## International Journal of Advanced Research in Biological Sciences ISSN: 2348-8069 www.ijarbs.com

**DOI: 10.22192/ijarbs** 

Coden: IJARQG(USA)

Volume 5, Issue 7 - 2018

**Research Article** 

2348-8069

DOI: http://dx.doi.org/10.22192/ijarbs.2018.05.07.004

# **Polycystic Ovary Syndrome**

Dr.Azhar safaa Alwardi

Gynecologest Dr.Rabiaa Adnan AlKaban

Gynecologest

Dr.Alaa Ghasoob Abid

Gynecologest Albatool Gyne. And Obest.Hospital / Baquba/ Iraq.

#### Abstract

Impaired glucose tolerance and other metabolic defects in women with polycystic ovary syndrome

Keywords: polycystic ovary, glucose tolerance, metabolic defect.

#### Introduction

Polycystic ovary syndrome (PCOS) is the most common endocrinopathy in women of reproductive age.<sup>1,2</sup> PCOS is mainly characterized by oligo- or clinical and/or anovulation. biochemical hyperandrogenism and polycystic ovaries and is the leading cause of anovulatory infertility.<sup>3,4</sup> However, PCOS is also associated with an array of metabolic disorders, among which impaired glucose metabolism has been a topic of intense research. Indeed, several cross-sectional and some prospective studies reported increased prevalence and incidence of impaired glucose tolerance (IGT) and type 2 diabetes mellitus (T2DM) in these patients.<sup>5.</sup>

### **Patients and Methods**

This was a case–control observational study carried out on women confirmed to have PCOS based on Rotterdam criteria (<sup>6</sup>) who attend gynecology consultation clinic in Alomara, Alazizeah, and Baqobq hospitals. Controls were selected from healthy woman attending birth control clinic.

For all patients detailed histories (menstrual, fertility, hirsutism, acne, greasy skin, acanthosis nigricance, scalp hair loss or thinning and family history of diabetes mellitus were taken. This was followed by a full examination including general and pelvic examinations, body weight (kg), and height (cm). The BMI was calculated by dividing the weight (in kg) by the height (in m) squared to assess obesity. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured with a sphygmomanometer.

Blood samples for baseline measurements were collected after an overnight fast on day 2 or day 3 of the menstrual cycle in the control group and after a spontaneous bleeding episode in the PCOS group or randomly in the case of amenorrhea. The circulating levels of total testosterone, estrogen and progesterone. Glucose, total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C) and triglycerides (TG) were also measured.

On the same day as blood samples were collected, transvaginal ultrasonography was performed and the volume of each ovary was determined, as well as the number of small follicles in each ovary.

Immediately after baseline blood sampling, an oral glucose tolerance test (OGTT) was performed in which glucose (75 g) was administered orally and serum glucose levels were determined at 0 and 120 min. A fasting plasma glucose (FPG) of <100 mg/dL (5.6 mmol/L) and 2-h glucose during OGTT <140 mg/dL (7.8 mmol/L) were accepted as normal values .

Categories of increased risk for diabetes (prediabetes) were defined as follows: impaired fasting glucose (IFG) was diagnosed when FPG was between 100 and 125 mg/dL (5.6–6.9 mmol/L) and IGT was diagnosed when the 2-h plasma glucose (2-h PG) value during a 75 g OGTT was between 140 and 199 mg/dL (7.8–11.0 mmol/L). Diabetes mellitus was confirmed by FPG 126 mg/dL (7.0 mmol/L), a 2-h PG value during a 75 g OGTT of 200 mg/dL (11.1 mmol/L), or a random PG concentration 200 mg/dL (11.1 mmol/L) in the presence of symptoms.<sup>7</sup>

The following exclusion criteria were applied: the presence of systemic disease that could alter insulin sensitivity (such as cardiovascular disease and diabetes mellitus); women on medication for 6 months prior to the study (including oral contraceptives, glucocorticoids, ovulation induction agents, and estrogenic or anti-androgenic drugs or any medication for dyslipidemia or anti-obesity drugs that could alter the patient's clinical presentation or hormonal profile); women with other endocrinological abnormalities such as primary hyperprolactinemia, thyroid dysfunction, and Cushing syndrome, adrenal congenital hyperplasia and androgen producing neoplasm.

#### Results

Finally we collect 536 patients with PCOS and 218 control. Glucose metabolism profiles were significantly different between the groups (P < 0.05); the PCOS group had higher values for 2-h glucose, and MetS than those for the control group. Lipid profiles also differed significantly between the two groups; the PCOS group had higher triglyceride, total cholesterol, low-density lipoprotein (LDL) levels and cardiovascular risk (TG/HDL) than the control group as shown in Table 1. The PCOS group was associated with a greater risk (2.5-times) of IR than the control group (P < 0.001)

Variables	PCOS	CONTROL	P-VALUE
Age (years)	$29.98 \pm 9.95$	27.43 ±8.56	
BMI (kg/m2)	$32.76\pm5.04$	24.65 ±6.97	< 0.001
Total testosterone (ng/mL)	$0.44\pm0.21$	$0.27\pm0.13$	< 0.001
Fasting glucose (FG) (mg/dL)	$93.79\pm79.55$	$60.85 \pm 39.24$	< 0.001
2-h glucose (2-h G)(mg/dL)	$126.44 \pm 46.75$	$107.86 \pm 23.12$	< 0.001
Normal GTT	62.6%	92.8%	< 0.001
Prediabetes (IFG and/or IGT)	32.8	6.1%	< 0.001
< 0.001			
Type 2 diabetes mellitus	4.6%	1.1%	< 0.001
Metabolic syndrome (MetS)	67.6%	27.7%	< 0.001
Total cholesterol (TC) (mg/dL)	$181.26 \pm 44.63$	$162.39 \pm 41.11$	< 0.001
Triglycerides (TG) (mg/dL)	$137.73 \pm 97.41$	$95.83 \pm 61.15$	< 0.001
HDL (mg/dL)	$31.79\pm9.89$	$43.69 \pm 9.68$	< 0.001
LDL (mg/dL)	$93.65\pm27.6$	$76.31 \pm 21.2$	< 0.001
Cardiovascular risk (TG/HDL)	65.7%	23.4%	< 0.001

Table 1The clinical, hormonal and metabolic parameters in patients with PCOS and control women

#### **Discussion and Conclusion**

In an early case-control study in 254 patients with PCOS and 80 age- and weight-matched controls, the prevalence of IGT was 2.7 times higher in the former (31.1 vs. 14.0%, respectively).6 Moreover, 7.5% of patients with PCOS had T2DM compared with none in the women in the control group.<sup>8</sup>

In a more recent large study in 11,035 patients with PCOS, the prevalence of T2DM was 2.45 times higher than in age matched controls.<sup>9</sup>

In a meta-analysis of 13 studies that compared the prevalence of IGT between patients with PCOS and controls, IGT was 2.48 times more frequent in the former.<sup>5</sup> Likewise, the prevalence of T2DM was 4.5 times higher in patients with PCOS than in controls in a meta-analysis of 15 studies5 and, importantly, these differences were similar in studies that included body mass index (BMI)-matched populations.<sup>5</sup> Of note, it has been estimated that 15.0-35.6% of all incident cases of T2DM in white women are attributable to PCOS.<sup>10</sup> Metabolic syndrome, which is associated with increased risk for T2DM,<sup>11</sup> is also more frequent in patients with PCOS,<sup>12.13,14.15</sup> while, in contrast, the prevalence of impaired fasting glucose or of HbA1c levels in the prediabetic range (i.e. between 5.7 and 6.4%) appears to be low in patients with PCOS.<sup>14,15-29</sup>.

Our data showed that impaired glucose tolerance, metabolic syndrome, and hyperlipidemia are prevalent in woman with POC, and need special attention and management to prevent future cardiovascular and macrovascular disease.

### References

1.Knochenhauer ES, Key TJ, Kahsar-Miller M, Waggoner W, Boots LR, Azziz R, 1998 Prevalence of the polycystic

ovary syndrome in unselected black and white women of the southeastern United States: a prospective study.

J Clin Endocrinol Metab 83: 3078-3082.

2. Diamanti-Kandarakis E, Kouli CR, Bergiele AT, et al, 1999 A survey of the polycystic ovary syndrome in the Greek island of lesbos: hormonal and metabolic profile. J Clin Endocrinol Metab 84: 4006-4011.

- 3. Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group, 2004. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. Fertil Steril 81: 19-25.
- 4. Azziz R, Carmina E, Dewailly D, et al, 2009 Task Force on the Phenotype of the Polycystic Ovary Syndrome of

The Androgen Excess and PCOS Society: the Androgen Excess and PCOS Society criteria for the polycystic

ovary syndrome: the complete task force report. Fertil Steril 91: 456-488.

- Moran LJ, Misso ML, Wild RA, Norman RJ, 2010 Impaired glucose tolerance, type 2 diabetes and metabolic syndrome in polycystic ovary syndrome: a systematic review and meta-analysis. Hum Reprod Update 16: 347-363.
- 6. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome.*Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group.Fertil Steril.* 2004 Jan; 81(1):19-25.
- 7. American Diabetes Association Standards of medical care in diabetes. Diabetes Care. 2013;1:S11. doi: 10.2337/dc13-S011
- 8. Legro RS, Kunselman AR, Dodson WC, Dunaif A, 1999 Prevalence and predictors of risk for type 2 diabetes mellitus and impaired glucose tolerance in polycystic ovary syndrome: a prospective. controlled study in 254 affected women. J Clin Endocrinol Metab 84: 165-169. 9. lo JC, Feigenbaum Sl, Yang J, Pressman AR, Selby JV, Go AS, 2006 Epidemiology and adverse cardiovascular risk profile of diagnosed polycystic ovary syndrome. J Clin Endocrinol Metab 91: 1357-1363. 10. Talbott EO, Zborowski JV, Rager JR, Kip KE, Xu X, Orchard TJ, 2007 Polycystic ovarian syndrome (PCOS): a significant contributor to the overall burden of type 2 diabetes in women. J Womens Health (larchmt) 16: 191-197.

11. Ford ES, Li C, Sattar N, 2008 Metabolic syndrome and incident diabetes: current state of the evidence. Diabetes Care31:1898-1904.

12. Cussons AJ, Watts GF, Burke V, Shaw JE, Zimmet PZ, Stuckey BG, 2008 Cardiometabolic risk in polycystic

ovary syndrome: a comparison of different approaches to defining the metabolic syndrome. Hum Reprod 23: 2352-2358.

13. Panidis D, Macut D, Tziomalos K, et al, 2013 Prevalence of metabolic syndrome in women with polycystic ovary syndrome. Clin Endocrinol (Oxf) 78: 586-592.

14. Lerchbaum E, Schwetz V, Giuliani A, Obermayer-Pietsch B, 2013 Assessment of glucose metabolism in polycystic ovary syndrome: HbA1c or fasting glucose compared with the oral glucose tolerance test as a screening method. Hum Reprod 28: 2537-2544.

15. Celik C, Abali R, Bastu E, Tasdemir N, Tasdemir UG, Gul A, 2013 Assessment of impaired glucose tolerance prevalence with hemoglobin A1c and oral glucose tolerance test in 252 Turkish women with polycystic ovary syndrome: a prospective, controlled study. Hum Reprod28:1062-1068.

16. Norman RJ, Masters I, Milner CR, Wang JX, Davies MJ, 2001 Relative risk of conversion from normoglycaemia to impaired glucose tolerance or non-insulin dependent diabetes mellitus in polycystic ovarian syndrome. Hum Reprod 16: 1995-1998.

17. Boudreaux MY, Talbott EO, Kip KE, Brooks MM, Witchel SF, 2006 Risk of T2DM and impaired fasting glucose among PCOS subjects: results of an 8-year follow-up. Curr Diab Rep 6: 77-83.

18. Legro RS, Gnatuk CL, Kunselman AR, Dunaif A, 2005 Changes in glucose tolerance over time in women with polycystic ovary syndrome: a controlled study. J Clin Endocrinol Metab 90: 3236-3242.

19. Gambineri A, Patton L, Altieri P, et al, 2012 Polycystic ovary syndrome is a risk factor for type 2 diabetes: results from a long-term prospective study. Diabetes 61: 2369-2374. 20. Morgan CL, Jenkins-Jones S, Currie CJ, Rees DA, 2012 Evaluation of adverse outcome in young women with polycystic ovary syndrome versus matched, reference controls: a retrospective, observational study. J Clin Endocrinol Metab 97: 3251-3260.

21. Carmina E, lobo RA, 2004 Use of fasting blood to assess the prevalence of insulin resistance in women with polycystic ovary syndrome. Fertil Steril 82: 661-665.

22. DeUgarte CM, Bartolucci AA, Azziz R, 2005 Prevalence of insulin resistance in the polycystic ovary syndrome using the homeostasis model assessment. Fertil Steril 83: 1454-1460.

23. Macut D, Micic D, Parapid B, et al, 2002 Age and body mass related changes of cardiovascular risk factors in women with polycystic ovary syndrome. Vojnosanit Pregl59:593-599.

24. Panidis D, Tziomalos K, Macut D, et al, 2012 Crosssectional analysis of the effects of age on the hormonal, metabolic, and ultrasonographic features and the prevalence of the different phenotypes of polycystic ovary syndrome.FertilSteril97:494-500.

25. Dunaif A, Segal KR, Futterweit W, Dobrjansky A, 1989 Profound peripheral insulin resistance, independent of obesity, in polycystic ovary syndrome. Diabetes 38: 1165-1174.

26. Macut D, Bjeki -Macut J, Raheli D, Dokni M, 2017 Insulin and the polycystic ovary syndrome. Diabetes Res Clin Pract 130: 163-170.

27. Stepto NK, Cassar S, Joham AE, et al, 2013 Women with polycystic ovary syndrome have intrinsic insulin resistance on euglycaemichyperinsulaemic clamp. Hum Reprod 28: 777-784.

28. Cassar S, Misso ML, Hopkins WG, Shaw CS, Teede HJ, Stepto NK, 2016 Insulin resistance in polycystic ovary syndrome: a systematic review and meta-analysis of euglycaemichyperinsulinaemic clamp studies. Hum Reprod31:2619-2631. 29. O'Meara NM, Blackman JD, Ehrmann DA, et al, 1993 Defects in beta-cell function in functional ovarian hyperandrogenism. J Clin Endocrinol Metab 76: 1241-1247.



How to cite this article: Azhar safaa Alwardi , Rabiaa Adnan AlKaban, Alaa Ghasoob Abid. (2018). Polycystic Ovary Syndrome . Int. J. Adv. Res. Biol. Sci. 5(7): 72-76. DOI: http://dx.doi.org/10.22192/ijarbs.2018.05.07.004