
FSH status in normal and obese individuals

AISHWARYA S¹, DR. ANITHA ROY², DR.T. LAKSHMI^{3*}

¹Graduate Student, Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Tamil Nadu, India

²Associate Professor, Department of Pharmacology, Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Tamil Nadu, India.

³Associate Professor, Department of Pharmacology, Saveetha Dental College & Hospitals, Saveetha Institute of Medical and Technical Sciences, Tamil Nadu, India.

*Corresponding author:

Email : lakshmi@saveetha.com

Abstract: Obesity is one of the major and common health problems in all developed and developing countries leading to infertility. It also acts as an important modifier of reproductive hormones. Follicle-stimulating hormone (FSH), a pituitary-derived glycoprotein hormone, is associated in reproductive activities such as formation of gonadal and germ cells and the development of sex hormones. The goal of this study was to assess the FSH level in normal and obese individuals. 60 subjects were chosen from those attending the outpatient department of a private hospital and divided into two groups (n=30). Group 1 served as control having healthy individuals, Group 2 served as study group with obese individuals. 5 ml of venous blood was collected from the participants after informed consent and was centrifuged at 2500 rpm for 10 minutes. The serum was separated and analysed for serum FSH by standard enzyme linked immunosorbent assay technology (ELISA) using Robonik Elisa reader. The mean BMI levels between the groups were 21.98±2.59 and 34.42±3.82 respectively. The mean FSH levels between the groups were 10.43±4.43 and 5.59±2.53 respectively. These results were analysed by student t-test and showed significant difference between these two groups (p <0.0001). Obese individuals had low FSH levels, when compared to the normal healthy individuals. The findings clearly indicate that there is an association between obesity and hormone changes in individuals.

Keywords: FSH, obesity, adipokin, BMI, gonadotropin

INTRODUCTION

Obesity is an important medical problem and is a typical issue among females of conceptive era. Robustness and obese include uneven and severe fat accumulation that adversely affects the body's adequacy (Zeynep Ozcan Dag, 2015). In obese females, gonadotropin secretion is impacted by the enhanced peripheral aromatisation of androgens to estrogens (Parihar, 2003).

Follicle-stimulating hormone (FSH) is a pituitary-derived glycoprotein hormone involved in reproductive processes such as gonadal and germ cell growth, including the production of sex hormones. (Xin-Mei Liu, 2017). Obesity is an important modifier of the reproductive hormone (Freeman, 2010). In a population based study, the body size (BMI) was found to be the important level of menopausal transition (Freeman et al., 2005). However there is a complex association between the BMI, post menopausal state, time and other hormone modifier making difficult to distinguish the contribution of BMI with FSH level. Although modified pulsatile gonadotropic hormone secretion is a well-defined mechanism in obese patients (De Pergola et al., 2006), it is not known whether fat and/or hyperinsulinemia may have autonomous impacts on the endocrine secretion of the ovary, such as estradiol, mainly in females with regular menstrual cycles and ordinary ovarian appearance, without hyperandrogenism. (Grenman et al., 1986). FSH fortifies follicle development and improvement in the ovaries and produces the greatest measure of develop spermatozoa in the testicles. FSH and its relating receptor (FSHR) have an essential capacity in different malignancies (Zhou, 2013).

The use of gonadotropin for sex gland stimulation is common for sterility management. Indications embrace biological process of ovulation induction in anovular subjects or for patients receiving intrauterine insemination and controlled sex gland stimulation for IVF. Close to simulation of the natural cycle with one dominant cyst is very important to avoid multiple gestation in programmes of ovulation induction whereas a satisfactory sex gland response is vital to attain an appropriate gestation rate while not an excessive sex gland response for COS (Loh and Wang 2002 ; Roest et al., 1996). Obstetricians and procreative researchers have witnessed a developing human society generative impacts as the next frequency of females diagnosed with diseases of emission, physiological state, Postpartum diabetes and substantial alternative sequelae (Norman, 2004). In addition, polycystic ovarian syndrome (PCOS), may be a disorder characterized by hyperandrogenism and catamenial disturbances, complicating the issue in addition (Sharpe and Franks, 2002). Finally, however best to

understand and retain weight loss through efficient weight management (Norma) are intriguing reports. Obesity is a major reproductive hormone modifier (Freeman, 2010). Body size (BMI) was discovered to be the significant level of menopausal transformation in a population-based research (Freeman et al., 2005). However, there is a complicated connection between the BMI, postmenopausal state, time, and other hormone modifier that makes it hard to differentiate BMI's contribution from FSH.

MATERIALS AND METHODS

60 subjects were chosen from the outpatient department of a private hospital. They were divided into two groups (n=30).

Group I (Control group) – Normal healthy female individuals

Group II (Study group) – Obese females

Inclusion Criteria –

1. Normal healthy females with normal BMI (19.9-24.9)
2. Obese females with BMI > than 30

Exclusion Criteria –

1. Subjects with systemic diseases like Diabetes Mellitus, CVD, Hypertension and endocrine disorders.
2. Females with irregular periods

Sample collection and procedure

5 ml of venous blood was collected from the participants was collected in plain collection tube and centrifuged at 2500 rpm for 10 minutes. The Serum was separated and analysed for serum FSH by Standard enzyme linked immunosorbent assay technology (ELISA) Using Robonik Elisa reader. All statistical analysis was carried out using student t- test.

RESULT

The BMI of the control group was 21.98±2.59 and the FSH level was 5.59 ± 2.53. The BMI and FSH level of obese women was 34.42 ± 3.82 and 10.43±4.43 respectively which was highly significant (p <0.0001) which is statistically significant. The obese women had two times higher FSH level than control group (Table.1, Fig. 1 and Fig. 2).

Table 1: Mean, SD and p value of Control and Study groups

PARAMETERS	Control		Obese		p value
	Mean	SD	Mean	SD	
BMI	21.98	2.59	34.42	3.82	0.0001
FSH(mIU/ml)	5.59	2.53	10.43	4.43	0.0001

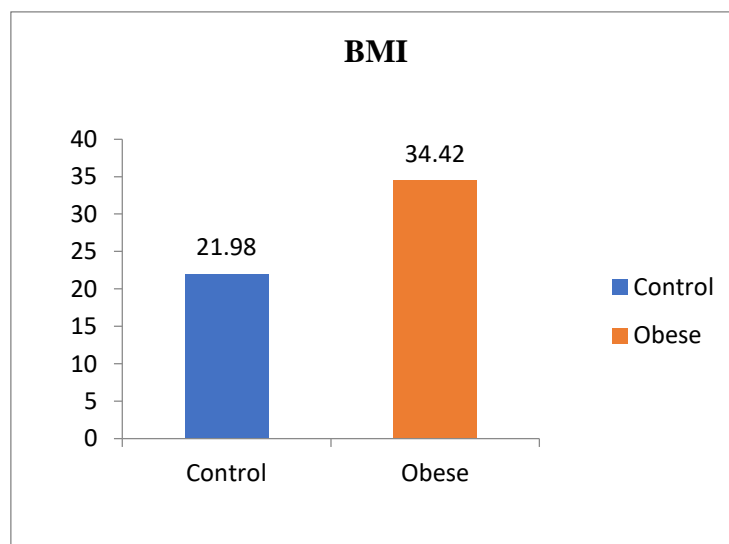


Fig.1: Graph showing BMI levels in study group

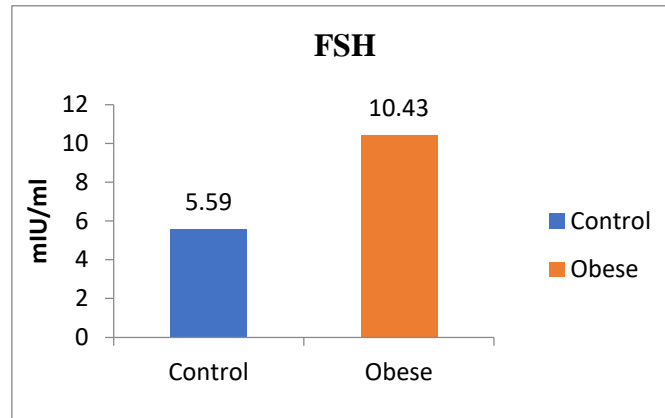


Fig.2: Graph showing FSH levels in study group

DISCUSSION

Although normal FSH level ranges from 0.3 U/ml- 10.0 U/ml, the FSH level of obese individuals in this study seems to be non variant to the normal value, but if the BMI increases further then FSH level may tend rise to higher levels. Fertility of obese females compared to standard weight females is lesser in natural cycles and sterility therapy cycles (Clark, 1996). Follicle-stimulating hormone is associated with lipodystrophy of fat tissue; this participation is further endorsed by the flexibility of gonadotropin to alter the degree of two main indices of lipid drop formation, especially leptin and adiponectin (ADPN). Leptin is an indicator of body fat mass; therefore, abnormal concentrations of leptin are associated in high blood pressure pathophysiology, artery induration, and heart disease (Trujillo et al., 2006; Lago et al., 2009; Sattar, 2009). Another adipokine obtained from adipocytes is ADPN; ADPN reduces fat and tends to increase after weight decreases. Diminished area unit ADPN concentrations probably associated with increased intra-abdominal fat (Yang et al., 2001; Asayama et al., 2003). BMI was discovered to be associated with changes in concentrations of inhibin B in the menopausal transition, with a significant connection between declining concentrations of inhibin B and raising independent body mass index (BMI). Obesity is a health burden on posh culture that is gradually prevailing. Most obese females are not incurable, but obesity and its adverse effect on reproductivity and fertility are well documented. Obese females are 3 times more probable than females with a normal body mass index to suffer from sterility (RichEdwards et al., 1993). Obese women experience fecundity impaired in both natural and supported cycles of conception (Zaadstra et al., 1993). An accepted indicator of visceral fat accumulation is a waist circumference > 80 cm in females (Tamer Erel et al., 2009). Obesity is a prevalent, rapidly-growing health, common health problem in the recent generation. Obesity modifies insulin sensitivity and gonadotrophins dynamics, and is associated with disorders of spontaneous ovulation (Sheik, 2015). Hence Healthy dietary habits and optimal physical activity has been always associated with disease prevention especially in cases of heart diseases, diabetes, blood pressure and obesity (Sheethalan et al., 2016).

CONCLUSION

The present study concludes that increase in BMI due to obesity leads to increase in FSH levels and various reproductive problems. Obesity seems to be one of the major problems prevailing among women leading to various health issues including infertility. Hence, maintenance of normal BMI is essential for a healthy lifestyle.

CONFLICT OF INTEREST

Nil

REFERENCE

1. Asayama K, Hayashibe H, Dobashi K, Uchida N, Nakane T, Kodera K, Shirahata A, Taniyama M, 2003. Decrease in serum adiponectin level due to obesity and visceral fat accumulation in children. *Obes. Res.* 11:1072–1079.
2. Clark AM, 1996. Weight loss in obese infertile women results in improvement in reproductive outcome for all forms of fertility treatment, *Fertility and foods*, 13(6) ;1502–1505.
3. De Pergola G, Maldera S, Tartagni M, Pannacciulli N, Loverro G, R Giorgino R (2006). Inhibitory Effect of Obesity on Gonadotropin, Estradiol, and Inhibin B Levels in Fertile Women, *Obesity*, 14(11): 1954–1960.
4. Freeman EW. 2010. Obesity and Reproductive Hormone Levels in the Transition to Menopause, *Menopause*, New York. 17(4): 718–726.

5. Freeman EW, Sammel MD, Gracia CR. Follicular phase hormone levels and menstrual bleeding status in the approach to menopause. *Fertil Steril* .2005;83:383–392.
6. Grenman S, Ronnema T, Irjala K, Kaihola HL, Gronroos M. Sexsteroid, gonadotropin, cortisol, and prolactin levels in healthy, massively obese women: correlation with abdominal fat cell size and effect of weight reduction. *J Clin Endocrinol Metab*. 1986;63:1257–61.
7. Lago F, Gomez R, Gomez-Reino JJ, Dieguez C, Gualillo O. 2009. Adipokines as novel modulators of lipid metabolism. *Trends Biochem. Sci*. 34:500–510
8. Loh S, Wang JX. 2002. The influence of body mass index, basal FSH and age on the response to gonadotrophin stimulation in non-polycystic ovarian syndrome patients. *Human reproduction*. 17(5): 1207–1211.
9. Norman RJ, Noakes M, Wu R, Davies MJ, Moran L, Wang JX 2004. Improving reproductive performance in overweight/obese women with effective weight management. *Hum Reprod Update*.10:267–80.
10. Norman RJ 2004. Improving reproductive performance in overweight/obese women with effective weight management. *Human Reproduction Update* .10(3) :267-280.
11. Norman RJ, Masters SC, Hague W, Beng C, Pannall P, Wang JX. 1995. Metabolic approaches to the subclassification of polycystic ovary syndrome. *Fertil Steril* 63:329-335.
12. Parihar M 2003. Obesity and infertility. *Reviews in Gynecological Practice*. 3: 120-126.
13. Pratt LA, Brody DJ 2014. Depression and obesity in the U.S. adult household population, NCHS Data Brief.167:1–8.
14. RichEdwards JW, Goldman MB, Willett WC, Hunter DJ, Stampfer MJ,
15. Colditz GA, Manson JE (1994). Adolescent body mass index and infertility caused by ovulatory disorder. *American Journal of Obstetrics and Gynecology* , 17(1): 171–177.
16. Roest J, van Heusden AM, Mous H, Zeilmaker, GH Verhoe A (1996). The ovarian response as a predictor for successful in vitro fertilization treatment at the age of 40 years. *Fertil. Steril*. 66, 969–973.
17. Sharpe RM and Franks S. 2002. Environment, lifestyle and infertility inter-generational issue. *Nature Cell Biol*. 4:33-40.
18. Sheik R, 2015. Awareness of Obesity as a Risk Factor for Polycystic Ovary Syndrome, *J. Pharm. Sci. & Res*. 7(7): 471-473.
19. Sheethalan MSR, Shankari, 2016. Physical fitness and dietary habit levels among different periodontal health conditions individuals - a cross sectional questionnaire study, *International Journal of Pharmaceuticals and Research*, 7(11):14401-14404.
20. Sattar N, Wannamethee G, Sarwar N, Chernova J, Lawlor DA, Kelly, Wallace AM, Danesh J, Whincup PH. 2009. Leptin and coronary heart disease: prospective study and systematic review. *J. Am. Coll. Cardiol*. 53:167–175.
21. Trujillo ME, Scherer PE 2006. Adipose tissue-derived factors: impact on health and disease. *Endocr. Rev*. 27:762–778
22. Tamer Erel C, Senturk LM. 2009. The impact of body mass index on assisted reproduction. *Current Opinion in Obstetrics and Gynecology*; 21: 228–235.
23. Xin-Mei Liu.2017. FSH regulates fat accumulation and redistribution in aging through the Gai/Ca2+/CREB pathway. *Aging Cell*.14;409–420.
24. Yang WS, Lee WJ, Funahashi T, Tanaka S, Matsuzawa Y, Chao CL, Chen CL, Tai, TY, Chuang LM. 2001. Weight reduction increases plasma levels of an adipose-derived anti-inflammatory protein, adiponectin. *J. Clin. Endocrinol. Metab*.86:3815–3819.
25. Zhou, 2013. Serum FSH level associated with her-2 and ki67 expression. *Oncology letter*. 6(4): 1128-1132