

Research Article

Altered Blood Glucose Levels in Polycystic Ovary Syndrome

Dr. Veena H.C¹, Dr. Reshmarani^{2*}, Dr. Shilpa. N³

¹Tutor, Department of Physiology, Hassan Institute Of Medical Sciences, Hassan, Karnataka, India

²Assistant Professor, Department of Physiology, Mall Reddy Medical College For Women, Hyderabad.

³Assistant Professor, Department of Physiology, Khaja Banda Nawaz Institute Of Medical Sciences, Gulbarga.

***Corresponding author**

Dr. Reshmarani

Email: reshmashivakumar@gmail.com

Abstract: Polycystic ovary syndrome (PCOS) is one of the most common endocrine disorder affecting 5-7% women of reproductive age. The etiology of PCOS is multifactorial. It is associated with menstrual dysfunction and subfertility. Hyperinsulinemia and insulin resistance are commonly associated with PCOS. Insulin resistance is now recognized as a major risk factor for development of hyperglycemia and type 2 diabetes mellitus. The Aims of present study is to know the blood glucose levels in women of younger age group with PCOS. A case control study was conducted in 50 non diabetic PCOS patients (15 to 25 years age) and 50 non diabetic normal menstruating women, matched for age and anthropometric data. 2 ml of fasting blood sample was collected for fasting blood sugar (FBS). 75 gram of glucose was given with 300ml of water to drink. After 2 hours, blood sample was collected for post prandial blood sugar (PPBS). Statistical analysis was done by using student 't' test. FBS and PPBS after 2 hour OGTT were significantly raised in PCOS patients compared to normal menstruating women. Periodic screening of PCOS patients should be carried out as they are more prone to develop glucose intolerance and diabetes mellitus and failing to do so the patient may end up in cardiovascular and other complication.

Keywords: Polycystic ovary syndrome (PCOS), diabetes mellitus, blood glucose, Hyperinsulinemia.

INTRODUCTION

Polycystic ovary syndrome (PCOS) is believed to constitute the most frequently encountered endocrinopathy in women of reproductive age present in 5 – 7% of women of reproductive age [1]. Although the first description of polycystic ovary syndrome (PCOS) is generally credited to Stein and Leventhal in 1935, it may have been observed as early as 1721, when the Italian scientist Antonio Vallisneri observed “young married women, moderately obese and infertile, with two larger than normal ovaries, bumpy and shiny, whitish, just like pigeon eggs. [2]. This depiction sounds strikingly similar to the subfertility and obesity commonly found in PCOS. It was not until 1921 that Achard noticed a relationship between hyperandrogenism and insulin resistance in their study of the “bearded diabetic woman.” And in 1935, Stein and Leventhal made the connection between amenorrhea and polycystic ovaries. In addition, they also noticed the occurrence of masculinizing changes, such as hirsutism and acne, in many patients with polycystic ovaries [3].

Diagnosis of PCOS is made according to Rotterdam criteria in presence of at least two of the following: 1) oligomenorrhea and/or anovulation; 2)

hyperandrogenism (clinical and/or biochemical); 3) polycystic ovaries with the exclusion of other etiologies. Women with PCOS demonstrate marked clinical heterogeneity; the commonly associated features of hirsutism, acne, polycystic-appearing ovaries, obesity and acanthosis nigricans are neither uniform nor universal [1]. Since its description in 1935 by Stein and Leventhal [3], much has been learned about the pathophysiology of PCOS from its neuroendocrine underpinnings [4] to an ever-growing understanding of the link between obesity, insulin resistance (IR) and PCOS [5]. In time the disorder may lead to onset of hyperinsulinemia, insulin resistance, gestational diabetes, early onset of type 2 diabetes mellitus (DM), dyslipidemia and cardiovascular disease (CVD) ¹. Hence this study is undertaken to know the blood glucose levels in PCOS subjects.

MATERIALS AND METHODS

The study was undertaken in Hassan Institute of Medical Sciences Hassan. It is a cross sectional study. The subjects for the study were selected from the out-patient and in –patient department of obstetrics and gynaecology. The women between 15-25 years who were fulfilling the criteria for PCOS were considered as cases. Age and anthropometrically matched women

with normal menstrual cycles were selected as controls. In both groups 50 subjects were selected. Women with disorders like pre-existing diabetes, hypertension, thyroid abnormalities or other ovarian or uterine problems were excluded from the study. For this study the ethical committee permission was taken. Informed consent was taken from the all the subjects. After obtaining a detailed history of all subjects the height and weight of the subjects were measured to calculate body mass index (BMI). The vital parameters like pulse and blood pressure were measured and ultrasonography of abdomen and pelvis was done to look for polycystic ovaries. Women fulfilling the criteria for PCOS were taken as cases. Women in both groups were advised to come empty stomach in the morning after overnight fasting. In the early morning under aseptic precaution 2ml of venous blood was taken for estimation of fasting blood sugar (FBS) level, this was followed by oral

glucose tolerance test(OGTT), by giving 75gm of glucose with 300ml of water. After 2 hours once again 2ml of venous blood was taken under aseptic precaution for estimation of post prandial blood sugar level(PPBS). The results were expressed in terms of mean \pm SD. The test of significance used was student 't' test and a p value less than 0.05 was considered statistically significant.

RESULTS

The study included 100 subjects, 50 cases and 50 controls. Table 1 shows the basic anthropometric parameters of both groups and there is no stastically significant difference of BMI between two groups. The table 2 shows the value of FBS and PPBS in cases and controls. The results of our study shows raised levels of FBS and PPBS in women with PCOS compared to controls.

Table 1: Anthropometric parameters of cases and controls

Parameters	Cases Mean \pm SD	Controls Mean \pm SD	't' value	'P' value
Age (years)	20.02 \pm 0.55	19.38 \pm 0.53	0.131	> 0.05*
BMI (kg/M ²)	22.36 \pm 0.73	22.43 \pm 0.62	0.123	> 0.05

Table 2: FBS and PPBS of cases and controls

Parameter	Group (Mean \pm SD)		Mean difference	95% CI of difference	t-value	p-value
	Study	Control				
FBS	92.11 \pm 5.21	87.16 \pm 5.57	4.95	2.8 – 7.09	4.58	P=0.0001
PPBS	129.15 \pm 7.6	120.11 \pm 8.23	9.04	5.89 – 12.18	5.7	P=0.0001

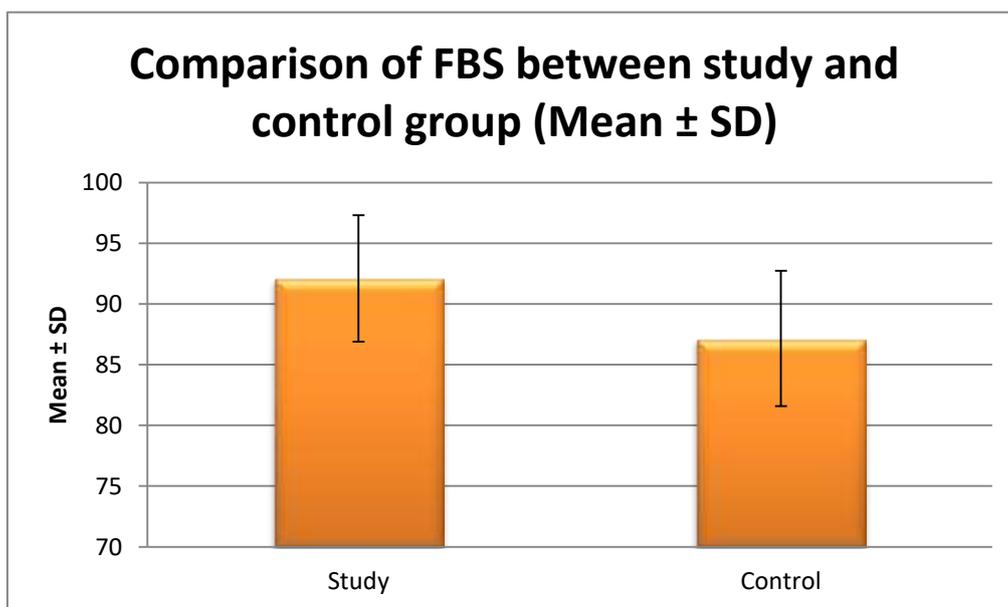


Fig-1: Comparison of FBS between study and control group

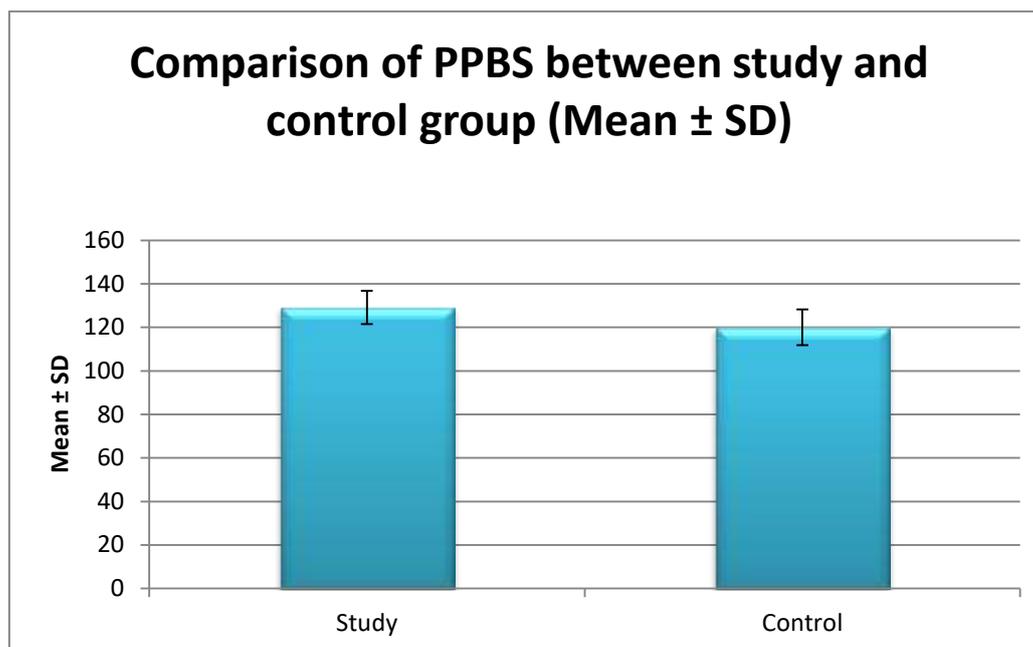


Fig-2: Comparison of PPBS between study and control group

DISCUSSION

In this study we evaluated the level of FBS and PPBS (2hrs after OGTT) in PCOS and compared the values with controls, and it clearly indicated that both the values are greater in PCOS and which was statistically significant. The results of our study are consistent with finding of Amruta Bennal *et al* [6], Farah Deeba *et al*[7], Jie Ping Jie *et al* [8], Richaard S Largo *et al* [9].

PCOS is a syndrome of ovarian dysfunction that is characterized by anovulation, hyperandrogenism, and/or the presence of polycystic ovary morphology[10]. Furthermore, PCOS is also associated with insulin resistance, pancreatic β -cell dysfunction, and obesity, abnormalities that confer a substantially increased risk for metabolic syndrome and type 2 diabetes mellitus[11]. They are also at increased risk for atherosclerotic cardiovascular disease (CVD) due to increased prevalence of obesity and central adiposity as well as to hypertension, hyperinsulinemia, type 2 DM, and dyslipidemia in these patients[12].

Although much remains unknown regarding the underlying pathophysiology of PCOS, a form of insulin resistance intrinsic to the syndrome appears to play a central role in its development. Among many women with PCOS, the observed insulin resistance is partially explained by excess adiposity; however, many studies have shown that even lean women with PCOS have increased insulin resistance compared with normal controls, as shown in our study. Although the nature of insulin resistance in PCOS is currently unclear, defects in insulin receptor or post-receptor signal transduction, altered adipocyte lipolysis, decreased glucose transporter 4 in adipocytes, and impaired release of a D-

chiro-inositol mediator have all been implicated. Furthermore, many women with PCOS exhibit β -cell dysfunction, rendering insulin response to a glucose load insufficient for the degree of insulin resistance in PCOS [13].

Hence women with PCOS irrespective of their BMI are at increased risk of developing glucose intolerance and type 2 diabetes mellitus, hence they have to be screened periodically with OGTT so as to detect diabetes mellitus and start treatment early. By this we can prevent the complication arising out of it. PCOS women should also be advised for life style modification and exercise. Androgen Excess Society of Virginia, prescribes, Patients with normal glucose tolerance should be rescreened at least once every 2 year, or more frequently if additional risk factors are identified. Those with IGT should be screened annually for development of type 2 Diabetes Mellitus [13].

CONCLUSION

Periodic screening of PCOS patients should be carried out as they are more prone to develop glucose intolerance and diabetes mellitus and failing to do so the patient may end up in cardiovascular and other complication.

REFERENCES

1. Fulghesu A, Magnini R, Portoghese E, Angioni S, Minerba L, Melis GB; Obesity-related lipid profile and altered insulin secretion in adolescents with polycystic ovary syndrome. *Journal of Adolescent Health*, 2010; 46(5):474-481.
2. Kovacs C, Smith J; *A Guide to the Polycystic Ovary: Its Effects on Health and Fertility*. Castle Hill Barns, U.K., TFM Publishing, 2002

3. Stein IF, Leventhal ML; Amenorrhea associated with bilateral polycystic ovaries. *Am J Obstet Gynecol*, 1935; 29:181–191.
4. Rebar R, Judd HL, Yen SS, Rakoff J, Vandenberg G, Naftolin F; Characterization of the inappropriate gonadotropin secretion in polycystic ovary syndrome. *J Clin Invest*, 1976;57:1320–1329.
5. Gambineri A, Pelusi C, Vicennati V, Pagotto U, Pasquali R; Obesity and the polycystic ovary syndrome. *Int J Obes Relat Metab Disord.* 2002; 26:883–896.
6. Bennal AS, Kerure SB; Effect of PCOS on glucose metabolism. *Natl J Physiol Pharm Pharmacol* , 2013; 3:167-170.
7. Butt K, Attique H, Deebea F, Khan SR; Polycystic Ovarian Syndrome: Determination of Alteration In Glucose Level and Lipid Profile. *Professional Med J*, 2012;19(6): 786-788.
8. Zhu JP, Teng YC, Zhou J, Lu W, Tao MF, Jia WP; Increased mean glucose levels in patients with polycystic ovary syndrome and hyperandrogenemia as determined by continuous glucose monitoring. *Acta Obstet Gynecol Scand*, 2012; 92(2): 165-171.
9. Legro RS, Kunselman 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 and Metab* 1999; 84(1):165-169
10. Azziz R; Diagnosis of polycystic ovarian syndrome: the Rotterdam criteria are premature. *The Journal of Clinical Endocrinology & Metabolism*, 2006; 91(3):781-785.
11. Biyasheva A, Legro RS, Dunaif A, Urbanek M; Evidence for Association between Polycystic Ovary Syndrome (PCOS) and TCF7L2 and Glucose Intolerance in Women with PCOS and TCF7L2; *J Clin Endocrinol Metab*; 2009; 94(7): 2617-2625
12. Conway GS, Agrawal R, Betteridge DJ, Jacobs HS; Risk factors for coronary artery disease in lean and obese women with the polycystic ovary syndrome. *Clinical Endocrinology (Oxf)*, 1992; 37:
13. Salley KE, Wickham EP, Cheang KI, Essah PA, Karjane NW, Nestler JE; Position Statement: Glucose Intolerance in Polycystic Ovary Syndrome—A Position Statement of the Androgen Excess Society. *J Clin Endocrinol Metab*, 2007; 92(12);4546-4556