A STUDY OF EVALUATION CLINICAL, BIOCHEMICAL AND ULTRASOUND PARAMETERS IN WOMEN WITH PCOD.

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CERTIFICATE

This is to certify that the dissertation entitled "**A STUDY OF EVALUATION CLINICAL, BIOCHEMICAL AND ULTRASOUND PARAMETERS IN WOMEN WITH PCOD** is a bonafide work done by **Dr.D.INDIRANI** at R.S.R.M. Lying in Hospital, Stanley Medical College, Chennai. This dissertation is submitted to Tamil Nadu Dr.M.G.R. Medical University in partial fulfillment of University rules and regulations for the award of M.S. Degree in Obstetrics and Gynaecology.

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I am very grateful to all professors for their invaluable advice, constant guidance and supervision during this study.

DECLARATION

I, **Dr.INDIRANI.D** solemnly declare that the dissertation titled **"A STUDY OF EVALUATION CLINICAL, BIOCHEMICAL AND ULTRASOUND PARAMETERS IN WOMEN WITH PCOD"** is a bonafide work done by me at Govt. R.S.R.M. Lying in Hospital, under supervision and guidance of **Prof.Dr.RAJALAKSHMI, MD DGO** in Department of Obstetrics and Gynaecology, Stanley Medical College, Chennai. This thesis is submitted to the Tamil Nadu Dr.M.G.R. Medical University in partial fulfillment of the rules and regulation for the M.S. Degree examination in obstetrics and Gynaecology to be held in April 2017.

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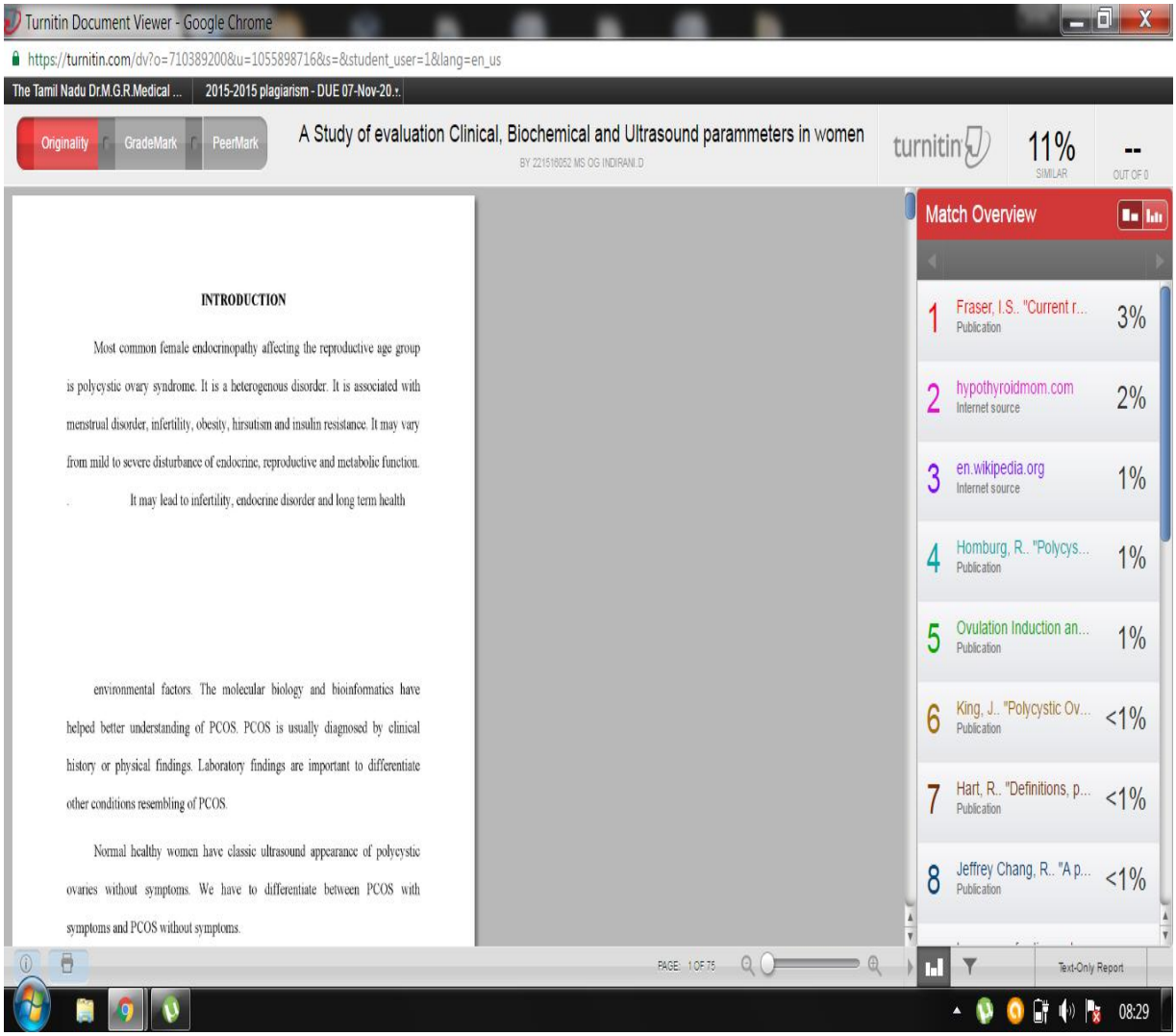
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INTRODUCTION
Most common female endocrinopathy affecting the reproductive age group is polycystic ovary syndrome. It is a heterogenous disorder. It is associated with menstrual disorder, infertility, obesity, hirsutism and insulin resistance. It may vary from mild to severe disturbance of endocrine, reproductive and metabolic function.
It may lead to infertility, endocrine disorder and long term health
environmental factors. The molecular biology and bioinformatics have helped better understanding of PCOS. PCOS is usually diagnosed by clinical history or physical findings. Laboratory findings are important to differentiate other conditions resembling of PCOS.
Normal healthy women have classic ultrasound appearance of polycystic ovaries without symptoms. We have to differentiate between PCOS with symptoms and PCOS without symptoms.
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CONTENTS

LIST OF ABBREVATIONS USED

PCO	-	Polycystic ovary
PCOS	-	Polycystic ovary syndrome
TVS	-	Trans vaginal sonograpny
TAS	-	Trans abdominal sonography
LH	-	Leutinizing hormone
FSH	-	Follicle stimulating hormone
NIH	-	National Institute of Health
USG	-	Ultrasonography
BMI	-	Body mass index
F-G Score	-	Ferriman – Gallwey Score
HDL	-	High densitylipoprotein
CAH	-	Congenital adrenal hyperplasia
ACTH	-	Adrenocorticortropic hormone
17OHP	-	17 hydroxy progesterone
RIA	-	Radio immunoassay
OCP	-	Oral Contraceptive pill
SHBG	-	Sex hormone binding globulin
DHEAS	-	Dehydroepiandrosterone sulphate
GnRH	-	Gonadotropin releasing hormone

INTRODUCTION

Most common female endocrinopathy affecting the reproductive age group is polycystic ovary syndrome. It is a heterogenous disorder. It is associated with menstrual disorder, infertility, obesity, hirsutism and insulin resistance. It may vary from mild to severe disturbance of endocrine, reproductive and metabolic function.

It may lead to infertility, endocrine disorder and long term health environmental factors. The molecular biology and bioinformatics have helped better understanding of PCOS. PCOS is usually diagnosed by clinical history or physical findings. Laboratory findings are important to differentiate other conditions resembling of PCOS.

Normal healthy women have classic ultrasound appearance of polycystic ovaries without symptoms. We have to differentiate between PCOS with symptoms and PCOS without symptoms.

AIMS AND OBJECTIVES

- To analyse various clinical presentation of PCOS in population in North Chennai.

- To establish the role of hormonal assay in diagnosing PCOS.

- To understand the correlation between clinical and endocrine changes with PCOS.

REVIEW OF LITERATURE

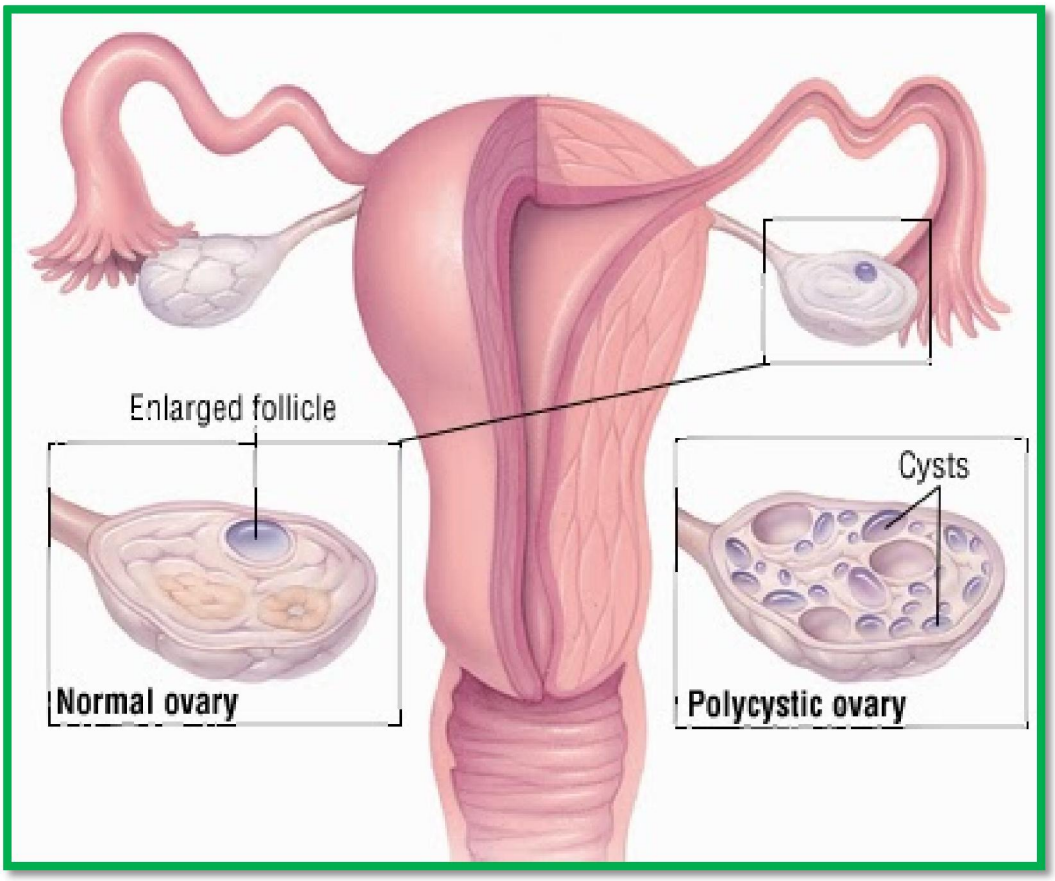


Figure 1: Polycystic Ovaries

Definition

A meeting was conducted recently by European society of Human Reproduction and Embryology regarding the redefinition of PCOS, which was agreed by Rotterdam consensus group 2003. It said that all clinicians and investigators should use this internationally agreed definition to be uniform in research and routine clinical management.

Meeting recommended at least two of the following 3 criteria in diagnosing PCOS.

1. Oligomenorrhoea / Anovulation

2. Laboratory finding / Clinical Findings suggestive of hyperandrogenism

3. Direct Inspection of polycystic ovaries by ultrasound. Other aetiologies for hyperandrogenism ruled out Eg: Cogential adrenal hyperplasia,Cushing's syndrome or Androgen -secreting tumours .

Androgen excess and PCOs Society (AE-PCOS) specified similar set of criteria for diagnosis of PCOs .They said that PCOs should be primarily a disorder of androgen excess or hyperandrogenism. The clinical criteria set forward as

1. Presence of hyperandrogenism (Clinical/Biochemical)
2. Ovarian dysfunction (Oligoanovulation/Polycystic ovaries)
3. Exclusion of related disorders.

NIH Criteria:

In 1990 a consensus workshop sponsored by NIH/NICHD suggested that a person has PCOs if they have all the following criteria

1. Oligoovulation
2. Signs of androgen excess
3. Exclusion of other disorder that can result in menstrual irregularity and hyperandrogenism

NIH 1990	ROTTERDAM 2003	AES 2006
MUST INCLUDE ALL 1.Chronic Anovulation 2.Clinical/Biochemical Signs of Hyperandrogenism	**TWO OF THE FOLLOWING** 1.Oligo/Anovulation 2.Clinical/Biochemical Signs of Hyperandrogenism 3.Polycystic Ovaries on USG	**MUST INCLUDE ALL** 1.Ovariam Dysfunction 2.Polycystic Ovaries On USG and Androgen Excess

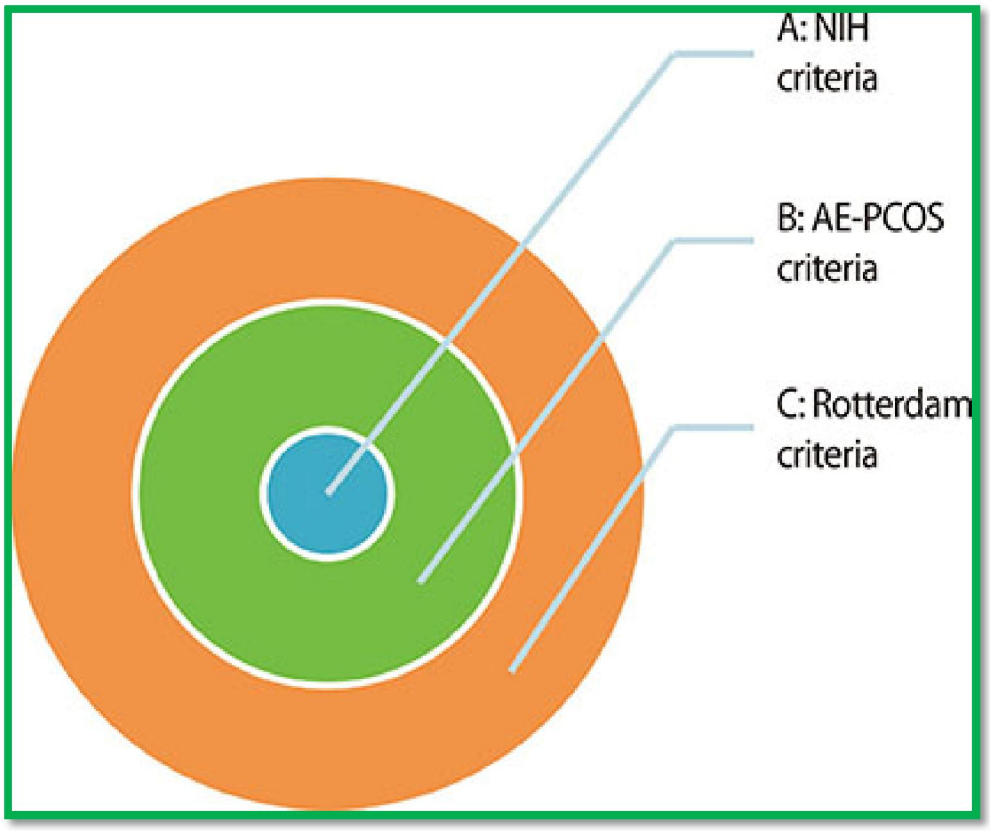


Figure 2 : CRITERIA OF PCOD

In UK –PCOs is diagnosed by ultrasound findings plus symptoms (Obesity, hyperandrogenism, menstrual cycle disturbances) and elevated serum concentration of testosterone/LH.In North America –Hyper androgenism plus menstrual cycle disturbances favour PCOs. In our country, PCOs is usually diagnosed by morphology seen in USG.

PCOS could be divided in 4 types

1. **Phenotype A:** Menstrual disturbances plus symptoms hyperandrogenism (Classical Symptoms)
2. **Phenotype B :** Menstrual irregularity with hyperandrogenism
3. **Phenotype C:** Hyperandrogenism with ovulatory PCOS.
4. **Phenotype D:** Menstrual irregularity + PCOS also called non hyperandrogenism PCOS.

PREVALENCE

PCOS is the most common gynaeocological condition affecting 5 to 10% of women in their reproductive years. It affects 20% of female population without any signs and symptoms. It is expressed some time during their fertile period.

AETIOLOGY

The underlying cause of PCOS is unknown. However, there is increasing evidence of genetic factors. It is a multifactorial disease. It is well documented by aggregation of the syndrome within the families. Increasing evidence has been noted between affected individuals and their sisters and mothers. It has been proved that it is transmitted as Autosomal Dominant. Environmental factors are also play a role in PCOS. It may be due to exposure of androgens during the fertile period. Identification of candidates with PCOS is a large research focus. It helps us for diagnosis and management of PCOs. It tells about the genes regarding androgen synthesis and insulin resistance. Experiment in Animals showed that exposure of fetus to excess androgens in uterus lead to these manifestations. PCOS is characterized by chronic anovulation. Ultrasound is the major tool for diagnosing PCOS. About 25% of normal women will demonstrate ultrasound findings typical of polycystic ovaries. Patients on oral contraceptive pills will also have polycystic ovaries.

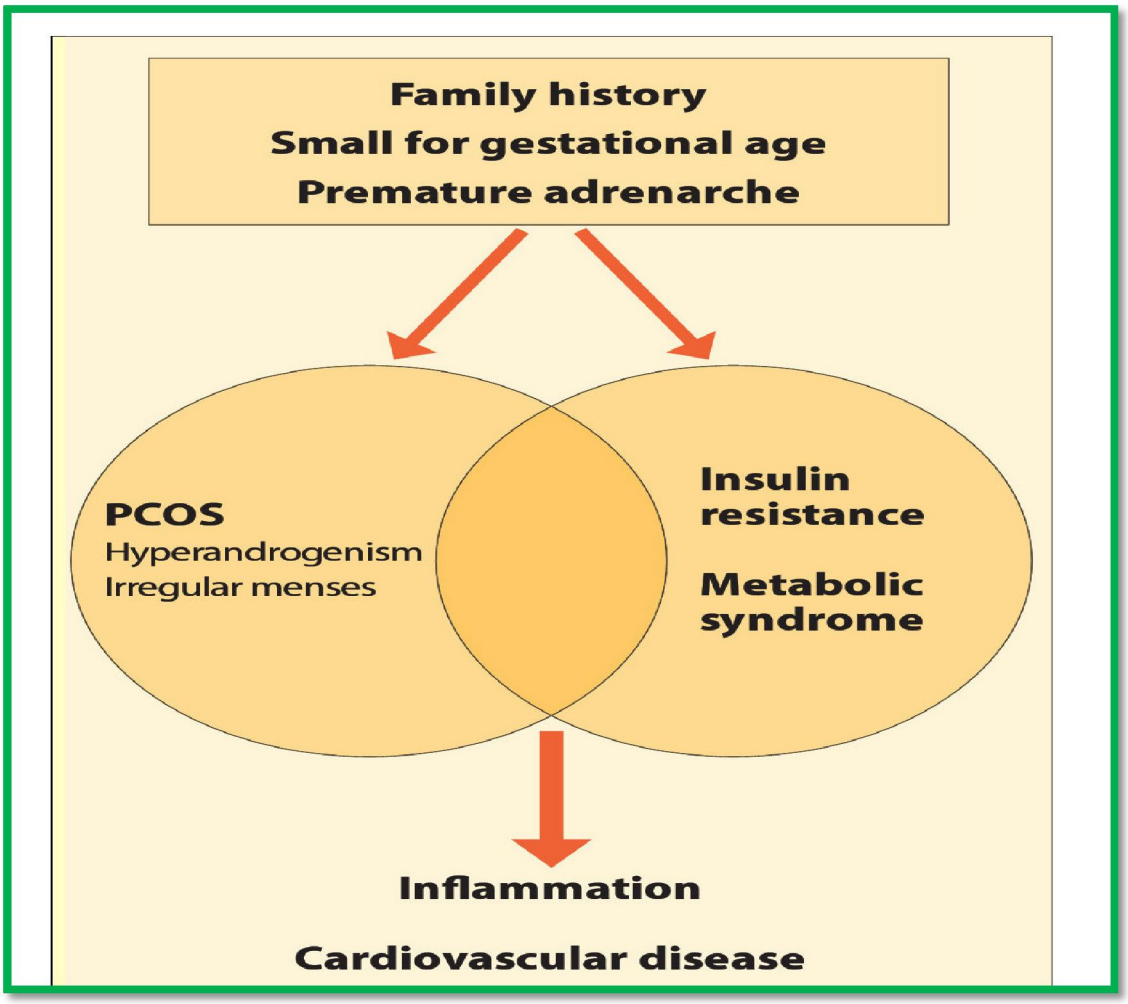


Figure 3: Relationship between PCOs and metabolic syndrome

PATHOPHYSIOLOGY

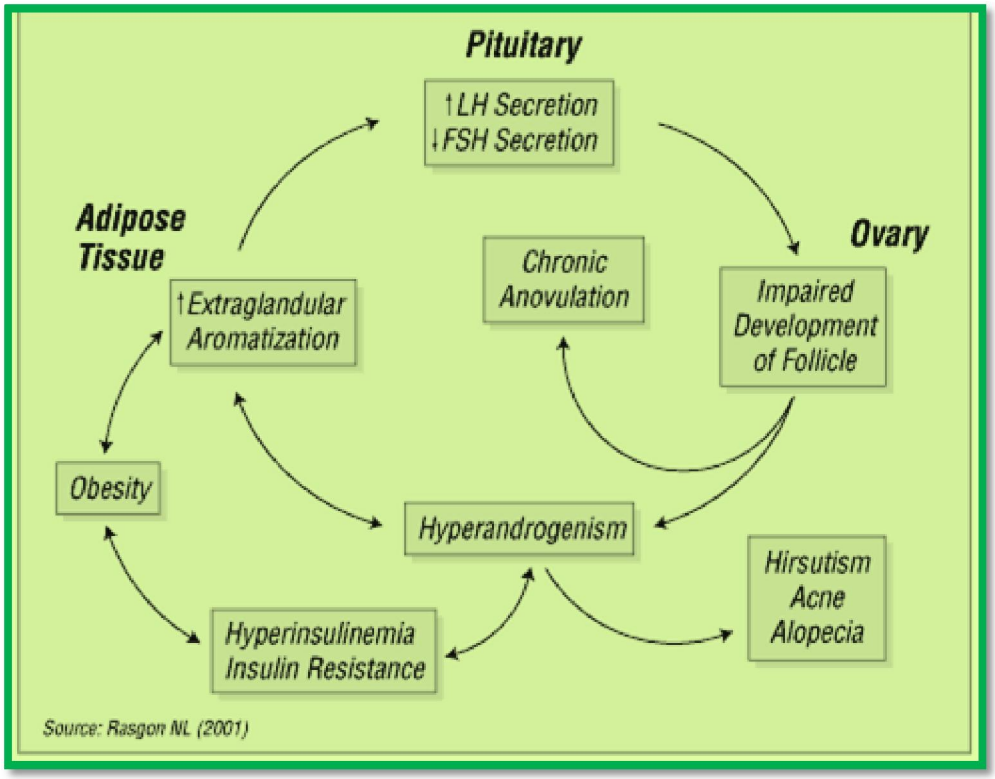


Figure 4: Pathophysiology of PCOs

There are four important pathophysiology in PCOS.

1. Abnormal ovarian morphology
2. Ovarian Androgen production
3. Hyperinsulinemia
4. Excess serum concentration of LH.

ABNORMAL OVARIAN MORPHOLOGY

6-8 times more preantral and small antral follicles are present in the polycystic ovary. The Follicles are arrested in development size of about 2-9 mm. There is slow rate of atresia. They are sensitive to exogenous follicle stimulating hormone. There may be enlarged stromal volume. Total ovarian volume greater than 10cc is seen. Excess androgen play a major role in abnormal ovarian morphology. Primary follices develop into the stages of preantral and small antral follicles in the presence of excess androgens. Follistatin, epidermal growth factor, androgens and antiapoptotic factors play a role in arrested growth of follicles. These give the appearance of typical polycystic ovarian morphology.

OVARIAN ANDROGEN PRODUCTION

Ovarian androgen production is the heart of PCOS. Androgen levels are typically elevated in PCOS. Progesterones levels are low due to anovulation. Both Luteinizing hormone and insulin play a key role in androgen excess.

Insulin Resistance

Women with PCOS have higher degree of insulin resistance with compensatory hyperinsulinemia. Patients with PCOS have post receptor uptake defect of glucose transport. Obesity is a major cause of PCOS. They have hyperinsulism leading to anovulation and hyperandrogenism.

Excessive serum concentration of LH are detected by blood sampling on 2^{nd} day of menstrual cycle. Increasing concentration is seen in lean PCOS. FSH level is not suppressed.

Homa IR Index

1. Homa model is used to yield an estimation of insulin sensitivity and Beta Cell function from fasting plasma insulin and glucose concentration.

2. Insulin resistance is a state in which the normal concentration of insulin produce a subnormal biological response.

Uses

1. It assesses the risk of development of Diabetes.
2. It allows assessment of inherent beta cell function and insulin sensitivity.
3. It characterizes those with abnormal glucose metabolism.
4. It can be used to assess the response to diet or oral drug therapy.

Limitations

1. Insulin glucose homa model cannot be used in those taking exogenous insulin.

2. Homa IR calculation version 2.2 accepts values in the following ranges of plasma insulin 2.9 to 57ml and blood sugar fasting 54.12 to 450 (mg/dl). Homa IR calculation is not possible if values are outside these ranges.

LH and FSH

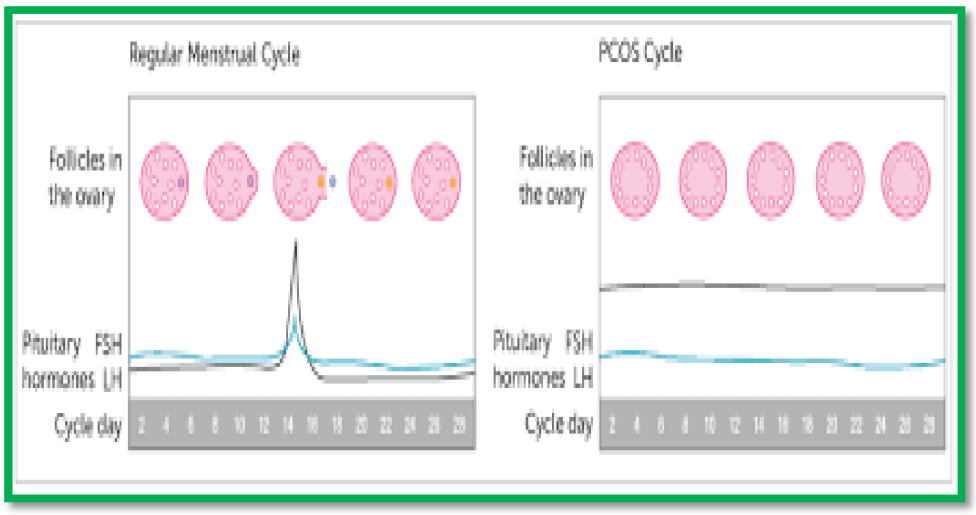


Figure No. 5 : Menstrual Cycle

LH and FSH that encourage ovulation .LH and FSH are secreted by Pituitary gland. At the early follicular phase LH and FSH levels are usually range between 5 to 20 mIU/ml. During follicular phase most women have equal amount of LH and FSH. Before 24 hrs of ovulation LH surge occur, in which the amount of LH increases to about 25 to 40 mIU/ml. LH levels are returns back when follicle ruptures.

Women with PCOs have LH levels 2 to 3 times that of FSH .They have elevated LH, FSH ratio. This elevated LH to FSH ratio disrupts ovulation. It is important in the diagnosis of PCOs. 40 to 50% of women

with PCOs have excess serum concentration of LH. High LH Concentration are more commonly seen in lean women than obese .There is intrinsic inhibition of FSH may present.

Pathogenesis of PCOD

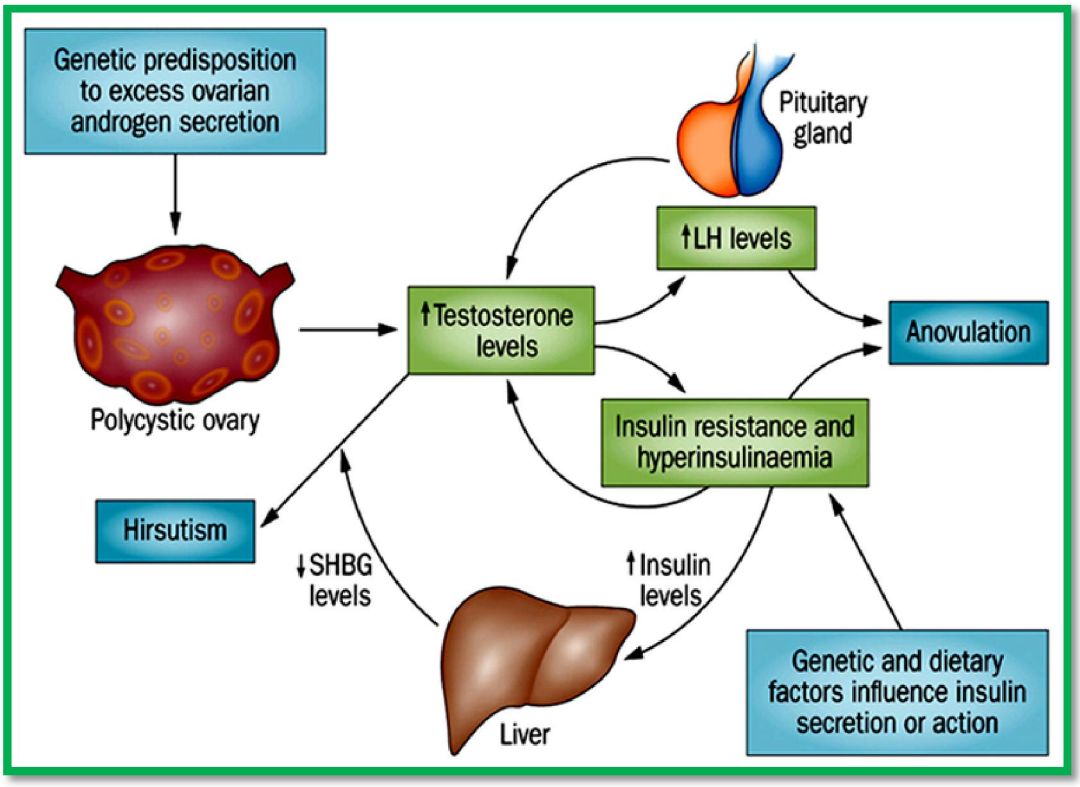


Figure 6: Pathogenesis of PCOD

Polycystic ovaries develop when the ovaries are stimulated to produce excess amount of androgenic hormone. The most important hormone is testostosterone with genetic susceptibility. The syndrome is popularly called as polycystic ovaries due to common sign in ultrasound. The follicles developed from primordial follicles. The

development has stopped at early antral stage due to disturbed ovarian function. The folliciles may be oriented along the peripheries of the ovary as a string of pearls on USG.

Women with PCOs have increased frequency of hypothalamic GNRH Pulses which inturn results in increase in LH & FSH Ratio. Most of the women with PCOs have insulin resistance and are obese. Elevated insulin level in PCOs causes abnormalities in hypothalamic pituitary ovarian axis that leads to PCOD. Hyperinsulinemia increases GNRH pulse frequency which in turn increases LH over FSH. It also increases ovarian androgen and decreases follicle maturation. Insulin resistance is common in obese and lean. Excess adipose tissue in obese women creates excess androgens which is responsible for virilisation.

Clinical Features

PCOs+Hyperinsulinaemia+Central Obesity

↓

Ovarian androgen Production +Serum Beta HCG +Free Testosterone+5-Alpha Reductase

↓

- **More FSH needed for ovulation induction**
- **Greater tendency to multi follicular response**
- **Inferior pregnancy and miscarriage rates**
- **Hirsutism**
- **Acne**

PCOS exhibits with different clinical features with variable presentation. By knowing the clinical features of particular patients, it helps the gynaecologist during workup , initial management and long term health risk.

Menstrual irregularity

The most common clinical presentation is oligo/amenorrhoea hence ovulation is inhibited. So it is usually associated with infertility. Patient usually attains menarche at right time followed by amenorrhoea associated with stressful life events. Unopposed estrogen may be associated with heavy bleeding that may cause endometrial hyperplasia. Period of amenorrhea is usually associated with elevated levels of LH with decreased levels of FSH. Serum prolactin and serum oestradiol level may be within normal value. The most important symptom is oligomenorrhea. There is hyperandrogenism followed by irregular ovulation. In PCOS, most of the patients have amenorrhea (47%) normal cycles (29%) Polymenorrhoea (2.7%). Patients with PCOS have prior irregular cycles may develop regular cycles as age advances. There may be decrease in antral follicle count.

In gynaecology outpatient department most of the patients have oligomenorrhoea. Menstrual disturbances are associated with obesity, insulin resistance and elevated androgens. Elevated LH, increases the ovarian volume. Regular cycles are present is women with PCOs if Body Mass Index (BMI) is less.

HYPERANDROGENISM

Hyperandrogenism is manifested clinically by hirsutism, acne and alopecia. Lab test in PCOS reveals hyperinsulinemia and Hyperandrogenism. Predominant symptom based on American society definition is symptoms of hyperandrogenism. Patients with insulin resistance may have acanthosis nigricans .

Hirsutism

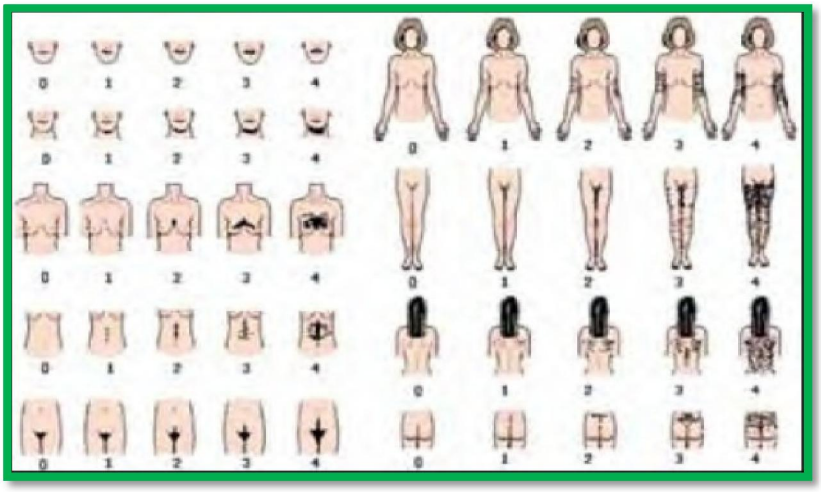


Figure 7: Ferriman-Gallwey score

In female hirsutism is male distribution of hair pattern. The number of hair follicle do not vary. The quality, length, diameter and pigmentation varies. It may be difficult to identify where normality ends and hirsutism begins. The most important androgen is DHTS,which is responsible for hirsutism features. PCOS is most common cause of hirsutism. The score of 6 -8 according to Ferriman-Gallwey score is suggestive of hirsutism ,which uses 9 areas.

In our country, hirsutism is considered if the score is 8 or above. Other causes of hirsutism are thyroid dysfunction, androgen secreting tumours of ovary and adrenals. Patients with hyperprolactinemia and acromegaly also present with hirsutism

ACNE

Figure 8: ACNE

Most of the PCOS patients also have acne. The controversy is also patients who have acne have PCOS. The most important culprit here is androgen which causes enlargement of sebaceous glands. Further enlargement sebaceous gland is caused by dihydrotestosterone which is active metabolite of androgen. The enlarged glands favour the growth of priopionibacterium . The bacterium which digests the sebum and makes it more viscous and stimulates inflammation. Comedons are the initial lesions of Acne. Distrupted follicles release inflammatory mediators into the dermis, resulting in papule, pustule and cyst formation.

Usually acne develops on face occasionally on back and chest. The lesions are tender and heal by scarring. Some studies say the relationship between severity of acne and androgen levels.

Scalp alopecia

Hair loss caused by PCOS called known as Androgen Alopecia. It is progressive patten of loss of scalp terminal hair without scarring. Hair loss is diffuse usually in the crown. Frontal hairline is usually preserved Although many hormones play role in androgen alopecia the main hormone is dihydrotestosterone.

Acanthosis Nigricans

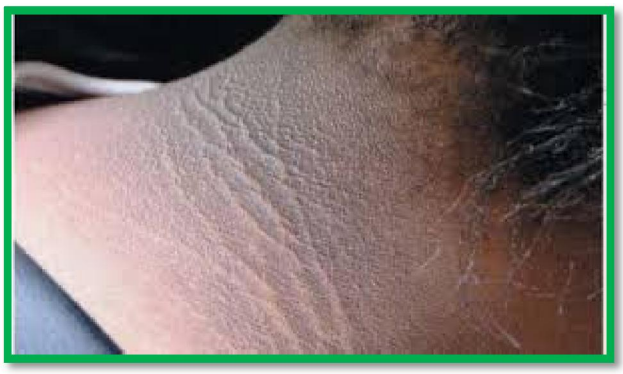


Figure 9: Acanthosis Nigricans

5% of PCOS have aconthosis nigricans. It is a skin manifestation leads to dark velvety appearance due to keratin deposition and pigmentation. The usual sites are nape, neck, axilla, under the breast. These are skin manifestations due to insulin resistance.

Clitromegaly

Goldzieher and Axelrod published a landmark review of the symptomatology and biochemical features of 1079 cases of surgically-proven PCOs and described a 21% incidence of mild virilization. PCOS usually manifest with mild degrees of clitromegaly. If there is

significant clitromegaly features of virilisation is looked for. If patient has adrenal or ovarian androgen secreting tumours there is rapid onset of virilisation with frontal balding, deepening of voice, broadening of shoulders, decrease in breast size and loss of vaginal rugae.

Hyperandrogenism with Hyperinsulinaemia

Hyperandrogensim is the most troubling symptom in PCOS. There is hyperinsulinism contributing to hyperandrogenism . There is great difficulty in measuring insulin resistance .Taking glucose sample frequently is difficult. Fasting blood glucose level is simple clinical tool. It has a great disadvantage to measure glucose level at single time .Some prefer to measure fasting blood sugar and 60 minute serum insulin. Usually the insulin resistance varies during female life time, with increasing age androgen level and insulin resistance increases.

Obesity

Obesity is the major cause of PCOD. Obesity increases the risk of PCOD by several factors. Obesity decreases Sex Hormone Binding Globulin and increases free testosterone and oestradiol. Dyslipidaemia in obesity which causes Coronary Heart Diseases. Insulin resistance in PCOD causes diabetes mellitus . Besides leading to diabetes mellitus it causes hyperinsulinemia leading to PCOS. Gynaecologist administer insulin senstiser for ovulation which also decreases androgen levels. Dyslipidaemia is one of the manifestation of PCOS is due to Insulin resistance. To asses obesity the most common used ratio is Body Mass Index which is ratio between weight in kilogram divided by height in meter^{2}square. BMI normal ranges (19-25), (26-29) is overweight, over 30 is being obese. Obesity is also measure by skin fold thickness and Waist Hip Ratio.

LONG TERM RISKS

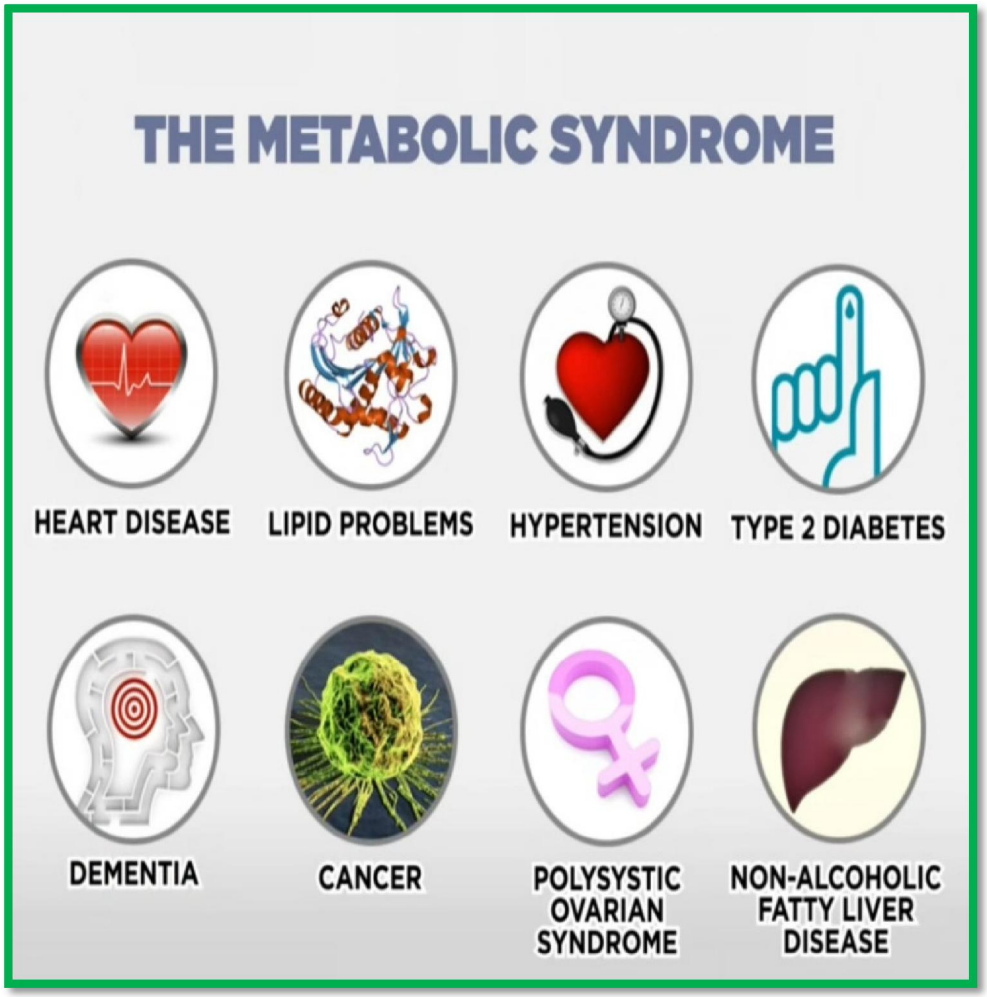


Figure 10 : Long term risk of PCOs

Glucose Intolerance

Dahlgren has proved that PCOS is the risk faster for Diabetes Mellitus.Diabetes Mellitus occurs only when pancreas could not

produce sufficient insulin. Even in lean PCOS there is Diabetes Mellitus and Insulin resistance. Insulin sensitisers should be given to Insulin resistance patients. In view of reducing long term complications of PCOS and minimizing cardiovascular risk, insulin senstiser is used. By giving insulin sensitiser androgen level decreased and complications of PCOS are decreased. Diabetes prevention programme campaigned by using metformin reduced the development of frank diabetes.

Cardiovascular Diseases

We have no proven evidence that PCOS is associated with long term cardiovascular diseases. Even though rising blood sugar and lipid profile increases the cardiovascular risk, there is no proven evidence that PCOS have cardiovascular Diseases. Study was made to prove Association between cardiovascular diseases and PCOS but there was no proven evidence.

Infertility

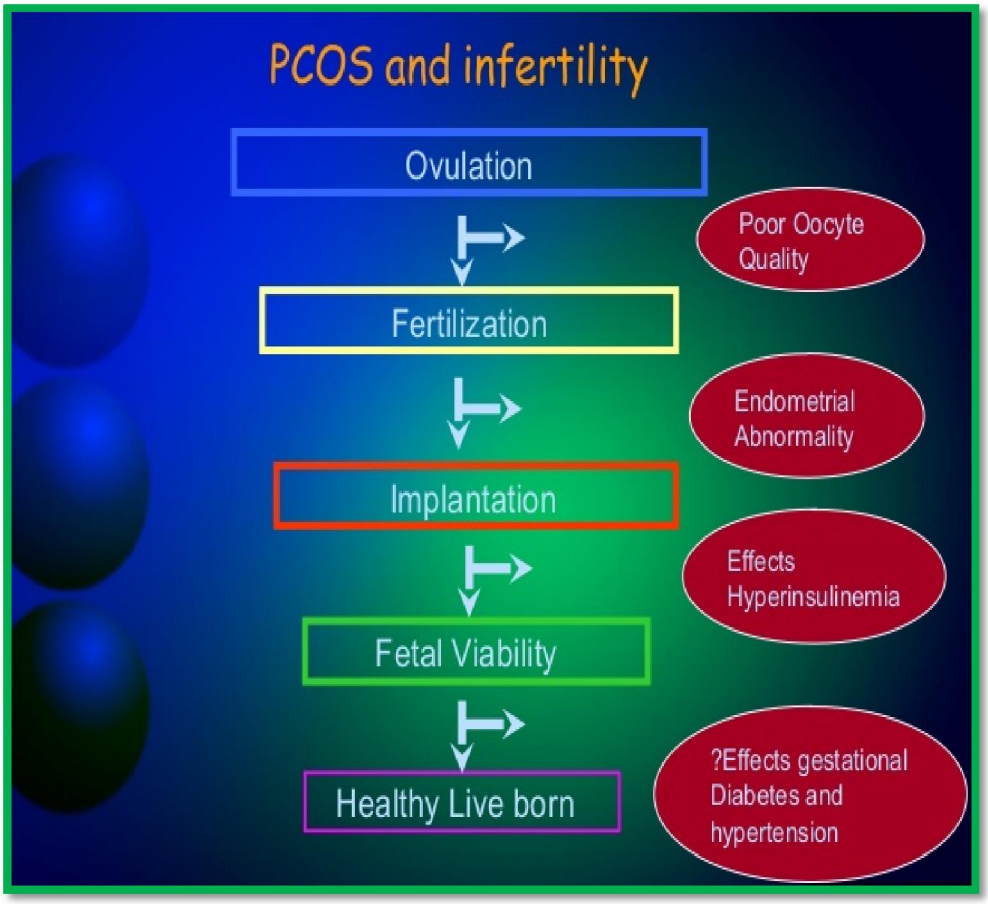


Figure 11 : PCOs Infertility

PCOS women present with sub fertility. This is because they have oligoamenorrhoea and amenorrhoea. Both of these are associated with anovolation. By giving insulin senstiser and inducing ovulation, fertility rate increases.

Recurrent Miscarriage

Women who have conceived on PCOS have recurrent miscarriage. The cause of recurrent miscarriage is unknown. But investigation should done to exclude other causes.

Endometrial Hyperplasia and Adenocarcinoma

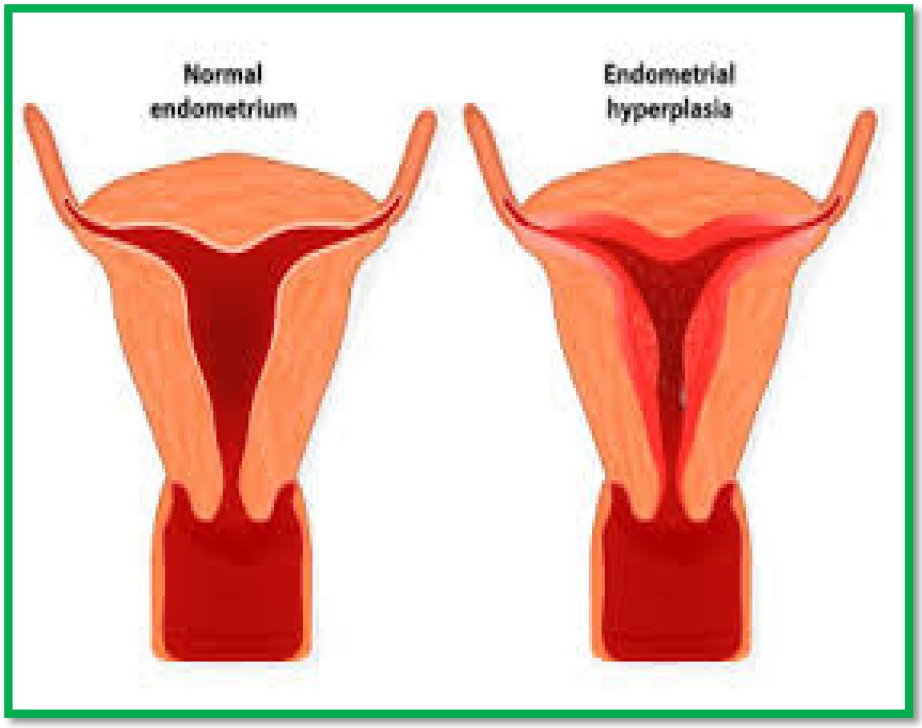


Figure 12 : Endometrial Hyperplasia and Adenocarcinoma

The patients who have PCOS have endometrial hyperplasia & adenocarcinoma. Dahlgren came to the conclusion that endometrial hyperplasia and adenocarcinoma is due to unopposed oestrogen. It is

mandatory to do endometrial sampling on hysteroscopy guided biopsy for patients with PCOS who have heavy and continuous bleeding.

Depression :

Screening with women with PCOs for depression and anxiety by history and if identified ,provide appropriate referral.

Sleep Disordered Breathing

Over weight and obese women with PCOs are screened for sleep disorder.If identified they are diagnosed by using plethysmography. Diagnosed patients should be referred for institution for treatment**.**

Non alcoholic fatty liver disease

There should be awareness of possibility of NAFLD and non alcoholic steatohepatitis in PCOD Patients.

Differential Diagnosis

a. Oligo/Amenorrhoea from other causes

1. Hypothalamic Amenorrhea
2. Hyperprolactinaemia
3. Premature ovarian failure
4. Drug therapy

b. Hyperandrogenism

1. Late onset congenital adrenal hyperplasia
2. Cushing's diseases
3. Androgen secreting tumors
4. Obesity for non PCOS reason
5. Non PCOS insulin resistance

Ovarian Hyperthecosis

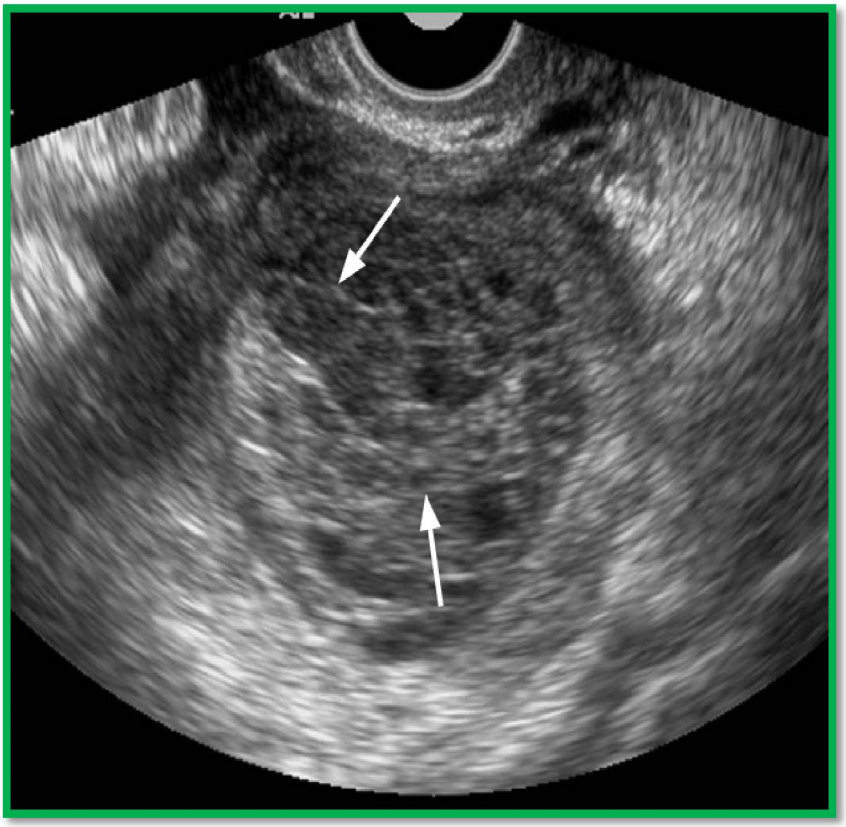


Figure 12 : Hyperthecosis

Ultra sound picture of hyperthecosis showing there is no follicle.

There is a table telling difference between hyperthecosis and PCOD.

	PCOs	Hyperthecosis
Inheritance	No	Autosomal Dominant
Ovarian Size	++ Volume > 10cm	+ Volume >7 cm
Follicles	Impaired Growth	Atretic
Stroma	Dense	Very Dense
Capsule	Thick	Thick
Appearance	Hirsutism	Virilisation
Menses	Oligo /Ameno	Oligo/Ameno
Hormones	Testosterone - N/↑ 17 OHP- N LH/FSH > 3	Testosterone ↑ 8 Fold 17 OHP ↑ twice
Biopsy	Theca cyst	No

Table 2: Difference between PCOs and Hyperthecosis

Hyperthecosis is a proliferative condition of ovary where nests of luteinizing theca cells scattered throughout the stroma. The ovary is enlarged due to fibroblast growth. Only differentiation between PCOS and hyperthecosis is absence of follicles. Patients with ovarian hyperthecosis high androgen level leading to severe virilisation. These

patients have Insulin resistance with circulating high insulin levels. Only the feature of insulin resistance is acanthosis nigricans.

Cushing syndrome

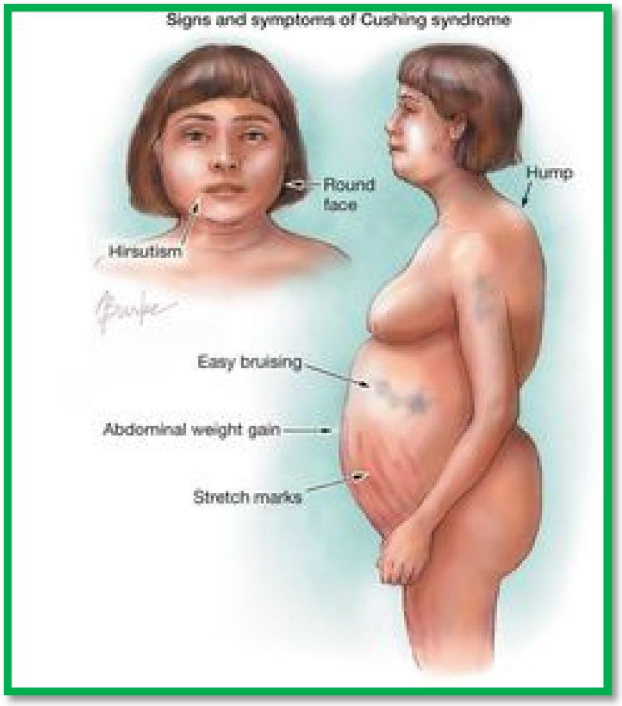


Figure 13 : Cushing's Syndrome

Cushing's syndrome is due to excessive cortisol production from adrenal neoplasm as Adreno Cortico Trophic hormone(ACTH). Over production of ACTH due to primary cause could be pituitary. Secondary cause may be adenocarcinoma of lung. Clinical feature of

cushings syndrome are obesity, hirsutism, acne and menstrual irregularity. They also have moonlike face, buffalo hump, hypertension, muscle wasting, abdominal striae and osteoporosis. It is usually manifested by increased free cortisol excretion in urine, loss of circadian rhythm and failure of suppression of dexamathasone. Ovaries do not have any features.

Androgen Producing Neoplasm

Very rarely androgen producing tumour are present in ovary and adrenal gland. The symptoms are very quick in onset and progress with in months like severe hirsutism, male body habits and clitromegaly. Because of rapid onset of symptoms it provides clue for diagnosis.

EVALUATION

Diagnostic evaluation

History

1. Menstrual History – includes age of onset of menarche, the present cycle, duration associated with menorrhoea. Periods of oligo / amenorrhea.

2. History of Androgenic symptoms such as hirsutism, alopecia, duration of onset of virilisation, acne and cliteromegaly.

3. Body mass index, obesity, eating & exercise habits

4. Questions regarding their lifestyle including alcohol, cigarette and smoking

5. History regarding infertility & recurrent miscarriage.

6. Family history of PCOS, Obesity, diabetes and hyper androgenism.

Examination

1. To evaluate obesity seeing weight, height, BMI, waist hip ratio.

2. Assessing hirsutism by ferriman & gallway score in face, chest & abdomen

3. General examination includes blood pressure, thyroid enlargement, abdominal striae, frontal balding, deepening of voice, broadening of shoulders, decrease in breast size and loss of vaginal rugae.

4. Pelvic examination: includes ovarian enlargement depending upon obesity ,loss of vaginal rugae and clitoral enlargement

LABORATORY INVESTIGATION

Gonadotropins

Abnormal gonadotropin secretion particularly increased LH concentration is seen in PCOS. The Patient have decreased or normal FSH, which causes abnormal LH/FSH ratio. LH and FSH is measured by radioimmuno assay. There was studies to suggest LH/FSH >3 as diagnostic criteria. It was taken as diagnostic criteria in absence of LH surge during ovulation or menopausal transition. Radio Immunoassay is based on polyclonal antibodies. The results obtained were not fully agreed. So the method was changed to immunometric assay. In immunometric assay, Monoclonal antibody is targeted to specific sites

and hence they were not accepted. So they developed new monoclonal assays. They found that LH/FSH ratio ≥ 1provided the diagnostic criteria of PCOS. In lean women there is rise in LH level which causes the features of PCOS. But in obese individuals the rise in androgen levels and insulin resistance leading to PCOS. So BMI play important role in diagnosing PCOS. Circulating oestradiol and progesterone could be measured. Oral contraceptive pills affect gonadotropins levels. By doing physiological and methodological evaluation of LH, LH could be used in diagnosing PCOD.

Androgens

There is increase in androgen production. To establish the diagnosis of PCOs clinical or biochemical signs of Hyperandrogenism must be present .Signs of hyperandrognism is hirsutism acne and hair loss. The measurement of androgen is most important step. The androgen measured are testosterone, Androstenedione, Dihydroepiandrosterone, Dihydroepi androsterone sulphate. They are produced from adrenal cortex and conversion of hormones by ovarian theca cells.

In premenopausal woman Dihydroepiandrosteronesulphate is produced by adrenal cortex. It is in free from without producing any virilisation. Dihydroepiandrosterone sulphate has two sources by ovary and adrenal cortex. Androstenedione is produced by 1) ovary 2) Adrenal cortex 3) from peripheral conversion of DHEA. Under the LH stimulation with negative feedback of oestradiol ovaries produce Androstenedione and testosterone. There is diurnal variation of Androstenedione. Diurnal variation is due to adrenal contribution and variation in ovarian contribution due to altered menstrual cycles. But it increased in PCOS.

Testosterone is most important androgen in PCOS and is produced by adrena,l ovaries and peripheral conversion from circulation.Testosterone is converted to active Dihydrotestosterone by 5α reductase in target tissue. It does not affected by dihydrotestoserone .Many studies concluded that testosterone level is elevated in PCOS. The study says total testosterone is diagnosis of PCOS. Estimation of free testosterone help in diagnosis of PCOS than bound testosterone. Female androgenism is measured by Free Androgen Index. It is most widely used to assess female androgenism. Sex Hormone binding globulin could also be used to diagnose PCOS. Sex Hormone Binding

globulin is decreased in PCOS. Testosterone binds tightly with Sex Hormone Binding globulin. In PCOS increased circulating insulin level decreases Sex Hormone Binding globulin with elevated Free Androgen Index.

Antimullerian hormone

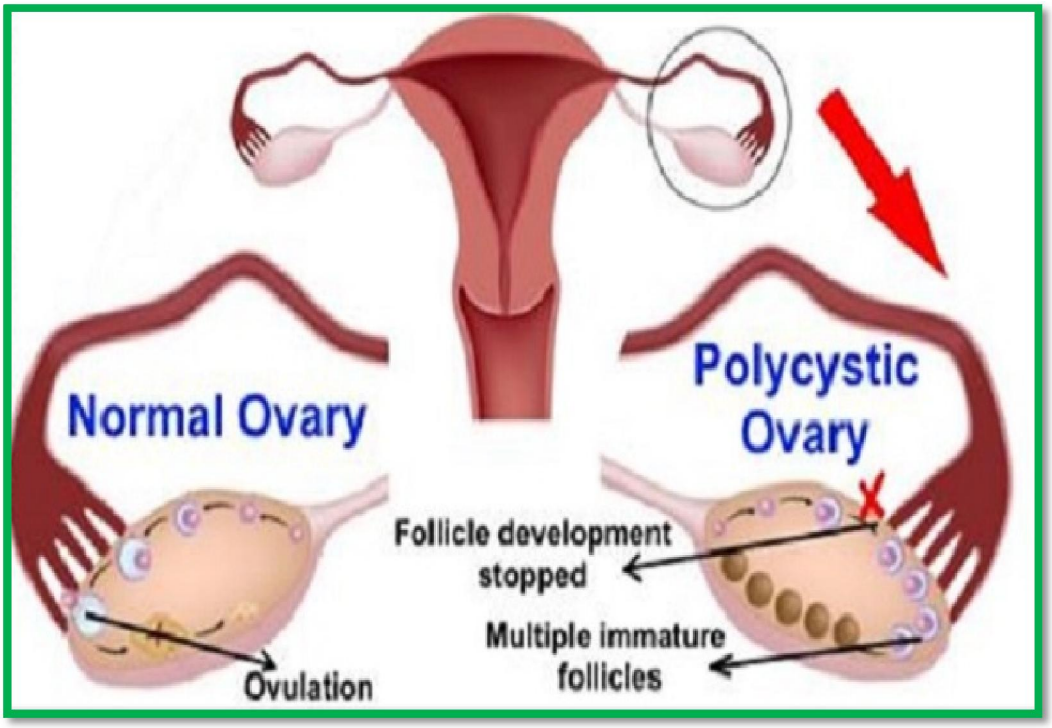


Figure 14: Multiple antral follicles

Anti mullerian Hormone is produced by growing antral follicle. This hormone is widely used invitro fertilization to know the Antal

follicale count. AMH helps for the growing follicle. AMH has high specificity in diagnosing PCOS. AMH is elevated PCOS. It is used in research purpose of PCOS not yet established for diagnosis.

We can investigate follicle reserve in PCOS. Since Antral follicle count is increased in PCOS AMH is measured. AMH could be most reliable marker of PCOS. AMH is used to research purposes but not established for lab diagnosis.

Laboratory test to exclude other causes of amenorrhoea and hyperandrogenism

Test	**To exclude**	**When to perform**
FT4	Thyroid Diseases	All cases
Thyroid stimulating hormone	Pregnancy	If clinical suspiciousun unplained low
LH/FSH, TSH	Hyperprolactinemia	All cases
Human Chorionic	Congenital Adrenal Hyperplasia	If testo sterone > 4nmol/l
Prolactin	Cushing syndrome	

PCOS occurs usually in reproductive age group. 10% of reproductive age group is affected by PCOS. Anovulation is usually caused PCOS. PCOS is diagnosed by menstrual disturbances with Anovulation and laboratory signs of hyperandrogenism. Other causes of menstrual disturbances is hyperprolactinemia. It incidence could be high as 17% in PCOS. In hyperprolactinemia also there is elevated androgen levels.

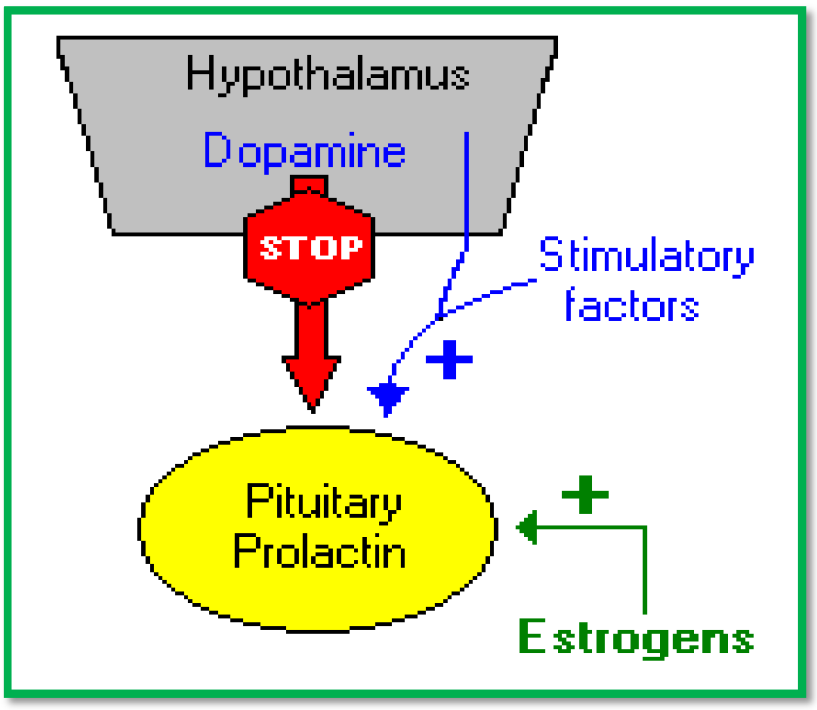


Figure 15 : Prolactin secretion

In 33%of PCOS has elevated prolactin. It may be due to lactotrope stimulation by chronic oestrogen exposure. Prolactin is anterior pituitary hormone. It is under chronic suppression due to dopamine produced in hypo thalamus reaches anterior pituitary via pituitary portal circulation. When the suppression is inhibited secretion increases. Prolactin has circardian rhythm. It alters throughout the cycle. So it should be measured at least twice to make the diagnosis. Hyperprolactinemia is caused by pregnanc,y lactation, stress, eating and thyroid disorder. So the cause of hyperprolactinemia is considered before making diagnosis of PCOs . Most common cause of hyperprolactinemiea is pituitary adenoma. Elevated levels of prolactin decreases gronadrotropin secretion which causes anovulation and alteration of menstrual cycle.

Estrogen causes high levels of prolactin which is invariably present in PCOS. Thyroid diseases also cause irregular cycles.

TSH Levels in PCOs

Thyroid dysfunction is present in PCOS. PCOS symptoms are aggravated by hypothyroidism. SHBG levels decreased in

hypothyroidism. This causes increase in free testosterone which causes hyperandrogenism. Gonadotropin levels are decreased in hypothyroidism which alters oestrogen and progesterone levels leads to infertility.

A study conducted on 2009 of 337 women with PCOs .All women were assessed with key markers of PCOs .The study said that women with PCOs has lowest levels of insulin resistance also had lowest TSH values.Womens with highest TSH Levels tend to have most severe insulin resistance. Intrestingly it was not related to weight.Sub cilinical hypothyroidism causes insulin resistance in female with all weight categories.The study concluded that TSH Level above 2 mIu/l was associated with insulin resistance in PCOs.

	PCOs	**Low Thyriod**
Difficulty in losing weight	Yes	Yes
Hair thinning	Yes	Yes
Depression	Yes	Yes
Difficulty in getting pregnant	Yes	Yes
Irregular menstrual cycles	Yes	Yes
Masculinisation	Yes	No
Cold Temperature	No	Yes
Blood Sugar Level	Yes	No

Table showing difference between hypothyroid and PCOD

Another study on women with PCOs found association between TSH, insulin resistance and androgen parameters. They reported that those who had TSH greater than 2.5 mIu/l had higher BMI ,higher fasting insulin levels and higher total testosterone.

Ovarian volume in Hypothyrodism

PCO like picture is induced by hypothyroidism .In hypothyroidism causes the deposition of mucopolysaccharides within various organs. Materials are also deposited in ovary. This hampers ovarian function and hormone synthesis resulting in disrupted menstrual cycles.

The study was conducted in 2011,Effect of hormone replacement therapy on ovarian volumes and androgen hormones with untreated primary hypothyroidism. The study discovered that providing thyroid replacement therapy reduces the size of ovaries and improves TSH, Prolactin, estradiol and testosterone.

ULTRASOUND IN PCOS

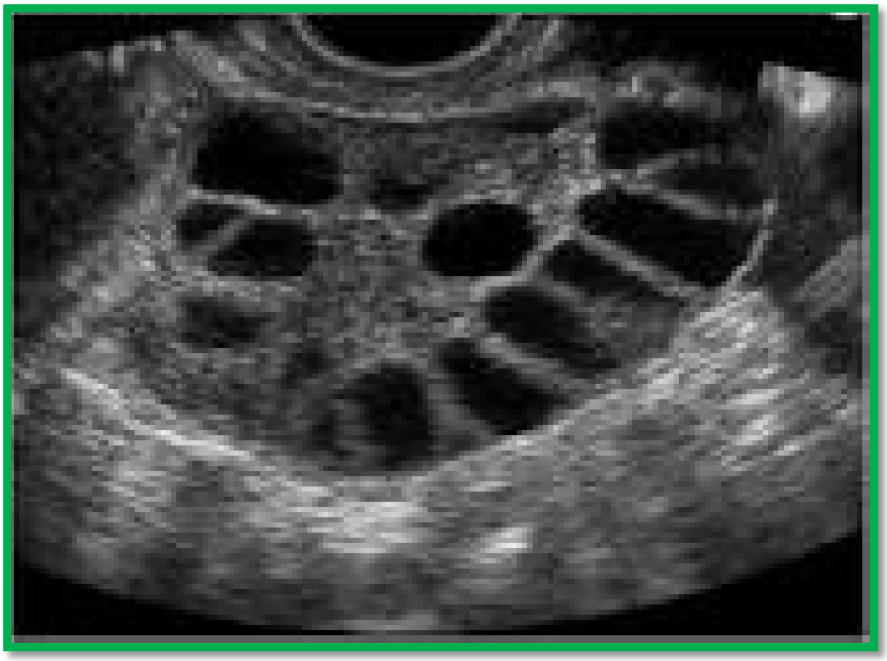


Figure 16: USG image of PCOs

For Diagnosis PCOs both TVS and TAS could be used. There are many USG criteria for diagnosing PCOD. The universally accepted criteria (i) increase size of volume due increase is number of follicles (2) increasing stoma volume. First real time USG was used by swanson et al 3.5 MHZ to describe of PCOS. Before this it was believed tiny cyst or follicles could be seen in USG. Both follicles number and increasing stromal volume was not recorded.

These come to end after high resolution USG. USG did not make diagnosis of PCOD. But gives a detailed appearance of Ovary in PCOD. Adams et al described TAS to diagnosis PCOD. He said atleast 10 follicles in one plane, around a dense core of ovaries stoma. Adam criteria was used thereafter for diagnosis PCOD.

Transvaginal USG

TVS is very useful in diagnosis of PCOS . TVS has greater and better resolution than TAS. TVS is better resolution and avoids full bladder. Ovaries could be visualized better and structure of ovary could be studied properly with TVS. In obese patient TVS is better choice. TVS is better because of (i) high resolution (2) less examination depth (3) Ovaries are close to vagina and uterus (4) presence of less fatty tissue.

USG Criteria of PCOS

1. For diagnosis PCOS there should be 12 or more follicles size about 2- 9 mm in diameter and increase ovarian volume. If

the cycle has dominant follicle or corpus luteal cyst the USG should be postpond to next cycle.

2. Measurement of Ovarian volume place key role in PCOD.

3. Presence of one criteria is sufficient to diagnosis of PCOD.

4. This is not true if women taking OC pills because size of ovary decreases.

5. If ultrasound pictures show PCOS but in absence of ovulation orhyperandrogenism diagnosis of PCOs should not be consider ed until more is known about this.

6. USG helps to measure ET

Technical Recommendation

1. USG is operated by trained person.

2. TVS is preferred than TAS especially in obese patients

3. If patients have regular cycles should be followed up in early follicular phase.

4. If patients is Amenorrohea give her withdrawal bleeding and then do USG.

5. Presence of dominant follicle or corpus luteal cyst postpond the USG in next cycle.

6. Ovarian volume must be estimated

7. Follicle number should be evaluated a longitudinal, transverse and AP cross sections.

Examination of Polycystic Ovary

It is very crucial to identify two ovaries separately. Maximum diameter is measured in all three planes. Calculation of ovarian volume in difficult because of irregular shape of ovary. Because of overlying sigmoid colon in side of left ovary,it is difficult to calculate ovarian volume.

Internal Features of PCOS

- Follicle size and Number
- Technical aspects

while describing polycystic Ovary, a oocyte containing Follicle was observed . In earlier it is believed that cyst are measured not follicle.

- Ovaries are measured in all views and number of follicles are also measured.

- TVS is used to measure size and number of follicles. The follicle are measured in all 3 diameters. PCOS patient have follicles of between 4 and 10 mm size.

Stroma volume and Echogenicity

Increase in stromal volume is not a good indicator of Pcod. TVS helps to identify stromal thickness. Stormal thickness difficult to measure and have subjective variation. Measuring ET is easy to

measure. Measuring stromal thickness along with follicle size and number is useful to diagnosis PCOS.

TAS, TVS, or both

Trams abdominal scan in used in virgin and those who did not prefer TVS.TVS help us to visualize ovaries better than TAS. TAS requires full bladder to visualize ovaries .Overfilled bladder gives false positive results due to compressing ovaries and falsely increasing length . Ovarian size is measured after partial micturition . Ovaries are usually found in near the ureters and iliac vessels.

TVS is best used to diagnosed PCOS. PCOS is diagnosed in 30% of women with TAS and 70% of women in TVS.

Management

1. Management of presenting symptoms
2. Life style modification
3. Weight reduction
4. Protect fertility

5. Prevent from endometrial hyperplasia
6. Prevent from Cardiovascular problems
7. Frequent screening for Diabetes Mellitus
8. Contraception
9. Treatment of Complications
10. Cyclical progesterone therapy
11. Fertility treatments
12. Antiandrogen therapy
13. .Insulin senstising therapy

Follow –up

1. Regular health checkup
2. Weight, BP, FBS, Lipids, (Breast examination) cervical smear, mammogram etc.
3. Contraception review
4. Follow up evey 3-6 months of Antiandrogen therapy.
5. Look for endometrial hyperplasia in Anovulatory dysfunctional uterine bleeding.

Gulekli Betal study found that hirsutism was higher in adult group by 15%. Menstrual irregularity was higher in adult group presenting with oligomenorrhoea. Body mass Index was higher in adult group. He found that there was positive correlation between ovarian volume, serum LH & total testosterone.

Robinsonsetal et al proved LH concentration and LH / FSH ratio was higher in patients with PCOS similarly testosterone was also higher in patients with PCOS.

Balen AH al studied about ultrasound features of PCOD. When ultrasound features suggest PCOD there is elevated LH concentration.

Dewailly Detal proved that there was no relation between LH and GNRH in cases with PCOD.

Clayton RN et al said the ultrasound features are present in 90% of PCOS. Same picture occurs in normal women about 20%. So only USG could not be taken for diagnosis of PCOD.

MATERIALS METHODS

Source of data

This study was carried from December 2015 to December 2016 on patient attending gynaecology outpatient department at Stanley Medical College and Hospital.

Methodology

1. 50 patients were studied in between ages of 18-35 years attending gynecology op with PCOD in ultrasound and clinical features.

2. Written informed consent was obtained.

3. History and clinical examination was done.

4. Performa was filled.

5. Patient who are excluded from study are those were on treatment for infertility and hormonal therapy.

6. History was taken in detailed manner regarding oligomenorrhoea, amenorrhoea and infertility.

7. Clinical finding was recorded including hirsutism, acne, alopecia, acanthosis nigricans and obesity.

8. Height and weight measured in OP, with help of it BMI was calculated.

9. Blood test were done in earlyfollicular phase on day 3 for serum LH, FSH, total testosterone, AMH, HOMA IR Index. It is done electro hemilluminiscence immune assay in Stanley Medical College.

Trans vaginal scan was done. If difficult arises, ovary is traced in relation to iliac vessels . Following parameters were located.

1. Patients with PCOD have atleast 12 or more follicles. Follicles should measure 2-9 mm in diameter.

2. Ovarian volume is measured with prolate ellipsoid method = 0.5 x length x breath x thickness > 10 cms^3

Hisutism was examined and graded by modified ferriman – gallwey score. Patient with score > 8 was hirsutism.

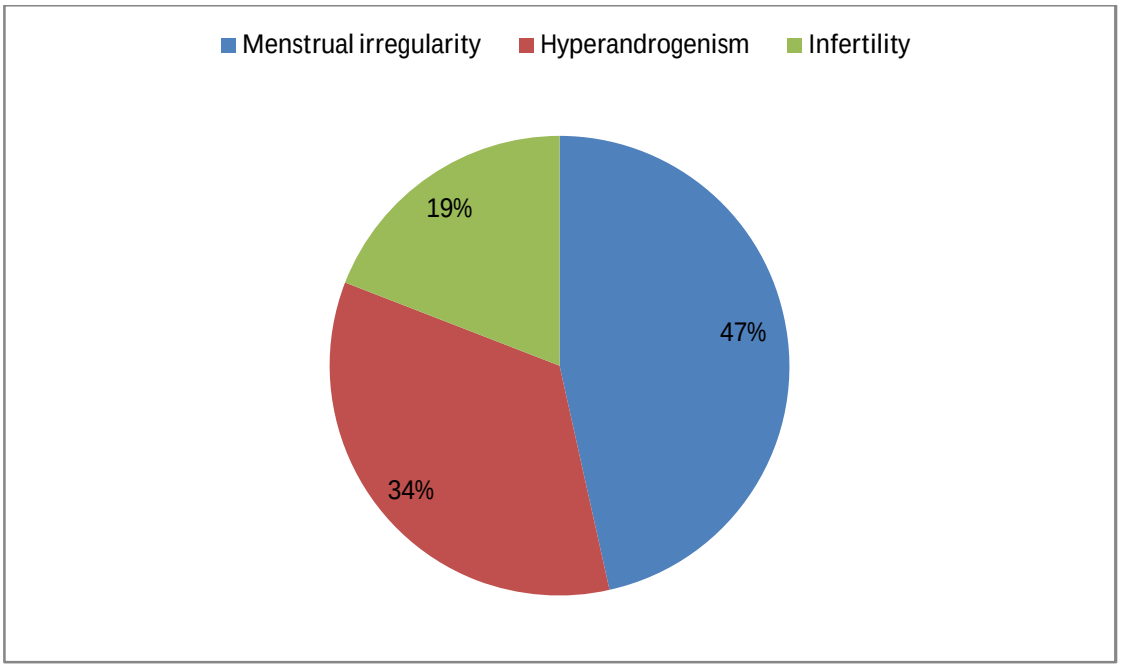
Statistical Analysis

The data is entered and calculated using SPSS version and software. The statistical significance will be tested. Mean and standard deviation were expressed using continuous variables as BMI, LH, FSH, LH/FSH, AMH, Home IR Index, sum LH, / FSH, menstrual irregularity, hirsutism , acne, , Ovarian volume, pearson correlation and coefficient calculated.

Relationship between LH level,LH/FSH ratio, serum testosterone, Oligomenorrhea / Amenorrhea, hirsutism, acne using Bi-serial correlation coefficient.

RESULTS

Pie chart showing Distribution of 50 cases of Present Study with symptoms

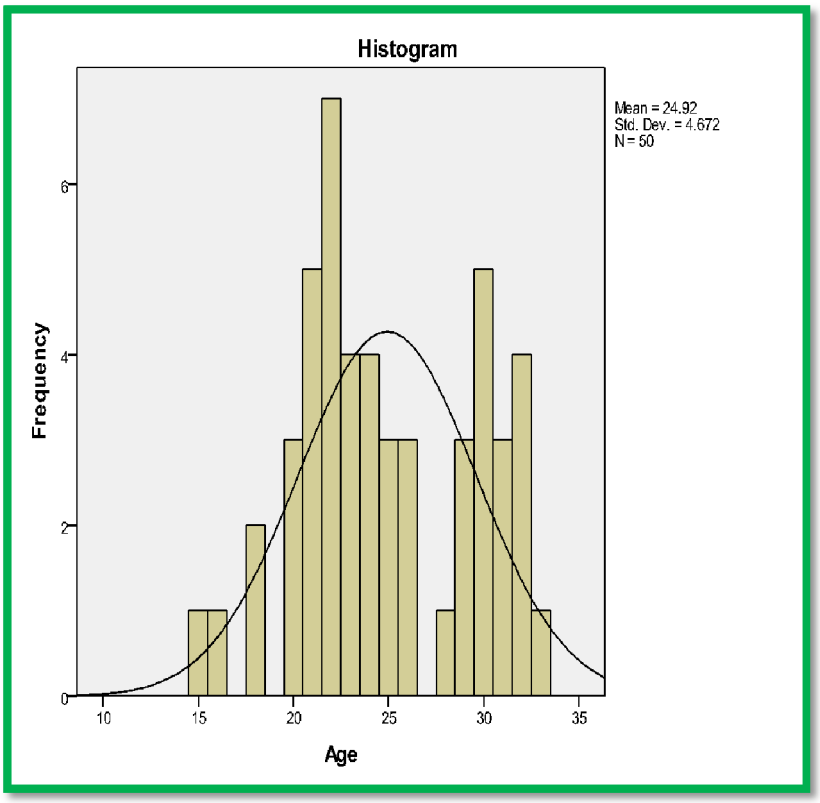


Graph 1: Pie chart showing Distribution of 50 cases of Present Study with symptoms

There are only 3 phenotypes of PCOS were seen in present study. Our study included patients with USG proven PCOs, the 4th phenotype could not be seen. Menstrual irregularity ,hyperandrogenism, plus USG

proven PCOS is seen in 36 cases. Hyperandrogenism + PCOs is seen in 15 cases and menstrual irregularity + PCOS in 27 cases.

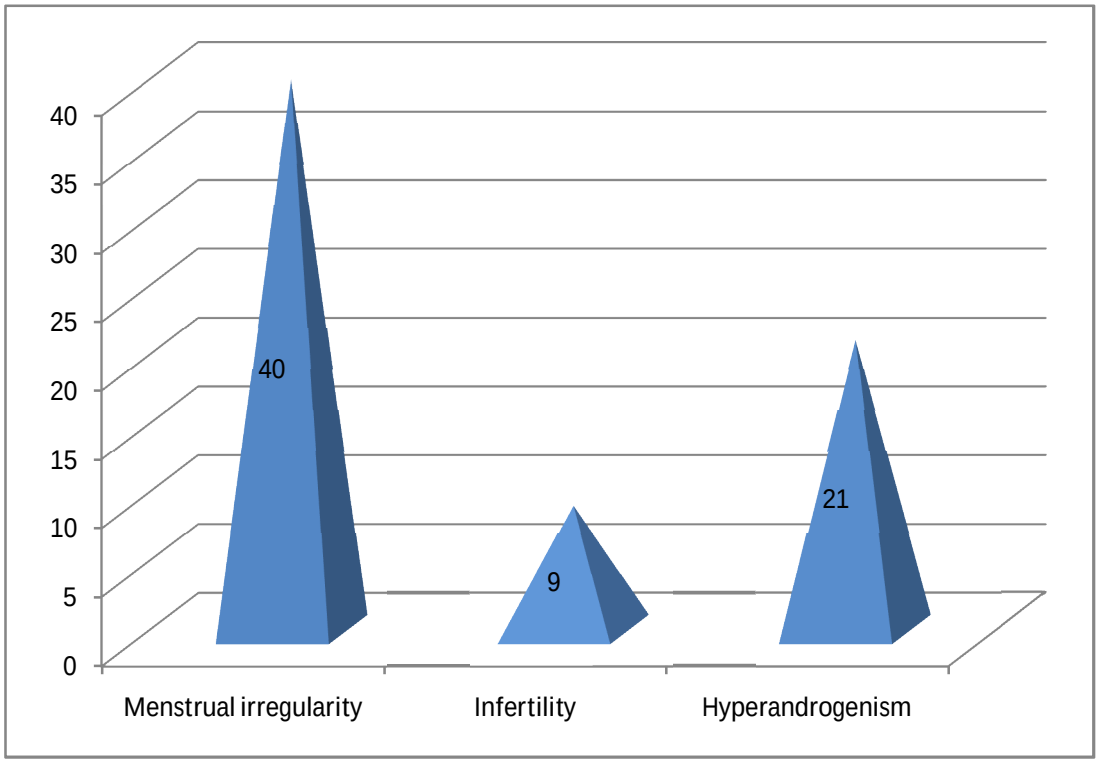
Age Distribution



Graph 2 : Age distribution in patients with PCOD

By age distribution graph the present study have age group maximum between 20-30 years (40 cases). Small group of patients was less than < 20 years > 30 years. The youngest age of patient with PCOs was 18 and oldest was 35.

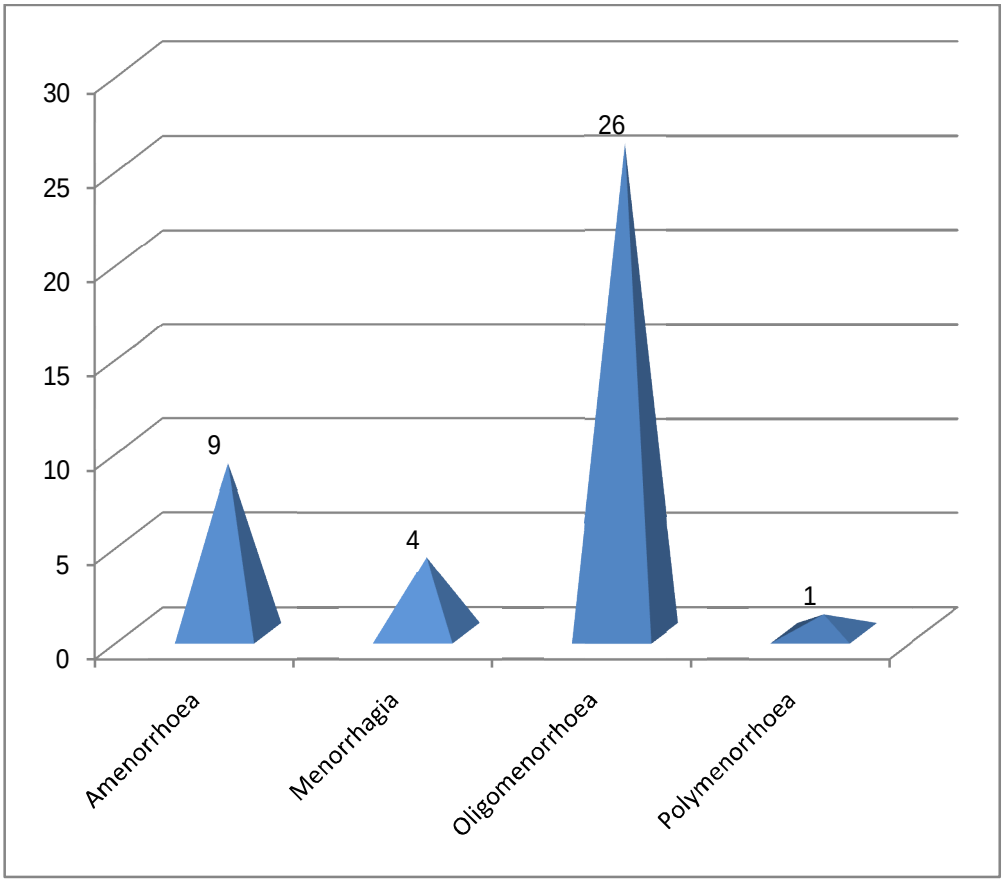
Distribution of Cases according to symptoms



Graph 3: Distribution of Cases according to symptoms

50 Patients were studied, out of this menstrual irregularity- 40 cases, Hyper androgenism- 21 cases, Infertility- 9 cases.

Menstrual Irregularities



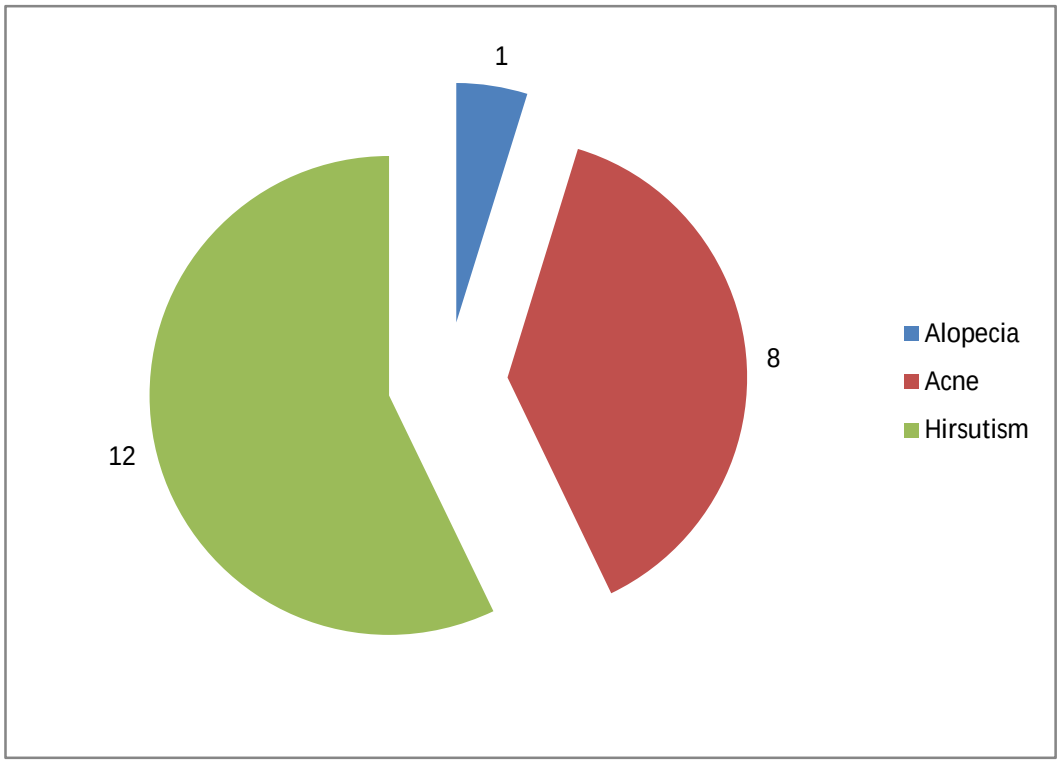
Graph 4 : Menstrual Irregularities

Menstrual Irregularity

The present study was done in 50 patients. The most common symptom is oligomenorrhoea in 26 cases, the least common symptom is polymenorrhoea.

Distribution of Hyperandrogenism

In this study, 21 cases have both clinical and biochemical features of hyperandrogenism.

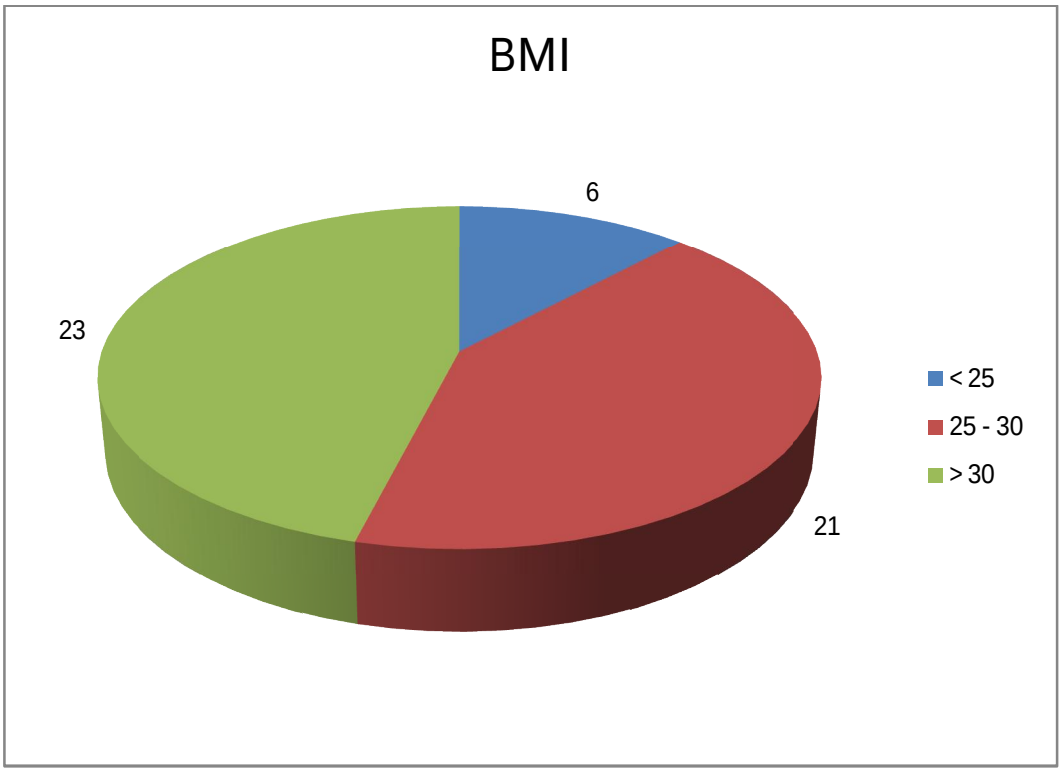


Graph 5 : Distribution of Hyperandrogenism

Symptoms of Hyperandrogenism

Hirsuitism is most common presentation of PCOS. Acne is present in small number. Androgenic alopecia only in one case.

Body Mass Index



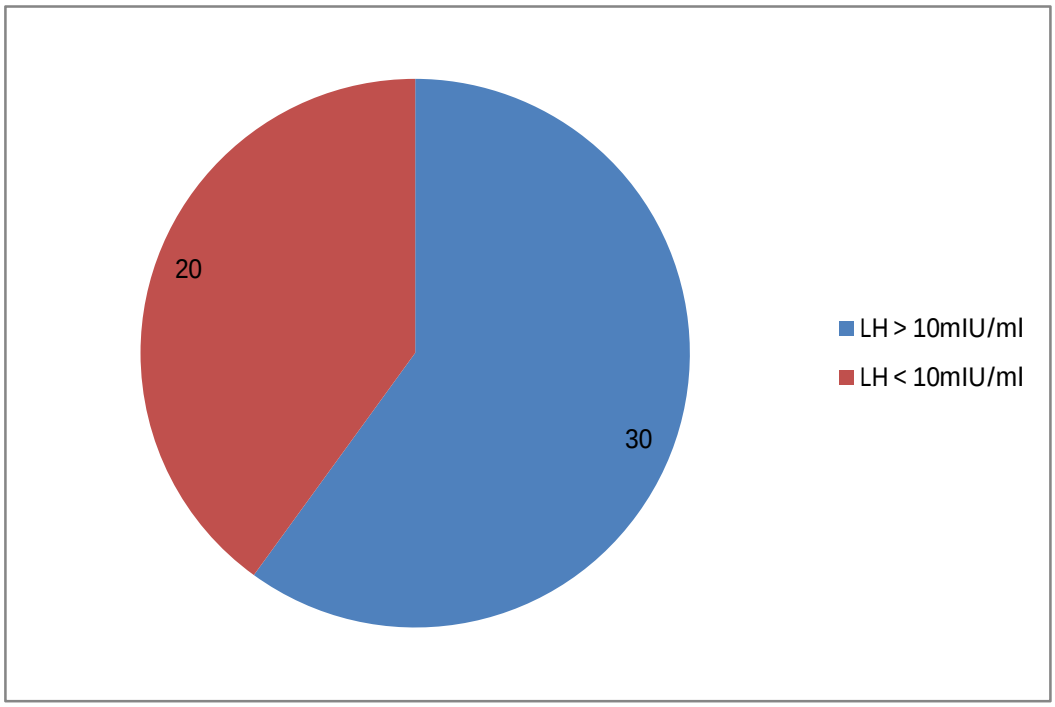
Graph 6 : Body Mass Index

The following study showed over weight was the major cause for PCOD. Next comes obesity.

Less than 25 body mass index in 6 cases, 25 to 30 in 21 cases, greater than 30 in 23 cases.

Serum LH in mIU/ml

Serum LH in mIU/ml



Graph 7 : Serum LH in mIU/ml

In the study, many patients have high LH values. 30 cases have LH > 10mIU/ml and 20 cases <10mIU/ml. There was positive correlation between LH, FSH and FT3 and negative correlation between LH/FSH and TSH.

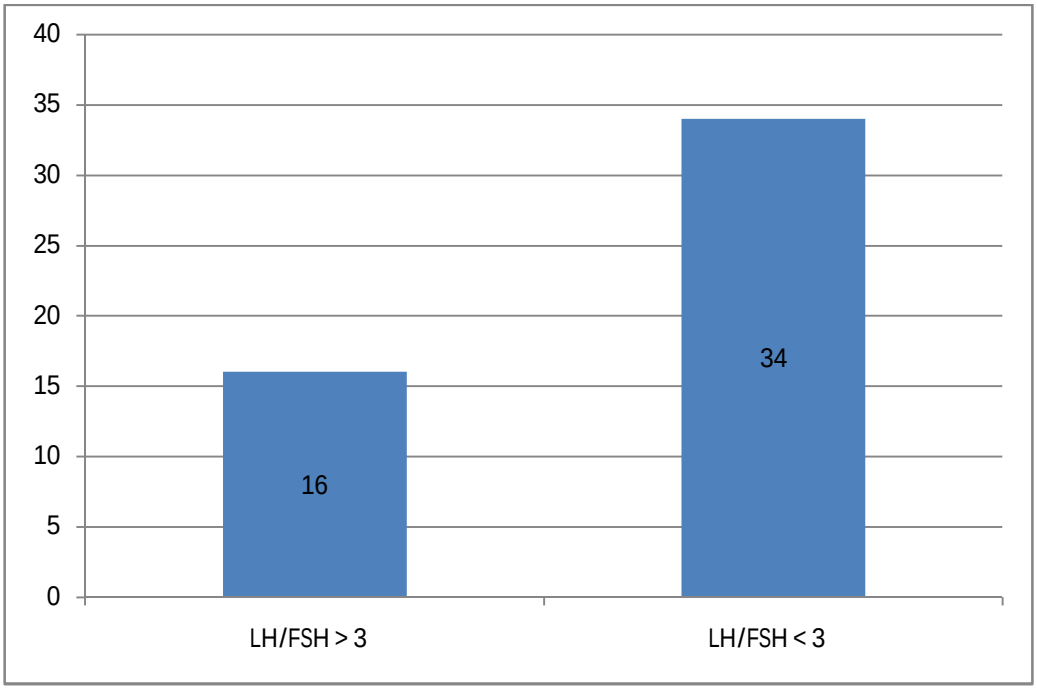
LH/FSH

Pearson correlation			
LH/FSH & FT3	Value= .303*	P Value = .033	Highly significant
LH/FSH & TSH	Value = -.344*	P value = .015	Highly significant

Positive Correlation between LH/FSH & FT3

Negative Correlation between LH/FSH and TSH

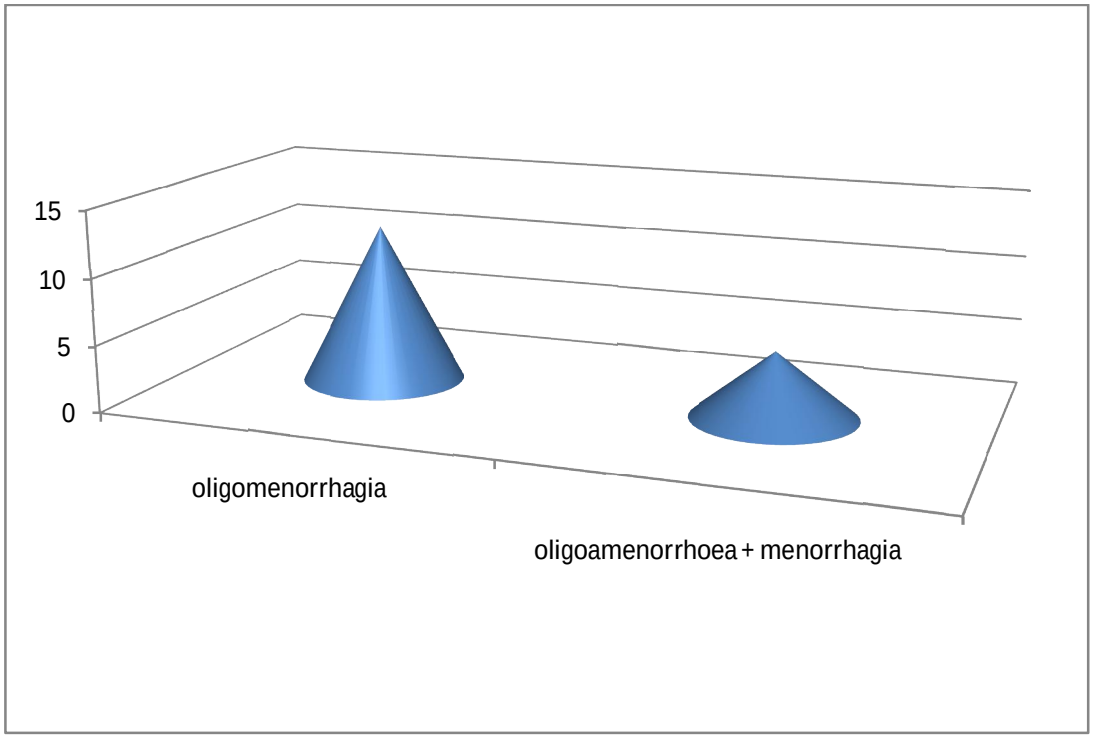
LH/FSH ratio



Graph 8 : Serum LH in mIU/ml

- LH/FSH > 3 in 16 cases
- LH/FSH <3 in 34 cases

Menstrual disturbances in Hypothyroidism



Graph 9 : Menstrual disturbances in Hypothyroidism

Thyroid profile is checked for all cases of PCOS. Those patients with hypothyroidism have major symptoms of oligomenorrhoea . 17 patients with PCOS have hypothyroidism in our study. They presented with Oligomenorrhoea.

Prolactin levels

Serum prolactin was checked for all cases. Patients with hyperprolactinemia had Oligomenorrohea.

Serum PROLACTIN

		PEARSON CORRELATION		
Serum prolactin & TSH	Pearson correlation	Value = -.282*	P value = .047	Highly Significant

There is Negative Correlation between Serum Prolactin and TSH.

DISCUSSION

The study is carried out in 50 subject with polycystic ovaries in USG. As per Rotterdam consensus study, clinical examination were used.

According to Rotterdam criteria 2003 PCOS is classified in 4 types.

1. Phenotype A : PCOs + menstrual irregularity + Hyperandrogensim

2. PhenotypeB : Hyperandrogenism + menstrual irregularity

3. Phenotype C : PCOs + Hyperandrogenism

4. Phenotype D : PCOs + menstrual irregularity

My study in stanley medical College showed only 3 phenotypes . The commonest phenotype in our study is phenotype A.

Dewailly detal said that phenotype D is the mildest form of PCOS with very little insulin resistance. These patients do not have long term risk as other phenotypes.

Symptoms of PCOS

In our study there 3 main symptoms are menstrual irregularity hyperandrogenism and infertility.

Symptoms of PCOs	No of cases
Menstrual irregularities	40
Hyperandrogenism	21
Infertility	9

History and Clinical Exam

In our study, amenorrhea was present in 10 cases, menorrhagia in 4 cases oligomenorrhoea in 23 cases . In the study conducted by ballenetal of about 1741 cases, 66 patients had menstrual irregularities. Only small number of cases have regular cycles. In study by franks et al

found oligomenorrhoea in 52% cases. Gold ziehertal said that 90% of cases were menstrual irregularity among them 30 to 40% had amenorrhoea. Most women with PCOS have abnormal menstrual cycle .The most common is amenorrhoea anovulation.

	Balen et al n = 1741 (%)	**Franks et al n = 300 (%)**	**Goldzieher et al n=1079 (%)**	**Present Study n = 50**
Oligomenorrhoea	47	52	29	26
Amenorrhoea	19	28	51	10
Polymenorrhoea	12	20	33	1
Menorrhagia	10	16	22	4

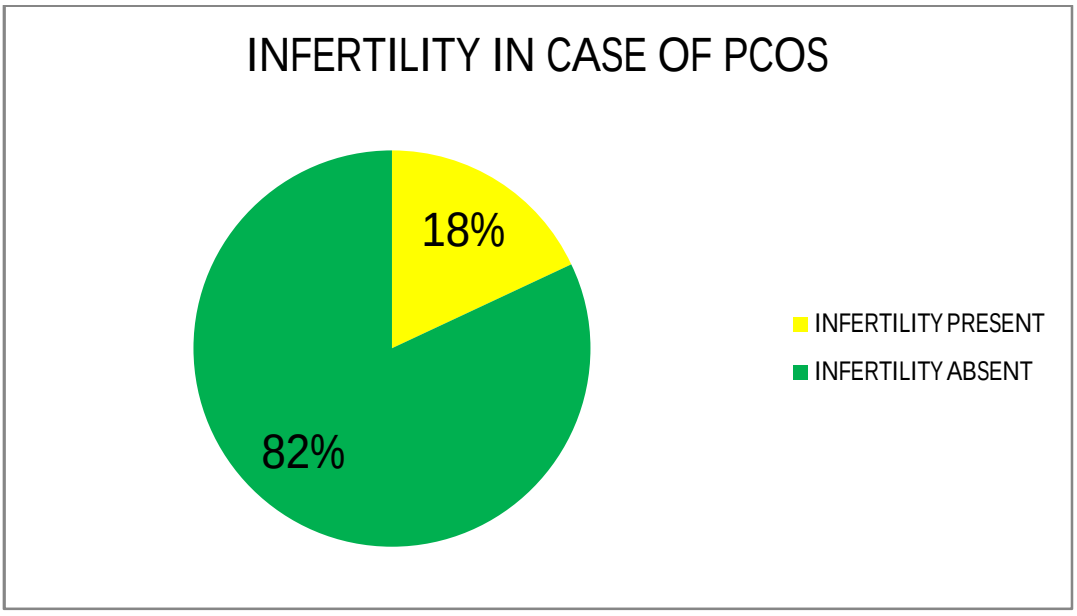
Goldzieher et al found there was Amenorrhoea more than other menstrual irregularities. Aarti Sharma said that the menstrual irregularity begins in puberty in the form Oligomenorrehoea, Amenorrhoea or polymenorrhoea. Some patients with PCOS have normal menstrual cycles. Studies proved that Oligomenorrehoea is present in 29-47 of female with PCOS , Amenorrhoea in 19.5% of cases.

Turhan No et al conducted study about 125 patients with ultrasound diagnosis of PCOS. 55.2% had menstrual disturbances, 23.2% had normal irregular cycles and 50.4% had infertility.

Gulekli B et al study found Oligomenorrehoea, in 42.8%, Amenorrhoea in 20% and normal cycles in 17.4% of cases.

INFERTILITY

Infertility



Graph 10 : Infertility & Alopecia

Correlation		Value	Approx. Sig.	
Infertility & alopecia	Phi Correlation	.305	.031	Highly Significant

Positive Correlation between Infertility & Alopecia

infertility and Hirsutism

Correlation		Value	Approx. Sig.	
infertility * hirsutism	Phi Correlation	.316	.026	Highly significant

Positive Correlation Between Infertility and Hirsuitism

Balenaetal had studied infertility about 20% of cases. Franks et al said that 42% o case had infertility. In the study by Aarthisharma because of Anovulation 20% of all couples with infertility.

Hyperandrogenism in PCOs

	Balen et al n = 1741 (5)	**Franks et al n = 300 (%)**	**Goldzieher et al n = 107 9 (%)**	**Present Study N = 50**
Hirsutism	66.2	64	69	12
Alopecia	6	3	-	1
Acne	35	27	-	8
Acnathosis	3	1	-	Nil

Balenetal study showed hirsutism in 66.2% , franks et al 64% , Goldzieher et al 69% and in our study12%. Small percentage of hirsutism in our study is due to Ferriman gallwey score of > 8 was taken for hirsutism. In all other studies above mention took FG score of > 6 for hirsutism.

The signs of hirsutism in PCOS beings in Menarche and progress gradually and rarely accompanied by other signs of virilisation. The most important clinical indicator of Androgen excess is hirsutism . The only presence of acne and androgenic alopecia could also be taken for Androgen excess. But they have not proved. These are poor markers of androgen excess.

Acne

In our study acne as found in 8% of cases. **Balen et al** had acne in 35% and 27% of cases, by study of franks et al. **Timpatanapong p et al** found in 51 patients with acne, 19 of them had PCOS.

Androgen alopecia

In our study Androgen alopecia is seen one case. Balen et al said 6% of PCOS .patients has androgen alopecia Ester Cela etal arrived a conclusion that clinical hyperandrogenism presented by hirsutism, acne or alopecia . Futterweit et al studied 109 women with alopecia and said 28% of women have PCOD.

Acanthosis Nigricans

In this study not even a single patient have Acanthosis Nigricans . Study by Balen et al had 3% of cases.

BMI in PCOS

BMI	**N = 50**
< 25	6
25- 30	21
> 30	23

In our study most of the cases were BMI > 30 whose considered Obese, so obesity is a major cause of PCOD. In Balen et al conducted a study and found 38% were obese . Series by franks et al and Goldzieher et al obesity is found 35% & 41% respectively. Aarti Sharma said obesity was seen in 10- 65% of women.

PCOS patient have body habitus which is typical. They are usually over weight with hirsutism. Some patient do not fit within the body habitus. They were called lean PCOS.

It was found that half the women PCOS have normal weight or lean. Insulin resistance is major role in PCOS. Gynaecologist disagree insulin resistance in lean PCOS. Lean of women also have dearrangement of insulin level and glucose metabolism. They did not express same degree of insulin resistance Vrlikova published a article on 2004 about Insulin sensitivity in women PCOS. He said that ovaries are sensitive to insulin in women with PCOS leading to hyperandrogenism.

Kravariti reported in September 2005 that lean women will also have same risk of Cardiovascular Symptoms as overweight women. He said that all of women with PCOS should be screened for CVS complication regardless of BMI.

Harmonal Evaluation of PCOS

Study	Balen et al n = 1741 (%)	Franks et al n = 300 (%)	Rajkhowa	Pankar et al	Present study n = 50
Elevated serum LH	40	51	33.6	-	28
Hypothyroidism	26	12	30	-	18
Hyper prolactinemia	10	8	8	-	8
Hypo prolactinemia	19	4	8	-	2

In our study levated serum LH level is seen in 28 cases, Hypothyroidism 18 cases, Hyper prolactinemia in 8. In the revised 2003 criteria there is elevated LH levels in PCOS in relation to FSH.

This is caused by increase amplitude of LH. PCOS women have elevated LH levels in 60% and LH / FSH ratio in 95% of women. Final said measurement of LH is not primary tool for diagnosis PCOS. But is should be used as second parameter for PCOS.

Prolactin

Several diagnostic criteria was used to diagnose PCOS. Among this is hyperprolactinemia is less common cause of menstrual irregularity. In hyperprolactinemia patients have elevated androgen levels. So Prolactin estimation should be done.

Prolactin is produced by anterior pituitary under the constant suppression of dopamine. Dopamine is produced in hypothalamus . It reaches anterior pituitary by pitutary portal circulation and decreases prolacltin release. When this cycles is altered hyperprolactin results. Prolactin values changes thorough out the menstrual cycles. So it is measured atleast twice. Prolactin is elevated by nipple stimulation, stress, eating and thyroid abnormalities. Pitutary adenoma cause hyperprolactinemia. GrRH is suppressed by prolactin which casus various menstrual disturbances.

Oestrogen causes increase prolactin levels in women with PCOS. They have high estrogen levels which leads to increase in a prolactin levels. This result tell us PCOS with hyperprolactinemia, other causes should be ruled out.

A study was conducted in Puduchery found out that a age advances there is elevated levels of TSH in PCOS patients. So PCOD patients must be evaluated for thyroid profile.

Ultrasonography

There was no significant correlation between hormonal estimation and ovarian volume.

Sikka P et al conducted study in 100 anovulatory infertile women with PCOD. Every women had amenorrhea, 70% had hirsutism, half of them were obese. He did TVS which showed increase ovarian volume. He concluded there was positive correlation between ovarian size, LH /FSH and hyperinsulinemia.

CONCLUSION

This study was conducted in Stanley Medical College at North Chennai in 50 cases with polycystic ovaries and concluded that.

1. PCOS in most common gynecological disorder of age 20 -30 years.
2. Phenotype A is the most common presenting type of PCOS.
3. Menstrual irregularity is the presenting symptom of 41 cases PCOS.
4. Among the menstrual irregularity oligomenorrhoea is present in 26 cases.
5. There are many features of hyperandrogenism among this Hirsutism is present 12 cases.
6. There was no correlation between serum prolactin and PCOs
7. TVS is the most the important diagnostic tool.
8. Serum LH/FSH ratio was secondary tool for diagnosing PCOs is 28 cases.
9. Obesity and overweight is a most important modifiable risk factor which causes 70% of cases.
10. The one of the important step is weight reduction.

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PERFORMA

Name :

Age :

Address :

OPD No. :

Chief Complaints :

- Amenorrhea / Olgomenorrhoea / menorrhagia / polymenorrhoe
- Hirsutism
- Infertility (Primary / Secondary)
- Acne
- Alopecia
- Weight gain

Any other complaints

Menstrual History :

Obstetric History :

Past History :

Treatment History :

Family History :

General Examination :

Height : Weight :

BMI :

Breast : Thyroid :

Acne :

Alopecia :

Acanthosis nigricans :

Local Examination :

Per Abdomen :

Per Speculum :

Per Vaginal :

Investigations

Serum LH Levels : Miu / ml

Serum FSH Levels : mIU / ml

Serum Prolaction Levels : ne / Dl

Thyroid Function Test : FTS: FT4: TSH:

Ultra Sound :

Right Left

- Ovarian Volume in cm3
- No of Follicles & Size
- Stroma

CONSENT FORM

I agree to participate in the study entitled **"Correlation of Clinical and Biochemical Parameters with Ultrasound Features in 100 Women with Polycystics Ovaries".**

I confirm that I have been told about this study in my mother tongue and have had the opportunity to clarify my doubts.

I understand that my participation is voluntary and I may refuse to participate at any time without giving any reasons and without affecting my benefits.

I agree not to restrict the use of any data or results that arise from this study.

Name of the participant:

Sign / Thumb print:

Name of the Investigator : Dr. Indirani.D

Sign of Investigator

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C¨£iUS

B´ÁõÍ¶ß øPö¯õ¨£® **÷{õ¯õÎ°ß øPö¯õ¨£®**

	Name	Age	Op.No.	Ameno	Meno	Oligo	Poly	Infertility	Acne	Alop	Hirsutism	BMI	USG PCO	OV (cm3)	LH	FSH	LH/FSH	FT3	FT4	TSH	S.PrI.
1	Pavithra	26	9874	No	No	Yes	No	No	No	No	Yes	28	Yes	12.46	7.6	4.4	1.7	4.2	0.2	5	9
2	Veena	21	7883	No	No	No	No	Yes	No	No	Yes	29	Yes	12.4	25	4.1	6.09	7.4	1.3	3	16
3	Reka	23	1971	No	No	No	No	No	No	No	No	30.4	Yes	12.3	28	1.6	15.5	5.6	0.66	0.6	13
4	Swapna	22	7886	No	No	No	No	No	No	No	Yes	26	Yes	14.2	13.8	2	6.9	6.4	1.33	0.6	49
5	Munisha	24	9447	No	No	No	No	No	Yes	No	No	28	Yes	14	6.7	11	0.6	2.3	1.7	6	13.6
6	Lakshmi	26	3478	No	No	Yes	No	No	No	No	No	24.01	Yes	10.2	11.1	2.3	4.82	4.6	2.33	5	0.3
7	Ganga	22	619	No	No	No	No	Yes	No	No	Yes	26.3	Yes	19.3	6.3	5	1.2	5.9	2.3	0.6	21
8	Varalakshmi	21	456	No	No	Yes	No	No	No	No	No	27	Yes	18.2	7.8	4.6	1.69	1.6	2.3	0.1	21.3
9	Saraswathi	23	7845	No	No	No	No	No	No	No	No	19.6	Yes	12.1	12.12	6.3	1.9	4.1	2.4	4.1	15
10	Rajeshwary	22	459	No	No	Yes	No	No	Yes	No	No	22.6	Yes	10.5	12	8	1.5	4.3	2.6	4.3	12.6
11	Roja	22	489	No	No	No	No	No	No	No	No	25.3	Yes	10.7	4.1	2.2	1.8	4.6	2.5	4.2	23.6
12	Yoga	21	4512	Yes	No	Yes	No	No	No	No	No	24.9	Yes	15.2	14.8	4.8	3.08	3.3	4.6	5	45.6
13	Baby	18	7840	No	No	Yes	No	No	No	No	Yes	30.2	Yes	20.6	12.4	1.2	10.3	6.2	1.3	1	30.2
14	Rajee	30	4600	No	No	Yes	No	No	No	No	No	35.1	Yes	15.6	14.2	7.2	1.9	4.3	3.1	2.2	16
15	Sharmila	32	4661	Yes	No	No	No	No	Yes	No	No	27.2	Yes	13	3.2	1.5	2.3	3.1	0.9	3.9	1.6
16	Poonam	30	4666	No	No	Yes	No	No	Yes	No	No	27.2	Yes	13.3	3.6	1.7	2.1	3.2	0.8	0.1	19.6
17	Jaya	29	4587	No	Yes	Yes	No	No	No	No	No	27.3	Yes	13.2	3.2	1.5	2.1	3.2	0.9	3.9	1.64
18	Divya	29	6488	No	No	No	No	Yes	No	No	Yes	23.1	Yes	15.1	16	1.2	13.3	6.6	3.4	4.2	2.3
19	Savitha	32	2346	No	No	No	No	No	No	No	Yes	21.7	Yes	16	6.1	8.3	0.73	3.2	1.5	2.1	2.4
20	Sathya	29	6613	No	No	Yes	yes	No	Yes	No	No	40	Yes	16	4.1	1.2	3.4	6.9	4.3	9.3	21.3
21	Malini	25	1164	No	No	No	No	No	No	No	No	34	Yes	16.2	6.1	2.8	2.1	6.7	3.1	2.1	21
22	Hindu	22	9170	No	No	Yes	No	No	No	No	No	20.1	Yes	17.1	8.8	4.1	2.1	4.5	0.8	6.2	26
23	Keerthi	31	78	No	Yes	Yes	No	No	No	No	No	31	Yes	10.2	12.3	6.8	1.8	5.2	0.7	4	29
24	Uma	30	4318	Yes	No	No	No	No	No	No	No	28.3	Yes	10.6	7.4	3.2	2.3	5.03	0.6	2.2	24.3
25	Ramya	21	4510	Yes	No	Yes	No	No	Yes	No	Yes	32.2	Yes	13.2	25.5	4.1	6.08	6.04	0.9	2.1	24

26	Arivu	20	9693	No	No	Yes	No	No	No	No	No	21.8	Yes	14.3	6.2	8.6	0.72	3.4	1.4	2.1	24
27	Sulthana	22	2889	No	No	No	No	Yes	No	Yes	No	25.8	Yes	16	17.2	2.3	7.4	9.3	1.6	0.4	22
28	Akila	24	8771	No	No	No	No	Yes	No	No	Yes	31.2	Yes	12.8	21.2	9.3	2.2	3	0.7	3.2	6.4
29	Narmadha	30	8114	No	No	No	No	Yes	No	No	No	25.4	Yes	10.9	11.3	3.2	3.5	1.1	0.6	4.1	21
30	Kavitha	26	6613	No	No	No	No	No	No	No	Yes	25.6	Yes	11.6	13.5	4.2	3.21	1.3	1.33	6	26
31	Indhu	24	9170	No	No	Yes	No	No	No	No	No	28.4	Yes	12.6	6.8	3.1	2.19	6.2	4.2	4.5	21
32	Keerthana	31	78	No	No	No	No	No	No	No	No	28.2	Yes	10.5	17.2	2.2	7.8	9.3	1.2	0.4	23.9
33	Selvi	21	4318	No	No	Yes	No	No	No	No	No	27.2	Yes	12.6	11.1	1.2	9.2	6.31	2.2	0.005	28
34	Poonam	31	4600	Yes	No	No	No	No	Yes	No	No	20.3	Yes	12.8	14.2	1.6	8.8	4.62	2.1	0.4	23.1
35	Lakshmi	28	4587	No	No	Yes	No	No	No	No	No	25.2	Yes	28.8	15.3	4.2	3.6	2.1	2.2	0.6	28.6
36	Durga	30	6488	No	No	Yes	No	No	No	No	No	23	Yes	21	13.3	3.2	4.1	4.3	2.3	0.3	27.6
37	Shakila	25	4789	No	No	Yes	No	No	No	No	Yes	26	Yes	12.3	15.8	1.3	12.1	1.36	1.2	1.02	26.1
38	Pathima	32	6799	No	No	Yes	No	No	No	No	Yes	23	Yes	10.2	17.1	2.2	7.7	2.8	2	1.02	22.46
39	Lekha	23	6971	No	Yes	Yes	No	No	No	No	No	27	Yes	14.3	12.2	4.3	2.8	4.7	2.2	3.2	21.4
40	Radha	18	5879	Yes	No	no	No	No	No	No	No	20.2	Yes	11.6	10.4	2.8	3.7	6.5	2.3	1.3	21
41	Sowmiya	20	7713	Yes	No	No	No	Yes	No	No	No	19.4	Yes	13.2	12.2	6.4	1.9	4.1	2.2	4.1	14
42	Sofia	15	1779	No	No	No	No	No	Yes	No	No	22.6	Yes	12.1	12.06	8.5	1.4	4.31	2.6	4.2	12.2
43	Yamuna	32	8639	No	No	Yes	No	No	No	No	No	26.2	Yes	10.5	4.2	2.2	1.9	4.2	2.2	4.3	23.3
44	Pavithra	24	9874	No	No	No	No	Yes	No	No	No	19.5	Yes	10.78	4.4	2.2	1.9	6.2	0.3	4.2	20.6
45	Geetha	33	4555	No	No	Yes	No	No	No	No	No	30.2	Yes	13	5.4	1.6	3.3	4.61	0.6	5	17.2
46	Kalpana	16	1010	Yes	No	Yes	No	No	No	No	No	20.6	Yes	12.8	10.3	4.9	2.1	5.1	0.6	4.2	19.6
47	Vaishnavi	23	2000	Yes	No	Yes	No	No	No	No	No	24.1	Yes	19.3	11.1	2.3	4.8	4.6	2.3	5	0.3
48	Bhuvana	22	4970	Yes	Yes	No	No	yes	No	No	Yes	26.3	Yes	10.4	6.3	5	1.26	5.9	2.3	0.6	21.3
49	Nivethitha	20	1331	No	No	No	No	No	No	No	No	29	Yes	18.2	7.4	1.6	4.6	4.26	2.2	2	24.6
50	Madhu	25	6974	No	No	yes	No	No	No	No	No	27	Yes	11.2	7.8	4.6	1.6	1.69	2.3	0.1	21.3