Study on Polycystic Ovarian Syndrome and Insulin Resistant Among Obese and Non-Obsess Patients in Tertiary Care Hospital, Cuddalore District, Tamilnadu

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I. INTRODUCTION

Polycystic ovarian syndrome has been becoming a common gynaecological and endocrinal disorder in females which starts from adolescent ages now a days. Obesity has stand as a global problem because of faculty culture, food habits, sedentary work and stress. Recent research shows that not only obese, even non-obese patients develop polycystic ovaries, leads to menstrual disturbances and then infertility. Polycystic Ovarian Syndrome (PCOS) is a common gynaceological disorder of reproductive age (12-45 years).

Nomenclature

Polycystic ovarian syndrome is also known as steinleventhal syndrome, functional ovarian hyperandrogenism, ovarian hyperthecosis, and sclerocystic ovary syndrome. There is substantial evidence that polycystic ovarian syndrome should no longer be considered purely a gynaecological disorder but rather a "complex endocrine disorder", characterised by hypergonadotrophism, hirsutism, obesity, oligomenorrhoea (or amenorrhoea). Polycystic ovarian syndrome affects approximately 5-10% of women of reproductive age group and is one of the most common cause of anovulatory infertility.

II. DIAGNOSIS OF POLYCYSTIC OVARY SYNDROME

Symptoms of androgen excess

- irregular menses
- acne, hirsutism

biochemical androgen excess

- Increased total / free testosterone, increased androstenedione and increased LH.

Pelvic ultrasound

- 1 or both ovaries enlarged, >12 peripheral follicles

III. OBJECTIVES

- To assess androgen, estrogen levels in obese and non-obese PCOS patients.

IV. METHODOLOGY

Setting

This study was done at Rajah Muthiah Medical College Hospital, Annamalai Nagar, Chidambaram. This is a very big teaching institution of health sciences in the Cuddalore district Tamil Nadu. There is a 5000 bedded hospital with all specialties in one campus.

Study subjects

50 current PCOS patients (25 obese; 25 normal) who came from in and around Chidambaram to the Rajah Muthiah Medical College and Hospital who has fulfilled the inclusion criteria were selected s experimental group. The primary data were collected during 2009-2010.

Sample Size

The universe of this study is very large, and solution of any percentage of the existing universe is near impossible and extensively time consuming so as a sample population, it was decided to select 50 respondents from PCOS of (Child bearing) age (20-30) fifty of both obese and normal (non-obese) PCOS.

Method of sample selection

Patients (PCOS) were selected from infertility clinic gynaecology OP assessed for their height, weight, anaemia, and vitamin and mineral deficiency. The respiratory system and cardiovascular system were found to be normal.

Criteria for sample selection

1. PCOS of age group 20-30.
2. History of menstrual irregularities.
3. History of infertility, obesity, hirsutism.
4. PCOS of both obese and non-obese.
5. Gynecological ultrasonography with PCOS.

Exclusion criteria

- Prolactin to rule out hyper prolactinemia.
- TSH and rule out hypothyroidism.

Body Mass Index (BMI) is a tool that helps to measure the amount of body fat based on height and weight. The body mass index can be calculated by the following method.

$$W/H = \frac{\text{Weight in Kg}}{\text{Height in m}^2} \text{ BMI}$$

<18.5	-	Under weight
18.5 – 24.9	-	Normal
25 – 29.9	-	Over weight
30 – 34.49	-	Obese

Gynaecological ultrasonography specifically looking for small ovarian follicles. There are believed to be the result of disturbed ovarian function with failed ovulation, reflected by the infrequent (or) absent menstruation that is typical of the condition.

1. Routine Investigation

Blood samples collected from biochemistry lab which were taken and sent by Gynaecology Department for other routine examinations Human Ethical Committee Clearance obtained. Hb%, TC, DC, ESR and for blood sugar, urea, creatinine, lipid profile done to rule out – anaemia, hypertension, renal pathology and diabetes mellitus.

2. Fasting blood of 5ml were collected and serum was separated and kept at 20oC for the estimation of

   a. Estrogen level
   b. Testosterone level
   c. Insulin level

3. Estimation of serum estradiol

17β estradiol is a C18 steroid hormone produced mainly by the ovary and placenta and in small amounts by adrenals and testes. Estradiol is in equilibrium

Serum estrodiol was estimated by the enzyme immunoassay

Calculation

Read the wells at 450nm against a reference filter set at 650 nm

$$B / BO (\%) = \frac{OD \text{ (Calibrator, Control (or) Sample)}}{OD \text{ (Zero calibrator}} \times 100$$

Reference Range

Menstrual Cycle pg / ml

Day 10	13 – 80
4	20 – 165
0	199 – 417
+2	22 – 154
+5	44 – 174
+10	13 – 146

Estimation of serum testosterone

Testosterone is a C-19 steroid hormone which is produced from androstenedione in the testes, adrenals and ovaries. Serum testosterone was estimated by the enzyme immunoassay.

Calculation

Read the wells at 450 nm against a reference filter set at 650 nm.

$$B / BO (\%) = \frac{OD \text{ (Calibrator, Control or Sample)}}{OD \text{ (Zero calibrator)}} \times 100$$

Reference Range

Males 1.21 – 6.91 ng/ml
Females ND 0.70 ng/ml

IV. RESULTS

INTERPRETATION

Testoterone Level

a. Level is high in both obese and non-obese polycystic ovarian patients than normal.
b. Comparison between obese and non-obese the testosterone is equally increased.

Estrogen Level

By finalizing the data the estrogen level

a. Is increased in both obese and non-obese polycystic ovaries patients with H/o menstrual disturbances and infertility, when compared with normal values (25 – 75 pg/ml).
b. Regarding obese and non-obese patients the estrogen level is comparatively high in obese patients.

V. DISCUSSION

Comparison of biochemical variables

While comparing the mean values of serum β estradiol level. Testosterone level and fasting serum insulin level of polycystic ovarian syndrome patients with normal reproductive age group women, which were significant.

Testosterone Level

Table 3 and 4 shows the raised testoterone level in both obese and non-obese polycystic ovarian patients. The underlying pathogenesis is the ovaries are stimulated to produce exercise amounts of male hormones (andogens), particularly testosterone either through the release of excessive leutinizing hormone (LH) by the anterior pituitary gland (or) though high levels of insulin in the blood (hyper insulinemia) in women whose ovaries are sensitive to this stimulus.
The abnormal ovarian androgen production that results from dysregulati dysregulation of key enzymes involved in theca cell androgen biosynthesis.

Hyperandrogenemia could be due simple to increased follicle number of theca cell hyperplasia. Both insulin and insulin like growth factors have been demonstrated to potentiate the actions of leutinizing hormone on theca cell androgen production.

Body Mass Index (BMI) is positively corrected to serum insulin and testosterone levels and is inversely related to sex hormone binding globulin (SHBG) levels.

Laboratory Findings

All the three hormonal parameters (estrogen, testosterone,) are increased when compared with normal. It may be caused by abnormalities in four endocrinologically active compartments.

i. The ovaries
ii. Adreneal glands
iii. The periphery (fat)
iv. Hypothalamic pituitary compartments.

Ovarian component

Table 1, 2, 3 and 4 shows increased estrogen and testosterone levels. Dysregulation of the cytochrone P 450c17, the androgen forming enzyme in both the adrenal and the ovaries may be the central pathogenetic mechanism underlying hyperandrogenesim in polycystic ovarian syndrome.

The ovarian stoma, theca and granulose contribute to ovarians hypoerandrogenism and are stimulated by leutinizing hormone.

The increased testosterone levels may reach 200ng/dl (or) more which inhibit follicular maturation which in turn results in inceptive granulose cells with minimal aromatasa activity.

Treatment with a gonadotrophin releasing hormone (GNRH) agonist effectively suppresses serum testosterone and androstenedione levels.

Adrenal Component

The polycystic ovarian syndrome patients were with H/o menstrual irregularities, Hirsutism – excessive and increased body hair typically in a male pattern affecting face, chest and legs.

Reasons

DHEAS –

Dihydroepiandro sterone is increased in about 50% of polycystic ovaries patients. This hyperresponsivenss of DHEAS is due to stimulation with (ACTH) adrenocorticotrophic hormone.

17, 20 lyase activation is a key event in adrenarche.

Obesity: Peripheral Compartment / Skin and Adipose tissue

From table 1 and 2 (estrogen level) and body mass index of 25 obese patients shows that increase estrogen level and body weight play a role in polycystic ovarian syndrome.

The presence of 5β reductase in the skin determines the presence (or) absence of hirsutism. Aromatase and 17β hydroxysteroid dehydrogenase activities are increased in fat cells and peripheal aromatization is increased with body weight.

The metabolism of estrogens, by way of reduced 2-hydroxylation and 17β oxidation is decreased. A chronic hyperestrogenic state results with reversal of E1: E2 ratio.

VI. CONCLUSION

In summary polycystic ovarian syndrome is a major disorder starts from adolescent age and producing menstrual disturbances leeds to infertility.

It is a treetable disease with good improvement in signal and symptoms. Patients are prone to cardiovascular diseases due to hypertension and dyslipidemia. Obesity and infertility leads to depression.

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