Chapter one

Introduction

Chapter One

1.1 Introduction

The polycystic ovary syndrome (PCOS) is a hyperandrogenic disorder associated with

insulin resistance and compensatory hyperinsulinemia, which is recognized as a major factor responsible for altered androgen production and metabolism [3]. Most women with PCOS are also overweight or obese, further enhancing androgen secreti

chronic oligo-anovulation and polycystic ovarian morphology (heartfoundation.org.au/SiteCollectionDocuments/MeredithFrearson.pdf). It is often associated with psychological impairments, including depression and other mood disorders and metabolic derangements, chiefly on while impairing metabolism and reproductive functions and possibly favoring the development of the PCOS phenotype. The definition of PCOS has led to an impressive increase of scientific interest in this disorder, which should be further directed to improve individualized clinical approaches and, consequently therapeutic strategies.PCOS was first identified by Stein and Leventhal in 1935 so that it can also be known as Stein – Leventhal Syndrome.

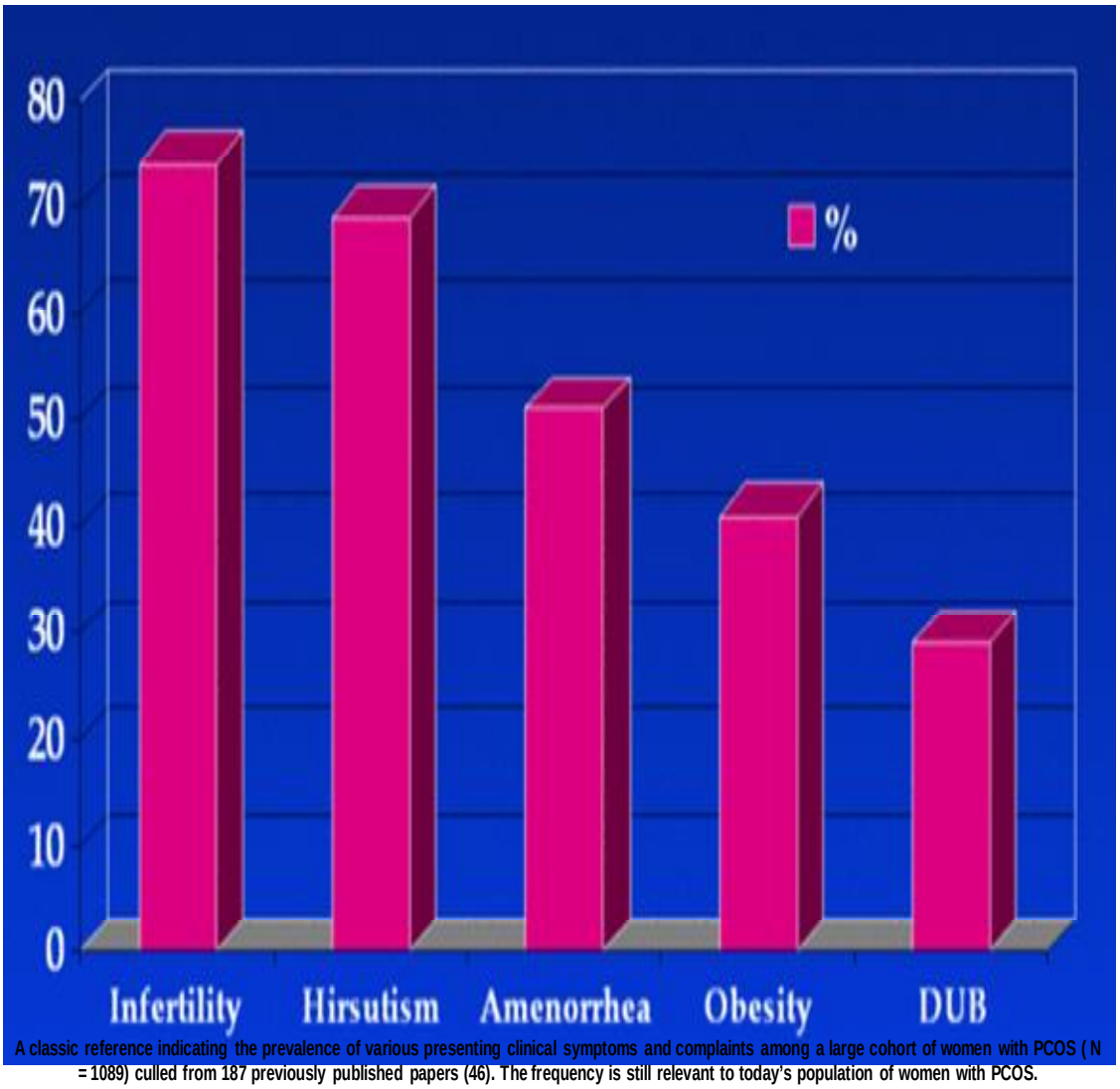
1.2 Prevalence of PCOS

Estimation of the 'true' prevalence has to be made with caution as many of the data available were collectedprior to the new Rotterdam diagnostic criteria. Most clinical data suggests a prevalence of 6–7% of thepopulation. The present Rotterdam criteria are current best practice but it is recognized that PCOSencompasses a wide spectrum of disorder, overlapping with normality.The prevalence of PCOS may differ according to ethnic background; for example, in women of South Asianorigin, PCOS presents at a younger age, has more severe symptoms and a higher prevalence.

1.3 Research Problem

Diagnosis of PCOSis not an easy issue as it requires the presence of two out of the following three criteria:

- Oligo- and/or Anovulation
- Hyperandrogenism (clinical and/or biochemical)
- Polycystic Ovaries,with the exclusion of other etiologies



A classic reference indicating the prevalence of various presenting clinical symptoms and complaints among a large cohort of women with PCOS (N = 1089) culled from 187 previously published papers (46). The frequency is still relevant to today's population of women with PCOS.
Source of Image: http://www.endotext.org/female/female6/female6.html

FIG (1) a Presents Sign and symptom of PCOS

1.4 SCOPE& LIMITATION OF THE STUDY

This study will focus particularly on SAQR Hospital, a 300 beds government hospital.A total number of 35 sample size were taken however, only 33 cases of PCOs were typically diagnosed and two patients among the samples were excluded because one was diagnosed as primary prolactinemia not merely associated to PCOS and the other case were confirmed on hypothyroidism and both were referred to specialized Physician respectively thus excluded in the inclusion criteria.

1.5 SIGNIFICANCE OF THE STUDY

PCOS affects 5% to 10% of women of reproductive age which approximately 4 million individuals. It's prevalence among infertile women is 15% to 20%..It is the most common endocrine disorder of women in this age group.It is often seen in the student health population and general medical practice but most often diagnosed when a women presents with infertility) Allabove mentioned made the proper diagnosis of PCOS mandatory.

1.6 Objectives:

1.7 GeneralObjective: To study the polycystic ovarian syndrome using ultrasound

1.8 Specific Objectives

1. To evaluate the presence of PCO according to the site and other ultrasound findings.
2. To evaluate the biochemical results of LH, FSH &Testosterone.
3. To correlate the biochemical analysis with ultrasound findings.
4. To correlate the findings with clinical data and patients history

1.9 Statement of the Problem

The diagnosis of PCO is not an easy thing as the can only be made when other etiologiesofhormonaldisturbances like thyroiddysfunction,

congenitaladrenalhyperplasia, hyperprolactinaemia, androgen-secreting tumors andCushing syndrome)

Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome.

The Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group

However, biochemical tests will be subjected to some errors as well as the ultrasound that depends on operator experience.

Having polycystic ovaries does not mean having PCOS as only 6-7% of women with polycystic ovaries have PCOS those patientsdiagnosed by the new Rotterdam criteria in use today, in particular where ultrasound was used as the main diagnostic criterion.

1.10 Organization of the study:

- **Chapter One:** Introduction.
- **Chapter Two:** Literature Review
- **Chapter Three:** Materials and Methods.
- **Chapter Four:** Results.
- **Chapter Five:** Discussion, Conclusion and Recommendations.
    - References
    - Appendix

Chapter TWO

Literatures Review

Theoretical Background

Previous Studies

Chapter Two

Literatures Review

2.1 Theoretical Background

Since the 1990 National Institutes of Health–sponsored conference on polycystic ovary syndrome (PCOS), it has become appreciated that the syndrome encompasses a broader spectrum of signs and symptoms of ovarian dysfunction than those defined by the original diagnostic criteria. The 2003 Rotterdam consensus workshop concluded that PCOS is a syndrome of ovarian dysfunction along with the cardinal features hyperandrogenism and polycystic ovary (PCO) morphology. PCOS remains a syndrome, and as such no single diagnostic criterion (such as hyperandrogenism or PCO) is sufficient for clinical diagnosis. Its clinical manifestations may include menstrual irregularities, signs of androgen excess, and obesity. Insulin resistance and elevated serum LH levels are also common features in PCOS. PCOS is associated with an increased risk of type 2 diabetes and cardiovascular events

One nutritional approach to PCOS is to take dietary steps to reduce insulin levels. This, in effect, means reducing in the diet carbohydrates that liberate significant quantities of glucose into the bloodstream (and therefore stimulate significant surges in insulin). At its heart, this is a ‘low-carb’ diet made up ostensibly of meat, fish, eggs, nuts, seeds, non-starchy vegetables and a little fruit such as berries. Not only does this sort of diet seem to help improve some of the biochemical imbalances typically found in PCOS, it often leads to weight loss too.

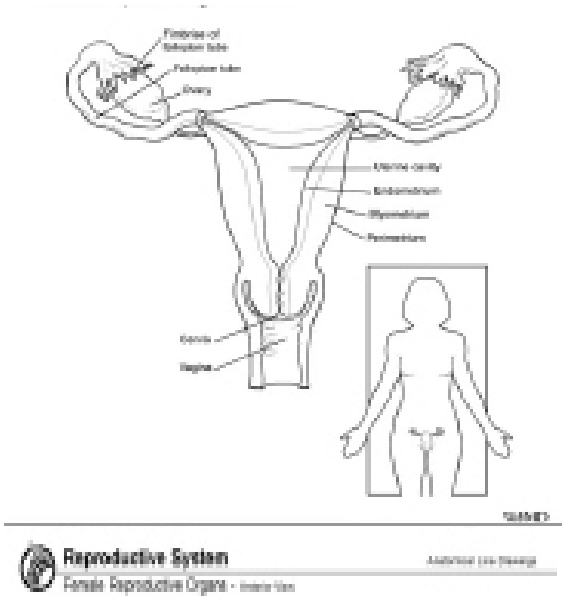
However, not all women with PCOS are overweight, but may nevertheless have features such as insulin resistance, raised testosterone and ovulation issues. I was very interested to read about a recent study in women with PCOS but normal bodyweightin

whom two different diets weretrialed [heartfoundation.org.au/SiteCollectionDocuments/Meredith-Frearson.pdf]. In this study, it was not the 'macronutrient' make-up of the diet that was being tested (e.g. low fat v low carb), but the phasing of food intake

2.1 **.1 Anatomy**

The ovaries are the female pelvic reproductive organs that house the ova and are also responsible for the production of sex hormones. They are paired organs located on either side of the uterus within the broad ligament below the uterine (fallopian) tubes. The ovary is within the ovarian fossa, a space that is bound by the external iliac vessels, obliterated umbilical artery, and the ureter. The ovaries are responsible for housing and releasing ova, or eggs, necessary for reproduction. At birth, a female has approximately 1-2 million eggs, but only 300 of these eggs will ever become mature and be released for the purpose of fertilization.

Anatomy of the ovaries is displayed in the images below.



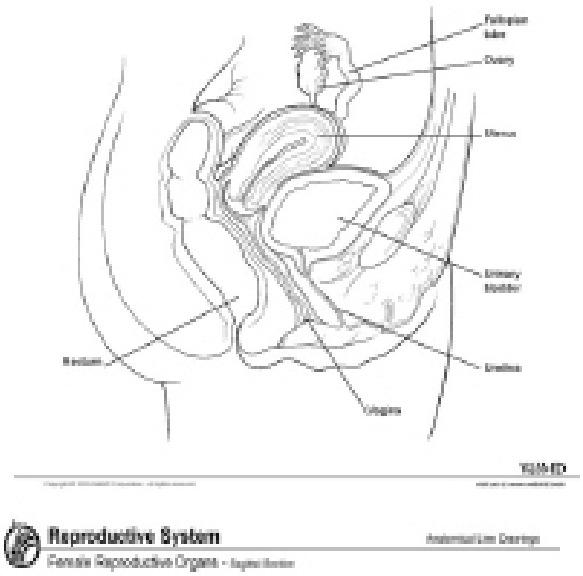


Fig (2.1):ShowsA-Veiw of Female Reportuctive system Fig (2.2):Shows Longitudinal Veiw of Female Reportuctive system

The ovaries are small, oval-shaped, and grayish in color, with an uneven surface. The actual size of an ovary depends on a woman's age and hormonal status; the ovaries, covered by a modified peritoneum, are approximately 3-5 cm in length during childbearing years and become much smaller and then atrophic once menopause occurs. A cross-section of the ovary reveals many cystic structures that vary in size. These structures represent ovarian follicles at different stages of development and degeneration.[heartfoundation.org.au/SiteCollectionDocuments/Meredith-Frearson.pdf]. The ovarian ligament Several paired ligaments support the ovaries connects the uterus and ovary. The posterior portion of the broad ligament forms the mesovarium, which supports the ovary and houses its arterial and venous supply. The suspensory ligament of the ovary (infundibular pelvic ligament) attaches the ovary to the pelvic sidewall. This larger structure also contains the ovarian artery and vein, as well as nerve supply to the ovary.

2.1 .1.1: Blood supply, nerve supply, and lymph drainage

Blood supply to the ovary is via the ovarian artery; both the right and left arteries originate directly from the descending aorta. The ovarian artery and vein enter and exit the ovary at the hilum. The left ovarian vein drains into the left renal vein, and the right ovarian vein empties directly into the inferior vena cava

2.1.2 Physiology

The ovaries are the organs in a woman's reproductive system that produces eggs (ova.) They are almond-shaped and about 3.5 cm (1.5 inches) long. The ovaries are deep in a woman's pelvis, on both sides of the uterus (womb), close to the ends of the Fallopian tubes.

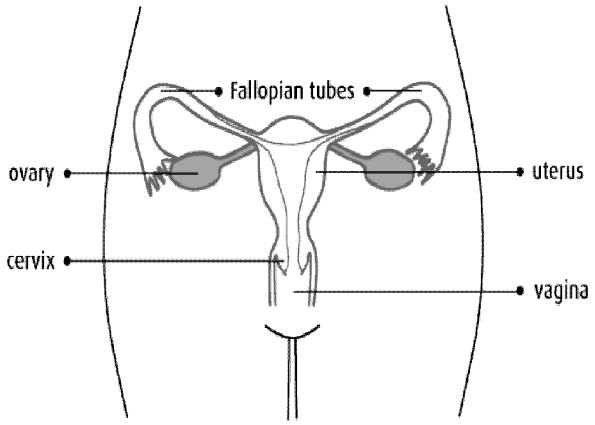


Fig (2.3):Shows Reportuctive system

http://www.cancer.ca/en/cancer-information/cancer-type/ovarian/anatomy-and-physiology/?region=on#ixzz3YEGFsVZM

2.1.2.1 Structure:

The ovaries are made up of 3 different types of cells:

- **Epithelial cells** make up the outer layer covering the ovary (epithelium).
- **Germ cells** are inside the ovary. They develop into eggs.
- **Stromal cells** form the supportive or connective tissues of the ovary (stroma).
- Each ovary is surrounded by a thin layer of tissue called the capsule.

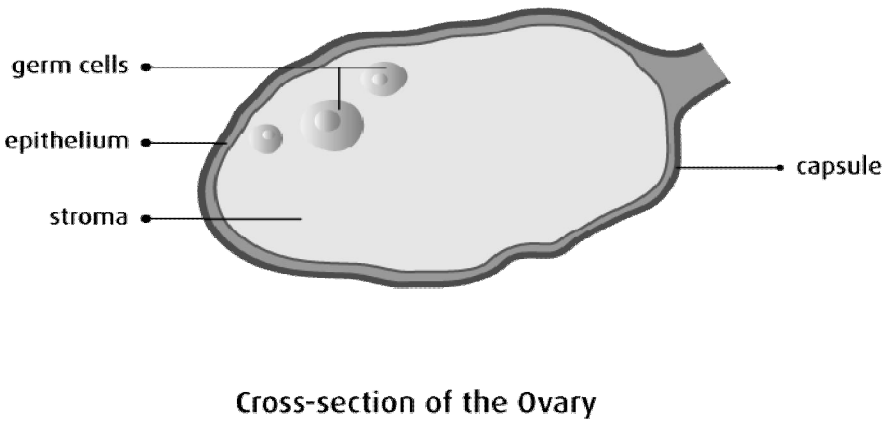


Fig (2.4):Shows Cross-sectionof the Ovary

http://www.cancer.ca/en/cancer-information/cancer-type/ovarian/anatomy-and-physiology/?region=on#ixzz3YEGFsVZM

2.1.2.2: Function

The ovaries have 2 main functions. They produce mature eggs. They also make the female sex hormones, which control reproduction and sexual development.

Estrogen is responsible for the development of secondary sex characteristics, such as the growth of breasts.

Progesterone prepares the body for conception by causing the buildup of the uterine lining (endometrium) and other changes.

The ovaries are the main source of estrogen in sexually mature women.

Each month during ovulation, an ovary releases a mature egg. The egg travels down the Fallopian tube to the uterus. If it is fertilized by a sperm, the egg implants into the lining of the uterus and begins to develop into a fetus. If the egg is not fertilized, it is shed from the body along with the lining of the uterus during menstruation.

During menopause, the ovaries stop releasing eggs and producing sex hormones.

2.1.3pathophysiology

The pathophysiology of PCOS, although still not entirely clear, is mainly due to the hormone imbalance caused by both hyperandrogenism and hyperinsulinemia, which are also effects of PCOS.

2.1.3 .1: Defining alterations of steroidogenesis in PCOS

In normal women, androgen production rate (PR) is the result of adrenal and ovarian secretion and conversion from precursors in peripheral tissues, particularly the adipose tissue and skin . Similarly, the metabolic clearance rate (MCR) of androgens may occur in both glandular and extraglandular tissues. Both PR and MCR of androgens in females depend on age and physiological status. All androgens exhibit a daily rhythm, less variable for androstenedione and testosterone than that of DHEA and cortisol. A few studies, all performed several decades ago, documented higher

PRs for both androstenedione and testosterone in women with PCOS, associated with a less pronounced increase of their MCR (*Achard/Thiers 1921)*. In addition, it was shown that testosterone MCR was higher in obese PCOS women and varied according to its PR, whereas MCR of androstenedione was marginally different with respect to normal weight affected women, suggesting that factors [peripheral conversion or possibly binding to sex hormone binding globulin (SHBG)] in addition to body size influenced testosterone MCR in PCOS women. Notably, there are no studies in PCOS women with different obesity phenotypes, although there is evidence that in women with simple obesity, those with abdominal fat distribution have higher testosterone PR, but not higher androstenedione, with respect to those with the peripheral phenotype*(Annals 2000)*. Similar studies should therefore be replicated in PCOS women with different obesity phenotypes.Estrogen and progesterone PRs in women with PCOS have been poorly investigated.One of the main problems in the diagnosis of hyperandrogenic states such as PCOS is the accurate measurement of androgens and particularly testosterone. Many radioimmunoassays, especially platform assays, for androgens are decidedly unsatisfactory. Most of these intrinsic methodological limitations are bypassed by the growing use of liquid chromatography-tandem mass spectrometry (LM/MS-MS), the modern gold standard for all steroid hormone measurement, particularly in women. By the use of LM/MS-MS it would be expected that additional kinetic studies in different phenotypes of this disorder may favor a better understanding of complex pathophysiological events leading to androgen excess in women with PCOS, as preliminary clinical studies seem to indicate.“Vicious cycle” Abnormal gonadotropin secretion, Excess LH and low, tonic FSH, Hypersecretion of androgens, Disrupts follicle maturation, Substrate for peripheral aromatization, Negative feedback on pituitary , Decreased FSH secretion, Insulin resistance, Elevated insulin levels.

2.1.3 .2: Ovaries: enlarged and/or polycystic ovaries

2.1.3.3:Endometrial: Lack of ovulation for an extended period of time may cause excessive thickening of the endometrium (the lining of the uterus).

2.1.3.4: Polycystic ovarian syndrome (PCOS) is a condition characterised by multiple cysts in the ovaries which can impair ovulation and therefore fertility. One common feature of the condition is raised levels of the 'male' hormone *testosterone*, which can lead to 'masculinising' side effects such as 'hirsutism' (abnormal, excessive hair growth), scalp hair loss, and acne.

Another key underlying biochemical feature of PCOS is raised levels of *insulin*, usually related to '*insulin resistance*' (impaired functioning of insulin). There is some thought that insulin acts on the ovaries to stimulate testosterone production

2.1.3.5: Significance of adrenal androgen production

It has been estimated that 25% of androstenedione and testosterone production is of ovarian origin, 25% is of adrenal origin and 50% is produced in peripheral tissues, while the adrenal cortex accounts almost uniquely for the synthesis of dehydroepiandrosterone (DHEA) and dehydroepiandrosterone sulfate (DHEAS) as well as that of androstenediol and 11β-hydroxyandrostenedione (*Abdel Gadir A et al 1992)*. In women androgens serve as precursors of estrogen biosynthesis, which starts to decrease 3–4 years before menopause . At the same time basal serum levels of ovarian androgens decrease only slightly and remain relatively stable until menopause, while the decrease of adrenal androgens can already be observed after the age of 30 years . Compared with healthy subjects, women with previous PCOS have an increased adrenal capacity to secrete androgens that remains until after menopause. These results confirm the adrenals contribute significantly to hyperandrogenism in PCOS and similarly to ovarian androgen secretion capacity, women with PCOS exhibit enhanced adrenal androgen production until their late reproductive years . The

pathophysiological mechanisms responsible for increased androgen production by the adrenals in women with PCOS remains, however, poorly elucidated and should require further investigation. Difficulty in obtaining viable and appropriate adrenal tissue has limited in vitro study of human tissue, but long term culture is possible, and the derivation of stem cell adrenal cortex tissue could significantly enhance studies of this important gland.

2.1.3.6: Specific steroidogenic enzyme defects in PCOS

The etiology of PCOS remains uncertain but intrinsic abnormalities in the synthesis and secretion of androgens are a plausible basis for the syndrome. There is clear evidence for constitutive hyper-secretion of androgen by ovarian theca cells but abnormalities of adrenal androgen production have also been implicated in the etiology. It is therefore reasonable to pose the question "are specific primary enzyme abnormalities in the steroidogenic pathway an important cause of PCOS. On the basis of currently available evidence, the answer to this question is probably "no". Amongst plausible candidate genes in genesis of hyperandrognemia are *CYP17* (coding for P450c17, and the associated P450 reductase) and, because of evidence for a global increase in steroidogenic enzyme activity in PCO theca cells, *CYP11a*(P450scc) . To date, case-control and family-based studies have shown no clear evidence that variants in these genes (or for that matter, many others involved in steroidogenesis) contribute to the pathogenesis of PCOS. Recent work has focused on metabolism of cortisol and adrenal androgens but, although specific enzyme defects may be associated with a PCOS phenotype (e.g. defects in cortisone reductase), the data from large association studies suggest that such defects are but a very minor contributor to the etiology of PCOS .In addition, extraglandular synthesis of androgens, particularly in the adipose tissue, has been found to be involved in the pathophysiology of PCOS. They involve alteration in the activity of 11β-hydroxysteroid dehydrogenase and both 5α-reductase and 5β-reductase . Alterations of these enzyme systems which are involved in peripheral cortisol metabolism may in turn activate the neuroendocrine drive to

support adrenal steroidogenesis and may partly explain the increased androgen production in specific subsets of women with PCOS.

2.1.3.7: Sympathetic nerve activity and hyperandrogenism

Many factors associated with polycystic ovary syndrome (PCOS) are also associated with increased activity in the sympathetic nervous system. The involvement of sympathetic nervous system in PCOS pathology is supported by the greater density of catecholaminergic nerve fibres in polycystic ovaries (PCO) . Increased ovarian sympathetic nerve activity might contribute to PCOS by stimulating androgen secretion. Nerve growth factor (NGF) is a strong marker for sympathetic nerve activity and recently it was demonstrated that women with PCOS has enhanced ovarian NGF production. In a transgenic mouse model overexpressing NGF in the ovaries, they found that that a persistent elevation in plasma LH levels is required for the typical morphological abnormalities to appear. These results suggest that overproduction of ovarian NGF is a component of PCO morphology.

Studies using indirect markers of autonomic function – heart rate variability and heart rate recovery after exercise – have shown that women with PCOS have increased sympathetic and decreased parasympathetic components . Recently, for the first time it was demonstrated that women with PCOS have high general activity in the sympathetic nervous system which may be relevant to the pathophysiology of the syndrome . Interestingly, testosterone was the strongest independent factor explaining high sympathetic nerve activity in women with PCOS. As the degree of androgen concentration can reflect the severity of PCOS, the relationship between sympathetic nerve activity and testosterone concentration indicates that the degree of sympatho-excitation is related to the degree of PCOS severity.

Recently, a randomized controlled trial demonstrated that low-frequency electro-acupuncture (EA) and physical exercise (both known to modulate sympathetic nerve activity) decreases high levels of circulating sex steroid precursors, estrogens, androgens, and glucuronidate androgen metabolites and improve menstrual bleeding

pattern in women with PCOS, and thus break the vicious circle of androgen excess [28]. In a subset of these women, low-frequency EA and physical exercise was shown to decrease high sympathetic nerve activity in women with PCOS, which may at least in part explain the beneficial effects of these therapies. It may also be hypothesized that therapies such as ovarian wedge resection or laparoscopic laser cauterization[30], utilize its effect by temporary disruption of ovarian sympathetic innervation, and thus increase ovulatory function and decrease androgen synthesis in women with PCOS.

2.1.4 Diagnosis

How is PCOS diagnosed?

Diagnosis of PCOS can only be made when other aetiologies have been excluded (thyroid dysfunction,congenital adrenal hyperplasia, hyperprolactinaemia, androgen-secreting tumours and Cushingsyndrome).A consensus definition using precise diagnostic criteria should be used when diagnosing PCOS tofacilitate effective patient care and robust clinical research.

The National Institutes of Health (NIH) 1990 preliminary consensus definition has now been replaced by amore recent definition by the Rotterdam European Society for Human Reproduction and Embryology(ESHRE) and the American Society of Reproductive Medicine (ASRM) PCOS Consensus Workshop Group.This has suggested a broader definition for PCOS, with two of the three following criteria being diagnostic ofthe condition:

- Polycystic ovaries (either 12 or more peripheral follicles or increased ovarian volume (> than 10 cm3)
- oligo- or anovulation
- Clinical and/or biochemical signs of hyperandrogenism.

A raised luteinising hormone/follicle-stimulating hormone ratio is no longer a diagnostic criteriafor PCOS owing to its inconsistency.12 It should be noted that the diagnosis of PCOS can only bemade when other aetiologies have been excluded. The

recommended baseline screening testsare thyroid function tests, a serum prolactin and a free androgen index (total testosterone dividedby sex hormone binding globulin (SHBG) x 100 to give a calculated free testosterone level). Incases of clinical evidence of hyperandrogenism and total testosterone greater than 5 nmol/l,17-hydroxyprogesterone should be sampled and androgen-secreting tumors excluded. If there isa clinical suspicion of Cushing syndrome, this should be investigated according to local practice.These new diagnostic criteria have affected the value of a number of systematic reviews, as the majority ofthe reviews are based on the NIH 1990 criteria, which may not be entirely representative of those patientsdiagnosed by the new Rotterdam criteria in use today, in particular where ultrasound was used as the maindiagnostic criterion.

2.2 Previous Studies:

2003 ESHRE/ASRM*(Rotterdam,Netherlands)* Consensus on the Dx of PCOS

Requires the presence of two out of the following three criteria:

Oligo- and/or Anovulation .Hyperandrogenism (clinical and/or biochemical)

Polycystic Ovaries, with the exclusion of other etiologies 1990 US NIH Consensus Conference: 2 minimal criteria

1. Menstrual Irregularity due to oligo- or anovulation
2. Clinical or biochemical hyperandrogenism
    a.Hirsutism,Acne,Male Pattern Baldness
    b.Elevated Serum Androgen Levels
3. Above not attributable to other causes(Diagnosis of exclusion)

Task Force Appointed by the Androgen Excess Society 2006

Reviewed all available data and recommended a new evidence-based definition

(*J ClinEndocrinol Metab.2006 Aug 29*)

The Task Force identified 4 key clinical features of PCOS:

1. Ovulatory and Menstrual Dysfunction
2. .Hyperandrogenism
3. Hirsutism, Acne and Androgenic Alopecia
4. Polycystic Ovaries

Plus the exclusion of other disorders of androgen like Congenital Adrenal hyperplasia (CAR) and adrenal tumors.

Chapter Three

Materials and Methods

Chapter Three

Materials & Methods

Study Area:

Saqr hospital is the only governmental hospital that serving all gynae and obstetric cases in Rasalkhaimah (RAK) and its border to Oman. RAK one of the 7th emirates of UnitedArab Emirates which located in the northern UAE. It has an area of 5800 km2 and an estimated population of approximately 25000 (2005).

3.1: Materials

3.1.1Machine Used:

3.1.1.1: Mass spectrometer.

3.1.1.2: Ultrasound –Hd I IXe(Complete Digital Imaging System) with linear convex curved array and 3D PROBS for abdominal vascular and ob/gyn.

3.1.2: Patients:

35Female patients, different age groups, nationalities anddifferent marital status were examined.

3.2: Methods:

3. 2.1: Technique used:

Both Trans abdominal andtranvaginalUltrasound were done.

Preparation for Trans abdominal Ultrasound (convex probe 3.5 – 5 MHz):

- Bladder should be full
- Patient should be in supine position; the probe should be covered with lubricant gel then move the probe longitudinal, transverse, right and left to visualize the internal pelvic organs.

- Philips IU 22 with high frequency tranvaginal probe (9 – 10 MHz)

Preparations for Transvaginal Ultrasound:

- Patient should remove her clothes and put on a gown or cover
- Bladder should be empty or partially filled
- Patient should lie down on an examination table in a supine position with feet placed in stirrups
- The ultrasound probe will be covered with condom and lubricant gel inserted inside the vagina longitudinally to view the uterus then transversely right and left
    - count the number of follicles
    - measure the volume of the ovary
    - measure the endometrial thickness

3.2.2 Labrotary Tests:

Hormonal assay was achieved by using liquid chromatography method avoiding the unsatisfactory results of radioimmunoassay.

Chapter Four

Results

Chapter Four

Results

The data was presented by the following tables and graphs

Table (4. 1): Demonstrates signs &Symptoms of PCOS

SIGNS AND SYMPTOMS	option	Frequencies	Percentage
BMI	High	11	33.3
	normal	22	66.7
	Total	33	100%
Irregular period	Yes	21	63.6
	No	12	36.4
	Total	33	100%
Rapid Weight Gain	Yes	6	18.2
	No	27	81.8
	Total	33	100%
Infertility	Yes	2	6.1
	No	31	93.9
	Total	33	100%
Abnormal Uterine Bleeding	Yes	0	0
	No	33	100
	Total	33	100%
Hirsutism/ Acne	Yes	14	42.4
	No	19	57.6
	Total	33	100%

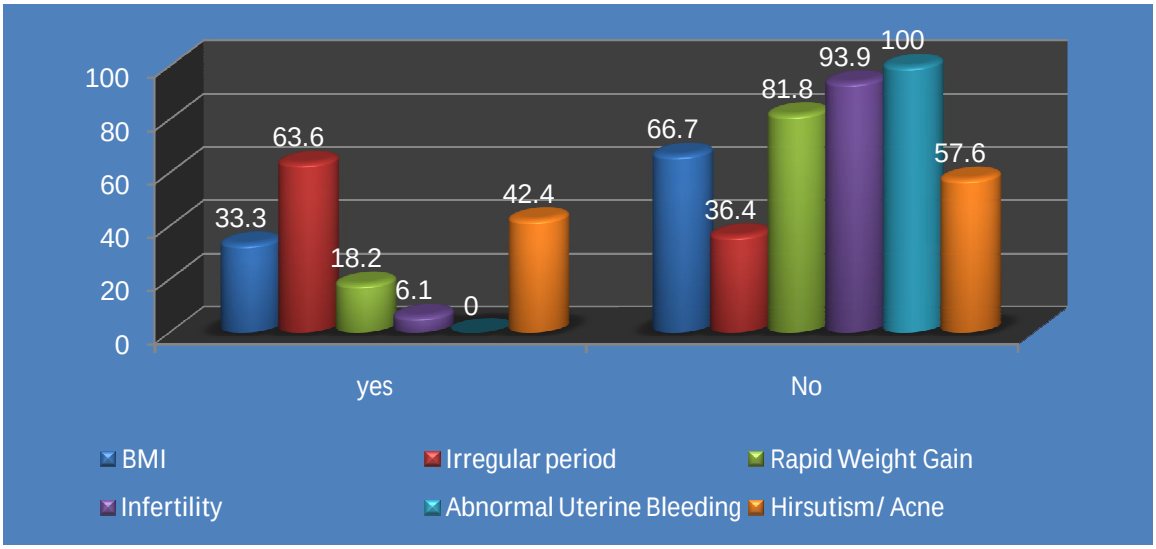


Fig. (4.1)Shows: Signs &Symptoms of PCOS

Table(4.2):Shows Labrotary Hormonal Analysis Of PCOS

LABORATORY RESULTS	Option	Frequencies	Percentage
Testosterone	Yes	21	63.6
	No	12	36.4
	Total	33	100%
Dehydroepiandosterone	Yes	10	30.3
	No	23	69.7
	Total	33	100%
Tyroid Function Test	Yes	6	18.2
	No	27	81.8
	Total	33	100%
High Prolactin	Yes	18	54.5
	No	15	45.5
	Total	33	100%
HF Insulin	Yes	11	33.3
	No	22	66.7
	Total	33	100%
Inverted LH/LSH	Yes	12	36.4
	No	21	63.6
	Total	33	100%

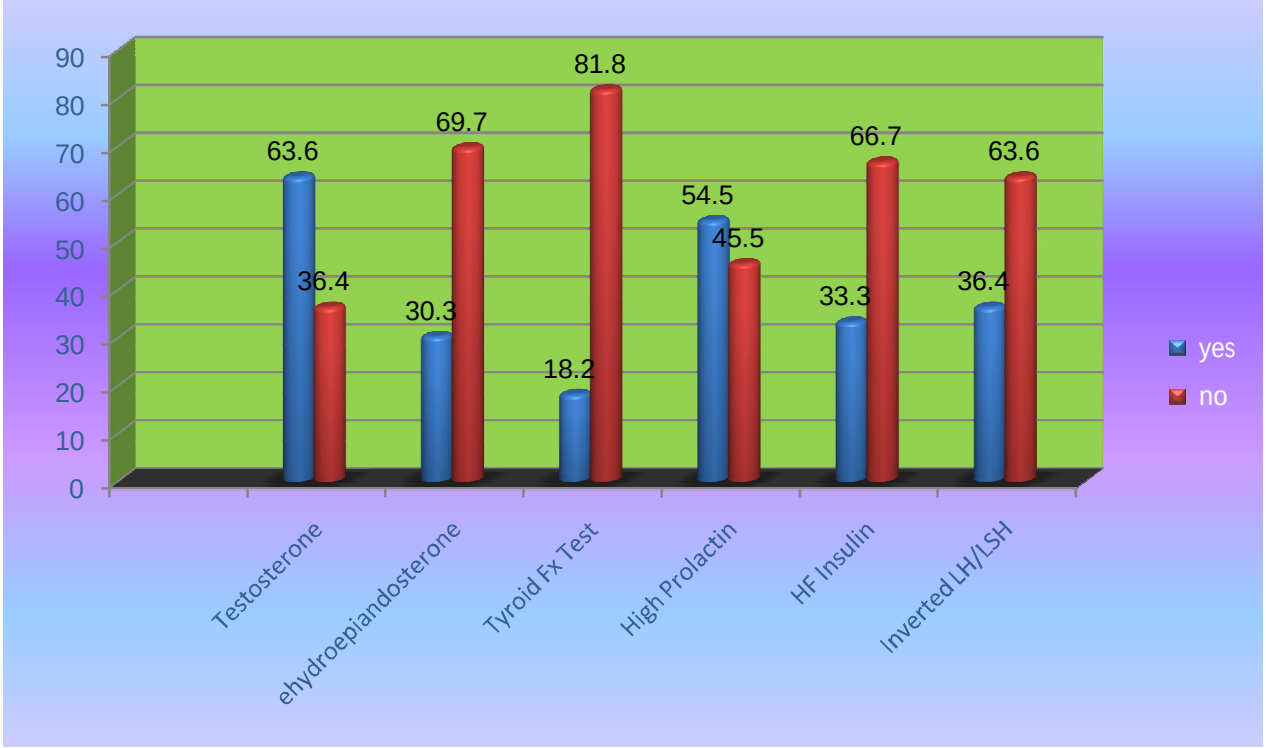


FIG (4. 2):Shows labrotary Hormonal Analysis Of PCOS.

Table(4.3) Shows Ultrasound Findings of PCOS

ULTRASOUND RESULT	option	Frequencies	Percentage
POLYCYSTIC OVARIES	Yes	18	54.5
	No	15	45.5
	Total	33	100%
OVARIAN VOLUME	Yes	14	42.4
	No	19	57.6
	Total	33	100%
PERIPHERAL FOLLICLES	Yes	19	57.6
	No	14	42.4
	Total	33	100%
ENDO. HYPERPLASIA	Yes	19	57.6
	No	14	42.4
	Total	33	100%

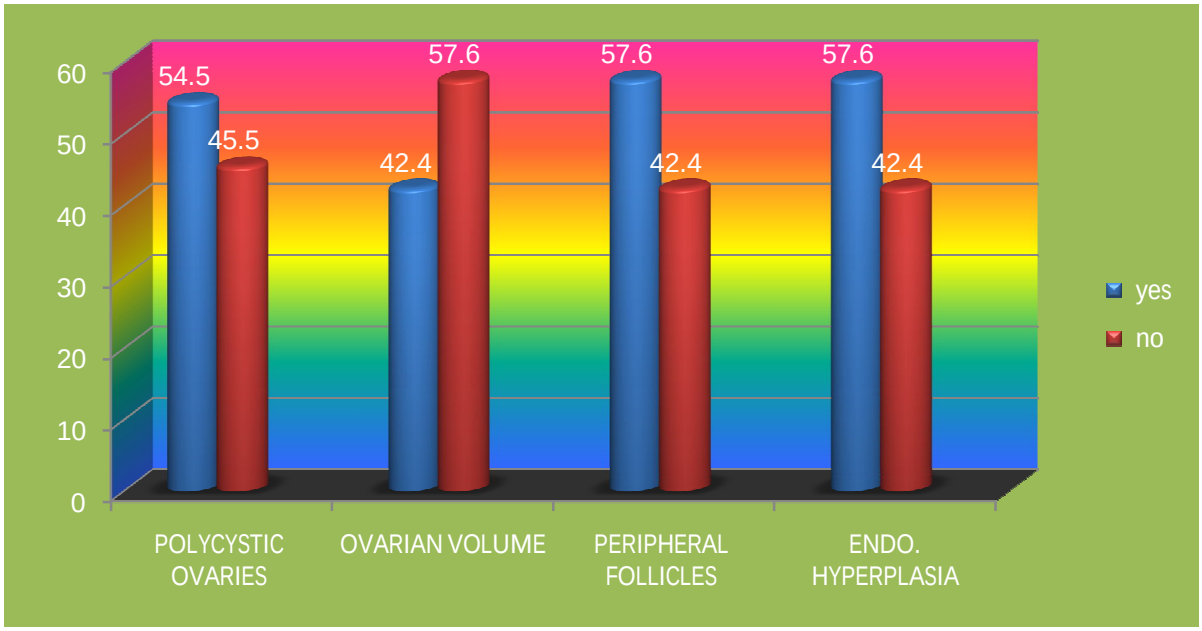


FIG (4. 3) Shows Ultrasound Findings of PCOS

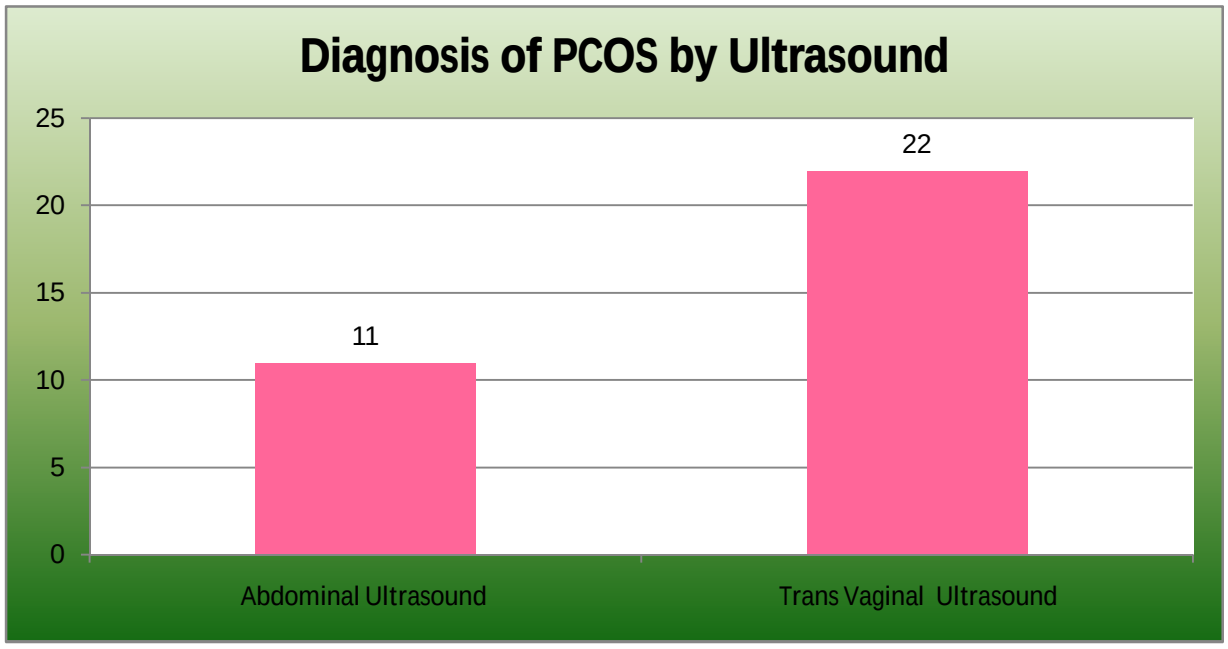


Fig (4.4) shows the comparison between abdominal ultrasound and TVS in Diagnosis

Chapter Five

Discussion, Conclusion and Recommendations

Chapter Five

Discussion, Conclusion and Recommendations

5.1: Discussion

PCOS first diagnosed in1935-Stein and Leventhal described the features of 7 hirsute, amenorrheic women based on the characteristic ovarian morphology from histological specimens taken at wedge resection of the ovaries (*Am J Obstet Gynecol. 1935;29:181-91.)*PCOS probably represents a spectrum of disease and variable presentations whichneccesate along list of investigations, The thing might be elusive to the generalist or specialist*(Nestler JE 1998).*

This study intended to investigate thirty three patients who attended our gynae clinic in saqr hospital –RasAlkhaima-United Arab Emirates.

Most of the patients in the study present with period irregularitythepercentage of 63.6%with a frequency of 21,Hirsutism , Acne 42.4% a frequency of 14,high BMI in a percentage of 33% frequency of 11&rapid gain weight 18%,frequency of 6 and 6.1% infertility with a frequency2.these results presented in **Table 4.1** as well as in **fig4.2.**While infertility in the literature represents 75%. 20%represents the period disturbance &95% with high BMI &rapid weight gain fig(1).(A classic reference indicating the prevalence of various presenting clinical symptoms and complaints among a large cohort of women with PCOS (N = 1089) culled from 187 previously published papers (46). The frequency is still relevant to today's population of woman with PCOs. Source of Image (*http://www.endotext.org/female/female6/female6.html*)

Early &accurate diagnosis is crucial as it may delay or possibly prevent some of sequelae associated with PCOS. (Class description of a bearded women with DM, Achard/Thiers 1921)& *Shoupe D et al1983* .A total of number of 35 sample size were taken however, only 33 cases of PCOs were typically diagnosed and two patients among the samples were excluded because one was diagnosed as primary prolactinemia not merely associated to PCOs and the other case were confirmed on hypothyroidism and both were referred to specialized Physician respectively thus excluded in the inclusion criteria.The following were the percentage of hormonal assay achieved by using liquid chromatography method avoiding the unsatisfactory results of radioimmmunoassay .The result presented in table (4.2) as well as in fig(4.2)The result showed that among the diagnosed cases testosterone is high to 63.6%

and 54.5% which is confirmed for high prolactin and 36.4% for inverted LH/FSH which both were lab confirmatory for the case. 57%with peripheral follicles &increased endometrial thickness, detecting 54% of In the study conducted, among the 33 samples of PCOs, 22 married cases were diagnosed by transvaginal ultrasound however 6 of them showed negative result but diagnosis were confirmed to both clinical and hormonal assay. Likewise, 11 unmarried samples were confirmed by clinical presentation and hormonal assay as they were not subjected for Trans Vaginal ultrasound , abdominal ultrasound were done which is less reliable for confirmatory diagnosis for the case. Therefore, Trans vaginal ultrasound is always recommended wherever possible. PCOS, then when all suspected cases of PCOS that were subjected to TVS, the diagnosis was established while abdominal ultrasound missed eighteen out of the thirty three cases(*Abdel Gadir A et al 1992)*

5.2: Conclusion:

The current study concluded that:

1. Diagnosis of PCOS is a complicated task .
2. Pelvic Ultrasound (Transvaginal is best); Endometrial thickness should always be assessed to exclude significant endometrial pathology.
3. Hormone Assays(to exclude other mimickers of PCOS)
4. Glucose Testing; 2 Hour Post Prandial
5. Lipid Status (to check Total Cholesterol, HDL and Triglyceride Levels)
6. Other investigations
7. Exclusion of other conditions that may mimic PCOSPCOS is the most common cause of anovulatory infertility.
8. PCOS is one of the commonest endocrinopathies to affect women(5-10%).
9. PCOS probably represents a spectrum of disease with variable presentations.
10. Is important to diagnose PCOS because of the potential long-term consequences.
11. Early diagnosis may delay or possible prevent some of sequelae associated with PCOS.
12. Further research is necessary in this syndrome

5.3: Recommendations

A consensus definition using precise diagnostic criteria should be used when diagnosing PCOS to facilitate effective patient care and robust clinical research.

Recommend to use Transvaginal ultrasound wherever possible and not to depend on trans abdominal one in confirming o

r refuting the diagnosis of PCOS

Recommend to do the radioimmunoassay using the liquid chromatography-tandem mass spectrometry (LM/MS-MS),

References

REFERENCES

http://www.heartfoundation.org.au/SiteCollectionDocuments/Meredith-Frearson.pdf

http://www.cancer.ca/en/cancer-information/cancer-type/ovarian/anatomy-and-physiology/?region=on#ixzz3YEGFsVZM

RCOG GUIDELINES 2000

Dehurts text book 2006 edition 6

January 2004Volume 81, Issue 1, Pages 19–25

Achard/Thiers 1921

20 June 2000 • Annals of Internal Medicine • Volume 132 • Number 12 991

Ann Intern Med. 2000;132:989-993.

Shoupe D, Kumar DD, Lobo RA. Insulin resistance in polycystic ovary syndrome. Am J Obstet Gynecol. 1983;147:588-92.

Abdel Gadir A, Khatim MS, Mowafi RA, Alnaser HM, Muharib NS, Shaw RW. Implications of ultrasonically diagnosed polycystic ovaries. I. Correlation with basal hormonal profiles. Hum Reprod. 1992;7:453-7.

Nestler JE. Polycystic ovary syndrome: a disorder for the generalist. FertilSteril. 1998;70:811-2)

Appendices

Appendix A

DEMOGRAPHIC PROFILE OF THE CASES REVIEWED ON THE STUDY

1.) Age in years :
- a) 21 - 30 ( )
- b) 31 - 40 ( )
- c) 41 - 50 ( )
- d) 51 - 60 ( )

2.) Nationality :
- a) Emirati ( )
- b) Other Arab Countries ( )
- c) Indian ( )
- d) Filipino ( )

3.) Marital Status :
- a) Single ( )
- b) Married ( )
- c) Widow ( )
- d) Divorced ( )

4.) Nutritional Status
- a) Underwieht ( )
- b.) Normal weif\ght ( )
- c.) fat ( )
- d,) Obese ( )

Appendix B

CHECKLIST USED IN THE STUDY

1. SIGNS AND SYMPTOMS	**YES**	**NO**
a. Irregular Period	( )	( )
b. Rapid Gain weight	( )	( )
c. Infertility	( )	( )
d. Abnormal l Uterine Bleeding	( )	( )
e. Hirsutism / acne	( )	( )

2. LABORATORY RESULTS

a. High Free Serum Testosterone Level	( )	( )
b. Slightly High or Normal	( )	( )
c. Dehydroepiandrosterone acetate	( )	( )
d. Normal Tyroid Function Test result	( )	( )
e. High Prolactin	( )	( )
f. High Fasting Insulin `	( )	( )
g. Inverted LH/FSH Ratio	( )	( )

3. ULTRASOUND RESULT(abdominal & TVS)

a. Polycystic Ovaries	(+)	( )	( )
b. Ovarian Volume > 10 cm		( )	( )
c. Peripheral Follicles ≥ 12		( )	( )
d. Positive for Endometrial Hyperplasia		( )	( )