

# PREDICTORS OF ABNORMAL GLUCOSE TOLERANCE AMONG WOMEN WITH POLYCYSTIC OVARY SYNDROME

A Ra Koh, MD, Se Jin Lee, MD, Sue Yeon Park, MD, Min Kyung Kim, MD, Ji Ye Kim, MD, Kyo Won Lee, MD, Kye Hyun Kim, MD

Department of Obstetrics and Gynecology, Kangbuk Samsung Hospital, Sungkyunkwan University School of Medicine, Seoul, Korea

## Objective

To determine the parameters associated with the risk for abnormal glucose tolerance (AGT) among women with polycystic ovary syndrome (PCOS) and to assess the optimal screening tests to predict AGT within this population.

## Methods

We evaluated 85 women with PCOS and 53 control women. All participants had an oral glucose tolerance test (OGTT) and hormonal blood profiles, including the measurement of follicle stimulating hormone, leutinizing hormone, estradiol testosterone, and serum lipid profiles.

## Results

Among the women with PCOS, those with AGT had significantly higher homeostasis model assessment of insulin resistance ( $P < 0.001$ ) values than those with normal glucose tolerance. The prevalence of impaired glucose tolerance (IGT) and/or impaired fasting glucose was 48.2% (41/85) in women with PCOS; 16 of 41 subjects with AGT were IGT. Six of 16 subjects (37.5%) with IGT had normal fasting plasma glucose (FPG < 100 mg/dL). Thus, the FPG failed to detect 37.5% of women with PCOS who were found to have AGT with the OGTT. Multivariate logistic regression analysis revealed that insulin, body mass index (BMI), age, and triglyceride (TG) were significant risk factors for abnormal glucose metabolism.

## Conclusion

Insulin, BMI, age, and TG predicted abnormal glucose metabolism in women with PCOS. The OGTT was a more reliable predictor of AGT than fasting plasma glucose. We recommend that women with PCOS undergo periodic screening for AGT using the OGTT, particularly if they have any of the above risk factors.

**Keywords:** Polycystic ovarian syndrome; Oral glucose tolerance test; Prevalence; Risk factors

Polycystic ovary syndrome (PCOS) is characterized by hyperandrogenism and chronic anovulation, and it is one of the most common endocrine disorders, affecting 5% to 10% of reproductive age women [1]. Although the etiology of the syndrome is complex, insulin resistance and hyperinsulinemia are thought to play a major role in the pathophysiology of PCOS. Insulin resistance is thought to be caused by obesity, although recent studies have found that underweight women with PCOS are more likely to show insulin resistance than a normal control group. These results suggest that PCOS is independent of body weight for underweight women, although it is widely accepted that obesity is a key factor in insulin resistance in the general population. Prevalence rates

Received: 2011.11.29. Revised: 2012.3.27. Accepted: 2012.4.26.  
Corresponding author: Kye Hyun Kim, MD, PhD  
Division of Gynecologic Endocrinology, Department of Obstetrics and Gynecology, Kangbuk Samsung Hospital, Sungkyunkwan University School of Medicine, 29 Saemunan-ro, Jongno-gu, Seoul 110-746, Korea  
Tel: +82-2-2001-2457 Fax: +82-2-2001-2187  
E-mail: khmd.kim@samsung.com

This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (<http://creativecommons.org/licenses/by-nc/3.0/>) which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

Copyright © 2012. Korean Society of Obstetrics and Gynecology

for insulin resistance, impaired glucose tolerance (IGT), and type 2 diabetes among women with PCOS are higher than expected for women of similar age, reaching 50% to 70%, 30% to 40%, and 10%, respectively [2,3]. Although glucose intolerant patients do not have symptoms, glucose intolerance accelerates the development of type 2 diabetes and cardiovascular diseases, so these patients should be screened to ensure early detection of these disorders. There is considerable disagreement on when and how screening should be conducted. The fasting glucose test has been widely used to detect abnormal glucose tolerance (AGT) because it is convenient and inexpensive. However, many women with PCOS who take the fasting glucose test have normal fasting glucose levels, and this test failed to detect 58% of those who were diagnosed with type 2 diabetes through the glucose test [4].

The purpose of this study was to determine the risk factors for AGT among women with PCOS and to assess the optimal screening tests to predict AGT within this population.

## Materials and Methods

### 1. Subjects

This study was conducted from August 2009 to May 2010 in the Department of Obstetrics and Gynecology at Kangbuk Samsung Hospital. We enrolled 85 women with PCOS, as well as 53 healthy women who were a similar age and body mass index (BMI) as the women with PCOS. The control group had regular menstrual cycles and no symptoms of hyperandrogenism, diabetes, and high blood pressure. They also had no history of cardiovascular disease and no family history of diabetes.

The diagnosis of PCOS was made according to the Rotterdam criteria, and patients were diagnosed with PCOS when at least two of the three following criteria were met: 1) the presence of cycle abnormalities, namely oligomenorrhea (6 or fewer menses per year) or amenorrhea (more than three months between menses); 2) clinical and/or biochemical evidence of hyperandrogenism; 3) enlarged ovaries containing at least twelve small (2 to 9 mm) follicles per ovary [5,6]. Other conditions with similar clinical manifestations, such as 21-hydroxylase deficiency, Cushing syndrome, hypothyroidism, hyperprolactinemia, and androgen-secreting tumors, were ruled out.

We used Choo's [7] categorization system for Asians to classify subjects according to BMI: underweight, less than 18.4 kg/m<sup>2</sup>; normal, between 18.5 kg/m<sup>2</sup> and 22.9 kg/m<sup>2</sup>; overweight, between 23.0 kg/m<sup>2</sup> and 24.9 kg/m<sup>2</sup>; obese, between 25.0 kg/m<sup>2</sup> and 29.9

kg/m<sup>2</sup>; extremely obese=over 30.0 kg/m<sup>2</sup>. Participants were excluded from the study if they had a history of glucose intolerance (including gestational diabetes) or non-insulin-dependent diabetes mellitus (NIDDM) hyperprolactinemia, thyroid dysfunction, late onset congenital adrenal hyperplasia, or Cushing's syndrome. We also excluded women who were taking medications that could alter their hormonal or biochemical profiles. No patients included in the study were pregnant. This study was approved by the Kangbuk Samsung Hospital Institutional Review Board, and all participants provided written informed consent.

### 2. Diagnostic method

Overnight fasting blood samples were taken between days 2 and 5 of the menstrual cycle, if present. Hormonal and biochemical analyses included the measurement of glucose, lipid, insulin, testosterone, sex hormone-binding globulin (SHBG), leutinizing hormone (LH), and follicle stimulating hormone (FSH). Blood samples for an oral glucose tolerance test (OGTT) were obtained at 30-minute intervals over two hours to measure glucose after ingestion of a standard 75 g of glucose.

The OGTT was performed in accordance with the criteria of the American Diabetes Association (ADA) [8]. Participants were considered to have AGT if they had either impaired fasting glucose (IFG), IGT, or overt diabetes mellitus (DM). IFG was defined as an elevated fasting plasma glucose (FPG) concentration between 100 mg/dL and 125 mg/dL, in accordance with the criteria of the ADA. IGT was defined as a plasma glucose level between 140 mg/dL and 200 mg/dL after a 75 g glucose load on the OGTT. Overt DM was defined as a plasma glucose level of 200 mg/dL or greater. Since the above definitions led to overlap between the two groups, we also determined which participants had combined glucose intolerance (CGI), which was defined as the presence of both IFG and IGT. Normal glucose tolerance (NGT) was defined as an FPG below 100 mg/dL and a 2-hour plasma glucose level below 140 mg/dL.

Insulin levels were measured with immunoradiometric assay (Dia Source, Nivelles, Belgium). Blood glucose was measured with the hexokinase method. Insulin resistance and  $\beta$ -cell function were calculated as: higher homeostasis model assessment (HOMA) of insulin resistance (fasting insulin [mU/L]  $\times$  fasting glucose [mmol/L] / 22.5). BMI was calculated as body weight (kg) divided by body height squared (m<sup>2</sup>). Serum total testosterone, free testosterone, dehydroepiandrosterone-sulphate (DHEA-S) SHBG, FSH, LH, and estradiol were measured with radioimmunoassay (RIA) methods (Siemens, Los Angeles, CA, USA). The free androgen index was

calculated as: (total testosterone/SHBG) × 3.47%. An enzymatic colorimetric test was used to measure total cholesterol, fasting triglyceride (TG), high-density lipoprotein cholesterol (HDL-cholesterol), and low-density lipoprotein cholesterol (LDL-cholesterol). High sensitivity C-reactive protein levels were measured with immunonephelometry (Dade Behring Co., Marburg, Germany).

### 3. Statistical analysis

Statistical analyses were performed with SPSS ver. 12.0 (SPSS Inc., Chicago, IL, USA). Mean values are ± SEM unless otherwise indicated. Differences between groups were assessed with the independent t-test, analysis of variance, and Mann-Whitney U test. Differences in frequencies were tested with the chi-square test. Multivariate logistic regression analysis was used to determine

which variables predicted glucose intolerance. *P*-values < 0.05 were considered statistically significant.

## Results

Table 1 depicts the clinical and biochemical characteristics of the two groups. Glucose metabolism profiles were significantly different between groups; the women with PCOS had higher values for fasting glucose, 2-hour post glucose, 2-hour post insulin, and HOMA than control women. Lipid profiles also differed between groups; the women with PCOS had lower HDL and higher triglyceride levels. Likewise, reproductive hormone profiles differed between groups; the women with PCOS had higher values for LH,

**Table 1.** Clinical and biochemical characteristics of women with PCOS and control women

Characteristics	PCOS (n = 85)	Control (n = 53)	<i>P</i> -value
Age (yr)	25 (21.5–28.5)	26 (24–31.5)	NS
BMI (kg/m <sup>2</sup> )	20.45 (18.79–22.87)	19.9 (18.7–21.3)	NS
Glucose metabolism profile			
Fasting glucose (mg/dL)	98 (91.5–103.5)	93 (89–96)	<0.001
2-hour post glucose (mg/dL)	111.45 ± 27.84	96.83 ± 15.50	<0.001
Fasting insulin (uIU/mL)	10.74 (7.75–14.20)	9.97 (7.40–11.93)	NS
2-hour post insulin (uIU/mL)	36.85 (25.02–59.22)	22.7 (14.5–29.1)	<0.001
HOMA	2.50 (1.77–3.42)	2.36 (1.51–2.72)	0.021
Lipid profile			
T-cholesterol (mg/dL)	183.55 ± 30.76	177.02 ± 27.53	NS
LDL-cholesterol (mg/dL)	102.08 ± 28.33	93.20 ± 21.98	NS
HDL-cholesterol (mg/dL)	59 (53.5–68)	66 (62–73.5)	0.002
Fasting triglycerides (mg/dL)	76 (60–98)	63 (49–79)	0.001
Reproductive hormone profile			
LH (mIU/mL)	8.87(5.72–12.18)	4.02 (2.38–6.29)	<0.001
FSH (mIU/mL)	4.64 (3.41–5.74)	4.41 (3.26–6.15)	NS
E2 (pg/mL)	75.52 (57.00–116.88)	82.4 (47.8–145.3)	NS
Total testosterone (ng/mL)	0.51 ± 0.26	0.28 ± 0.13	<0.001
Free testosterone (pg/mL)	1.50 (0.74–2.30)	0.62 (0.47–1.00)	<0.001
DHEA-S (umol/L)	222.91 ± 117.87	162.12 ± 73.49	<0.001
SHBG (nmol/L)	67.04 ± 35.04	85.23 ± 21.99	0.001
FAI	2.54 (1.45–4.79)	1.06 (0.75–1.61)	<0.001
HS-CRP (mg/dL)	0.06 (0.02–0.17)	0.02 (0.01–0.04)	<0.001

Values are presented as mean ± standard deviation or median (25th to 75th percentile).

PCOS, polycystic ovary syndrome; BMI, body mass index; NS, not significant; HOMA-IR, homeostasis model assessment of insulin resistance; T-cholesterol, total-cholesterol; LDL, low-density lipoprotein; HDL, high-density lipoprotein; LH, leutinizing hormone; FSH, follicle stimulating hormone; E2, estradiol; DHEA-S, dehydroepiandrosterone-sulfate; SHBG, sex hormone-binding globulin; FAI, free androgen index; HS-CRP, high-sensitivity C-reactive protein.

total testosterone, free testosterone, high-sensitivity C-reactive protein (HS-CRP), and DHEA-S than control women. Table 2 depicts the prevalence of AGT among women with PCOS

and control women. AGT was present in 48.2% (41/85) of the women with PCOS. Among the women with PCOS who had AGT, 60.9% (25/41) had IFG, 14.6% (6/41) had IGT, and 24.4% (10/41)

**Table 2.** Prevalence of AGT among women with PCOS and control women

Group	PCOS (n=85)	Control (n=53)	P-value
NGT	44 (51.8)	47 (88.7)	<0.001
AGT			
Isolated IFG	25 (29.4)	5 (9.4)	<0.001
Isolated IGT	6 (7.0)	1 (1.9)	<0.001
CGI	10 (11.8)	0 (0)	<0.001

Values are presented as frequency (%).

AGT, abnormal glucose tolerance; PCOS, polycystic ovary syndrome; NGT, normal glucose tolerance; IFG, impaired fasting glucose; IGT, impaired glucose tolerance; CGI, combined glucose intolerance.

**Table 3.** Clinical and biochemical characteristics of the women with PCOS in relation to glucose tolerance status

Characteristics	AGT (n=41)	NGT (n=44)	P-value
Age (yr)	27 (21–30)	24.5 (22–27.7)	0.048
BMI (kg/m <sup>2</sup> )	21.3 (18.94–25.48)	20.01 (18.30–21.25)	0.017
Glucose metabolism profile			
Fasting glucose (mg/dL)	104 (100.5–106.5)	93 (89.25–96.75)	<0.001
2-hour post glucose (mg/dL)	125.83 ± 30.912	98.05 ± 15.63	<0.001
Fasting insulin (uIU/mL)	12.82 (8.58–15.88)	8.49 (6.93–12.39)	<0.001
2-hour post insulin (uIU/mL)	45.8 (30.7–106.55)	29 (20.9–50.9)	0.001
HOMA	3.29 (2.17–3.94)	1.81 (1.59–2.79)	<0.001
Lipid profile			
T-cholesterol (mg/dL)	186.10 ± 33.60	178.42 ± 27.18	0.150
LDL cholesterol (mg/dL)	104.45 ± 32.70	95.69 ± 22.00	0.064
HDL-cholesterol (mg/dL)	58 (51–65)	66 (58–73)	0.001
Fasting triglycerides (mg/dL)	86 (67–118)	66 (50–80)	<0.001
Reproductive hormone profile			
LH (mIU/mL)	7.10 (5.10–10.11)	10.21 (7.47–13.99)	0.006
FSH (mIU/mL)	4.46 (3.12–5.46)	4.83 (3.87–6.02)	0.122
E2 (pg/mL)	75.95 (62.89–117.52)	75.25 (54.08–105.62)	0.356
Total testosterone (ng/mL)	0.54 ± 0.290	0.48 ± 0.23	0.273
Free testosterone (pg/mL)	1.55 (0.80–2.37)	1.20 (0.65–1.90)	0.123
DHEA-S (umol/L)	217.11 ± 122.17	228.32 ± 114.90	0.668
SHBG (nmol/L)	65.67 ± 30.44	68.20 ± 39.12	0.766
Free androgen index	2.63 (1.83–4.47)	2.40 (1.25–5.24)	0.804
HS-CRP (mg/dL)	4.46 (3.12–5.46)	0.04 (0.02–0.11)	0.034

Values are presented as mean ± standard deviation or median (25th to 75th percentile).

PCOS, polycystic ovary syndrome; AGT, abnormal glucose tolerance; NGT, normal glucose tolerance; BMI, body mass index; HOMA-IR, homeostasis model assessment of insulin resistance; T-cholesterol, total-cholesterol; LDL, low-density lipoprotein; HDL, high-density lipoprotein; LH, leutinizing hormone; FSH, follicle stimulating hormone; E2, estradiol; DHEA-S, dehydroepiandrosterone-sulfate; SHBG, sex hormone-binding globulin; HS-CRP, high-sensitivity C-reactive protein.

had both IFG and IGT (i.e., CGI). In addition, 16 women had IGT (isolated IGT+CGI). Of the women with PCOS who had IGT, 6 had isolated IGT with normal fasting glucose levels. The fasting glucose test identified 25 women with isolated IFG and 10 women with CGI, but it did not identify the remaining 6 women with AGT.

Table 3 depicts the clinical and biochemical characteristics of the 44 women with PCOS who had NGT and the 41 (48.2%) women with PCOS who had AGT. The glucose metabolism profiles were significantly different between groups. In addition, the women with AGT had significantly lower values for HDL and significantly higher values for TG than the women with NGT. With regards to reproductive hormones, women with AGT had significantly lower values for LH and significantly higher values for HS-CRP.

Multivariable logistic regression analysis identified several risk factors for AGT among women with PCOS (Table 4), including age (OR, 1.137; 95% CI, 1.022-1.266), BMI (OR, 1.358; 95% CI, 1.057-1.744), insulin (OR, 1.268; 95% CI, 1.070-1.502), and TG (OR, 1.022; 95% CI, 1.002-1.042).

## Discussion

In our study of 85 women with PCOS, the prevalence of IGT (isolated IGT + CGT) was 18.9% (16/85). This prevalence rate is similar to that of Chinese (20.5%), Thai (20.3%), and Mediterranean (15.7%) women with PCOS but lower than that of American women with PCOS (31%) [3,9-11].

In a previous study, 48.6% (17/35) of women with PCOS and NGT developed IGT at a rate of 16% per year, and 2% developed DM every year [12]. Another study followed up 67 PCOS patients for 6.2 years and found that 9% (5/54) of women who were normoglycemic at baseline developed IGT and 8% (4/54) developed DM. Of

the women with IGT at baseline, 54% (7/13) had developed DM at the end of the follow-up period [13]. In another recent study of 83 women with PCOS, 24.1% developed AGT during a median follow-up of 3 years, including 3.6% who developed DM [14].

The prevalence of glucose intolerance and type 2 diabetes is high among both adults and adolescents with PCOS [15]. In a previous study of 141 adults and 62 adolescents with PCOS, the prevalence of AGT among adolescents was more than twice that of adults (19.1% and 9.7%, respectively;  $P=0.03$ ). In addition, many of the adolescents with PCOS experienced deterioration of glucose tolerance before the age of forty, even though these individuals had NGT during adolescence [16]. These results highlight the importance of periodically screening women with PCOS for AGT.

The ADA has recommended that practitioners consider screening women with PCOS for abnormal glucose metabolism [15,17]. Our study indicates that such widespread screening is indeed warranted. Screening is especially important because recent data suggest that early intervention can prevent DM and can mitigate the effects of insulin resistance and attendant cardiovascular risk factors [17]. If prediabetes and diabetes could be diagnosed earlier, this could lead to better treatments and improved outcomes. In prediabetes, there is irrefutable evidence suggesting that lifestyle changes and pharmacological therapies can reduce or delay the development of type 2 DM by 30% to 50% [18,19]. However, for screening to be practical in a clinic setting, simple techniques are needed to screen the large population of individuals with PCOS for IGT and/or DM.

Both the ADA and WHO recommend using FPG for the primary screening process because FPG is more convenient to patients, less costly, more reproducible, and easier to administer than the 2-hour OGTT [17]. Our study compared the clinical and metabolic characteristics of women with PCOS who had NGT or AGT.

**Table 4.** Logistic regression analysis of the risk factors for AGT among women with PCOS

Characteristics	Odds ratio (95% confidence interval)	P-value
Age (yr)	1.137 (1.022–1.266)	0.019
BMI (kg/m <sup>2</sup> )	1.358 (1.057–1.744)	0.016
Insulin	1.268 (1.070–1.502)	0.006
HDL	0.993 (0.948–1.040)	0.755
HS-CRP	6.662 (0.092–484.664)	0.386
LH	1.011 (0.896–1.141)	0.857
TG	1.022 (1.002–1.042)	0.027

Values are presented as median and inter-quartile range.

AGT, abnormal glucose tolerance; PCOS, polycystic ovary syndrome; BMI, body mass index; HDL, high-density lipoprotein; HS-CRP, high-sensitivity C-reactive protein; LH, leutinizing hormone; TG, triglyceride.

We found that the fasting glucose test failed to identify 6 (7%) women with IGT; these women would have been considered NGT if we had not performed the OGTT. Thus, our study highlights the importance of using another screening test in addition to the fasting glucose test to identify women with IFG, IGT and CGT. Similar results were obtained in another study showing that most women with PCOS have fasting glucose levels within the normal range [4]. In that study, the fasting glucose test failed to detect 58% of women diagnosed with type 2 diabetes with the glucose tolerance test. Likewise, the authors of another study argued that the FPG is insufficient as a screening test because the sensitivity of any screening test should be at least 80%, yet the FPG was unable to detect 52% of women with PCOS who had AGT [20].

Thus, the fasting glucose test appears to be too insensitive to determine which patients have IGT. Women with PCOS could instead be screened for abnormal glucose metabolism with an OGTT using the 2-hour glucose concentration after a 75 g glucose challenge. However, the OGTT is controversial because it is more time consuming and costly. One possible solution to this problem is to screen women with the OGTT only when they have particular risk factors for AGT because this would increase detection accuracy while decreasing expenses.

In large population studies, older age, BMI, an unfavorable body fat distribution, and parameters of insulin sensitivity and secretion during the OGTT predict the development of DM [12,21]. Compared with the general population, our study population was leaner and did not include many obese women. A previous study found that obesity, high blood pressure, high TG level, high waist-to-hip ratio, and a family history of diabetes are important risk factors for AGT [20,22].

In another study, women with PCOS and AGT had higher testosterone levels and lower SHBG levels than patients with NGT. The higher androgen and lower SHBG levels may result from their more pronounced insulin resistance, since there is good evidence that compensatory hyperinsulinemia amplifies androgen production in PCOS and inhibits SHBG synthesis by the liver [23]. However, our study indicates that age, BMI, insulin and TG are significant risk factors for AGT among women with PCOS. If women with PCOS are stratified by these risk factors, then the screening guidelines for diabetes could be refined to increase detection accuracy and reduce costs.

In conclusion, we found that age, insulin, BMI and TG were predictors of abnormal glucose metabolism in women with PCOS. We also found that an OGTT was a more reliable predictor of abnormal glucose metabolism than fasting plasma glucose. We

recommend periodic screening for AGT using an OGTT for women with PCOS who have any of the above risk factors. Future studies are needed to clarify the optimal timing and best practices for AGT screening among women with PCOS.

## References

1. Azziz R, Woods KS, Reyna R, Key TJ, Knochenhauer ES, Yildiz BO. The prevalence and features of the polycystic ovary syndrome in an unselected population. *J Clin Endocrinol Metab* 2004;89:2745-9.
2. Ehrmann DA, Barnes RB, Rosenfield RL, Cavaghan MK, Imperial J. Prevalence of impaired glucose tolerance and diabetes in women with polycystic ovary syndrome. *Diabetes Care* 1999;22:141-6.
3. Legro RS, Kunesman AR, Dodson WC, Dunaif A. 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* 1999;84:165-9.
4. Salley KE, Wickham EP, Cheang KI, Essah PA, Karjane NW, Nestler JE. Glucose intolerance in polycystic ovary syndrome: a position statement of the Androgen Excess Society. *J Clin Endocrinol Metab* 2007;92:4546-56.
5. Rotterdam ESHRE/ASRM-Sponsored PCOS consensus workshop group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome (PCOS). *Hum Reprod* 2004;19:41-7.
6. Ferriman D, Gallwey JD. Clinical assessment of body hair growth in women. *J Clin Endocrinol Metab* 1961;21:1440-7.
7. Choo V. WHO reassesses appropriate body-mass index for Asian populations. *Lancet* 2002;360:235.
8. American Diabetes Association. The American Diabetes Association (ADA) has been actively involved in the development and dissemination of diabetes care standards, guidelines, and related documents for many years. Introduction. *Diabetes Care* 2009;32 Suppl 1:S1-2.
9. Weerakiet S, Srisombut C, Bunnag P, Sangtong S, Chuangsoongnoen N, Rojanasakul A. Prevalence of type 2 diabetes mellitus and impaired glucose tolerance in Asian women with polycystic ovary syndrome. *Int J Gynaecol Obstet* 2001;75:177-84.
10. Chen X, Yang D, Li L, Feng S, Wang L. Abnormal glucose tolerance in Chinese women with polycystic ovary syndrome. *Hum*

- Reprod 2006;21:2027-32.
11. Gambineri A, Pelusi C, Manicardi E, Vicennati V, Cacciari M, Morselli-Labate AM, et al. Glucose intolerance in a large cohort of mediterranean women with polycystic ovary syndrome: phenotype and associated factors. *Diabetes* 2004;53:2353-8.
  12. Legro RS, Gnatuk CL, Kunselman AR, Dunaif A. Changes in glucose tolerance over time in women with polycystic ovary syndrome: a controlled study. *J Clin Endocrinol Metab* 2005;90:3236-42.
  13. Norman RJ, Masters L, Milner CR, Wang JX, Davies MJ. Relative risk of conversion from normoglycaemia to impaired glucose tolerance or non-insulin dependent diabetes mellitus in polycystic ovarian syndrome. *Hum Reprod* 2001;16:1995-8.
  14. Pesant MH, Baillargeon JP. Clinically useful predictors of conversion to abnormal glucose tolerance in women with polycystic ovary syndrome. *Fertil Steril* 2011;95:210-5.
  15. Palmert MR, Gordon CM, Kartashov AI, Legro RS, Emans SJ, Dunaif A. Screening for abnormal glucose tolerance in adolescents with polycystic ovary syndrome. *J Clin Endocrinol Metab* 2002;87:1017-23.
  16. Bhattacharya SM. Abnormal glucose tolerance in polycystic ovary syndrome. *J Obstet Gynaecol Res* 2008;34:228-32.
  17. American Diabetes Association. Standards of medical care in diabetes: 2007. *Diabetes Care* 2007;30 Suppl 1:S4-41.
  18. Knowler WC, Barrett-Connor E, Fowler SE, Hamman RF, Lachin JM, Walker EA, et al. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med* 2002;346:393-403.
  19. Tuomilehto J, Lindström J, Eriksson JG, Valle TT, Hämäläinen H, Ilanne-Parikka P, et al. Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *N Engl J Med* 2001;344:1343-50.
  20. Gagnon C, Baillargeon JP. Suitability of recommended limits for fasting glucose tests in women with polycystic ovary syndrome. *CMAJ* 2007;176:933-8.
  21. Saad MF, Knowler WC, Pettitt DJ, Nelson RG, Mott DM, Bennett PH. The natural history of impaired glucose tolerance in the Pima Indians. *N Engl J Med* 1988;319:1500-6.
  22. Lillioja S, Mott DM, Spraul M, Ferraro R, Foley JE, Ravussin E, et al. Insulin resistance and insulin secretory dysfunction as precursors of non-insulin-dependent diabetes mellitus. Prospective studies of Pima Indians. *N Engl J Med* 1993;329:1988-92.
  23. Möhlig M, Spranger J, Ristow M, Pfeiffer AF, Schill T, Schlösser HW, et al. Predictors of abnormal glucose metabolism in women with polycystic ovary syndrome. *Eur J Endocrinol* 2006;154:295-301.

## 다낭성 난소증후군을 가진 여성에서 비정상적인 당내성을 일으키는 예측인자

성균관대학교 의과대학 강북삼성병원 산부인과교실

고아라, 이세진, 박수연, 김민경, 김지예, 이교원, 김계현

### 목적

다낭성 난소증후군 여성에서 비정상적인 당내성을 일으키는 위험인자를 알아보고 비정상 당내성을 예측하기 위한 적절한 선별검사를 평가하고자 한다.

### 연구방법

2009년 8월부터 2010년 1월까지 다낭성 난소증후군 85명과 정상대조군 53명의 여성을 대상으로 경구당부하검사를 실시하여 혈당과 인슐린, 혈중 생식호르몬 농도와 혈중 지질 profile을 측정하였다.

### 결과

다낭성 난소증후군 환자에서 비정상 당내성을 가진 41명 중 6명에 해당하는 14.6%는 공복혈당검사에서 정상 소견을 보였다. 비정상 당내성의 의미 있는 위험인자는 인슐린, body mass index, 나이, 그리고 중성지방임을 평가할 수 있었다.

### 결론

비정상 당내성을 가진 환자의 선별검사로는 공복혈당검사보다 경구당부하검사가 더 신뢰할 수 있다. 따라서 위험인자를 가진 다낭성 난소증후군 여성은 비정상 당내성을 선별하기 위해서 주기적으로 경구당부하검사를 실시하여야 한다.

**중심단어:** 다낭성 난소증후군, 비정상 당내성, 경구당부하검사, 위험인자