

Journal homepage: http://www.journalijar.com

INTERNATIONAL JOURNAL OF ADVANCED RESEARCH

RESEARCH ARTICLE

Association of body mass index (BMI) and reproductive hormones with polycystic ovary syndrome in Iraqi patients

Abdul Hussein Moyet Al-Faisal and Tabark Sabah Al – Rubiay

Institute of Genetic Engineering and Biotechnology for Postgraduate Studies, University of Baghdad, Iraq

Manuscript Info	Abstract		
Manuscript History:			
Received: 25 September 2014 Final Accepted: 26 October 2014 Published Online: November 2014	Polycystic ovarian syndrome (PCOS) is one of the most common endocrine diseases in women. This syndrome is characterized by hyperandrogenism, chronic an ovulation, infertility and obesity. In this prospective study, blood samples from two groups of females patients PCOS (70) and control were		
Key words PCOS, FSH, LH, Testosteron, BMI	taken for estimation of levels of testosterone (T), luteinizing hormone (LH), follicle-stimulating hormone (FSH), LH/FSH clinical symptoms of PCOS. Body mass index (BMI) and other parameters were also recorded. The data		
*Corresponding Author Abdul Hussein Moyet Al- Faisal, alfais2000@yahoo.com	obtained from hormonal study revealed that significant levels were detected in LH, LH\FSH and testosterone in patients (5.78 ± 0.42 , 1.165 ± 0.08 and 0.491 ± 0.07 respectively) comparing to healthy control (2.78 ± 0.26 , $0.693 \pm$ 0.10 and 0.205 ± 0.04 respectively) with non-significant level in FSH hormone among patients (5.41 ± 0.37). These results were not significant according to age groups. On the other hand, body mass index of patients were with significant prevalence of overweight\obesity (0.0033 ± 0.0001). This could be due to the androgen excess detected in patients.		

Copy Right, IJAR, 2014,. All rights reserved

Introduction:

Polycystic ovary syndrome (PCOS) is a common hormonal disturbance in women affecting between 7-10% of reproductive-age (Kaiser, 2011). The syndrome is often associated with obesity, insulin resistance and metabolic syndrome (Balen and Michelmore, 2002; Kaiser, 2011). It has been recognized that PCOS has a genetic and environmental basis (Kandarakis *et al.*, 2006).

The level of hypothalamic gonadotropin-releasing hormone (GnRH) regulate the secretion of pituitary luteinizing hormone (LH) and follicle-stimulating hormone (FSH) which vary throughout the menstrual cycle and regulate (**King, 2006**). Therefore, the levels of FSH and LH are critical for follicular development and subsequent ovulation (**Hillier, 2001**). The understanding of the underlying PCOS pathogenity is still not clear because the heterogeneity of this disorder (**King, 2006**). An increase in gonadotropin releasing hormone (GnRH) secretion which results in increased (LH) secretion is one of the reasons could be behind POCS arising (**Tsilchorozidou** *et al.*, **2004**). The increase in LH could be the primary factor driving the increase in testosterone secretion in PCOS (**Hill, 2003**). The obese and overweight PCOS women also found to correlate with the increase levels of T hormone and LH (**Benson** *et al.*, **2008**). The high prevalence of overweight/obesity in PCOS women was found to associate with another factors such as insulin resistance, decreased SHBG in addition to increase in circulating testosterone (**Freytag, 2003**; **Azziz** *et al.*, **2004**). The aim of the current work is to find the association between reproductive hormones and obesity in Iraqi POCS patients.

Materials and Methods

Seventy PCOS patients and 20 healthy females (aged between 20 to 40 years) were included in the current study. Venous blood sample (5 ml) was collected from each woman of both PCOS and healthy control. The serum obtained

by putting the blood samples in a clean dry plain plastic tube and allowed to clot at 37C for 30 minutes. The tubes centrifuged at 5000 rpm for 5 minutes, serum was collected and kept in freezer until used. Hormonal analysis was performed by using Addendum-Mini VIDAS apparatus (VIDAS) 12 mode 10, 1992, BioMerieux Company, France, through an enzyme linked fluorescent assay (ELFA) technique. Body Mass Index (BMI) for each female was also estimated. PCOS patients and healthy control with other diseases were excluded.

Statistical Analysis

The Statistical Analysis System- SAS (2010) and least significant difference –LSD test were used to analyze different factors include in the study.

Results and Discussion

The data obtained from hormonal study revealed that significant levels were detected in LH, LH\FSH and testosterone in patients (5.78 ± 0.42 , 1.165 ± 0.08 and 0.491 ± 0.07 respectively) comparing to healthy control (2.78 ± 0.26 , 0.693 ± 0.10 and 0.205 ± 0.04 respectively) with non-significant level in FSH hormone among patients (5.41 ± 0.37) (Table I).

Parameters	Mean ± SE		LSD
	Patients	Healthy (control)	
	(n=70)	(n=20)	
FSH (IU/ml)	5.41 ± 0.37	4.44 ± 0.23	1.404 NS
LH (IU/ml)	5.78 ± 0.42	2.78 ± 0.26	1.576 *
LH/FSH (IU/ml)	1.165 ± 0.08	0.693 ± 0.10	0.352 *
Testosterone (ng/ml)	0.491 ± 0.07	0.205 ± 0.04	0.267 *
BMI (Kg/m ²)	0.0033 ± 0.0001	0.0026 ± 0.0001	0.0004 *

 Table 1: The hormonal and BMI profiles of PCOS patients and healthy control.

* (P<0.05), NS: non-significant

These results were not significant according to age groups (Table II). On the other hand, body mass index of patients were with significant prevalence of overweight/obesity (0.0033 ± 0.0001). This could be due to the androgen excess detected in patients. PCOS develops when the ovaries overproduce androgens (eg, testosterone). Androgen overproduction often results from overproduction of LH (luteinizing hormone) which is produced by the pituitary gland. Excess of LH will stimulate ovaries to produce more testosterone when insulin level in the blood is high (Yousouf et al., 2012). That is, the combination of having ovaries which are responsive to insulin and high insulin levels in the blood, can result in the overproduction of testosterone. Such association between androgen hormones and obesity was detected by others. Low level of FSH in PCOS patients was also detected by Beydoan et al. (2009), and Liou et al., (2009) while the obese and overweight PCOS women who had increase levels of T hormone and LH was detected by **Benson** et al.(2008). Others reported that there was a high prevalence of overweight/obesity in PCOS women which associated with insulin resistance and correlated with decreased SHBG which caused an increase in circulating testosterone (Chang,2004). Obesity is defined as a body mass index (BMI) exceeding 25 or 27. It is estimated that 50% or more of women with PCOS are obese, which is higher than the prevalence rate of obesity in the general population (Franks, 1995; Azizz et al., 2004). This may due to metabolic abnormalities. These are often associated with many factors such as hyperandrogenism, IR, dyslipidaemia and glucose intolerance. Others proposed that the metabolic consequences of obesity may precipitate a genetically susceptible individual to express the PCOS phenotype (Laitinen et al., 2003; Ehrmann, 2005; Yildiz et al., 2008).

	Age group (year)			
Parameters	Less than 25	25-30	More than 30	LSD
FSH(mIU/mI)	5.13 ± 0.34	5.06 ± 0.34	6.02 ± 0.98	1.822 NS
LH(mIU/mI)	6.27 ± 0.67	6.01 ± 0.75	5.06 ± 0.74	2.041 NS
LH/FSH	1.35 ± 0.18	1.19 ± 0.13	0.953 ±0.13	0.435 NS
Testosterone(ng/ml)	0.487 ± 0.11	0.441 ±0.11	0.541 ± 0.14	0.350 NS
BMI (Kg/m ²)	0.0034±0.0002	0.0031±0.0001	0.0032±0.0001	0.0005 NS

Table 2: The hormonal and BMI profiles of PCOS patients and healthy control according to age groups.

* (P<0.05), NS: non-significant

The association between high level of androgens- hyperandrogenaemia- and obesity was also detected by Lee *et al.*, (1988), Dnuaif, (1997) and Freytag (2003) who suggested that hyperandrogenaemia possibly causes android fatadrenocortical obesity- after hirsutism in PCOS women. Results obtained revealed that there is a no significant FSH level in all age groups of PCOS in relation to control groups as shown in Tables 1&2. The current results agreed with Nawras, (2010) who showed that no significant differences (p>0.05) in level of FSH. The reduction levels of FSH can be explained by the increase of the conversion of androstenedione in adipose tissue which additionally stimulates LH and inhibits FSH (Marx and Mehata, 2003). High levels of inhibin and Follistatin have been found in the PCOS women which leads to FSH reduction and increase of ovarian androgen production (Karkanaki *et al.*, 2011). On the other hand, the FSH/LH ratio was significantly high in PCOS patients comparing to healthy group. In contrast the ratio was not significant between POCS age groups and healthy control. This is in agreement with Taylor *et al.*, (1997) and Mukherjee *et al.*, (1996) and Nawras, (2010) who detect non significant FSH\LH ratio and in contrast with Taylor *et al.*, (1997) who suggested an increased FSH:LH ratio may predict an elevation in FSH alone.

Refrences:

Azziz, R., Woods, K.S., Reyna, R., Key, T.J., Knochenhauer, E.S.and Yildiz, B.O. (2004). The prevalence and features of the polycystic ovary syndrome in an unselected population. J. Clin. Endocrinol. Metab., 89:2745–2749.

Balen, A.and Michelmore, K.(2002). What is polycystic ovary syndrome? Are national views important? Human Reproduction 17(9): 2219-2227

Benson,S.,Janssen,O.E.,Hahn,S., Tan,S., Dietz,T. and Man,K.(2008).Obisity,depression, and chronic low-grade inflammation in women with polycystic ovary syndrome.Brain,behave.& immune.,22:177-184.

Beydoun,H., Stadtmauer,L.,Russell, H.,Zhaol,Y and Oehninger,S. (2009).Polycystic ovary syndrome, body mass index and outcomes of assisted reproductive technologies . Repro. BioMed. Online. 18(6):856-863

Chang. R. J.(2004). A practical approach to the diagnosis of polycystic ovary syndrome. Amer.J.Obst& Gynecol., 191:713–717.

Dunaif, A. (1997). Insulin resistance and the polycystic ovary syndrome: mechanism and implications for pathogenesis.Endocr.Rev.18:774-800.

Ehrmann, D. A. (2005). Polycystic ovary syndrome. Nat Engl J Med 352:1223-1236.

Franks, S. (1995). Polycystic ovary syndrome. N. Engl. J. Med., 333(13): 853-861.

Freytag, U. (2003): Subcutaneous adipose tissue pattern in lean and obese women with PCOS. Diploma-Francens-University

Freytag, U. (2003): Subcutaneous adipose tissue pattern in lean and obese w omen with PCOS. Diploma-Francens-University

Hill ,K.M. (2003). The pathogenesis and treatment of PCOS. Nurse Pract., 28:8-25.

Hillier, S.G. (2001) Gonadotropic control of ovarian follicular growth and development. Mol. Cell Endocrinol., 179:39-46.

Kaiser, U.B. (2011). Gonadotropin Hormones. 3th .ed. Elsevier Inc. Pp:205-251.

Kandarakis, E., Piperi, C., Argyrakopoulou, G., Spina, J., Papanastasiou, L., Bergiele, A. and Panidis. D. (2006). Polycystic Ovary Syndrome: The influence of environmental and genetic factors. Hormones. **5** (1):17-34.

King.J. (2006) .Polycystic Ovary Syndrome. Journal of Midwifery & Women's Health, 51(6):1-4.

Laitinen, J., Taponen, S., Martikainen, H., Pouta, A., Millwood, I., Hartikainen, A.L., *et al.*(2003). Body size from birth to adulthood as a predictor of self-reported polycystic ovary syndrome symptoms. Int. J. Obes. Relat. Metab. Disord, 27:710–5.

Lee, S.J., Lenton, E.A., Sexton, L.and Cooke, I.D. (1988): The effect of age on the cyclical patterns of plasma LH, FSH, estradiol and progesterone in women with regular menstrual cycles. Hum. Reprod., 3:851-5.

Liou, T., Yang, J., Hsieh, C., Lee, C., Hsu, C., and Hsu, M. (2009). Clinical and biochemical presentations of polycystic ovary syndrome among obese and nonobese women. Fertil & Steril., **92**(6):1960-1965.

Marx.T.L.and Mehta.A.E.(2003).Polycystic ovary syndrome: pathogenesis and treatment over the short and long term. Cleve.Clin.j.Med.,70(1):31-41

Mukherjee, T., Copperman, A.B., Lapinski, R. et al. (1996). An elevated day 3 follicle stimulating

hormone:luteinizing hormone ratio (FSH:LH) in the presence of a normal day 3 FSH predicts a poor response to controlled ovarian hyperstimulation. Fertil. Steril., 65:588-93.

Nawras N. (2010). Study of the Hormonal Reproductive exchanges associated with Polycystic Ovary Syndrome in women of reproductive age in Najaf city. J. Kuffa Uni. Dep. Biol., 2(2):135-142.

SAS. (2010). Statistical Analysis System, User's Guide. Statistical. Version 9.1th ed. SAS. Inst. Inc. Cary. N.C. USA.

Taylor, A.E., McCourt, B., and Martin, K.A.(1997). Determinants of abnormal gonadotropin secretion in clinically defined women with polycystic ovary syndrome. J Clin Endocrinol Metab; 82:2248–2256.

Tsilchorozidou,T., Overton, C., and Conway, G.S.(2004). The pathophysiology of polycystic ovary syndrome. Clin. Endocrinol., 60: 1–17.

Yildiz B.O., Knochenhauer E.S.and Azziz, R.(2008). Impact of obesity on the risk for polycystic ovary syndrome. J. Clin. Endocrinol. Metab., 93:162–8.

Yousouf, R., Khan, M., Kounsar, Z., Ahangar, S. and Lone, W.A. (2012). Polycystic Ovarian Syndrome: Clinical Correlation with Biochemical Status. Surgical Science, 3: 245-248