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Research Article

**IMPORTANCE OF GLUCOSE TO INSULIN RATIO (G/R) IN
NON-OBESE AND OBESE PATIENTS WITH POLY CYSTIC
OVARIAN SYNDROME**¹Dr Anam Nazir, ²Dr Shahwaza Ajmal, ³Dr Ayesha Zafar¹Fatima Jinnah Medical University, Lahore²Avicenna Medical College, Lahore³Avicenna Medical College, Lahore**Article Received:** June 2020**Accepted:** July 2020**Published:** August 2020**Abstract:**

Polycystic ovary syndrome is a common endocrine disorder in women of childbearing age. This syndrome has a family predisposition. The main problem is the hypothalamic-pituitary axis leading to an increase in the LH / FSH ratio. Insulin resistance and the resulting hyperinsulinemia are common symptoms that lead to altered steroid hormone metabolism and other symptoms of the syndrome.

***Aim:** This study was conducted on sixty women with PCOS, half obese and half non-obese, taken from OPD gynecology and obstetrics department.*

***Place and Duration:** In the Gynecology and Obstetrics department of Jinnah Hospital Lahore for one-year duration from May 2019 to May 2020.*

***Methods:** They were diagnosed on the basis of an interview, and clinical examination increased the LH / FSH ratio. Forty women, half obese and half non-obese, were taken for control. Fasting glucose and insulin levels were determined and the ratio (GIR) was calculated. Both cases and the control group received 75 g of glucose in 200 ml of water. Two hours after the glucose load, glucose and insulin levels were re-measured and the ratio of glucose to insulin was calculated. Fasting glucose was not significantly high in both obese and non-obese groups.*

***Results and Conclusion:** However, significantly higher insulin levels were observed especially in the obese group. Thus, the GIR index decreased (assumed to be 4.5) in 33% non-obese and 60% obese. After being loaded with glucose. Glucose levels were not significantly elevated, but marked hyperinsulinemia was observed in obese subjects. The GIR decreased and was below the cut-off of 4.5 in 80% of non-obese and 86.6% obese cases. These are patients who need insulin sensitizers.*

***Key words:** obesity, polycystic ovary syndrome, hypothalamic-pituitary axis*

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INTRODUCTION:

Polycystic ovary syndrome (PCOS) is one of the most common endocrine disorders in women, affecting approximately 5-10% of women of reproductive age (12-45 years) and is one of the leading causes of infertility. In 2003, the Rotterdam Consensus Workshop identified PCOS if 2 of the 3 criteria are met. Oligo ovulation and / or anovulation. Excessive activity of androgens. Polycystic ovaries (in gynecological ultrasound) and other endocrine disorders are excluded. About 50% of women with PCOS are overweight or obese, and most of them have an abdominal phenotype. Serum (blood) levels of androgens (male hormones), including androstenedione, testosterone and dehydroepiandrosterone sulfate, may be elevated. adolescent girls and is often associated with an irregular menstrual cycle. In most cases, due to immaturity of the axis of the hypothalamus pituitary ovarian. PCOS may be associated with chronic inflammation, with several researchers correlating inflammation mediators with anovulation and other symptoms of PCOS. Obesity, which is often associated with PCOS, appears to enhance insulin resistance. Adiponectin, a recently discovered adipokine specific to adipose tissue, is said to be involved in obesity and diabetes. Most PCOS patients are insulin resistant and / or obese. Their elevated insulin levels contribute to or cause the abnormalities seen in the ovarian hypothalamic-pituitary axis that lead to PCOS. Insulin malfunctions are poorly detected simply by measuring glucose or insulin levels. The ratio of fasting insulin ($\mu\text{IU} / \text{L}$) to fasting glucose (mmol / L) has been found to be a simple and accurate marker of insulin resistance at values above $4 \mu\text{L} / \text{mmol} / \text{L}$. Measurement of impaired glucose tolerance often indicates abnormalities in the fasting and fasting state. 2 hours of glucose challenge with 75 g glucose each or fasting glucose to insulin ratio.

MATERIALS AND METHOD:

Sixty patients from the OPD of the Gynecology and Obstetrics department of Jinnah Hospital Lahore for one-year duration from May 2019 to May 2020 were participated in this study. They were of childbearing age already diagnosed and documented as polycystic ovary syndrome based on a history and thorough physical examination, blood tests (LH / FSH), and pelvic ultrasound

results. Half of them were obese with a body mass index (BMI) of 30 and over 30, and the other half were obese with a BMI <29.9 . Forty healthy women, half obese and half obese, were taken as controls. Both patients and controls were fasted for 10-12 hours. Baseline blood analysis including insulin and glucose levels was obtained. Patients and controls then drank 75 grams of glucose in 200 ml of water. Two hours later, the insulin and glucose levels were repeated and by examining the baseline level and stimulated insulin and glucose levels, the ratio was calculated and a diagnosis was made. 5 ml of venous blood was collected from a vein in the arm. Place 1.5 ml in a test tube with sodium fluoride and EDTA to assess the glucose concentration. The rest may clot in the plastic tube. After centrifugation, the clear serum was removed and stored in plastic cups at -20 degrees Celsius for insulin evaluation. Glucose was determined by enzymatic oxidation in the presence of glucose oxidase using the Randox glucose kit. Insulin determination was performed on stored serum by enzyme immunoassay (EIA).

RESULTS

The mean fasting glucose level in the obese group was $79.50 \text{ mg} / \text{dl}$, and in healthy subjects it was $72.8 \text{ mg} / \text{dl}$. Similarly, the fasting insulin level was $16.86 \mu\text{IU} / \text{L}$ in obese people and $12.4 \mu\text{IU} / \text{L}$ in the healthy group, when the mean ratio was calculated, which was $4.72 \mu\text{IU} / \text{L}$ in obese subjects and $5.8 \mu\text{IU} / \text{L}$ in the healthy group. The mean fasting blood glucose level in obese healthy (control) subjects was $78 \text{ mg} / \text{dL}$, while in obese healthy subjects it was $81.0 \text{ mg} / \text{dL}$. The mean fasting insulin level in healthy obese subjects was $7.14 \mu\text{IU} / \text{L}$, and in normal weight healthy subjects it was $6.3 \mu\text{IU} / \text{L}$. GIR in the obese control group was $10.9 \mu\text{IU} / \text{L}$, and non-obese subjects in the control group 12 , 8. Two hours after the glucose load, the mean glucose level in the obese group was $128.6 \text{ mg} / \text{dl}$, and in the non-obese group it was $118.2 \text{ mg} / \text{dl}$. Similarly, the level of insulin in the obese group was $74.1 \mu\text{u} / \text{l}$, and in healthy people - $54.8 \mu\text{u} / \text{l}$. When calculating the glucose-to-insulin ratio (GIR) in the obese group it was 2.6, and in healthy people it was 3.1, where, as in the control group, glucose was $125.7 \text{ mg} / \text{dl}$. The insulin level was 19.5 and the GIR was 6.9. In the non-obese group, blood glucose was $117 \text{ mg} / \text{dl}$. The insulin level was $14.4 \mu\text{IU} / \text{L}$ and the GIR was 8.3.

Fasting Levels of Glucose, Insulin and Glucose/ Insulin ratio (Non-Obese & Obese Group)

| Fasting Levels | | N | Non-Obese | Obese |
|-----------------------|---------|----|-----------|-------|
| | | | Mean | Mean |
| Glucose | Cases | 30 | 72.8 | 79.50 |
| | Control | 20 | 81.0 | 78.00 |
| Insulin | Cases | 30 | 12.4 | 16.86 |
| | Control | 20 | 6.3 | 7.14 |
| Glucose insulin ratio | Cases | 30 | 5.8 | 4.72 |
| | Control | 20 | 12.8 | 10.9 |

Two Hours Levels of Glucose, Insulin and Glucose/Insulin ratio (None-Obese & Obese Group)

| | | N | Non-Obese | Obese |
|-----------------------|---------|----|-----------|-------|
| | | | Mean | Mean |
| Glucose | Cases | 30 | 118.2 | 128.6 |
| | Control | 20 | 117.0 | 125.7 |
| Insulin | Cases | 30 | 54.8 | 74.1 |
| | Control | 20 | 14.4 | 19.5 |
| Glucose insulin ratio | Cases | 30 | 3.1 | 2.6 |
| | Control | 20 | 8.3 | 6.9 |

DISCUSSION:

In this study, the results for glucose and insulin in both obese and non-obese PCOS (values shown in the results) are comparable to a similar study by Legro and all in 1998 in which fasting glucose did not differ, but women with PCOS have significantly higher fasting insulin levels than women in the control group ($p = 0.001$). Obese families with a low GIR than the cut-off (<4.5) have been observed to be more insulin resistant than non-obese PCOS. When their mean values were calculated, 86.6% of obese subjects (26 out of 30), while 80% (24 out of 30) of non-obese subjects were insulin resistant. Falcone et al. Found that 63% of their healthy patients were insulin resistant. Glucose-dependent hyperinsulinemia and decreased GIR were more pronounced in obese subjects (86.6%) than in healthy subjects (80%). In a study of 83 women by Robert Kaufman et al. (2002), and before insulin resistance valence, 54.5% of their PCOS population broken down by ethnic insulin resistance was found in 43.8% of white and 73.1% of Mexican American women with PCOS.

CONCLUSION:

Fasting insulin and GIR are sensitive measures of insulin resistance. 60% of obese and 33% of non-obese PCOS were insulin resistant ($GIR <4.5$). After glucose load, 86.6% of obese and 80% of non-obese PCOS were insulin resistant. ($GIR <4.5$) that can be selected and selected for the use of insulin lowering drugs in addition to traditional ovulation induction and hormone therapy.

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