

**POLYCYSTIC OVARY SYNDROME-A LIFESTYLE RISK FACTOR OF MODERN
WORLD****¹Dr. T. Reena, ² Anisha. D. L**

Assistant Professor, Department of Microbiology, Malankara Catholic Mariagiri, Kaliyakkavillai, Kanyakumari district¹-PG student, Department of Microbiology, Malankara Catholic Mariagiri, Kaliyakkavillai, Kanyakumari district²

reenavictor06@gmail.com¹

ABSTRACT

The polycystic ovary syndrome is a familial disorder, but the genetic basis of the syndrome remains controversial. Determining the mode of inheritance of this syndrome is difficult because there has been no clearly described male phenotype and because it is a disorder that affects principally women of reproductive age. There is a lack of information about further PCOS development and prognosis, considering the environmental and individual factors. No systematic studies were reported regarding the genetic integrity, especially the DNA damage in PCOS. Hence the present study was undertaken to evaluate a case study in Trivandrum Kerala district and the DNA damage in women with PCOS by investigating the biochemical and molecular cytogenetic alterations.

Keywords: Polycystic ovary, DNA damage, Phenotype, Metabolic abnormality.

1. INTRODUCTION

Polycystic ovarian syndrome (PCOS) is a major cause of infertility in women affecting 6-10% of women of childbearing age (Kai *et al.*, 2008; March *et al.*, 2010). It is related to the absence of ovulation (anovulatory infertility). The metabolic profile noted in women with PCOS is similar to the insulin resistance syndrome, a clustering within an individual of hypersulinemia, mild glucose intolerance, dyslipidemia and hypertension. Insulin resistance plays a role in the pathogenesis of PCOS & dyslipidemia may be the most common metabolic abnormality in PCOS, with a prevalence of up to 70% by the National Cholesterol Education program criteria (LEGRO 2001). PCOS is classically associated with an atherogenic lipoprotein profile, characterized by elevated triglyceride-rich lipoproteins, accumulation of small dense low density lipoprotein (LDL) and depressed high density lipoprotein (HDL). All these changes were reported to be due to insulin resistance, although androgens only contribute to small HDL size by stimulating hepatic lipase activity (Rajhoo *et al.*, 1997). Early subclinical atherosclerotic disease, as evidenced by carotid intimal media thickness (Talbot *et al.*, 2000) and increased coronary artery calcification (Christian *et al.*, 2003; Talbot *et al.*, 2004) were reported in women with PCOS.

2. MATERIALS AND METHODS

Twenty nine study subjects with a clinical diagnosis of PCOS referred from various maternity hospitals of Kerala to Genetika, Center for Advanced Genetic studies, Trivandrum for genetics studied; twenty one age matched healthy fertile women formed the control group. Informed consents were obtained from all the subjects of the study. Relevant information was recorded using Performa.

3 ml of blood was collected in sodium heparinized vacutainers for quantifying the extent of somatic DNA damages by Cytokinesis-block micronuclei (CBMN) assay.

The remaining 8 ml of venous blood was collected aseptically from all the subjects by vein puncture after overnight fasting. Five ml of blood was allowed to clot, serum separated immediately, blood sugar and lipid profile was estimated using semi analyzer.

2.1. BIOCHEMICAL ANALYSIS (Benjamin *et al* 2020)

1. Glucose
2. Total cholesterol
3. High density lipoprotein cholesterol (HDL)
4. Low density lipoprotein cholesterol (LDL)
5. Triglycerides

3. OBSERVATION AND RESULTS

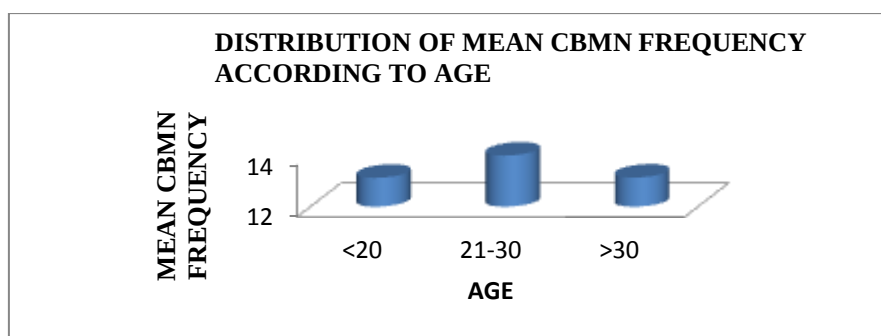


Figure: 1

The study showed that the mean CBMN frequency was increasing with the increase of age. The highest mean CBMN frequency (13.14) was observed in subjects with age greater than 30.

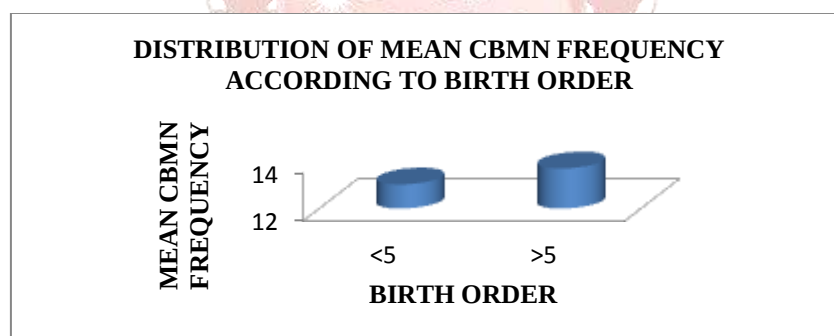


Figure: 2

Increased mean CBMN frequency was observed in subjects with birth order greater than 5. The mean CBMN frequency was found to be 13.68.

Distribution of mean CBMN frequency according to Residence

Table: 1

Area of Residence	Number	Percentage	Mean CBMN Frequency
Rural	20	71.4%	13.09
Urban	7	25%	13.21
Costal	1	3.57%	12.46

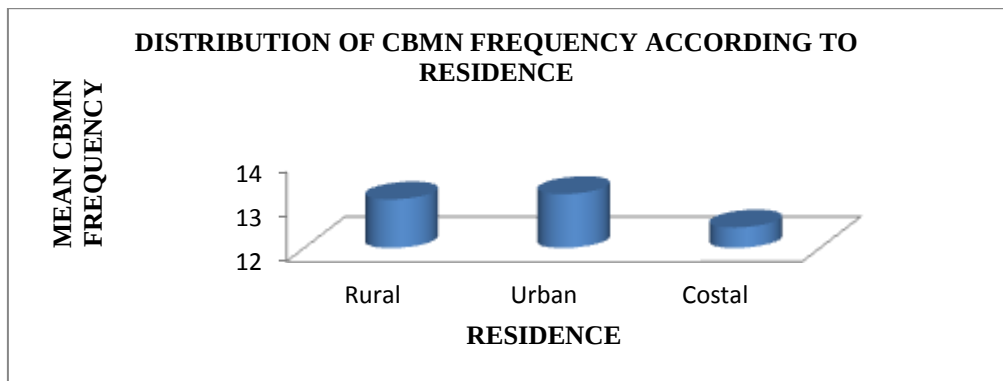


Figure: 3

According to the area of residence, increased mean CBMN frequency (13.21) was showed by subjects in urban area by the rural and costal area.

Distribution of CBMN frequency according to Religion

Table: 2

<i>Religion</i>	<i>Number</i>	<i>Percentage</i>	<i>Mean CBMN Frequency</i>
<i>Hindu</i>	20	71.42%	13.13
<i>Christian</i>	4	14.28%	13.18
<i>Muslim</i>	4	14.28%	13.27

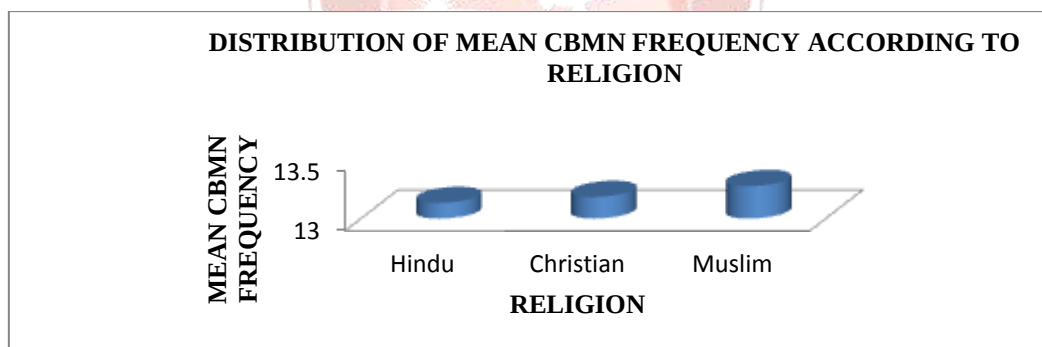


Figure: 4

On the basis of religion, highest mean CBMN frequency (13.27) was showed by the subjects belonging to Muslim category followed by Christian (13.18) and Hindu (13.13)

Distribution of mean CBMN Frequency according to Educational achievements.

Table: 3

<i>Educational Achievements</i>	<i>Number</i>	<i>Percentage</i>	<i>Mean CBMN Frequency</i>
<i>Graduate/PG</i>	12	42.85%	13.28
<i>Higher secondary</i>	13	46.42%	13.15
<i>primary</i>	3	10.71%	13.04

Based on the educational qualification, study subjects were classified as those with graduation/PG, higher secondary and primary level education. Subjects with graduation showed high CBMN frequency (13.28) followed by higher secondary and primary.

Distribution of Mean CBMN Frequency according to H/O Infertility or sub infertility

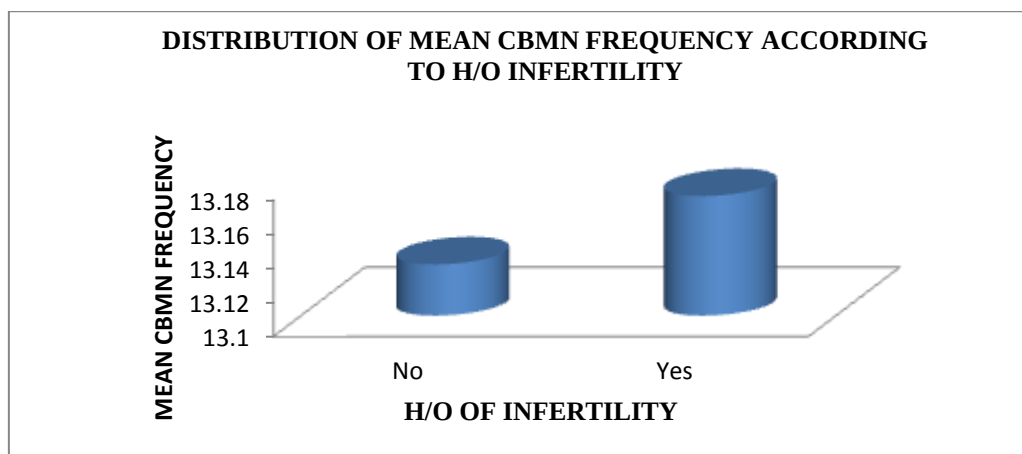


Figure: 5

Based on the H/O infertility and sub fertility, majority of the subjects (92.8%) were fertile. Highest mean CBMN frequency (13.17) was showed by the subjects having no family h/o infertility/subfertility.

Distribution of Mean CBMN frequency according to Clinical Conditions.

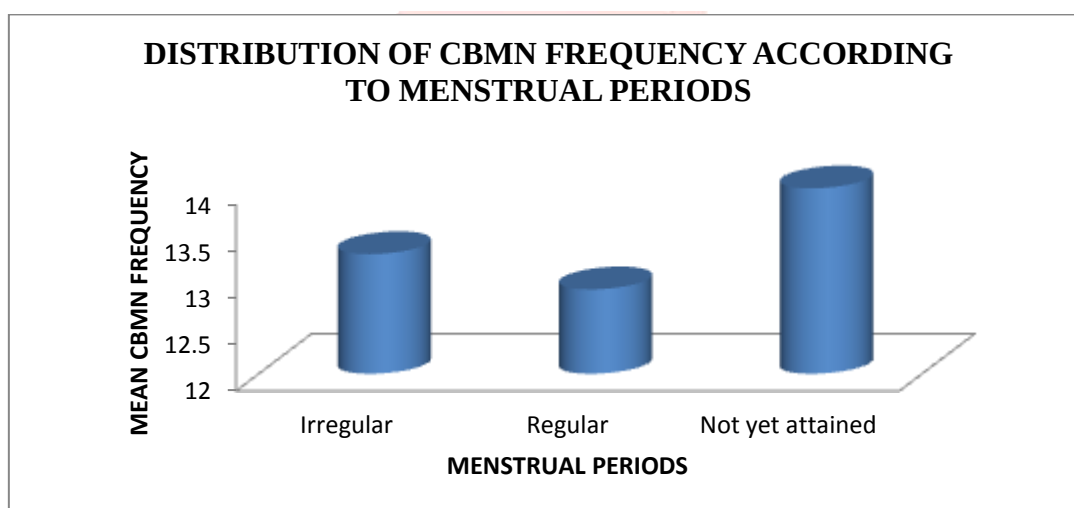


Figure: 6

Among the subjects with regular or without menstrual periods, highest mean CBMN frequency (14) was showed by the subjects having no menstrual period.

Distribution of mean CBMN frequency according to Menarche

Table: 4

Menarche	Number	Percentage	Mean CBMN Frequency
<15	24	85.71%	13.06
>15	3	10.71%	13.64
Not yet attained	1	3.57%	14

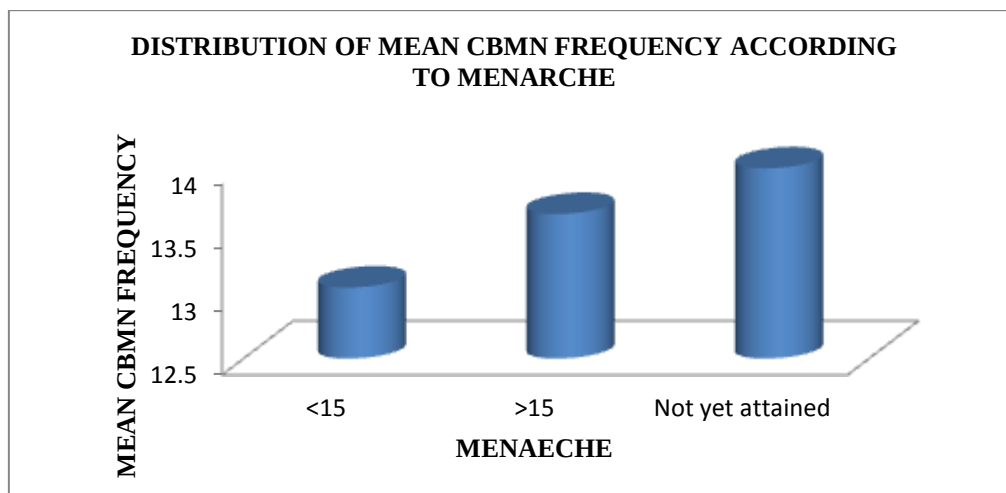


Figure 7

Highest CBMN frequency (14) was showed by the subject who did not attained Menarche, so far.
Distribution of Mean CBMN frequency according to economic status

Table: 5

<i>Menarche</i>	<i>Number</i>	<i>Percentage</i>	<i>Mean CBMN Frequency</i>
<i>High</i>	4	14.28%	13.4
<i>Low</i>	3	10.71%	12.88
<i>Medium</i>	21	75%	13.15

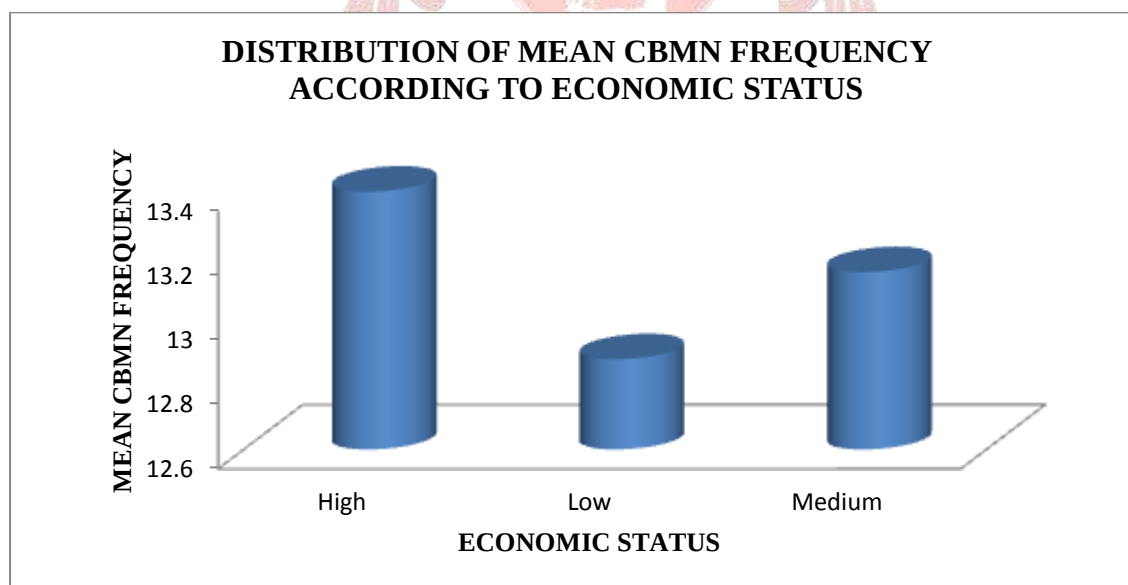


Figure: 8

Study subjects were classified as per their economic status. Highest mean CBMN frequency (13.4) was showed by the subjects belonging to high economic status followed by medium and low.

Distribution of mean CBMN frequency according to FBS

Table: 6

<i>FBS</i>	<i>Number</i>	<i>Percentage</i>	<i>Mean CBMN Frequency</i>
<i>Normal</i>	9	32.14%	13.04
<i>Abnormal</i>	19	67.85%	13.21

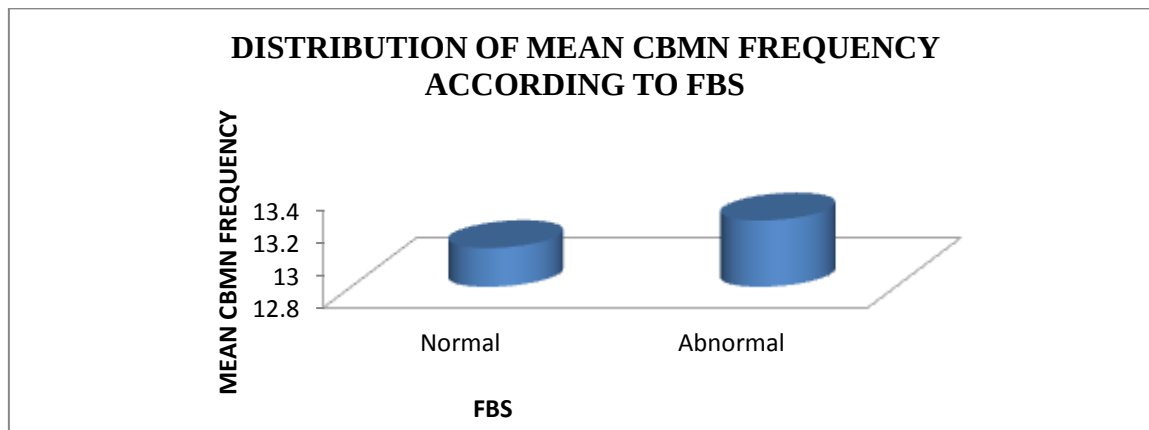


Figure: 9

Majority of the study subjects (96.78%) have abnormal range of FBS. The distribution of mean CBMN frequency according to fasting blood sugar (FBS) was recorded. Subjects having elevated FBS level showed increased CBMN value (13.21) than the subjects with normal FBS.

Distribution of mean CBMN frequency according to total cholesterol

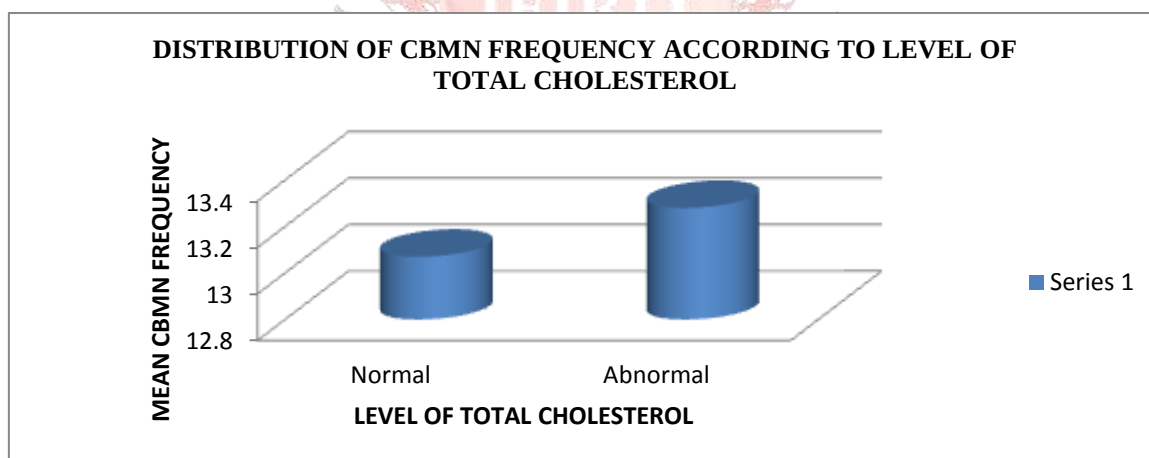


Figure: 10

The distribution of the mean CBMN frequency according to cholesterol level was observed among the study subjects. Majority of the study subjects (60.71%) have abnormal range of cholesterol level. Subjects with abnormal cholesterol level showed increased CBMN frequency (13.28) than those with normal cholesterol level.

The distribution of the mean CBMN frequency according to LDL cholesterol level was observed among the study subjects. 67.85% of the study subjects showed high levels of LDL. Increase mean CBMN frequency (13.22) was observed in subjects having abnormal LDL level.

Distribution of mean CBMN frequency according to LDL

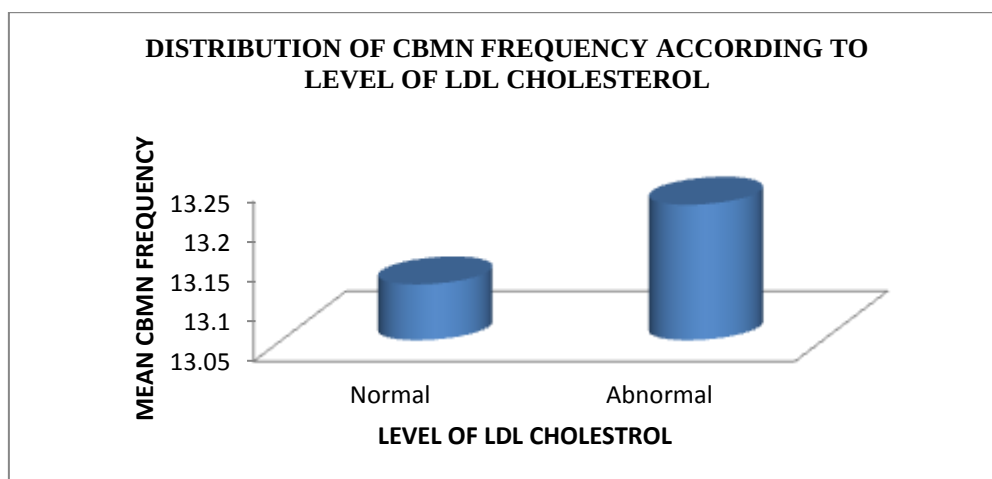


Figure: 11

Distribution of mean CBMN frequency according to TG

Table.7

<i>TG</i>	<i>Number</i>	<i>Percentage</i>	<i>Mean CBMN Frequency</i>
<i>Normal</i>	11	39.28%	13.07
<i>Abnormal</i>	7	60.71%	13.28

Regarding the concentration of Triglyceride (TG), 60.71% of the study subjects showed high levels for TG. The subjects with abnormal TG level showed a mean CBMN value 13.28 than those normal TG level.

4. SUMMARY AND CONCLUSION

Twenty nine subjects with a clinical diagnosis of PCOS referred from various maternity hospital of Kerala, Centre for advanced Genetic studies, Trivandrum for genetic studies formed the study group; twenty one age matched healthy fertile women formed the control group. Informed consents were obtained from all the subjects of the study. Relevant information was recorded using performance. Study subjects with PCOS showed various clinical conditions.

The present study reveals various physiological, demographical, biochemical and genetic characteristics in subjects with PCOS. The mean CBMN frequency measure the extent of somatic DNA damages among the PCOS subjects. The findings of the present study include:

- The mean CBMN frequency was found to be increased with an increase in age.
- Birth order showed a direct influence on the mean CBMN frequency.
- An increased mean (CBMN frequency was observed among subjects residing at urban area (13.12) followed by rural and coastal.
- Subjects with H/o infertility or sub fertility showed an increased frequency of DNA damages.
- Highest CBMN frequency (14) was showed by the subjects who did not attained menarche, so far.
- Highest mean CBMN frequency was found to be higher in subjects belonging to high socioeconomic status (13.4).
- The mean CBMN frequency was found to be higher among subjects with an abnormal range of fasting Blood sugar, Total cholesterol, LDL and triglyceride.

The present study observed an increased risk of PCOS subjects towards the CVD, mostly mediated through increased lipid profiles. The study also indicates the increased risk for type 2 Diabetes Mellitus in subjects suffering from PCOS.

An increased frequency of DNA damage in study subjects was indicated by the increased mean CBMN frequency among them, which play an important role in PCOS patients. The study provides an important insight into the causation of infertility / sub fertility. The high incidence of genetic instabilities in subjects with PCOS clearly warrants periodical examinations and counseling. Proper diet control and better life style modifications can keep the individual devoid of other complications of polycystic ovarian syndrome. Dietary alternation (decrease in calories by restricting daily intake to 1400 kcal, avoiding sugary drinks (water is preferable), have more low glycogenic index vegetables and fruits. Avoiding snacking between meals .A low glycaemic diet is preferable and regular moderate exercise which will provide fresh oxygenic mobilization to the body. Strictly Stop smoking and Moderate caffeine intake are the beneficial regular habits that can reduce the risks of polycystic ovary syndrome. Today the modern generation is very lazy to follow a good life style practice to manage a healthy life. Prevention is better than cure and good healthy practice leads a happy life.

REFERENCES

- [1] Arslanian.S.A. Witchel S.F.(2002),Polycystic ovary syndrome in adolescents: Is there an epidemic? Current opinion in Endocrinology and Diabetes; vol 9:32-42.
- [2] Asuncion M, Calvo RM, San Millen J.L, Sancho J, Avila S, Escobar-Morale H.F (2000). A prospective study of the prevalence of the polycystic ovary syndrome ,in unselected Caucasian women from Spain. J Clin Endocrinol Metab. 85:2434-8.
- [3] Azziz R, Carmina E, Dewily D, (2006);, positions statement: criteria for defining polycystic ovary syndrome as a predominantly hyper androgenic syndrome: An androgen Excess society guideline. J Clin Endocrinol Metab. 91; 4237-4245.
- [4] Bill Slater and Nancy Dunne, (2005.) Control PCOS and Infertility: Ten Essential Tips, Health-and-Fitness: Women –Issues,
- [5] Elenikandaraki; Charikleia christakou; Evanthia Diamanthi (2009). Kandarakis Metabolic syndrome and polycystic ovary syndrome and vice versa Arq Bras Endocrinol Metab; vol 53 no 2.
- [6] Francesco orio Jr., Stefano palomba, letizia spinelli,(2004) the cardiovascular risk of young women with polycystic ovary syndrome: An observational analytical, prospective case-control study. Journal of clinical endocrinology& metabolism, vol.1(89)3696-3701.
- [7] Franks S, Gherani N, McCarthyM. Candidate genes in polycystic ovary syndrome (2008); Report issue 7: 405-410.
- [8] Grundy SM (2007), metabolic syndrome: a multiples cardiovascular risk factor. J clin Endocrinol Metab; 92 : 399-404.
- [9] Halpern, Marcio C.,Maria Eliance C., Magalhaes (2010) metabolic syndrome, dyslipidemia, hypertension and type 2 diabetes in youth: from diagnosis to treatment. Diabetology & Metabolic syndrome 2:55.