Obesity, Glycemic and Hormonal Criteria Of Polycystic Ovary Syndrome

Tawfeeq F. R. AL-Auqbi M.B, Ch.B FICMS/CM

ABSTRACT

Background: Polycystic ovary syndrome (PCOS) is the most common endocrinopathy affecting women, at reproductive age. PCOS is a chronic hyperandrogenic state that has many significant short-term and long-term implications for patients such as oligomenorrhea, amenorrhea, infertility, diabetes mellitus, cardiovascular disease, increased risk of endometrial cancer, and hirsutism.

Objectives: To evaluate the obesity and glycemic criteria among women with polycystic ovary syndrome.

Method: A case control designed study was carried out at the National Diabetes Center (NDC) / Al-Mustansiryia University; on 50 participants formed the PCOS group and 50 healthy control participants. Data collected about age, age at menarche and BMI. Also, blood samples examined for FPG and 2-h OGTT test carried out for all the participants. Moreover hormonal assessment for the LH, FSH and total testosterone were done for all participants.

Results : PCOS group women age and age at menarche were 27.62 ± 5.74 and 12.0 ± 1.06 years; vs. the control group were 25.42 ± 4.94 and 11.64 ± 0.69 years respectively. All PCOS group were experienced signs and symptoms of ovarian dysfunction, and infertility while the control group did not experience any of such evidences. BMI showed significant difference between study groups (t-test ($P < 0.05$)); 98% of the PCOS group was obese vs. 58% of the control group. The waist/hip ratio showed insignificant difference (t-test ($P > 0.05$). Fasting plasma glucose (FPG) showed insignificant difference between the study groups (t-test ($P > 0.05$); about 18% of the PCOS group was prediabetic (Impaired Fasting Glucose, IFG) (100-125mg/dl) and 6% was diabetic (>126mg/dl) whereas the entire control group was normoglycemic. The OGTT showed significant difference between the study groups (t-test ($P < 0.05$)); about 22% of the PCOS group was prediabetic (Impaired Glucose Tolerance, IGT) (140-199mg/dl) and 6% was diabetic (>200mg/dl) whereas the entire control group was normoglycemic. PCOS group showed highly significant elevation in the LH level vs. the control group by about four folds (t-test ($P < 0.05$)). Also, FSH showed similar elevation by about two folds among the PCOS group vs. the control group (t-test ($P < 0.05$)). Moreover the LH/FSH ratio was elevated by about two folds among the PCOS group vs. the control group (t-test ($P < 0.05$)). Total serum testosterone of the PCOS group showed significant raise vs. the control group (t-test ($P < 0.05$)). Whole the PCOS group had A total testosterone level >60 ng/dL and in contrary the entire control group didn't show any elevation >60ng/dl.

Conclusion: obesity of android (central) type was frequent and prevalent among PCOS women. About 20-28% of PCOS women was prediabetic or diabetic due to insulin resistance and decreased insulin sensitivity. Hormonal assay of LH, FSH, LH/FSH ratio and total testosterone were all significantly elevated by two folds or more among the PCOS women. It is not essential that a woman who had polycystic ovaries by ultrasound to have PCOS.

Key Words: polycystic ovary syndrome (PCOS), BMI, Waist/hip ratio, FPG, OGTT, LH, FSH, LH/FSH ratio, total testosterone.

Al- Kindy Col Med J 2009; Vol .5 (1) P: 33-39-

Introduction

Polycystic ovary syndrome (PCOS) is one of the most common endocrine disorders in women of reproductive age. The overall prevalence of PCOS among women in the reproductive age is ranged between 4 - 8% [1] and 5 - 10% as well; it is the most common endocrinopathy affecting women. [2, 3, 4] Moreover, the prevalence may be as high as 30% in women with secondary amenorrhea, 75% in women with oligomenorrhea, and 90% in women with hirsutism. [5]

Stein and Leventhal first described PCOS in 1935 when they published their report of seven women with amenorrhea, hirsutism, obesity, and enlarged polycystic appearing ovaries. [6, 7] But in fact, PCOS had been initially described in 1844 by Chereau and Rokitansky.[8] The definition of PCOS, most commonly used today, arose from the proceedings of an expert conference sponsored by the National Institutes of Health (NIH) in April 1990 in the United States (*i.e.* NIH 1990 criteria); They summarized these criteria into the following major research criteria (in order of importance) hyper-and-organism and/or hyperandrogenemia, oligoovulation and exclusion of known disorders such as Cushing's syndrome, hyperprolactinemia and CAH 59%.[9] A fourth criterion was considered polycystic ovaries on ultrasound which been particularly controversial.[8, 9] But PCOS, as defined by ultrasonography (the presence of 10 or more cysts, 2-8 mm in diameter, arranged either peripherally around a dense core of stroma or scattered throughout an increased amount of stroma, and/or ovarian volume > 10 mL) should also be considered as one of the possible diagnostic criteria for PCOS.[10, 11]Another expert conference was organized in Rotterdam in May 2003, sponsored in part by the European Society for Human Reproduction and Embryology and the American Society for Reproductive Medicine (*i.e.* Rotterdam 2003 criteria); The proceedings of the conference noted that PCOS

could be diagnosed, after the exclusion of related disorders, by two of the following three features ovarian dysfunction evidenced by oligomenorrhea or amenorrhea, clinical evidence of androgen excess (e.g., hirsutism and acne) and polycystic ovaries according to ultrasonic diagnostic criteria.[1, 10, 11, 12, 13]

Nowadays, it is well accepted that PCOS is a chronic hyperandrogenic state that has many significant short-term and long-term implications for patients such as oligomenorrhea, amenorrhea, infertility, diabetes mellitus, cardiovascular disease, increased risk of endometrial cancer, and hirsutism.[7]

Obesity, which is seen in 50% to 65% of PCOS patients, may increase the insulin resistance and hyperinsulinemia, the correlation between hyperandrogenism and insulin resistance has been recognized in both obese and nonobese anovulatory women. It is also clear that adiposity plays a essential role in maintaining and presumably in generating PCOS; evidence for this includes the often dramatic improvement in menstrual regularity and in both symptoms of hyperandrogenism and ovulatory function in response to modest weight loss of 5% body weight in women with PCOS.[14, 15] Furthermore, Many women with PCOS (between 38% and 88%) are overweight or obese;[16, 17] because obesity per se also contributes to features of hyperandrogenism even in women with normal ovaries.[17] The link between obesity, hyperandrogenism, insulin resistance and hyperinsulinemia in PCOS women likely to determined genetically by candidate genes for PCOS include those involved in the regulation of ovarian steroidogenesis but also those genes that influence body mass index (BMI) and adiposity.[19]

Of this study the objective is to evaluate the obesity , glycemic and hormonal criteria of women with polycystic ovary syndrome.

Methods

A case control designed study was carried out at the National Diabetes Center (NDC) / Al-Mustansiryia University in the second half of 2007. Total of 100 participants were studied in two different groups, age and sex matched, formed of 50 women had PCOS and 50 healthy control women; ultrasonographic criteria were depended for selection of PCOS group. Oral consent was taken from all enrolled people, after obtaining their agreements according to the medical research and ethical regulations.

Data were collected from all participants regarding their age, BMI, gynecological and endocrinological history. Fasting blood samples were taken for Fasting Plasma Glucose (FPG); 75 grams oral glucose tolerance tests (OGTT) were conducted for all participants. Also their serum were investigated for the following hormones LH, FSH and testosterone.

Statistical analysis and recording of obtained data were carried out by using Microsoft Excel - Windows XP professional program. Differences are considered to be of significance according to the t-test at level of $P \leq 0.05$.

Results

Women of the studied PCOS group age and age at menarche were 27.62 ± 5.74 and 12.0 ± 1.06 years vs. the control group were 25.42 ± 4.94 and 11.64 ± 0.69 years respectively, (t-test ($P < 0.05$)).

All women of PCOS group were experienced signs and symptoms of ovarian dysfunction, evidenced by oligomenorrhea or amenorrhea, and clinical evidence of androgen excess as hirsutism, and infertility while the control group did not experience any of such evidences. (Table-1)

Prevalence of obesity among the two groups was studied by comparing their BMI and waist/hip ratio as parameters of obesity. (Table-1) BMI showed significant difference between the two study groups (t-test ($P < 0.05$)); about 98% of the PCOS group was obese vs. 58% of the control group. Whilst the waist/hip ratio showed insignificant difference (t-test ($P > 0.05$)); despite this fact, the 0.72% cut-off point for diagnosis of central obesity showed that about half the PCOS group was obese; while the depending of 0.85% as a cut-off point of central obesity associated with risk of cardiovascular disease, was prevalent in 8% of PCOS group. (Table-1)

Fasting plasma glucose (FPG) and 2 hours oral glucose tolerance tests (OGTT) were used to evaluate glycemic state of the study population, (Table-2). FPG showed insignificant difference between the study groups (t-test ($P > 0.05$)); about 18% of the PCOS group was Impaired Fasting Glucose (IFG, 100-125mg/dl) and 2% diabetic (>126mg/dl) whereas the entire control group was normoglycemic. Similarly, the 2 hours OGTT test was used to assess the glycemic state showed significant difference between the two study groups (t-test ($P < 0.05$)); about 22% of the PCOS group was Impaired Glucose Tolerance (IGT, 140-199mg/dl) and 6% was diabetic (>200mg/dl) whereas the entire control group was normoglycemic.

The following hormones LH, FSH and total serum testosterone were investigated among whole the participants, (Table-3). Elevation in LH level was highly significant in PCOS group vs. the control group by about four folds 11.90 ± 3.35 and 2.93 ± 0.70 IU/l respectively (t-test ($P < 0.05$)). Similar elevation of FSH founded among the PCOS group vs. the control group by about two folds 7.57 ± 3.01 and 3.79 ± 0.87 IU/l respectively (t-test ($P < 0.05$)). Moreover the LH/FSH ratio was elevated by about two folds among the PCOS group vs. the control

group 1.71± 0.71 and 0.80 ± 0.22 respectively (t-test ($P < 0.05$)); elevation of LH/FSH ratio by <2 folds noticed among 86% of the PCOS group and 14% showed elevation by >2 folds, in contrary the entire control group didn't show any elevation. Total serum testosterone raised significantly, about five times, among the PCOS group vs. the control group 112.4 ± 44.4 and 23.2 ± 10.5 ng/dl respectively (t-test ($P < 0.05$)); all the PCOS group had a total testosterone level >60 ng/dL, which is considered elevated, and 10% of them had total testosterone level >200ng/dl, in contrary the entire control group didn't show any elevation (>60ng/dl) (Table-3).

Discussion

All the NIH 1990 criteria and Rotterdam 2003 criteria were applied on the participants of PCOS group since all of them experienced ovarian dysfunction, infertility and hirsutism; **(Table-1)** in addition to the biochemical and ultrasonic changes which considered as golden standard for the diagnosis of PCOS. [20]

Different groups of investigators found that obesity is a common feature among PCOS women; they found in population of their studies, 10–38% of women with PCOS have been reported to be obese (BMI ≥30 kg/m^2, grade II obesity) [21, 22, 23, 24] which was less than among our study; we found that 78% of PCOS group were obese (BMI ≥30 kg/m^2, grade II obesity) vs. 8% among the control healthy group. But we noticed that our results were in consistence with Dunaif et.al findings, who stated that at least one-half of women with PCOS are obese compared to normal women, [25] and Ricardo Azziz who found that the most common abnormalities in women with PCOS were increased their BMI in 72.3% of them, [26] adding together with earlier findings which shows that between 38% and 88% of PCOS women are overweight or obese, [15, 16] **(Table-1).** A likely explanation for the mechanisms underlying the development of obesity in women with PCOS is the combined effect of a genetic predisposition to obesity in the context of an obesogenic environment (poor unbalanced diet and reduced exercise); the development of obesity in women with PCOS in turn amplifies and may even unmask the biochemical and clinical abnormalities characteristic of this condition, the implication of that was observed in many obese women with PCOS may remained asymptomatic and did not become obese. [19]

Furthermore, the PCOS women tend to have android type of obesity (central obesity) rather than the gynecoid type. Central obesity, waist/hip ratio > 0.72%, found in about 54% of the PCOS group vs. 24% among the control group, [20] **(Table-1)**. Rosenfield found that PCOS is the single most common endocrine obesity syndrome in females; and obesity begins in mid-childhood and typically is android in type, with a waist/hip ratio > 0.85%.[7, 27] Our study showed waist/hip ratio > 0.85% among PCOS group was 8% vs. 2% among the healthy group, **(Table-1)**. Central obesity with a waist/hip ratio of > 0.85 is associated with cardiovascular disease and is a marker for PCOS. [7, 27]

However, our understanding of adipose tissue physiology and pathophysiology explain its complex role in the regulation of appetite, weight, metabolism and reproductive capacity; also adipose tissue plays a key role in the pathogenesis of various common and complex disorders such as type 2 diabetes mellitus and polycystic ovary syndrome (PCOS) .[19]

Women of PCOS group in our study classified as Impaired Fasting Glucose (IFG) and diabetics according to their FPG (ADA criteria) [28] as 18% and 2% respectively; according to their 2 hours OGTT (WHO criteria) classified as Impaired Glucose Tolerance (IGT) and diabetics as 22% and 6% respectively; [29] while all the control group women were normoglycemic, normal FPG and OGTT, (Table-2). Findings of present study regarding FPG and OGTT were less than that obtained from western studies. It was well-known that Insulin resistance precede the development of type 2 diabetes mellitus; Glucose tolerance testing is important; as many as 35-45% of PCOS patients will have impaired glucose testing and about 7-10% will have type 2 diabetes mellitus.[30, 31] Studies shown that 30-40% of women with PCOS have impaired glucose tolerance, and as many as 10% develop type 2 diabetes mellitus by the age of 40.[8,32] These observations indicate that insulin resistance and type 2 diabetes is a common finding in women with PCOS independent of obesity and that insulin resistance in obese PCOS is composed of dual contributions, one unique to PCOS and the other obesity-specific.[33] Both obese and nonobese women with PCOS have a higher incidence of insulin resistance and hyperinsulinemia than age-matched controls; however, obese women with PCOS have significantly decreased insulin sensitivity compared with nonobese women who have PCOS.[34] Moreover, women with PCOS are more likely to develop gestational diabetes; [35] and in postmenopausal women with a history of PCOS found a 15% prevalence of type 2 DM .[36]

The present study proved that LH level, FSH level and LH/FSH ratio of PCOS group vs. the healthy control group were elevated by four folds, two folds and two folds respectively; in contrary the entire control group didn't show any elevation. Also total serum testosterone of all PCOS group was raised, >60 ng/dL, and 10% of PCOS group had total testosterone levels of >200 ng/dL which suggest a virilizing tumor, (Table-3). LH hypersecretion is a characteristic hallmark of PCOS, women with PCOS have an

increase in both the LH pulse frequency and amplitude, Increased LH, in turn, leads to an increase in androgen production by the theca cells within the ovary.[11, 32] Because of the pulsatile nature of gonadotropin (GnRH) secretion, there is a wide "normal range" for both LH and FSH; therefore, a single blood sample from a patient with PCOS will frequently be in this normal range, [23, 24]but when it be as high as our finding certainly, it could be depended for diagnosis. The reason for these elevations was that Insulin acts synergistically with LH to enhance androgen production in the ovarian theca cells, insulin also decreases hepatic synthesis and secretion of sex hormone-binding globulin (SHBG), the protein that binds testosterone in the circulation, thus increasing the amount of free testosterone that is biologically available.[32,37] Women with PCOS and hyper-insulinemia typically have elevated free testosterone, but the total testosterone concentration may be at the upper range of normal or only modestly elevated;[32] but very high total testosterone level are not generally seen in PCOS and suggest a virilizing tumor. [38, 39]

Although all the PCOS group were diagnosed according to the ultrasonic criteria and they were experienced symptoms of ovarian dysfunction such as infertility, oligomenorrhea and/or amenorrhea and hirsutism, some of them were not obese, normoglycemic and their hormonal assay was normal or a higher normal level. We should keep in mind the golden statement, it is not essential that a woman have polycystic ovaries to have the PCOS; polycystic ovaries observed on ultrasound are a sign of PCOS and not by themselves diagnostic of the disease, furthermore the Polycystic ovaries are seen 67-86% of the time in patients who have PCOS.[2, 4, 7, 40, 41]

Conclusions

Obesity was frequent and prevalent among PCOS women, at least three quarters are obese compared to normal women; obesity was commonly presented in android (central) type.

About 20-28% of PCOS women were prediabetic (IFG and/or IGT) or diabetic due to insulin resistance and decreased insulin sensitivity; it may need to pay more attention as far as they are at higher risk to develop type 2 diabetes mellitus, gestational diabetes at the reproductive age and even after age of forty and post menopause.

Hormonal assay of LH, FSH, LH/FSH ratio and total testosterone were all significantly elevated by two folds or more among the PCOS women.

It is not essential that a woman who had polycystic ovaries by ultrasound to have PCOS, because polycystic ovaries are a sign of PCOS and not diagnostic for the syndrome, so detailed investigations required to confirm the professional diagnosis.

References

1. Chang R. A practical approach to the diagnosis of polycystic ovary syndrome. Am J Obstet Gynecol 2004;191:713-7.
2. Slowey MJ. Polycystic ovary syndrome: new perspective on an old problem. South Med J. 2001;94:190-6.
3. Ahles BL. Toward a new approach: primary and preventive care of the woman with polycystic ovarian syndrome. Prim Care Update Ob/Gyns. 2000;7:275-8.
4. Kidson W. Polycystic ovary syndrome: a new direction I treatment. Med J Aust. 1998;169:537-40.Available at http://www.mja.com.au/public/issues/nov16/kidson/kidson.html. Accessed July 17, 2002
5. Adams J, Polson DW, Franks S. Prevalence of polycystic ovaries in women with anovulation and idiopathic hirsutism. Br Med J 1986;293:355-9.
6. Stein IF, Leventhal ML. Amenorrhea associated with bilateral polycystic ovaries. Am J Obstet Gynecol. 1935;29:181-91.
7. Mac Pannill, MPAS, PA-C,. Polycystic Ovary Syndrome: An Overview. Advanced Practice Nursing eJournal 2(3), 2002.
8. Ricardo Azziz. (2006) Diagnosis of Polycystic Ovarian Syndrome: The Rotterdam Criteria Are Premature. Journal of Clinical Endocrinology and Metabolism, 91 No3, 781-5.
9. Zawadzki JK, Dunaif A 1992 Diagnostic criteria for polycystic ovary syndrome: towards a rational approach. In: Dunaif A, Givens JR, Haseltine FP, Merriam GR, eds. Polycystic ovary syndrome. Boston: Blackwell Scientific; 377–84.
10. Adams, J., Franks, S., Polson, D.W., Mason, H.D., Abdulwahid, N., Tucker, M., Morris, D.V., Price, J. & Jacobs, H.S. (1985) Multifollicular ovaries: clinical and endocrine features and response to pulsatile gonadotropin releasing hormone. Lancet, 2, 1375-9.
11. Ehrmann DA. Medical progress: Polycystic ovary syndrome. J Engl J Med 2005;352:1223-36.
12. The Rotterdam ESHRE/ASRM-sponsored PCOS consensus workshop group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polysystic ovary syndrome (PCOS). Hum Reprod 2004;19:41-7.
13. Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group 2004 Revised 2003 consensus on diagnostic criteria and long-term healnth risks related to polycystic ovary syndrome. Fertil Steril 81:19–25.
14. Kiddy, D.S., Hamilton-Fairley, D., Bush, A., Short, F., Anyaoku, V., Reed, M.J. & Franks, S. (1992) Improvement in endocrine and ovarian function during dietary treatment of obese women with polycystic ovary syndrome. Clinical Endocrinology, 36, 105-11.
15. Holte, J., Bergh, T., Berne, C., Wide, L. & Lithell, H. (1995) Restored insulin sensitivity but persistently increased early insulin secretion after weight loss in obese women with polycystic ovary

16. syndrome. Journal of Clinical Endocrinology and Metabolism, 80, 2586-93.
17. Legro, R.S. (2000). The genetics of obesity: lessons for polycystic ovary syndrome. Annals of the New York Academy of Sciences, 900, 193-202.
18. Balen, A.H., Conway, G.S., Kaltsas, G., Techatrasak, K., Manning, P.J. & West, C. (1995). Polycystic ovary syndrome: the spectrum of the disorder in 1741 patients. Human Reproduction, 10, 2107-11.
19. Taponen, S., Martikainen, H., Jarvelin, M.R., Laitinen, J., Pouta, A., Hartikainen, A.L., Sovio, U., McCarthy, M.I., Franks, S. & Ruokonen, A. (2003) Hormonal profile of women with self-reported symptoms of oligomenorrhea and/or hirsutism: Northern Finland Birth Cohort 1966 Study. Journal of Clinical Endocrinology and Metabolism 88,141-7.
20. Barber T. M., McCarthy M. I., Wass J. A. H., Franks S.; (2006) Obesity and Polycystic Ovary Syndrome. Clin Endocrinol. 2006;65(2):137-45.
21. Barbara S. Snyder. Polycystic Ovary Syndrome (PCOS) in the Adolescent Patient: Recommendations for Practice. Pediatr Nurs. 2005;31(5):416-21.
22. Saara Taponen, Hannu Martikainen, Marjo-Riitta Järvelin, Jaana Laitinen, Anneli Pouta, Anna-Liisa Hartikainen, Ulla Sovio, Mark I. McCarthy, Stephen Franks, Aimo Ruokonen. Hormonal Profile of Women with Self-Reported Symptoms of Oligomenorrhea and/or Hirsutism: Northern Finland Birth Cohort 1966 Study. The Journal of Clinical Endocrinology & Metabolism Vol. 88, No. 1 141-7.
23. Knochenhauer ES, Key TJ, Kahsar-Miller M, Waggoner W, Boots LR, Azziz R Prevalence of the polycystic ovary syndrome in unselected black and white women of the Southeastern United States: a prospective study. J Clin Endocrinol Metab 1998 83:3078–82.
24. Asuncion M, Calvo RM, San Millan JL, Sancho J, Avila S, Escobar-Morreale HF 2000 A prospective study of the prevalence of the polycystic ovary syndrome in unselected Caucasian women from Spain. J Clin Endocrinol Metab 85:2434–8.
25. Diamanti-Kandrakis E, Kouli CR, Bergiele AT, Filandra FA, Tsianateli TC, Spina GG, Zapanti ED, Bartzis MI 1999 A survey of the polycystic ovary syndrome in the Greek island of Lesbos: hormonal and metabolic profile. J Clin Endocrinol Metab 84:4006–11.
26. Dunaif, A, Segal, KR, Futterweit, W, Dobrjaosky, A. Profound peripheral insulin resistance, independent of obesity, in polycystic ovary syndrome. Diabetes 1989; 38:1165.
27. Ricardo Azziz. How Prevalent is Metabolic Syndrome in Women With Polycystic Ovary
28. Syndrome. Nat Clin Pract Endocrinol Metab. 2006;2(3).
29. Rosenfield Robert L,. Clinical features and diagnosis of polycystic ovary syndrome in adolescents. UpToDate of Medicine. version 10.3, August 2002.
30. The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Follow-up Report on the Diagnosis of Diabetes Mellitus. Diabetes Care 2003; 26: 3160-7.
31. Definition and diagnosis of diabetes mellitus and intermediate hyperglycemia: report of a WHO/IDF consultation. I. World Health Organization. II. International Diabetes Federation. ISBN 92 4 159493 4 (NLM classification: WK 810). ISBN 978 92 4 159493 6. © World Health Organization 2006.
32. Dahlgren E, Janson PO, Lapidus L, Oden A. Polycystic ovary syndrome and risk for myocardial infarction. Acta Obstet Gynecol Scand. 1992;71:599-604.
33. Ehrmann DA, Cavaghan MK, Barnes RB, et al. Prevalence of impaired glucose tolerance and diabetes mellitus in women with polycystic ovary syndrome. Diabetes Care. 1999;22:141-6.
34. Tsilchorozidou T, Overton C, Conway GS. The pathophysiology of polycystic ovary syndrome. Clin Endocrinol (Oxf) 2004;60:1-17.
35. Tasoula Tsilchorozidou, Caroline Overton and Gerard S. Conway. The Pathophysiology of Polycystic Ovary Syndrome. Clin Endocrinol 60(1):1-17, 2004.
36. Joyce King. Polycystic Ovary Syndrome. J Midwifery Womens Health. 2006; 51(6):415-22.
37. Lanzone, A., Caruso, A., Di Simone, N., De Carolis, S., Fulghesu, A.M. & Mancuso, S. (1995) Polycystic ovary disease. A risk factor for gestational diabetes? Journal of Reproductive Medicine, 40, 312-6.
38. Dahlgren, E., Johansson, S., Lindstedt, G., Knutsson, F., Oden, A., Janson, P.O., Mattson, L.A., Crona, N. & Lundberg, P.A. (1992) Women with polycystic ovary syndrome wedge resected in 1956-65: a long-term follow-up focusing on natural history and circulating hormones.Fertility and Sterility,57,505-8.
39. Carmina E. Diagnosing PCOS in women who menstruate regularly. Contemp Obstet Gynecol 2003;53-64.
40. Guzick DS. Polycystic ovary syndrome. Obstet Gynecol 2004;103:181-93.
41. Legro RS. (2003) Diagnostic criteria in polycystic ovary syndrome. Semin Reprod Med 2003;21:267-5.
42. Speroff L, Glass R, Kase NG. Anovulation and the polycystic ovary. Chapter 13. Clinical Gynecologic Endocrinology and Infertility, Fifth Edition. Philadelphia, Pa: Williams & Wilkins; 1994:457-82.
43. Barberi RL. Induction of ovulation in infertile women hyperandrogenism and insulin resistance. Am J Obstet Gynecol. 2000;183:1412-8

(Table-1)
The clinical and orthopometric studied parameters of PCOS and control groups.

Parameters	PCOS group	Control group	Notes
Infertility	100%	-----	
Hirsutism	100%	-----	
	`(Obesity) BMI		
Mean ± SD (kg/m^2)	**33.62 ± 5.59**	**25.40 ± 3.50**	**t-test (P < 0.05)**
< 25	2%	42%	Normal
25 – 29.9	26%	50%	Grade I obesity
30 – 39.9	64%	8%	Grade II obesity
> 40	8%	-----	Grade III obesity
	(Obesity) Waist/hip ratio		
Mean ± SD (%)	**0.710 ± 0.111**	**0.678 ± 0.068**	**t-test (P > 0.05)**
	0.72 % cut-off point (central obesity)		
< 0.72	46%	76%	No Central obesity
> 0.72	54%	24%	Central obesity
0.85 % cut-off point (central obesity associated with risk of cardiovascular disease)			
< 0.850	92%	98%	No Central obesity
> 0.850	8%	2%	Central obesity

(Table-2)
The biochemical glycemic parameters of PCOS and control groups.

Parameters	PCOS group	Control group	Notes
	FPG		
Mean ± SD (mg/dl)	**90.26 ± 12.45**	**82.86 ± 7.11**	**t-test (P > 0.05)**
< 100	80 %	100 %	Normal
100 - 125	18 %	-----	IFG, Prediabetic (ADA)
> 126	2 %	-----	Diabetic (ADA)
	OGTT (2 hours)		
Mean ± SD (mg/dl)	**124.18 ± 42.10**	**91.34 ± 6.85**	**t-test (P < 0.05)**
< 140	72 %	100 %	Normal
140 – 199	22 %	-----	IGT, Prediabetic (WHO)
> 200	6 %	-----	Diabetic (WHO)

(Table-3)
The hormonal assay of PCOS and control groups.

Parameters	PCOS group	Control group	Notes
Luteinizing Hormone (LH)			
Mean ± SD (IU/l)	**11.90 ± 3.35**	**2.93 ± 0.70**	**t-test (P < 0.05)**
Follicle-Stimulating Hormone (FSH)			
Mean ± SD (IU/l)	**7.57 ± 3.01**	**3.79 ± 0.87**	**t-test (P < 0.05)**
LH/FSH ratio			
Mean ± SD (%)	**1.71± 0.71**	**0.80 ± 0.22**	**t-test (P < 0.05)**
< 2	86%	100%	
> 2	14%	-----	
Testosterone			
Mean ± SD (ng/dl)	**112.4 ± 44.4**	**23.2 ± 10.5**	**t-test (P < 0.05)**
60 ng/dl (the upper normal limit)			
< 60	-----	100%	
> 60	100%	-----	
200 ng/dl (high level and sign of virilism or virilizing tumor)			
< 200	90%	100%	
> 200	10%	-----	

Al- Kindy Col Med J 2009; Vol .5 (1): P: 39

*From the Department of Medicine, National Diabetes Center (NDC), AL-Mustansiryia University, Baghdad

Correspondence Address to: Dr. Tawfeeq F. R. AL-Auqbi

Received at : 26-5-2008 **Accepted at:** 20-9-2008