers (14).

This study aims to estimate testosterone and prolactin levels in chronic Sudanese smoker regardless of their fertility functions.

MATERIALS & METHODS

The study involved two groups: a control group of apparently healthy non-smokers (N= 50) matched for age with a test group of smokers (N=50). The age range of both groups was 20-60 years. All volunteers

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were recruited from Omdurman area –Khartoum state-Sudan during the period from December 2012 to March 2013. The study was approved by Alneelain University Ethics Committee and all subjects gave informed consent (Based on Helsinki Declaration).

Venous blood samples (5.0 ml) were collected from each volunteer in appropriate containers. Testosterone and prolactin level were measured using Fortress Diagnostic Reagents (UK), and determined using ELISA full automated human analyzer (Human-Germany).

Statistical evaluation was performed using the Microsoft Office Excel for windows; (2007) and SPSS for windows version (19). Normal distribution of the studied variables was examined using kolmogrov-smirnova and Shapiro-wilk tests were used to assess significant difference in the means of the studied variables in smokers and non-smokers. Correlation between serum testosterone level and duration of smoking were assessed using bivariate correlation. $P < 0.05$ was considered statistically significant.

RESULTS

The age of smokers [$M \pm SD = 33.8 \pm 10$] years was comparable with control group [$M \pm SD = 34.9 \pm 10.1$] years old. The $M \pm SD$ of the duration of the smoking = [12.7 ± 9.4] years. The testosterone levels were lower in smokers [$M \pm SD = 3 \pm 1.9 \text{ ng/ml}$] compared with control group [$M \pm SD = 6.1 \pm 2.8 \text{ ng/ml}$], this difference was considered statistically significant [$P = 0.03$ which is less than 0.05]. While there is no significant differences in prolactin levels in smokers [$M \pm SD = 7.3 \pm 2 \text{ ng/ml}$] compared with control group [$M \pm SD = 7.6 \pm 1.8 \text{ ng/ml}$ $P = 0.83$ which is higher than 0.05].

Finally there was negative correlation between the duration of smoking and concentration of testosterone ($CC = -0.291, P = 0.006$). However there was no significant correlation between duration of smoking and prolactin levels ($CC = -0.091, P = 0.16$).

DISCUSSION

Despite considerable amount of research devoted to study on the effects of cigarette smoking on reproductive system, in this study we concern about the effects of chronic smoking on testosterone

Table-1: the mean of prolactin and testosterone levels in studied group

Parameter	Study Group (n=100)		95% confidence interval for mean in smokers group		P.value
	Smokers (n =50)	Control (n =50)	Lower	Upper	
	Mean±SD	Mean±SD			
Age (years)	33.8±10	34.9±10.1	20	60	$P = 0.53$
Prolactin (ng/ml)	7.3±2	7.6±1.8	1.8	17	$P = 0.83$
Testosterone (ng/ml)	3.0±1.9	6.1±2.8	2.5	10	$P = 0.03$

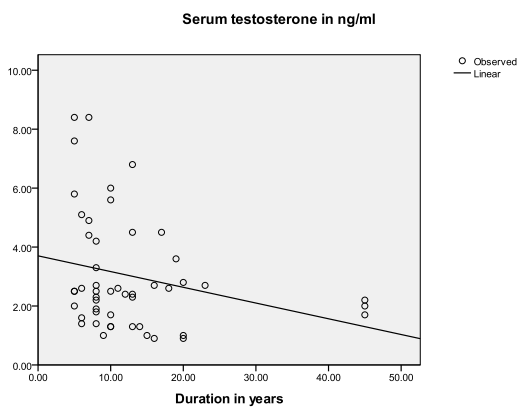


Figure 1. Correlation between duration of smoking and testosterone level (CC = -0.291, P = 0.006)

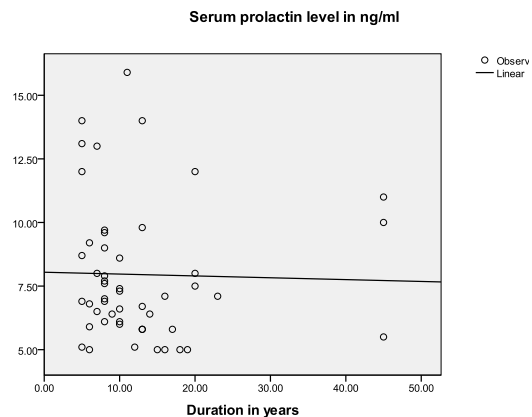


Figure 2. Correlation between duration of smoking and prolactin level (CC = -0.091, P = 0.16).

and prolactin hormones which were considered as part of the main causes of endocrine related infertility in male(15;16).

Recently there is interest in studying the association between low androgen levels and the metabolic syndrome, and coronary heart disease (17). Various studies examining the effects of smoking on serum testosterone levels have reported conflicting findings largely due to difficulties in the hormonal assays. Testosterone circulates into two forms, the major is bind to sex hormone binding globulins (SHBG) which represents about (65-80 %) of the total, and free form. Thus levels of total testosterone can be affected by changes in the levels of SHBG and other plasma proteins (18). The SHBG and free testosterone levels have been measured in few studies and are reported to be higher among smokers (19;20). The increase of total testosterone can be explained by the effect of nicotine on SHBG synthesis which is positively correlates with nicotine level(21). Significantly decreased levels in smokers was also reported (22), this is in accordance with our finding. It would seem likely that the effects of smoking on testosterone levels are due to changes in plasma-binding capacity rather than a direct effect of nicotine on testosterone. However it was reported by Svartberg et al., that there is a positive association between testosterone and smoking even after adjusting of SHBG level (23).

The results of the recent study revealed lower levels of prolactin in smokers compared with non-smokers, but this decrease did not a achieve statistical significant values. This finding confirmed what had been reported previously (24).

Nicotine was shown to down regulate prolactin gene expression so baseline prolactin levels are thus lower in chronic smokers than non-smokers (24). Suggest that the reduced serum PRL levels that result from smoking may originate in part from decreased transcription of the PRL gene resulting from a direct effect of nicotine on pituitary PRL-secreting cells.

This study limited by the fact that the current results uncover some of detrimental effects of smoking on male infertility causes such as the effects of nicotine and nicotinic receptors in a differential manner hypothalamic – pituitary - gonadal axis, sex steroid hormones metabolism and prostate or seminal vesicle functions which are influenced by cigarette nicotine (25).

CONCLUSION

From the results of the present study, it may be concluded that, cigarette smoking during adolescent period may induces disturbance in fertility state, abnormal low testosterone level, so it is strongly recommended to avoid smoking for the benefit of reproductive health.

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