

# Research Journal of Pharmaceutical, Biological and Chemical Sciences

## Status of Leptin, Lipid Profile in Oligomenorrhic and Eumenorrhic Adolescence Girls: It's Comparison.

Minu priya P<sup>1</sup>, Prasanna KB<sup>2</sup>, Kalai Selvi VS<sup>3</sup>, Prabhu K<sup>4\*</sup>, Devaki P<sup>5</sup>, and Jaya Kumari<sup>4</sup>.

<sup>1</sup>Department of Obstetrics & Gynecology, Sree Balaji Medical College and Hospital, Chrompet, Chennai – 600 044, Tamil Nadu, India.

<sup>2</sup>Department of General Medicine, Tagore Medical College and Hospital, Chennai, Tamil Nadu, India.

<sup>3</sup>Department of Biochemistry, Sree Balaji Medical College and Hospital, Chrompet, Chennai – 600 044, Tamil Nadu, India.

<sup>4</sup>Department of Anatomy, Sree Balaji Medical College and Hospital, Chrompet, Chennai – 600 044, Tamil Nadu, India.

<sup>5</sup>Department of Physiology, Sree Balaji Medical College and Hospital, Chrompet, Chennai – 600 044, Tamil Nadu, India.

### ABSTRACT

The presence of obesity, hyperandrogenism, hyperinsulinemia, hirsutism, polycystic ovaries are well established features in PCOS subjects. The adolescence girls experience menstrual irregularity in first five years after menarche. The purpose of this study is to analyse the status of lipid profile and leptin at the end of hypothalamo hypophyseal maturation that is eight years after menarche. The subjects in this study includes two groups. Group 1 includes regularly menstruating adolescence girls and group 2 includes irregularly menstruating girls. Student t test was performed to analyse the test of significance between the two groups. The mean value for leptin, total cholesterol and triglycerides were statistically significant between the two groups. This study suggest that the status of adiposity marker that is leptin in irregularly menstruating adolescence girls precede towards the aetiogenesis of PCOS in later adulthood life.

**Keywords:** leptin, lipid profile, Oligomenorrhic and Eumenorrhic.

*\*Corresponding author*

## INTRODUCTION

Oligomenorrhoea is common in adolescent girls and is defined as cycles less than nine menstrual periods per year. The variations in the length of the menstrual cycle suggests that the irregular menstrual cycles in the first five years after menarche shows physiological irregularity rather than the reason for clinical or endocrine evaluation [1]. Due to rise in overweight among adolescent girls, it is important to investigate the role of adiposity on its reproductive status and to analyze the strength of association with menstrual irregularity.

Obesity is a complex multifactorial disease that develops from the integration of social, behavioural, cultural, physiological, metabolic and genetic factors [2]. It is directly or indirectly associated with various diseases like cardio vascular diseases, hypertension, Diabetes mellitus, sleep apnea, osteoarthritis, fatty liver disease gall bladder disease and cancer and poses a threat to health [3]. The initial body mass of adipose tissue is essential for the normal development of female reproductive function. Polycystic ovarian syndrome with presence of obesity is a disorder characterized by chronic anovulation, hyperandrogenism and hyperinsulinemia.

Insulin resistance with the altered insulin action precedes the increase in androgen in PCOS and cause hyperandrogenism by inhibiting hepatic synthesis of SHBG and by binding insulin like growth factor (IGF -1) receptors in the ovary leading to increase in androgen production by the thecal cells. So there is decreased insulin dependent receptor autophosphorylation of tyrosine residue and increased serine phosphorylation. These defects may cause insulin resistance in women with PCOS- ser [4]. This chronic effect exerts stimulatory effect on adipocytes to increase leptin synthesis and in turn diminishes glucose oxidation, lipogenesis and increased fat utilization [5,6] which leads to hyperinsulinemia and hyperandrogenism which is established features in PCOS women.

Clinical studies have established that adiposity is increased and suggest the role of ovarian dysfunction with menstrual irregularity in PCOS subjects. The purpose of this study is to examine the role of adiposity like lipid profile and leptin in regular and oligomenorrhoeic adolescence girls that is eight years after menarche that is towards the end of hypothalamic hypophyseal maturation.

## MATERIALS AND METHOD

After obtaining the institutional ethical committee clearance and informed consent from the subjects, the study includes adolescent girls with the age in the range between 17-19 years. They were divided into two groups. The group 1 includes regularly menstruating girls and group 2 includes irregularly menstruating girls. Menstrual cycle is defined as regular when an average length of the cycle was between 22 and 41 days. The menstrual cycle is defined irregular when the length of the cycle length was between 22 and 41 but with two or more cycles with a length <22 or >41 days during the year. After recording the general profile of the subjects like age, BMI for the both groups, 3 ml of intravenous blood was taken from each participant, the serum samples were kept at -20 c and hormones like FSH on day 3 and for adiposity markers lipid profile and hormone like Leptin were assessed and calculated.

The FSH levels were analysed by MONOBIND INC, E2 by Biosource belgium. Leptin were measured using enzyme-linked immunoassay kit and Lipid profile was measured by Standard Biochemical method. The test of significance of the above variables were performed between the two groups using SPSS( social package for statistical sciences).

## RESULTS

The mean value of age in adolescent girls with regular cycle was and in adolescent girls with irregular cycle was 18.7years and 19.06 years. The mean value of height, weight, BMI in Group 1 were 155.6cm, 48.7kg, and 20.15kg/m<sup>2</sup>. The mean value of height, weight, and BMI in adolescent girls with irregular cycles were 155.7cm, 51.8kg, and 21.3kg/m<sup>2</sup>. The mean value of lipid profile in Group 1 (total Cholesterol =149.4, triglycerides= 112.46, HDL=48, LDL=71.4, VLDL=22.4 ratio= 2.9) the mean value of Lipid profile in adolescent girls with irregular cycle were (Total cholesterol=207.5, triglycerides=116.7, HDL=46.4, LDL= 79.6, VLDL= 23.4 Ratio=3.1) The mean value of leptin and insulin in adolescent girls with regular cycles were 18.2 and, 13.6 and

its mean value in Group 2 were 38.0 and 14.1. The variables whose mean showed a test of significance between regular and irregular cycled adolescent girls were total cholesterol P=.071, triglycerides=.067, Leptin p= p=0.001) and their p- value is shown in bracket.

**Tables showing test of significance between the Variables in two groups.**

	Group 1	Group 2	P- Value
Age ( years)	18.7±0.888	19.06±1.75	0.516
Height (cm)	155.6±6	155.7±.5	0.976
Weight(kg)	48.7±6.8	51.8±6.7	.226
BMI(kg/m2)	20.15±2.7	21.3±2.5	.228
Total cholesterol	149.4±16.8	207.5±20.9	.071
Triglycerides	112.4±15.5	79.3±9.4	.067
HDL	48±4.4	46.4±3.4	.373
LDL	71±19.3	79.6±15.6	.733
VLDL	22.4±3.06	23.4±1.84	.047
Ratio	2.9±.5	3.1±.3	.175
Leptin (ng/ml)	18.2±10.6	38.0±23.2	.001**
Insulin(mU/ml)	13.6±4.4	14.1±5.10	.947

\*\* Statistically significant.

### DISCUSSION

The present work associate the relation of adiposity marker that is leptin with reproductive status in oligomenorrhic adolescent girls and it was highlighted that leptin concentration , total cholesterol and triglycerides were significantly different between regularly and irregularly menstruating adolescent girls.

Previous study indicates that leptin levels were increased in obese individuals than in normal weight individuals and seems to correlate better with subcutaneous fat rather than with visceral fat in both obese and non-obese subjects[7,8,9,10]. The current study also highlights that leptin concentration is related to fat content and its level may be decreased by reduction of body fat even though BMI values remain unchanged [11]. It also shown that subcutaneous fat produces more leptin than visceral fat and relative insulin deficiency is also an important stimulator of leptin production. It is also noted that subcellular and tissue distribution of leptin is different in human adipocytes and can be altered by effectors such as insulin [12,13]. Above reports highlights that leptin is marker for adiposity rather than body mass and this study highlights its higher value in oligomenorrhic girls which is responsible for menstrual irregularity in adolescent girls.

Women with oligomenorrhea tend to have less good cholesterol and more bad cholesterol, in addition triglycerides levels another component of lipid profile is found to be also higher in women with oligomenorrhic girls which further contributes to the risk of heart disease. PCOS women have been reported to have higher serum concentration of leptin than those with weight matched controls. The expression of the obese gene in white fat cells is stimulated by insulin, glucocorticoids , nor adrenaline and by nutrients in human [14,15]. Circulating serum leptin concentration and OB – gene expression are increased in obese human. The raised circulating leptin concentration of obese individuals reflect the amount of fat – tissue in PCOS women.

Insulin stimulated leptin secretion is limited by insulin resistance in adipocytes of women with PCOS. Overweight results in progressively severe insulin resistance with decompensating of reproduction and metabolic function. High leptin concentration may affect ovarian function by reducing the response to gonadotrophin stimulation. Long or irregular cycles are likely to be a marker of PCOS with increased body mass index may preside of PCOS. In this study leptin concentration were increased in adolescent girls with irregular cycles than girls with regular cycles and serve as an indicator of fat content and is directly associated with insulin resistance.



## CONCLUSION

This study suggest that the status of adiposity marker that is leptin in irregularly menstruating adolescence girls precede towards the aetogenesis of PCOS in later adulthood life.

## REFERENCES

- [1] Gardner J. Ann Hum Biol 1983;10: 31- 40.
- [2] Al-Quaiz , AL-Joharah M. Saudi Med J 2001;22:205-10.
- [3] Shigeta H, Shigeta M, Nakazawa A, Nakamura N, Yoshikawa T. Diabetes Care 2001;24(3):608.
- [4] Dunaif A. Endocrinol Metab Clin North Am 1999;28:341-59.
- [5] Caro JF, Sinha Mk, Kolaczynski Jw. Diabetes 1996;45:1455-62.
- [6] Bryson Jm, Phuyal JL, Swan V et al. Am J Physiol Endocrinol Metab 1999;277:E417-E422.
- [7] Considine RV, Sinha MK, Heiman ML et al. N Engl J Med 1996;334:292-5.
- [8] Ruhl CE, Everhart JE. Am J Clin Nutr 2001;74:295-301.
- [9] Cnop M, Landchild MJ, Vidal J et al. Diabetes 2002;51:1005-15.
- [10] Masoud AY, Adel AA. Sultan Qaboos Med J 2006;6:28-31.
- [11] Abdelgadir M, Elbagir M, Eltom M, Berne C, Ahren B. Metabol 2002;51:304-6.
- [12] Russel CD, Ricci MR, Brolin RE, Magili E, Fried SK. Am J Physiol Endocrin Metab 2001;280:E399-404.
- [13] Minocci A, Savia G, Lucantoni R et al. Int J Obes Relat Metab Disord 2000;24:1139-44.
- [14] Rohner-Jeanrenaud F and Jeanrenaud, B. N Eng J Med ;334:324-325.
- [15] Saladin R De Vos , P, Guerre-Millo M et al. Nature 1995 ;377: 527-529.