EUROPEAN ACADEMIC RESEARCH
Vol. IV, Issue 5/ August 2016

Impact Factor: 3.4546 (UIF)
DRJI Value: 5.9 (B+)

ISSN 2286-4822
www.euacademic.org

Impact of Obesity on Serum Testosterone, Luteinizing Hormone and Follicular Stimulating Hormone levels in Sudanese Obese Male

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Abstract:

Obesity contributes to infertility by reducing semen quality, changing sperm proteomes, contributing to erectile dysfunction and inducing other physical problems related to obesity. The aim of this study was to provide current scenario linking obesity and male fertility. Obesity has been linked to male fertility because of lifestyle changes, internal hormonal environment alterations.

The present study included 40 obese males as test group and the control group consists of 40 healthy males with normal weight, age was matched in two groups (30 ± 10 years). Estimation of testosterone, LH, and FSH levels was done by Enzyme Linked Immune Sorbent assay using an Instrument MAPLAP plus (ITALY). Statistical analysis was done by SPSS computer program and results showed a negative correlation of increasing BMI on testosterone level with a mean concentration of (1.0550 ± 0.62755 ng/ml) in obese male and (2.9575 ± 0.64127 ng/ml) in control subjects with (P. value =0. 00). However the results revealed a positive correlation of increasing BMI on FSH level with mean concentration of (4.0875 ± 3.808 mlU/ml) in

obese male and (2.9875 ± 1.81266 mlU/ml) in control subjects with (P. value =0,103) and on LH level with mean concentration of (3.7975 ± 1.44160 mlU/ml) in obese male and (2.5250 ± 1.28017 mlU/ml) in control subjects with (P. value = 0.00). Results also showed a negative correlation between BMI and testosterone level (P. value = 0.00, r = -0.675), and a positive correlation between BMI and both LH and FSH levels (P. value = 0.00, r = 0.442) and (P. value = 0.264, r = 0.126) respectively.

In conclusion: male obesity causes marked decrease of testosterone level and slightly increase of follicular stimulating hormone and luteinizing hormone levels, and there is an inverse correlation between BMI and testosterone whereas LH and FSH levels are positively correlated with BMI.

Key words: Fertility, Male obesity, Male infertility, Testosterone, FSH and LH.

1. INTRODUCTION:

Male fertility both directly and indirectly has been proposed to be affected by obesity, which inducing variation in erectile dysfunction, hormonal profiles behavior, scrotal temperatures and sleep (1). About 76% of men who reported with an erectile dysfunction or decrease in libido are overweight or obese (2). Regarding the hormonal profile, there is a complicated net work of hormones negative and positive feedback mechanisms which include testosterone, FSH and LH (3). The severity of obesity determines the degree to which levels of estradiol are increased and testosterone decreased (4). The incidence of obesity is rapidly rising in almost every region of the world. Although obesity affects women more than men, male obesity is an issue of serious concern. In Europe, the International Obesity Task Force has indicated that obesity rates in adult men range from 10 to 27%, with this prevalence rising significantly in the last 10 years (5). The adverse influence of obesity on various

aspects of female reproduction and fertility has been realized for some time (6), and management guidelines are now available (7). More recently, data regarding male obesity and infertility have been accumulating (8, 9). There are now several population-based studies showing that overweight and obese men have an up to 50% higher rate of sub-fertility when compared with normal weight men (9, 10). Male infertility could be related to confounding factors such as male age, smoking and alcohol use, and female partner obesity. However, once these factors have been excluded it was shown that for every three-point increase in a man's BMI, couples were 10% more likely to be infertile (11).

2. MATERIALS AND METHODS:

2.1. Materials:

2.1.1. Study design: This is a descriptive analytical case control study.

2.1.2. Study area: This study was conducted in Khartoum state.

2.1.3. Study populations: The study included 40 obese males as test group and 40 apparently healthy males with normal weight as control group (mean age of 30 ± 10 years for both groups).

2.1.3.1. Inclusion criteria: Healthy obese males were included in this study.

2.1.3.2. Exclusion criteria: Obese males with chronic diseases, Liver cirrhosis and psychiatric Disease and obese males who taking drugs which are known to interfere with serum testosterone level for example, glucocorticoids, and obese males with other possible secondary causes for male infertility were excluded from this study.

2.1.4. Ethical consideration: individuals involved in this study were informed by the study and its importance. This study was approved by the Research Committee of Medical Laboratory Science College Al-Neelain University.

2.1.5. Statistical analysis: Data were summarized, presented and analyzed using statistical package for the social science (SPSS) software program version 16. Chi-sqare test was conducted to study the association between nominal variables such as the association between groups and BMI classes. Unpaired t-test was used to compare the difference in mean numeric variables between two groups. P-value of ≤ 0.05 was considered significant.

2.1.6. Sampling: 5 milliliters of venous blood was collected from each male enrolled in the study in plain tube, serum was separated immediately after coagulation then stored frozen at -20 c. The deep frozen serum samples were thawed, kept to reach room temperature, and brought for the estimation of hormones.

2.2. Methods:

Estimation of testosterone, LH, and FSH was done by Enzyme Linked Immune Sorbent assay using an Instrument MAPLAP plus (ITALY).

2.2.1. Estimation of Follicle Stimulating Hormone:

The FSH Quantitative test Kit is based on the principle of a solid phase enzyme linked immunosorbent assay. The assay system utilizes a polyclonal anti -FSH antibody for solid phase (micro titer wells) immobilization and a mouse monoclonal anti-FSH antibody in the antibody-enzyme (horseradish peroxidase) conjugate solution. The test sample was allowed to react simultaneously with the antibodies, resulting in FSH molecules being sandwiched between the solid phase and enzyme-linked antibodies. After 60 minutes incubation at room temperature, the wells are washed to remove unbound labeled antibodies. A

solution of TMB was added and incubated for 20 minutes, resulting in the development of a blue color. The color development was stopped with the addition of 2N HCL, and the color was changed to yellow and measured spectrophotometrically at 450nm. The concentration of FSH was directly proportional to the color intensity of the test sample.

2.2.2. Estimation of Luteinizing Hormone: The LH Quantitative test Kit is based on the solid phase enzyme linked immunosorbent assay. The assay system utilizes one anti- LH antibody for solid phase (micro titer wells) immobilization and another mouse monoclonal anti-LH antibody in the antibody-enzyme (horseradish peroxidase) conjugate solution. The test sample was allowed to react simultaneously with the antibodies, resulting in LH molecules being sandwiched between the solid phase and enzyme-linked antibodies. After 60 minutes incubation at room temperature, the wells are washed to remove unbound labeled antibodies. A solution of TMB was added and incubated for 20 minutes, resulting in the development of a blue color. The color development was stopped with the addition of 2N HCL, and the color was changed to yellow and measured spectrophotometrically at 450nm. The concentration of LH was directly proportional to the color intensity of the test sample.

2.2.3. Estimation of Testosterone: The testosterone Quantitative test Kit is based on the principle of a solid phase enzyme linked immunosorbent assay. The assay system utilizes a polyclonal anti –testosterone antibody for solid phase (micro titer wells) immobilization and a mouse monoclonal anti-testosterone antibody in the antibody-enzyme (horseradish peroxidase) conjugate solution. The test sample was allowed to react simultaneously with the antibodies, resulting in testosterone molecules being sandwiched between the solid phase and enzyme-linked antibodies. After 60 minutes

incubation at room temperature, the wells were washed to remove unbound labeled antibodies. A solution of TMB was added and incubated for 20 minutes, resulting in the development of a blue color. The color development was stopped with the addition of 2N HCL, and the color was changed to yellow and measured spectrophotometrically at 450nm. The concentration of testosterone was directly proportional to the color intensity of the test sample.

2.2.4. Calculations of body math index: The BMI was calculated by dividing the subject mass by the square of his height, typically expressed in metric (Kg/m2) (12).

3. RESULTS:

The statistical analysis was done by SPSS and the results were as follow:

3.1. Effect of obesity on testosterone level: Comparison of means showed a significantly decrease testosterone level in case versus control groups (Figure 1).

3.2. Effect of obesity on LH level: Comparison of means showed a significantly increase in LH level in case versus control groups (Figure 2).

3.3. Effect of obesity on FSH: Comparison of means showed a significantly increase in FSH level in case versus control groups (Figure 3).

3.4. Person's Correlation Results:

3.4.1. Testosterone level correlated negatively with BMI as presented in Figure 4.

3.4.2. Luteinizing hormone correlated positively with BMI as presented in Figure 5.

3.4.3. Follicular stimulating hormone correlated positively with BMI as presented in Figure 6.

3.4.4. Testosterone and Luteinizing hormone levels were correlated negatively as presented in Figure 7.

3.4.5. Testosterone and follicular stimulating hormone levels were correlated negatively as presented in Figure 8.
3.4.6. Luteinizing hormone correlated positively with follicular stimulating hormone level as presented in Figure 9.

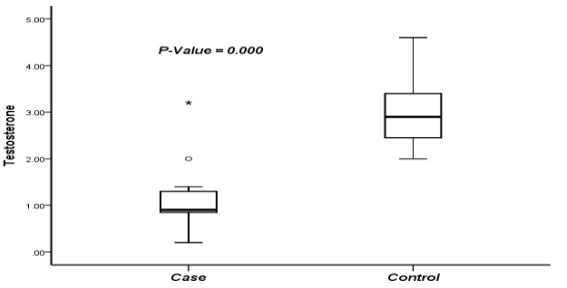

Figure 1: Testosterone level in case versus control groups.
Significant difference consider as P.value $\leq$ 0.05.

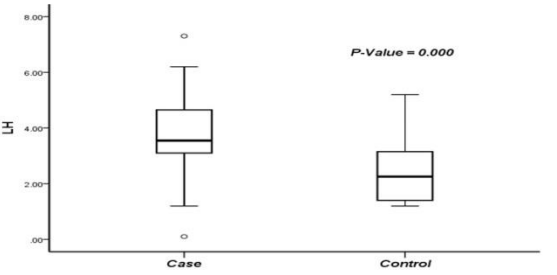

Figure 2: LH level in case versus control groups.
Significant difference consider as p-value $\leq$ 0.05.

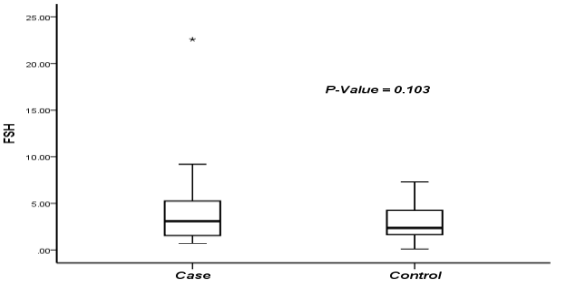

Figure 3: FSH level in case versus control groups.
Significant difference consider as p-value $\leq$ 0.05.

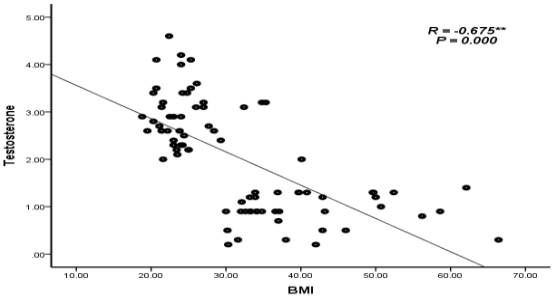

Figure 4: Correlation between testosterone level and BMI

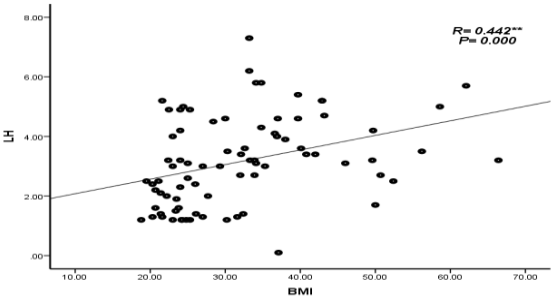

Figure 5: Correlation between lutenizing hormone level and BMI

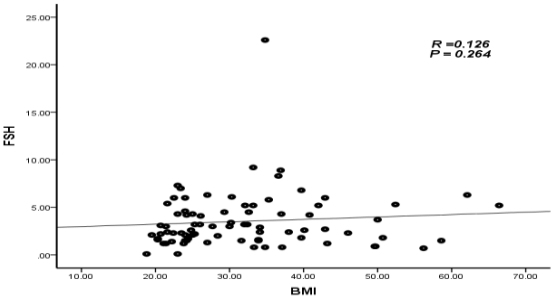

Figure 6: Correlation betweenFollecular stimulating hormone level and BMI

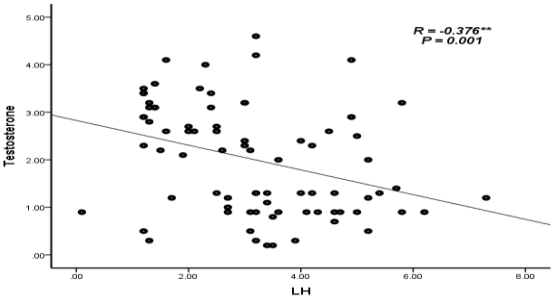

Figure 7: Correlation between testosterone and Lutenizing hormone levels.

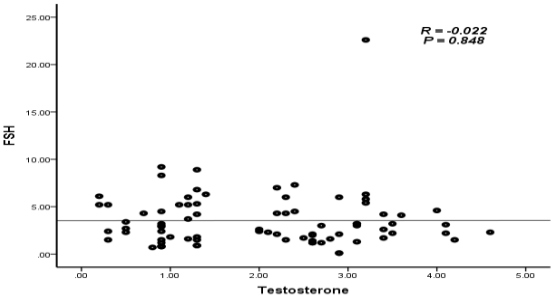

Figure 8: Correlation between testosterone and follicular stimulating hormone levels.

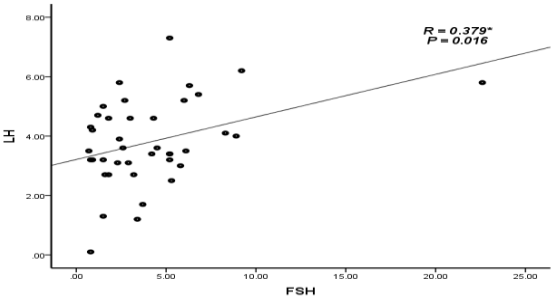

Figure 9: correlation of Luteinizing hormone with follicular stimulating hormone levels.

5. DISCUSSION:

The interaction between obesity and fertility has received increased attention owing to the rapid increase in the prevalence of obesity in the developed world (13). And the mechanisms that explain the relation between obesity and male infertility are not fully understood (14).

The results of this study showed a statistically significant effect (a negative correlation) of increasing BMI on testosterone with a mean concentration of (1.0550±0.62755ng/ml) in obese male and (2.9575±0.64127ng/ml) in control subjects with (P. value =0, 00). Low testosterone level are thought to be the result of; decreased sex hormone-binding globulin (SHBG) binding capacity, direct action of leptin and other adipocyte derived hormones leydig cell function (15) and impaird functioning of the hypothalamic-pituitary-testicular (HPT) axis(15). Our findings agreed with those previously described with P. value < 0.001 (16). However we observed a significant (positive correlation) of increasing BMI on FSH with mean concentration of (4.0875±3.808 mlU/ml) in obese male and (2.9875±1.81266 mlU/ml) in control subjects with (P. value =0,103) and on LH with mean concentration of (3.7975±1.44160 mlU/ml) in obese male and (2.5250±1.28017 mlU/ml) in control subjects with (P. value= 0.00) this agreed with previous studies results with (P. value< 0.001 and P. value< 0.001) respectively (16, 17).

6. CONCLUSIONS:

Male obesity causes a marked decrease in testosterone level and slightly increases of follicular stimulating hormone and luteinizing hormone levels; there was inverse correlation between BMI and testosterone whereas LH and FSH were positively correlated with BMI.

REFERENCES

1. Hammoud, A. O., Gibson, M., Peterson C. M., et al. "Obesity and male reproductive potential". J. Androl. (2006), 27, 619-626.
2. Cheng, J. Y. & Ng, E. M. "Body mass index, physical activity and erectile dysfunction: an U-shaped relationship from population based study". Int. J. Obes. (2007), 31, 1571-1578.
3. Globerman, H., Shen-Orr, Z., Karniel, E., et al." Inhibin B in men with severe obesity and after weight reduction following gastroplasty". Endocr. Res.(2005), 31, 17-26.
4. Aggerholm, A. S., Toft, G., ET AL. "Is overweight arisk factor for reduced semen quality and altered serum sex hormone profile." Fertile. Steril. (2008). 90, 619-626.
5. Lobstein T, rigby N, Leach r. EU platform on diet, physical activity and health. International Obesity Task Force (March 2005). www.iaso.org.
6. Mahmood T. Obesity: a reproductive hurdle. Br J Diabetes Vasc Dis, (2009);**9**:3-4.
7. Balen AH, Anderson r. Impact of obesity on female reproductive health: British Fertility society, policy and practice guidelines. Hum Fertil (2007);**10**:195-206.
8. Hammoud AO, Gibson M, peterson CM et al. Impact of male obesity on infertility: a critical review of the current literature. Fertil Steril (2008);**90**:897-904.
9. shayeb AG, Bhattyachary s. review: Male obesity and reproductive potential. Br J Diabetes Vasc Dis (2009);**9**:7-12
10. ramlau-Hansen C, Thulstrup A, Nohr E et al. subfecundity in over- weight and obese couples. Hum Reprod (2007);**22**:1634-7.
11. sallmen M, sandler D, Hoppin J, Baird D. reduced fertility among weight and obese men. Epidemiology (2006);**17**:520-3

12. MacKay, N. "Scaling of human body mass with height: the body mass index revisited," . J Biomech, (Mar 3 2010) vol. 43, pp. 764-6.
13. Robert G. Global obesity epidemics out of control. The New Zealand Herald; (2004) . Available: www.nzherald.co.nz.
14. Mamdoh Eskandar et al. Impact of Male Obesity on semen Quality and S erum Sex Hormone . Advances in Urology. (2012); Article ID 407601.
15. Jorge E. Chavarro, Thomase L. Toth, Diane L. Wright, John D, Meeke and Russ Hauser Body mass index in relation to semen quality, sperm DNA integrity and serum reproductive hormone level among men attending an infertility clinic. Fertil Steril. (2010);93(7) :2222-2213 . doi:10.1016/j .
16. paul EM, Legro RS, Demers LM, Kunselman AR, Dodson WC, Lee PA. Diminshed paternity and gonadal function with increasing obesity in men. Fertil Steril. (2008); 90:346-51(PubMed: 18291378).
17. Hammod AO, Gibson M, Peterson CM, Meikle AW, Carrell DT. Impact of male obesity on fertility: a critical review of the current literature. Fertile Steril. (2008); 90(4) :897-904.