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Effect of obesity on some reproductive hormones in Iraqi men

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Abstract

This study was conducted to investigate the relationship between obesity or high body mass index (BMI) and some reproductive hormones in Iraqi men, during the period from February 2012 to July 2012. Subjects were exposed through medical history evaluation. Serum was collected from 80 Iraqi men who involved in this study, the men were divided into two groups .One group was considered as control (BMI of < 30). The other group was considered as test group (BMI of >30). BMI was calculated and serum follicle stimulation hormone (FSH), luteinizing hormone (LH), testosterone (T), prolactin (PRL), estrogen (E2) was estimation. The results revealed significant (P<0.05) increase in mean BMI, serum E2, FSH, and LH and significant decrease in serum T in test groups compared with control group. Serum PRL was significantly (P<0.05) unaffected between two groups. In conclusion, the association explored between BMI and some reproductive hormones, as well as different patterns of this association between obese and non-obese Iraqi men, will be of help to broaden our understanding of the effect of obesity on some male reproductive physiologic characteristics.

Keywords: Obesity, Body mass index, Reproductive hormones, Iraqi men.

Introduction

Obesity is considered now as an epidemic disease that is rapidly progressing in developed and underdeveloped world. The effects of obesity not only relate to chronic medical conditions but also have been strongly related to reproductive problems (Must *et al.*, 1999; Clark *et al.*, 1998). The obese male is generally characterized as having greater than 25% body fat of total body mass with a BMI in excess of 30Kg/ m^2 (Oken and Gillman, 2003). BMI is used as the chief indicator of obesity, with stratified BMI categories as follows: 18.5-24.9Kg/m^2 (normal), 25Kg/m^2 and above (overweight), 30Kg/m^2 (obese) (Pauli *et al.*, 2008). Nguyen *et al*. (2007) reported that excess weight has been related directly or indirectly to biologic changes that could reduce male infertility. In addition, obesity was demonstrated to be associated with a significant disturbance in the hormonal milieu. Several reports showed that obese men exhibit significant decrease in androgen and sex hormone - binding globulin (SHBG) levels and significant elevated in E2 levels, correlating with the degree of obesity (Hammoud *et al.*, 2008; Magnusdottirm *et al.*, 2005). Tchernof and Després (2000) showed that, sex steroid hormones in males have been closely related to the regulation of adiposity, either through direct or indirect physiological mechanisms. Chavarro, *et al.* (2010) suggested that body mass index (BMI) was positively related to estradiol levels and inversely related to total testosterone and sex hormone-binding globulin (SHBG) levels. There was also a strong inverse relation between BMI and inhibin B levels and a lower testosterone LH ratio among men with a BMI > or = 35kg/m (Derby *et al.*, 2006). Obesity was shown to affect the GnRH-LH/ FSH pulse that may impair Leydig and Sertoli cell function and interfere with the release of sex hormones with consequent effect on sperm maturation (Belanger *et al.*, 2002). Potential effects of increased body mass index (BMI) in Iraqi men on male fertility have not been subjected to the same degree of research as Iraqi female. Therefore, this study was designed to evaluate whether plasma levels of some reproductive hormones are primarily related to obesity in Iraqi men.

Materials and Methods

This retrospective study conducted from different regions of Baghdad during the period from February 2012 to July 2012. We studied the medical history evaluation of the participants. A total of 80 Iraqi men were collected and divided into two groups: First group was considered as control (BMI of < 30) .While, the second group was considered as test group (BMI of >30). BMI was calculated dividing weight in kilograms by squared height in meters (kg/m^2). To analyze the data, the BMI was categorized as < 20.0kg/m^2 labeled as underweight, 20.0–25.0kg/m^2 labeled as normal weight, 25.1–

30.0kg/m^2 labeled as overweight, and >30.0kg/m^2 labeled as obese. (WHO, 2012). Venous blood samples (5 ml) were withdrawn from each participant and centrifuged, the resulting sera were preserved at −4C°. Serum FSH, LH and PRL levels were estimated by the ELISA method (Diagnostics Systems Laboratories, Webster, Tex, USA). Serum total T and E2 levels were estimated by enzyme immunoassay (Diagnostics Systems Laboratories). Data were analyzed using SPSS (IBM, Armonk, NY, USA) version 16 statistical software. Mean serum sex-hormone levels of FSH (mIu/ml), LH (mIu/ml), E (Pg/ml), PRL (Ng/ml), and testosterone (Ng/ml), using Student's T-test to compare values from test and control groups. Differences between values were considered significant at (P<0.05).

Results and Discussion

The present study showed that the mean BMI in test group was significantly (P<0.05) higher than in control group (Figure 1). BMI is used as the chief indicator of obesity. In this present study, we showed that obesity is associated with an altered hormonal profile by significant decrease (P<0.05) in T level and significant increase (P<0.05) in serum LH, FSH and E2 levels (Table 1). Hofny *et al*. (2010) reported that BMI had significant positive correlation with LH and non-significant correlation with serum FSH in obese infertile males compared with obese fertile males. On the other hand, Jensen *et al*. (2004) reported that BMI had no effect on serum FSH or LH in men. Nguyen *et al*. (2007) showed that excess weight has been related directly or indirectly to biologic changes that could reduce male infertility. In addition, obesity was demonstrated to be associated with a significant disturbance in the hormonal milieu, which can affect the reproductive system. Obesity was shown to affect the GnRH – LH / FSH pulse that may impair Leyd and Sertoli cell function and interfere with the release of sex hormones with consequent effect on sperm maturation (WHO, 2012).

Adipose tissue has endocrine function and synthesize chemical messenger known as adipocytokines or adipocyte - derived hormones, in addition to the aromatization of androgens to estrogens. Basal aromatase activity decreases during adipocyte maturation with tenfold less aromatase activity in mature adipocytes compared to preadipocytes (McTernan *et al*., 2002). Tchernof and Després (2005) showed that, sex steroid hormones in males have been closely related to the regulation of adiposity, either through direct or indirect physiological mechanisms. Adipocyte derived estrogen in obese men provide feedback inhibition to hypothalamic – pituitary – testicular axis (HPT axis) modulated by the presence of estrogen receptors localized to the hypothalamus and pituitary shown in mouse (Couse *et al.*, 1997) and rat (Laflamme *et al.*, 1998). A plausible biological mechanism for obesity–induced hypogonadotropic– hypogonadism may result, in part, from increased feedback inhibition of the HPT axis due to high serum levels of estrogens in obese males (Strain and Zumoff, 2006). Wherein an increased adipose tissue mass represents a significant peripheral source of estrogens, which, in turn, suppress the HPT axis and increase central adiposity. The subsequent reduction in circulating testosterone lead to increased deposits of visceral/ abdominal adipose tissue (Marin *et al.*, 1995; Kapoor *et al.*, 2008), and subfertility, whereas the increased production of circulating estrogens support differentiation of adipocytes (Price *et al.*, 1998). Several reports showed that the accumulation of fatty tissue in men was associated with decrease total, free T, and increased E2 serum levels (Pauli *et al.*, 2008; Hanafy *et al.*, 2007). Zohdy *et al*. (2007) reported significant negative correlation between BMI and serum total T. On other hand, Chavarro, *et al.* (2010) suggested that body mass index (BMI) was positively related to estradiol levels and inversely related to total testosterone and sex hormone-binding globulin (SHBG) levels. Our results meet with researchers (Mara *et al.*, 2008) from Reproductive Biology Associates report that a high BMI in men correlates with reduced testosterone levels. Low testosterone levels in obese (McTernan *et al.*, 2002) men are due to lower sex hormone-binding globulin, the enhancement of negative feedback on gonadotropin by the increased E2, insulin resistant.

In the present study, we investigated the association between BMI and serum prolactin level. It was found that serum prolactin level was significantly unaffected (P>0.05) between two groups (Table 1). It has recently been shown that increased body weight is associated with prolactinomas and that weight loss occurs with normalization of prolactin levels .On the other hand, decreased dopaminergic tone in humans is well correlated with obesity (Mirjana *et al.*, 2002). In a Nigerian study on 120 men, researchers found statistically significant associations between BMI and serum levels of progesterone and E, but these were not associated with PRL, T, or LH serum concentrations (Egwurugwu *et al.*, 2010). In humans, outside pregnancy, PRL secretion is altered by increasing body weight in adults and children. However, no molecular basis has been found which links PRL with increasing body fat, weight and appetite, although some data suggest the involvement of PRL with leptin (Kopelman, 2000).

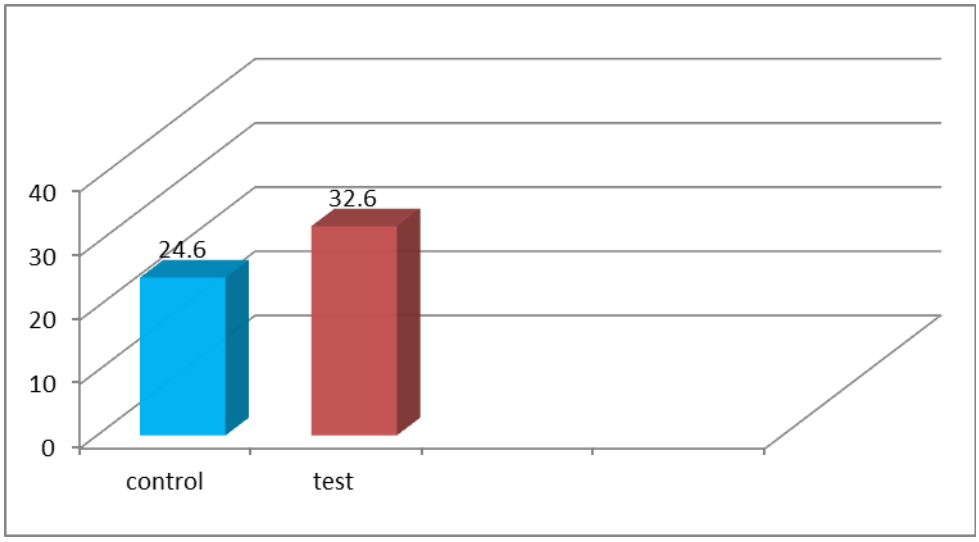

Figure (1): The mean of BMI in both control and test groups.

Table (1): Clinical hormonal in both control and test groups (Mean ± Standard Error)

Hormone levels	Test group	Control group
FSH(mIu/ml)	12.16 ± 0.02*	9.01 ± 0.03
LH(mIu/ml)	8.32 ± 0.02*	5.09 ± 0.01
T (ng/ml)	6.05 ± 0.02*	24.27 ± 0.03
E2 (pg / ml)	28.27 ± 0.19*	19.49 ± 0.03
PRL (ng/ml)	11.34 ± 0,04	9.49 ± 0.03

*Significantly (P<0.05) different from control group

Conclusions

In conclusion, the association explored between BMI and some reproductive hormones, as well as different patterns of this association between obese and non-obese Iraqi men, will be of help to broaden our understanding of the effect of obesity on some male reproductive physiologic characteristics. However, considering the available information in order to clarify the existing facts, it is recommended for future studies to consider assessing the role of weight loss on improving male fertility status through prospective cohort or interventional studies.

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