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Incidence of Polycystic Ovarian Disease in Adolescent: A Prospective Study at Rims Ranchi

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Abstract: *Recently there has been an increase in the incidence of adolescent PCOS1. This is more common in urban as compared to those in rural areas. In this context persistence of physiological hyperinsulinemia may be a cause but environmental factors play significant role .The aim of my study wasto study the incidence of polycystic ovarian disease among adolescent age group girls in age group between 11 to 19 years attending GOPD in Department of Obstetrics and Gynaecology RIMS Ranchi and to study levels of serum DHEAS, serum LH/FSH Ratio, serum testosterone, serum LDL and LDL Levels and serum Insulin levels .This was a prospective study . Detailed history taking,general examination including ferryman galway's scoring was done followed by routine investigations and specific investigation.During a time period of May2016 to Sep2017 incidence was calculated to be 9.15%.This concludes that there is a rising incidence of polycystic ovarian disease among adolescent age girls.*

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I. Introduction

Adolescence represents the transitional period linking childhood to adulthood and involves physical , biological and psychosexual changes in a girl[1].

Endocrinological changes during puberty consist of sudden hyperactivity of all the pituitary tropic hormones with consequent over production and release of target gland hormones. A close relationship between normal pubertal endocrine changes and abnormal hormone profile seen in women with PCOS has been observed.

Recently there has been an increase in the incidence of adolescent PCOS . This is more common in urban as compared to those in rural areas. In this context persistence of physiological hyperinsulinemia may be a cause but environmental factors play significant role.

Though pathophysiology of PCOS is still not very clear, it is well accepted that hyperandrogenicity is almost always associated with PCOS. Though hyperandrogenicity is a common association of PCOS , the exact mechanism is not very clear . One hypothesis is dysregulation of ovarian hormone production by pituitary gonadotropine hypersecretion causing hyperandrogenism. The other and which is gaining popularity is that hyperinsulinemia is the most significant cause of hyperandrogenicity[10,11].

Hyperinsulinemia leads to decline in hepatic synthesis of SHBG and IGFBP1 Decline in the level of SHBG will lead to excess bioavailability of free androgen,while decline in the level of IGFBP-1 will help in increasing the level of circulating IGF-1.IGF-1 and insulin are structurally and chemically similar receptors of IGF-1 are present in the ovary (theca cells).Elevated levels of insulin – through IGF-1 receptors will amplify LH mediated thecal androgen production. Hence the ultimate consequence of hyperinsulinemia is hyperandrogenicity. Insulin also acts as a co-gonadotropin. There are insulin receptors in the pituitary .Therefore excess insulin accelerates LH hypersecretion (a common gonadotropin dysregulation in PCOS) which in turn will increase ovarian theca – cell androgen production.

There are clinical and biochemical markers by which PCOS in adolescent girls can be identified. The clinical markers are obesity or increased BMI >25 and features of hyperandrogenism like hirsutism, acne, acanthosis-nigricans, premature puberty. The biochemical markers of adolescent PCOS are fasting insulin /fasting glucose ratio less than 4.5, elevated free testosterone, DHEASO4, 17 hydroxy-progesterone suggested as diagnostic parameters of adolescent PCOS [21]. LH/FSH ratio may be normal and it is not true that high BMI and insulin resistance are always associated with an increase in LH levels or increased LH/FSH ratio.

Metabolic and endocrinological changes common in normal puberty and in PCOS which has already been outlined are:

a) Hyper pulsatile gonadotropin.
b) Excess ovarian and adrenal androgen production.

c) Insulin resistance /hyperinsulinemia
d) As a consequence of hyperinsulinemia –hyperandrogenism

Because of these shared features it has been speculated that puberty triggers PCOS in predisposed girls. Prepubertal pubarche before the age of 8 years may be a clinical marker for PCOS in adolescent and adult life.

II. Material And Method

This was a prospective study carried out in the Department of obstetrics and gynaecology at Rajendra Institute of Medical Sciences, Ranchi from May 2016 to September 2017

During this period all adolescent girls aged 11 to 19 years coming with gynaecological problems either in outpatient department or admitted in the indoor were selected for the study

The approach to history taking and examination of the adolescent is different from that of an adult. Privacy and comfort were maintained and the girls were interviewed alone on a one to one basis which allowed a rapport to be established and privacy maintained. This overcame the un-welcomed effects of a dominant mother and also allowed her to feel that she has been treated as an adult.

At the end of the interview the problem and any additional information necessary was discussed with the girl's parents. In case of a young girl or those who are with mental handicap, the mother's presence was essential.

History taking was started by asking general questions like her schooling and interests rather than moving directly to the problem. Regarding the gynaecological problem, direct questioning and detail history were taken. Accurate details of a previous medical problem(Mothers were more reliable historian)

In general gynaecological examination of the adolescent girls was performed simply with patience and with respect and honesty with some modification of the adult techniques.

On completion of the general and specific examination, the nature of the problem, its management were discussed in details with the girl. After this, mother was brought and the situation was explained in the girl's presence.

INCLUSON CRITERIA

- Girls attending GOPD in adolescent age group.
- Adolescent girls with menstrual problems.
- Hirsutism and acne.
- Obesity (central with raised waist to hip ratio)

BMI-

Underweight	Normal	Overweight	Obese
Below 18.5	18.5-24.9	25-29.9	30 or higher

EXCLUSION CRITERIA

- Women with signs and symptoms of PCOD who do not correspond to adolescent age group
- Idiopathic hyperandrogenism
- Patients with adrenal gland hyperplasia or tumor
- Patients who were in adolescent age group but were pregnant

THE STUDY WILL BE WORKED UP ACCORDING TO THE FOLLOWING PROTOCOL
HISTORY

- Name
- Age
- Sex
- Address
- Marital Status

CHIEF COMPLAINTS

- Menstrual irregularities.
- Acne.
- Hirsuitism
- Obesity**.**

MENSTRUAL HISTORY
- Last Menstrual Period and cycle History
- amenorrhea/ Oligomenorrhea

Past Medical History
- History of any chronic disease such as (tuberculosis, hypertension, asthma, diabetes mellitus, hematological disorder etc)
- History of previous surgery

Personal History
- History of any type of addiction or any drug
- History of food habits and life style
- History of bowel and bla**General Examination**

- Built
- Nutrition
- Height
- Weight
- Pallor
- Icterus
- Edema
- Cyanosis
- Tongue
- Neck
- Gum
- Tonsil
- Neck Veins
- Lymph nodes
- Leg veins
- Pulse
- Respiration
- Body Temperature
- Blood pressure
- Cardiovascular system: auscultation of heart
- Respiratory system: Auscultation of chest
- Abdominal examination: Liver, Spleen
- Examination of breast

EXAMINATION FOR BODY HAIR DISTRIBUTION [6]
Ferriman and Galway scoring for hirsuitism

A score of 1to 4 is given for nine areas of body Total score less than 8 is normal score, 8 to 15 is mild hirsuitism score more than 15 is moderate to severe

AREAS INCLUDED IN FERRIMAN AND GALWAY SCORING
- Upper Lip
- Chin
- Chest
- Upper Back
- Lower Back
- Upper Abdomen
- Lower Abdomen
- Upper Arms
- Forearms
- Thighs
- Legs

PER ABDOMINAL EXAMINATION
PELVIC EXAMINATION
- Inspection

INVESTIGATIONS
ROUTINE INVESTIGATION
- ABO Rh typing
- HIV1&2
- HBSAg
- VDRL
- Blood Sugar (Fasting And Pp)
- Routine Urine
- Culture And Sensitivity Urine
- TC, DC, Hb%, BT, CT, ESR etc

IMAGING- USG WHOLE ABDOMEN AND PELVIS
SPECIFIC INVESTIGATION
- VLDL, -FT4,FT3,TSH
- LDL, FFA,S. Cholesterol, HDL, Apoprotein A-I
- S.LH,S.FSH
- S. Testosterone (Total &Free)
- S.DHEAS
- Sex hormone binding globulin
- GTT
- Serum Insulin
- S.ANDROSTENIDIONE
- Albumin

DIAGNOSIS –
- Signs and symptoms
- Clinical features
- Investigations

III. Results

Study during May 2016 to September 2017 carried out in the Department of Obstetrics and Gynaecology at Rajendra Institute of Medical sciences Ranchi, Jharkhand. Outpatient adolescent girls aged b/w 11 to 19 years were selected to study the incidence of PCOD among total no of adolescence patient visiting GOPD during the study period b/w May 2016 to september2017. Total no of new cases - 90Total no of enrolment of adolescent age group patient - 984Incidence of PCOD in adolescent age group patient during study period Of May 2016 to September 2017= No of new cases/Total no of patient at risk multiplied by 100. Incidence=90/984*100=9.15%

TABLE-1
INCIDENCE OF PCOD IN ADOLESCENT AGE GROUP PATIENTS

TOTAL NO OF PATIENTS	TOTAL NO OF NEW CASES	INCIDENCE
984	90	9.16%

TABLE-2
DISTRIBUTION ACCORDING TO DIFF AGE GROUP

AGE	NO OF PATIENTS	PERCENTAGE
11 TO 13 YRS	2	2.2%
14 TO 16 YRS	34	37.8%
17 TO 19 YRS	54	60%

Out of 90 PCOD patients there were total 54 patients in age group between17 to 19years and there were 34 patients of PCOD in age group 14 to 16 years , there were 2 patients in age group 11 to 13 years.

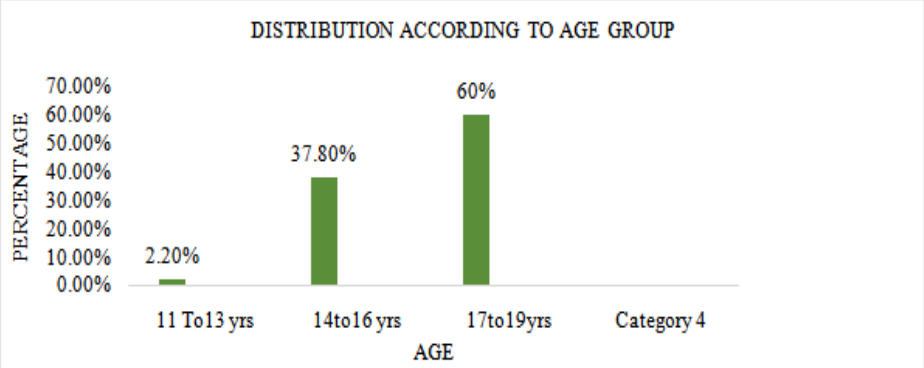


TABLE-3
DISTRIBUTION OF PATIENTS ACCORDING TO THE RESIDENCE

RESIDENCE	NO OF PATIENTS	PERCENTAGE
URBAN	69	76.67%
SEMIURBAN	16	17.78%

RURAL	5	5.55%
TOTAL	90	100%

Majority of the PCOD patients belonged to urban area followed by semi -urban and rural areas this is because the life style and food habits in urban area predisposes to obesity and increased BMI thus leading to increased chances to develop PCOD in adolescent belonging to urban area.

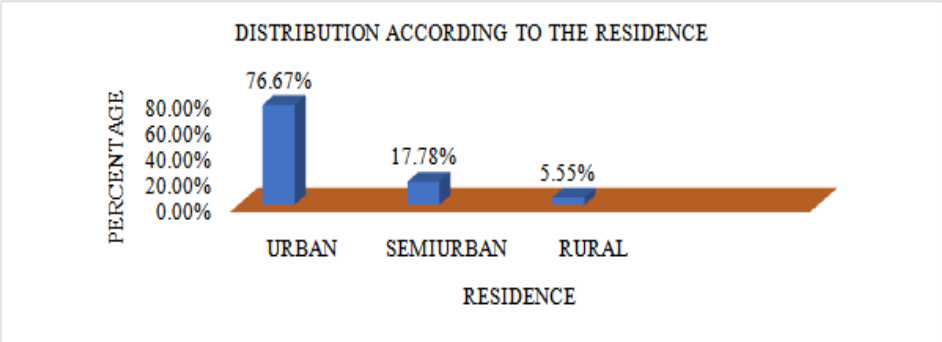


TABLE-4

DISTRIBUTIION OF PATIENTS ACCORDING TO THE TYPE OF FAMILY AND PCOD

TYPE OF FAMILY	NO OF PATIENTS	PERCENTAGE
NUCLEAR	59	65.56%
JOINT	31	34.44%
TOTAL	90	100%

Majority of patients of PCOD were living in nuclear family i.e. about 65.56% patients.

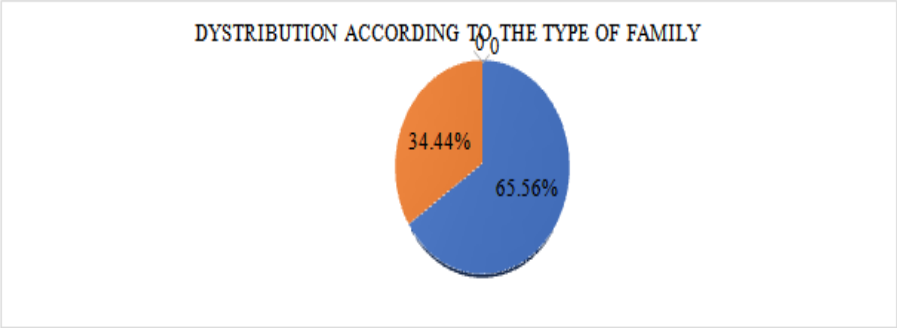


TABLE-5

DISTRIBUTION OF PATIENTS ACCORDING TO THE SOCIO-ECONOMIC STATUS.

SOCIO-ECONOMIC STATUS	NO OF PATIENTS	PERCENTAGE
LOW	17	18.89%
MIDDLE CLASS	73	81.11%
TOTAL	90	100%

Maximum cases of PCOD (81.11%)were from middle class socio – economic status and only 18.89 % belonged to low socio-economic status. PCOD is more common in middle and high socio-economic status though high socio-economic status patients number visiting rims is very less so no data was available.

DISTRIBUTION OF PATIENTS ACCORDING TO MENSTRUAL PROBLEMS

TYPE OF PROBLEM	PATIENTS	PERCENTAGE
IRREGULAR MENSTURATION	34	37.78%
SCANTY MENSES	20	22.22%
SECONDARY AMENORRHEA	22	24.44%
REGULAR CYCLE	14	15.56%
TOTAL	90	100%

Commonest menstrual problem in patients of PCOD was irregular cycle, followed by secondary amenorrhea and then scanty menses.

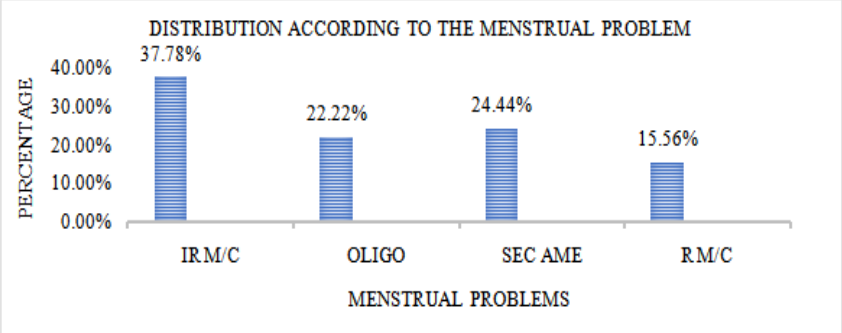


TABLE-6

DISTRIBUTION OF PATIENTS ACCORDING TO THE FOOD HABITS

FOOD HABITS	NO OF PATIENTS	PERCENTAGE
NON VEGETERIAN	42	46.67%
VEGETERIAN	20	22.22%
JUNK FOOD	28	31.11%
TOTAL	90	100%

Majority of patients were non vegeterian by diet and there was about 31.11% case who took junk foods which shows an increasing association of junk food with obesity and increased BMI which increases the risk of developing polycystic ovarian disease.

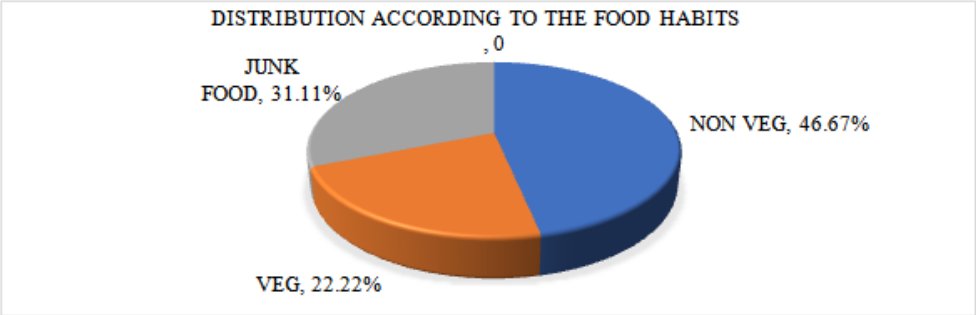


TABLE-7

DISTRIBUTION OF PATIENTS ACCORDING TO THE VALUE OF BMI

BMI	NO OF PATIENTS	PECENTAGE
NORMAL (18.5-24.9)	40	44.44%
OVERWEIGHT (25-29.9)	40	44.44%
OBESE (>30)	10	11.11%

There were about 55.55%PCOD patients who were overweight and obese and there were about 44.44% who were categorized under thin PCOS having BMI between normal range

TABLE-8

DISTRIBUTION ACCORDING TO DIFFERENT CLINICAL FEATURES

CLINICAL FEATURES	NO OF PATIENTS	PERCENTAGE
OLIGO+ I R M/C	3	3.33%
HIRSUITISM	2	2.22%
IR M/C	7	7.78%
SCANTY MENSES +OBESITY	10	11.11%
SEC AME+ACNE	2	2.22%
ACNE+HIRSUITISM	5	5.55%
OBESITY	4	4.44%
I R M/C + OBESITY+HIRSUITISM	4	4.44%
SEC AME	9	10%
SEC AME +OBESITY	5	5.55%
SEC AME +HIRSUITISM	4	4.44%
OLIGO+ACNE	4	4.44%
OLIGO+ACNE+OBESITY	1	1.11%
ACNE+OBESITY	4	4.44%
I R M/C + ACNE	5	5.55%
OLIGO	4	4.44%

IR M/C +HIRSUITISM	4	4.44%
OLIGO+HIRSUITISM	4	4.44%
I R M/C + OBESITY+ACNE	1	1.11%
I R M/C + OBESITY	10	11.11%

TABLE-9

DISTRIBUTION OF PATIENTS ACCORDING TO VALUE OF SERUM HDL

S.HDL	NO OF PATIENTS	PERCETAGE
35-45MG/DL	12	13.33%
>45MG/DL	72	80%
<35MG/DL	6	6.67%

Values of serum HDL were deranged only in 6.67% and was in borderline in 13.33% , it shows that in adolescent patients lipid profile was not so deranged.

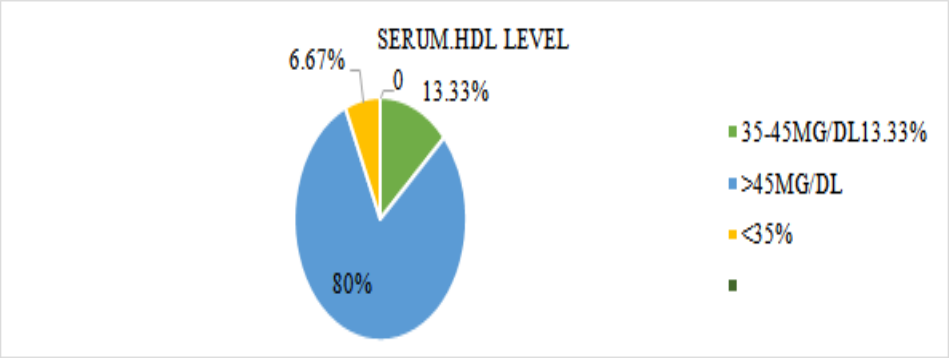


TABLE-10

DISTRIBUTION OF PATIENTS ACCORDING TO THE VALUE OF S.LDL

VALUES	NO OF PATIENTS	PERCENTAGE
NORMAL (<130 MG/DL)	62	68.89%
BORDERLINE (130-159MG/DL)	24	26.67%
HIGH RISK(160-189MG/DL)	4	4.44%

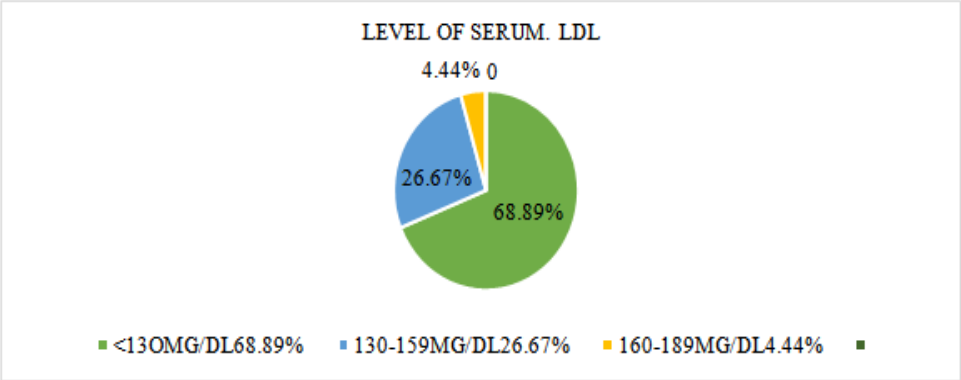
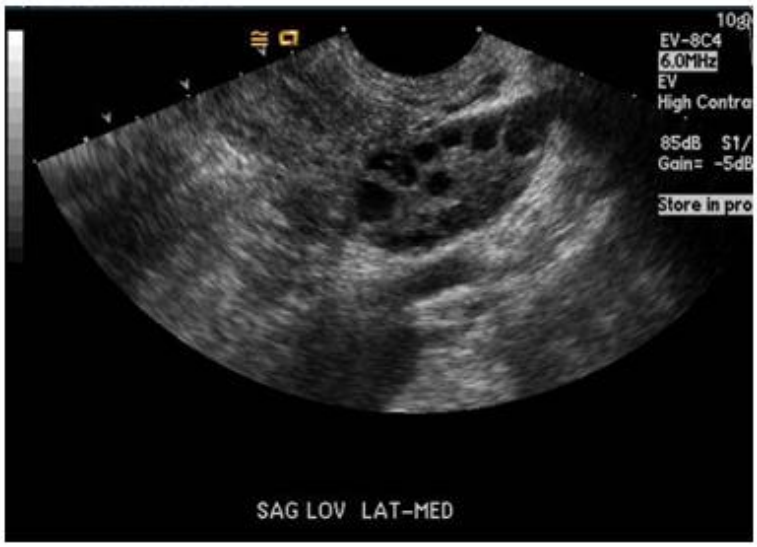


TABLE -11

DISTRIBUTION OF PATIENTS ACCORDING TO THE ULTRASOUND FINDING

ULTRASOUND FINDINGS	NO OF PATIENTS	PERCENTAGE
POLYCYSTIC OVARIES	27	30%
OVARIAN VOLUME>10ML	30	33.33%
USG-NORMAL	33%	36.67%

Ovaries were either polycystic or with increased volume in about 63.33% patients



POLYCYSTIC OVARIES

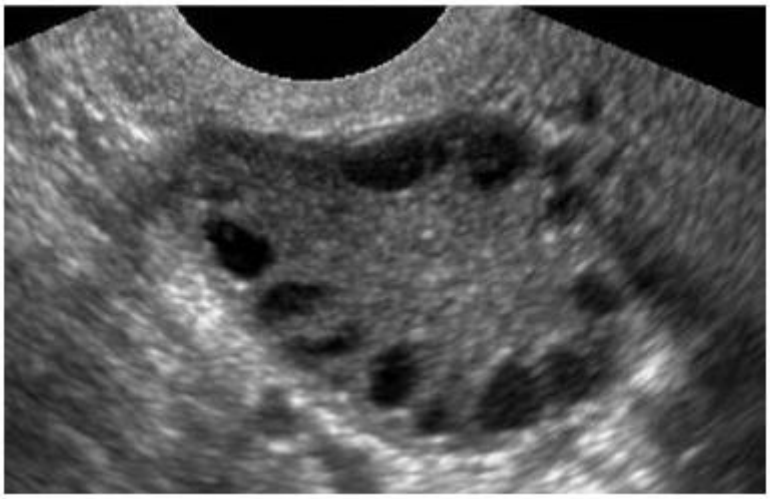


TABLE-12

DISTRIBUTION ACCORDING TO THE FERRIMAN GALWAY SCORE FOR HIRSUITISM

SCORING	PATIENTS	PERCENTAGE
NORMAL SCORE (<8)	24	26.67%
MILD HIRSUITISM (B/W 8 TO 15)	40	44.44%
MODERATE TO SEVERE (>15)	26	28.89%

Scoring more than 8 was present in about 73.33% of patients of PCOD.

TABLE-13

DISTRIBUTION OF PATIENTS ACCORDING TO THE LIFE STYLE

LIFE STYLE	NO OF PATIENTS	PERCENTAGE
SEDENTRY	60	66.67%
ACTIVE	30	33.33%

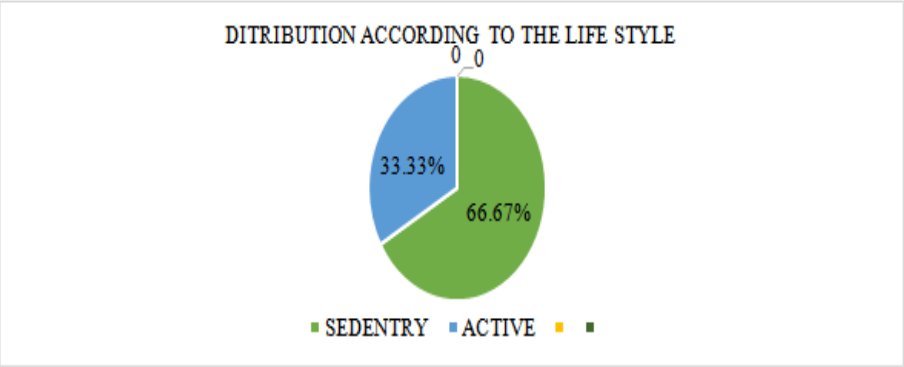


TABLE -14

DISTRIBUTION ACCORDING TO THE S. DHEAS LEVEL

VALUE OF S. DHEAS	NO OF PATIENTS	PERCENTAGE
NORMAL RANGE (3.92 TO 10.66MIC MOL/L)	29	32.22%
RAISED (>10.66 MIC MOL/L)	61	67.78%

67.78% of the adolescent PCOD patients had raised serum DHEAS level

TABLE- 15

DISTRIBUTION ACCORDING TO THE LH/FSH RATIO

VALUES	PATIENTS	PERCENTAGE
NORMAL (<2:1)	55	61.11%
RAISED (>2:1)	35	38.89%

Only 38.89% of the patients had raised levels of LH/FSH ratio in comparison to other biochemical marker. Majority of cases had raised levels of s. DHEAS, S. Insulin and S. Testosterone level

TABLE-16

DISTRIBUTION ACCODING TO THE S. INSULIN LEVEL(FASTING) IN PCOD PATIENTS

S. INSULIN (FASTING)	NO OF PATIENTS	PERCENTAGE
NORMAL LEVEL (<25miu/l)	22	24.44%
INSULIN RESISTANCE (>25miu/l)	68	75.56%
TOTAL	90	100%

In majority of the patients there was hyperinsulinemia

TABLE-17

DISTRIBUTION OF PATIENTS ACCORDING TO THE S. TESTOSTERONE VALUE

S. TESOSTERONE(TOTAL)	NO OF PATIENTS	PERCENTAGE
NORMAL (6 TO 26 MG/DL)	35	38.89%
RAISED (>86 MG/DL)	55	61.11%
TOTAL	90	100%

Majority of the patients had a raised level of serum testosterone level.

There were only 4 adolescent PCOD patients who had a raised level of s. prolactin and there were 7 patients who had associated hypothyroidism

IV. Discussion

The present study was conducted in the department of Obstetrics &Gyn ecology, Rajendra Institute of Medical Sciences, Ranchi, Jharkhand from May, 2016 to Sep, 2017. The adolescent girls of age group between 11-19 yrs were included in my study.

Out of total 984 adolescent girls in accordance to the inclusion and exclusion criteria there were 90 newly diagnosed cases of Polycystic Ovarian disease.

The incidence calculated was 9.16%. The age range in my study was 11-19 yrs although 10-19 yrs is the defined age as per the WHO and IAP recommendations. According to the Indian journal of Endocrinology growth of pubic hairs and breast development starting at age between 8-13 yrs.

The mean age of menarche is typically between 12-13 yrs i.e. 12.50 yrs. In my study there were 18.89% of the patients who attained menarche at the age of 13 yrs. About 20% attained menarche at the age of 11 yrs. There were only 2.22% of the patients attained menarche at the age of 15 yrs. This shows that the age of menarche is in a declining trend.

If PCOS is defined by the ultrasonographic appearance of the Polycystic Ovaries, the prevalence varies. Polycystic Ovaries are seen in 92% of the women with idiopathic hirsutism, 87% of the women with

oligomenorrhea, 21-23% of randomly selected women, 23% of women who consider themselves normal and who report regular menstrual cycles and in 17% of women participating in routine PAP smear[21].

In my study there were 37.78% patients who had irregular menstrual cycle 22.22% had scanty menses about 24.44% of the patients had secondary amenorrhea and about 15.56% patients had regular menses. In my study there 13.33% patients who had borderline deranged S. HDL level and there were 6.67% of the patients who had markedly deranged level of S. HDL.

There were 26.67% patients who had S. LDL borderline deranged and there were only 4.44% patients who were included in high risk category i.e. S. LDL between 160-189 mg/dl. In my study there were 30% of the patients with Polycystic Ovaries and there were 33.33% of the patients who had increased volume of ovaries. There were 36.67% patients who had normal ultrasonography. In my study there were 21.11% of the patients who had hirsutism along with Polycystic Ovaries or Ovaries with increased volume.

When Poison and Colleagues**[23]** examined a large group of volunteers from the general population, they found that 22% of 257 women had Polycystic ovaries by ultrasound examination, however 1/3rd of these had regular menstrual cycles. In my study about 63.33% of the patients had either Polycystic Ovaries or increased Ovarian volume and out of these 22 patients had regular menstrual cycle.

Clinically the most common sign of hyperandrogenism in PCOS women is hirsutism. The range of the prevalence of hirsutism in PCOS women varies between 17 to 83%[28]. Hirsutism may develop Peripubertal or during adolescence or it may be absent until the 3rd decade of life. There is strong evidence of a peripubertal onset of the PCOS the symptoms of which has been used as the diagnostic criteria[29]. In my study there were 16.65% of cases with hirsutism. Other signs of hyperandrogenism present in my study was acne. 17.76% of the patients had acne.

Obesity in particular central obesity plays a key role in the development of PCOS and the majority of the women with PCOS are either overweight or obese. Obesity is an independent factor associated with insulin resistance and sex steroid disturbance, which may lead to an increased risk of menstrual irregularities and hyperandrogenism[32]. Insulin resistance is associated with an increased risk of developing impaired glucose tolerance or manifest type to diabetes, lipid disturbance and cardiovascular disease. The well known obesity associated disturbances in the glucoses and insulin metabolism leading to IGT may however be different from those in women with PCOS, in particular lean women with PCOS[33]. In my study there were 44.44% patients who were overweight and there were 10% of the cases who had BMI more than 30 i.e. were obeas. In PCOS patients there were 75.56% cases who had insulin resistance with raised level of fasting level serum insulin. 61.11% of the patients who raised level of serum testosterone and there were 38.89% of the patients who had normal levels of total serum testosterone. There were 67.78% of the patients who had raised level of serum DHEAS i.e. (>10.66 μ mol/l).

Rapid urbanization and changes in life style in many developing countries is causing an increase in incidence of PCOS. On an average PCOS affects 5-10% of the women in the productive age group worldwide. Prevalence of PCOS is rapidly rising among Indians. Estimate of PCOS in migrant indians had been estimated at 52% level. And about 37% among the north indian women have been estimated to suffer from PCOS[54, 55]. In my study there were 76.67% of the PCOD patients who belonged to urban areas and there were 17.78% patients who belonged to semi-urban areas. Only 5.55% of PCOS patients belonged to rural areas. There were 66.67% of the patients who lead a sedentary life style rest 33.33% had active lifestyle.

V. Conclusion

There is rising incidence of polycystic ovarian disease among adolescent age girls, this rising incidence is attributed to the changing life style, food habits and increasing incidence of obesity.

There were deranged levels of serum. DHEAS, serum Testosterone , serum fasting insulin levels, serum LDL and HDL levels this concludes that there is hyperandrogenism and hyperinsulinemia in adolescent age PCOD patients which is a risk factor to develop metabolic syndrome in future . This is also a hyperestrogenic stage which in future may predispose the development of endometrial hyperplasia

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