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EFFECT OF CHRONIC CIGARETTES SMOKING ON SERUM TESTOSTERONE, LUTINIZING HORMONE AND PROLACTIN LEVELS AMONG SUDANESE SMOKERS

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ABSTRACT

Cigarette smoking is highly prevalent among men. Many studies have evaluated the effect of cigarette smoking on levels of male reproductive hormones; however, the findings still remain controversial. The main objective was to study the effect of cigarette smoking on Luteinizing, Prolactin and Testosterone hormones in male. Hundred blood samples were collected from apparently healthy Sudanese individuals, smoker (N= 50) and non smoker as control (N=50). All volunteers were recruited from Omdurman area- Khartoum- Sudan. The samples measured using ELISA full automated hormone analyzer (Human-Ouno, Germany). This study showed that Luteinizing hormone levels among smokers and non-smoker were (mean ± SD 3.16±1.09 ng/ml, and 3.17±1.23 ng/ml), respectively with no significant difference between means*P* =>0.05), also it showed 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). While there is significant lower levels in testosterone [M±SD =3.0±1.9ng/ml] compared with control group (M±SD = 6.1±2.8ng/ml), *P* = 0.03. The study concluded that there was significant low level of testosterone in smokers compared to non-smokers, while there were no significant differences on serum levels of Luteinizing Hormone and prolactin.

INTRODUCTION

The health consequences of cigarette smoking and of the use of other tobacco products are well known. They are an important cause of increased mortality and morbidity in developed countries and the prevalence is increasing in the developing world as well [1].

Smoking has an effect on the various metabolic and biological processes in the body including secretion of hormones. Cigarette smoking has major effects on the reproductive potential of humans; it has anti-estrogenic effect in women. In males, the effect of smoking is on androgen levels; given the recent interest in the association between androgen levels and metabolic syndrome and coronary heart disease [2].

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Impotence also known as erectile dysfunction which characterized by men's inability to achieve or maintain an erection [3]. If he is a cigarette smoker, nicotine may be the culprit since it acts as vasoconstrictor (4) 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 vessel [4, 5].

Cigarette smoking stimulates the release of several anterior and posterior pituitary hormones [6]. Smoking acutely increases the plasma levels of prolactin, adrenocorticotrophin (ACTH), growth hormone (GH) and arginine vasopressin (AVP) without significant changes in TSH, luteinizing hormone (LH) and follicle-stimulating hormone (FSH). These effects are directly proportional to the nicotine content of cigarettes, with greater hormonal responses observed in high content cigarettes [7].

Cigarette smoking is a known risk factor for many diseases such as respiratory, cardiovascular, and neoplastic and also affects the male's fertility functions.

Studies show that various hormones including sex steroid hormones can modulate 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 are influenced by cigarette nicotine [8]. Changes at cellular and molecular levels in various tissues including reproductive tissues have also been reported following cigarette smoking [9].

High nicotine cigarette smoking stimulated significant increases in LH release that were temporally correlated with increases in plasma levels of nicotine [10].
In the male, LH acts upon the Leydig cells of the testis and is responsible for the production of testosterone, an androgen that exerts both endocrine activity and intratesticular activity on spermatogenesis [11].

The release of LH at the pituitary gland is controlled by pulses of gonadotropin-releasing hormone (GnRH) from the hypothalamus. Those pulses, in turn, are subject to the estrogen feedback from the gonads.
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 [12].

Prolactin is the main hormone involved in the formation and production of milk. It is secreted by the anterior pituitary gland.

The hormonal response to cigarette smoke inhalation has been studied with somewhat equivocal results in the recent past. Under some condition activity of the adrenal cortex and the adrenal medulla, and secretion of growth hormone, gonadotropin and testosterone have all shown some response to cigarette smoke [13]. The result appear to be conflict, we therefore, decided to restudy the acute endocrine effects of cigarette smoking under controlled conditions. The aim of this study is to evaluate the effects of chronic smoking on the LH, Prolactin and Testosterone levels in men.

MATERIALS AND METHODS

This study is a descriptive cross sectional laboratory based study. It was conducted during the period from March to May 2013 in Altegana private laboratory Khartoum-Sudan.

Subjects: 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 were recruited from Omdurman area – Khartoum – Sudan. The study procedures were explained and any questions or concerns were discussed.

Sample collection procedure: Venous blood samples (5 *ml*) for hormone analysis were collected in Vacutainer tubes without preservative. All samples were immediately centrifuged, and serum was removed.

Assay Procedures: Serum LH, Prolactin and Testosterone Assay were determined by ELISA Full automated Human Ouno machine using Kits (Fortess reagents)

Data Analysis: Statistical evaluation was performed using Microsoft office excel for windows 2007 and SPSS (SPSS for windows version 17). Unpaired T-test was used to assess significant difference in the means of the studied variables in smokers and non-smokers. Correlations between serum LH, Testosterone & Prolactin and the duration of smoking were assessed using bivariate correlations. $P < 0.05$ was considered statistically significant

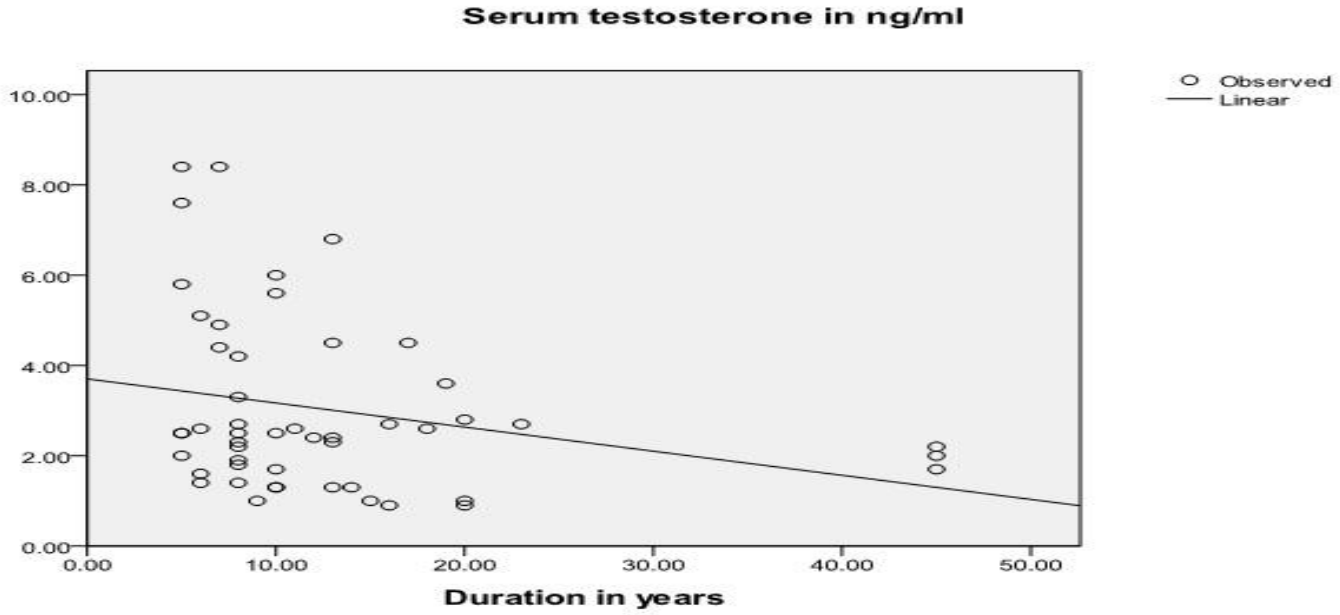
RESULTS

The study of population included 100 men, with age of (20 -60) years. Fifty people were heavy smokers and fifty non- smokers. Baseline data of the study groups are shown in (Table .1). The testosterone levels were lower in smokers [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]. While No significant differences were noticed in LH level (M±SD 3.16± 1.09 ng/ml, 3.17±1.23 ng/ml) and prolactin level [M±SD = 7.3±2 ng/ml, 7.6±1.8 ng/ml] among smokers and non smokers respectively at p>0.05), also the correlation analysis showed significant negative correlation between duration of smoking and testosterone levels, CC = -0.291, P = 0.006).

Table 1.LH,testosterone and prolactin levels (ng / ml) at both smokers and non – smokers

Parameter	Study Group (n=100)		95% confidence interval for mean in smokers group		P.value
	Smokers (n =50) Mean±SD	Control (n =50) Mean±SD	Lower	Upper	
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
LH (ng/ml)	3.16± 1.09	3.17±1.23	1.1	7	*P* = 0.489

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



DISCUSSION

Cigarette smoking is an important modifier of hormones and a detailed smoking history is essential when assessing patients with endocrine disorders. The direct toxic effect of environmental toxins present in cigarette smoking which contains a lot of known toxins that may have detrimental effects on fertility in both sexes [14]. Nicotine was shown to down regulate prolactin gene expression so baseline prolactin levels are thus lower in chronic smokers than non-smokers, this finding as similar as our result but this decrease did not a achieve statistical significant values. In our study, the data indicates that tobacco smoking has no significant effect on the biological active function of LH in smokers compared with non-smokers group which was reported before. A similar also study done on rats that resulted in serum level of LH was not significantly changed, while, other researchers have observed a positive significant correlation between LH level and tobacco smoking, this may be due to the time factor of sample collection, LH levels increased significantly within 14 min after high nicotine cigarette smoking and it remain higher than baseline for 40 min, but after that LH levels did not changed significantly from baseline [15]. In few studies, sex hormones bindingglobulins (SHBG) and free testosterone levels, have been measured and are reported to be higher among smokers [16, 17]. Nicotine has an effect on SHBG synthesis that lead to increase of total testosterone which is positively correlates with nicotine level. but others reported that there is a significantly decreased level in smokers, this is in accordance with our finding [18]. 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 that there is a positive association between testosterone and smoking even after adjusting of SHBG level [19].

CONCLUSION

Tobacco smoking has no significant effect on levels of LH and prolactin, but testosterone level is significantly decreased in smokers when compare with non smokers.

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REFERENCES

1. Al-Azzawy A. (2011). The Impact of Cigarette Smoking on Levels of Sex Hormones and Zinc in Blood of Smokers. *ibn al- haitham j. for pure & appl. Sci*, (24), 2.
2. Morris PD, Channer KS. (2012). Testosterone and cardiovascular disease in men. *Asian J Androl*, 14(3), 428-35.
3. Harte CB, Meston CM. (2008). Acute effects of nicotine on physiological and subjective sexual arousal in nonsmoking men: a randomized, double-blind, placebo-controlled trial. *J Sex Med*, 5(1), 110-121.
4. Black CE, Huang N, Neligan PC, Levine RH, Lipa JE, Lintlop S, et al. (2001). Effect of nicotine on vasoconstrictor and vasodilator responses in human skin vasculature. *Am J Physiol Regul Integr Comp Physiol*, 281(4), 1097-1104.
5. Tostes RC, Carneiro FS, Lee AJ, Giachini FR, Leite R, Osawa Y, et al. (2008). Cigarette smoking and erectile dysfunction: focus on NO bioavailability and ROS generation. *J Sex Med*, 5(6), 1284-1295.

6. Mello NK and Mendelson JH. (2002). Cocaine, hormones and behavior: clinical and preclinical studies, Hormones, Brain and Behavior. *Academic Press: New York*, 665–745.
7. Kapoor D, Jone TH. (2005). Smoking and hormones in health and endocrine disorder. *Eur J Endocrinol*,152(4), 491-499.
8. Moreira DM, Antonelli JA, Presti JC, Jr., Aronson WJ, Terris MK, Kane CJ, et al. (2010). Association of cigarette smoking with interval to biochemical recurrence after radical prostatectomy: results from the SEARCH database. *Urology*, 6(5), 1218-1223.
9. Heidary F, Ahmadi R and Lotfi A. (2012). The Effects of Cigarette or Hookah Smoking on Serum Levels of LH, FSH or Testosterone in Male Rats. *International Conference on Medical, Biological and Pharmaceutical Sciences (ICMBPS) Bangkok*, 17-18.
10. Mello NK. (2010). Hormones, nicotine, and cocaine: clinical studies. *Horm Behav*, 58(1), 57-71.
11. Welsh M, Saunders PT, Atanassova N, Sharpe RM, Smith LB. (2009). Androgen action via testicular peritubularmyoid cells is essential for male fertility. *FASEB J*, 23(12), 4218-4230.
12. Chin KY, Soelaiman IN, Mohamed IN, Ngah WZ (2012). Serum testosterone, sex hormone-binding globulin and total calcium levels predict the calcaneal speed of sound in men. *Clinics (Sao Paulo)*, 67(8), 911-916.
13. Winternitz WW and Quillen D. (1977). Acute hormonal response to cigarette smoking. *J ClinPharmacol*, (17), 389–397.
14. Trummer H, Habermann H, Haas J, Pummer K.(2002). The impact of cigarette smoking on human semen parameters and hormones. *Hum Reprod*, 17(6), 1554-1559.
15. Mendelson JH, et al. (2003). Effects of intravenous cocaine and cigarette smoking on luteinizing hormone, testosterone, and prolactin in men. *J Pharmacol Exp Ther*, 1, 29-134.
16. English KM, Pugh PJ, Parry H, Scutt NE, Channer KS, Jones TH. (2001). Effect of cigarette smoking on levels of bioavailable testosterone in healthy men. *Clin Sci (Lond)*, 100(6), 661-665.
17. Field AE, Colditz GA, Willett WC, Longcope C, McKinlay JB. (1994). The relation of smoking, age, relative weight, and dietary intake to serum adrenal steroids, sex hormones, and sex hormone-binding globulin in middle-aged men. *J Clin Endocrinol Metab*, 79(5), 1310-1316.
18. Shaarawy M, Mahmoud KZ. (1982). Endocrine profile and semen characteristics in male smokers. *Fertil Steril*, 38(2), 255-257.
19. Svartberg J, Midtby M, Bonaa KH, Sundsfjord J, Joakimsen RM, Jorde R. (2003). The associations of age, lifestyle factors and chronic disease with testosterone in men: the Tromso Study. *Eur J Endocrinol*, 149(2), 145-152.
20. Arabi M, Shareghi B. (2005). Anti-fertility effect of nicotine. *Zhonghua Nan Ke Xue*, 11(5), 323-330.
21. Ambrose JA, Barua RS. (2004). The pathophysiology of cigarette smoking and cardiovascular disease: an update. *J Am Coll Cardiol*, 43(10), 1731-1737.
22. Spira A, Beane J, Shah V, Liu G, Schembri F, Yang X, et al. (2004). Effects of cigarette smoke on the human airway epithelial cell transcriptome. *Proc Natl Acad Sci U S A*, 101(27), 10143-10148.
23. Mahadeva R, Shapiro SD. (2002). Chronic obstructive pulmonary disease, Experimental animal models of pulmonary emphysema. *Thorax*, 57(10), 908-914.