METABOLIC SYNDROME AND INSULIN RESISTANCE SYNDROME AMONG POLYCYSTIC OVARY SYNDROME

DISSERTATION SUBMITTED TO

In partial fulfillment of the requirement for the degree of

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BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled "**METABOLIC SYNDROME AND INSULIN RESISTANCE SYNDROME AMONG POLYCYSTIC OVARY SYNDROME"** submitted by **Dr.SRI SUBBULAKSHMI.R** to the Tamilnadu Dr. M.G.R Medical University, Chennai, in partial fulfillment of the requirement for the award of M.S. Degree Branch – II (Obstetrics and Gynaecology) is a bonafide research work carried out by her under direct supervision & guidance.

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This is to certify that **"METABOLIC SYNDROME AND INSULIN RESISTANCE SYNDROME AMONG POLYCYSTIC OVARY SYNDROME"** presented here in by **Dr.SRI SUBBULAKSHMI.R** is an original work done in the Department of Obstetrics and Gynaecology, Tirunelveli Medical College Hospital, Tirunelveli for the award of Degree of M.S. (Branch II) Obstetrics and Gynaecology under my guidance and supervision during the academic period of 2019 -2022.

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DECLARATION

I solemnly declare that the dissertation titled "**METABOLIC SYNDROME AND INSULIN RESISTANCE SYNDROME AMONG POLYCYSTIC OVARY SYNDROME"** is done by me at Tirunelveli Medical College hospital, Tirunelveli. I also declare that this bonafide work or a part of this work was not submitted by me or any others for any award, degree, or diploma to any other University, Board, either in or abroad.

The dissertation is submitted to The Tamilnadu Dr. M.G.R.Medical University towards the partial fulfilment of requirements for the award of M.S. Degree (Branch II) in Obstetrics and Gynaecology.

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Dear Dr. SRI SUBBULAKSHMI.R, The Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during The IEC meeting held on 07.01.2020.

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1.	TIREC Application Form	☐
2.	Study Protocol	☐
3.	Department Research Committee Approval	☐
4.	Patient Information Document and Consent Form in English and Vernacular Language	☐
5.	Investigator's Brochure	☐
6.	Proposed Methods for Patient Accrual Proposed	☐
7.	Curriculum Vitae of the Principal Investigator	☐
8.	Insurance /Compensation Policy	☐
9.	Investigator's Agreement with Sponsor	☐
10.	Investigator's Undertaking	☐
11.	DCGI/DGFT approval	☐
12.	Clinical Trial Agreement (CTA)	☐
13.	Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA)	☐
14.	Clinical Trials Registry-India (CTRI) Registration	☐

THE PROTOCOL IS APPROVED IN ITS PRESENTED FORM ON THE FOLLOWING CONDITIONS

1. The approval is valid for a period of 2 year/s or duration of project whichever is later
2. The date of commencement of study should be informed
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STANDS APPROVED UNDER SEAL

Dr.K.Shantaraman, MD
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CERTIFICATE – II

This is to certify that this dissertation work titled **"METABOLIC SYNDROME AND INSULIN RESISTANCE SYNDROME AMONG POLYCYSTIC OVARY SYNDROME"** of the candidate **Dr.SRI SUBBULAKSHMI.R** with registration Number **221916361** for the award of **M.S.** Degree in the branch of **OBSTETRICS AND GYNAECOLOGY (II).** I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion page and result shows **11% percentage** of plagiarism in the dissertation.

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INTRODUCTION

Polycystic ovarian syndrome (PCOS) is associated with chronic anovulation, insulin resistance and androgen excess. It is considered one of the most common endocrinopathies among reproductive-age women. It affects approximately about 6-10% of reproductive-age women in the USA. Some clinical manifestations of PCOS are oligomenorrhea or amenorrhea, hyperandrogenism, hirsutism and chronic anovulation. Women with this syndrome are at elevated risk of metabolic syndrome (MBS: X syndrome & insulin resistance syndrome). Metabolic syndrome consists of a constellation of metabolic abnormalities that confer an advanced risk of cardiovascular disease and diabetes mellitus[1].

"National Cholesterol Education Programme Adult Treatment Panel III (NCEPATP III) guidelines defines Metabolic sydrome as having 3 or more of the following abnormalities:

1. waist circumference in females > 88 cm,

2. fasting serum glucose level at least 110mg/dl,

3. fasting serum triglycerides level at least 150mg/dl,

4. serum high-density lipoprotein cholesterol (HDL-C) < 50 mg/dl and

5. blood pressure at least 130/85mmHg"[2]. Although insulin vallues are not used to diagnose either PCOS or MBS. However, insulin resistance and

compensatory hyper-insulinemia are the key pathogenic factors in the pathogenesis of these disorders[1]. It seems the prevalence of Metabolic syndrome in PCOS patients is higher than in the general population. USA studies confirmed the prevalence of the MBS in PCOS women (43- 46%) were nearly two-fold higher than that reported for aged-matched women in the general population[3]. Two different analyses which were conducted in Iran country had some controversy. The first study was conducted by Lankarani et al. manifested the criteria for Metabolic syndrome are frequently present in young women with PCOS and are more useful as a prognostic factor than insulin resistance. Meanwhile, they also suggested the evaluation of insulin resistance in older age women with PCOS[4]

Long-term health consequences of the syndrome are currently being investigated. Still, multiple studies indicate that women with the syndrome are at increased risk for the development of glucose intolerance or frank type 2 diabetes mellitus (DM2), hypertension, dyslipidemia [decreased plasma high-density lipoprotein cholesterol (HDL-C) and increased plasma triglycerides], and atherosclerosis. It has been postulated that the insulin resistance of PCOS contributes to these long-term comorbidities. Insulin resistance also appears to play a pathogenic role in metabolic syndrome[5]. The National Cholesterol Education Programme Adult Treatment Panel (NCEP ATP III)[2] guidelines define the MBS as having three or more of the

following abnormalities: waist circumference in females greater than 88 cm; fasting serum glucose at least 110 mg/dl; fasting serum triglycerides at least 150 mg/dl; serum HDL-C less than 50 mg/dl; and blood pressure at least 130/85 mm Hg. The MBS is associated with a heightened risk for developing DM2 and cardiovascular disease[6], as well as with cardiovascular mortality. The NCEP ATP III criteria were used to ascertain the prevalence of the MBS in a representative U.S. adult sample, using data from the Third National Health and Nutrition Examination Survey (NHANES III). In this sample, the prevalence of the MBS among women in age groups 20 –29 and 30 –39 yr was 6 and 15%, respectively[7]. Cardiovascular and DM2 risk factors defining the MBS are prevalent in PCOS[7]. Recently, some studies assessed the prevalence of metabolic abnormalities or MBS in women with PCOS. Korhonen et al.[8] conducted a cross-sectional population-based study and reported that serum concentrations of some sex hormones differed between premenopausal women with and without ATP III-defined MBS. Glueck et al.[3] studied the prevalence of the MBS, using ATP III criteria, in 138 PCOS patients and found it to be 46%. Legro et al.[9] compared metabolic abnormalities and cardiovascular risk factors between women with PCOS and control women and reported them to be more frequent in the former group.

Metabolic syndrome includes central obesity, insulin resistance, hypertension, and atherogenic dyslipidaemia. Understanding metabolic syndrome involves 2 distinct concepts: cardiovascular factors and endocrinological factors, with emphasis on insulin resistance and its sequelae. Polycystic ovary syndrome affects 10–18% of women of reproductive age.1 Insulin resistance appears to be important in the pathogenesis of PCOS and subsequent metabolic syndrome. The prevalence of the metabolic syndrome is as high as 33% in women with PCOS. It is associated with long-term consequences such as cardiovascular disease, diabetes mellitus type II, sleep apnoea, cancers, and psychological problems. Conventionally, management of polycystic ovarian syndrome has focused on infertility, anovulation and hirsutism; thus, there is a need to increase clinicians awareness of the metabolic syndrome. The iniquity of the health burden of metabolic syndrome defines that accurate detection and early intervention are extremely important.

Metabolic syndrome is a combination of risk factors, including abdominal obesity, dyslipidemia, glucose intolerance, and hypertension. The clustering of these factors is often attributed to Gerald Reaven, who popularised the term 'Syndrome X' in 1988. However, these factors have been investigated in various combinations for more than 80 years. The aggregation of these features into a single entity provides clinicians with a

tool by which they can identify a significant segment of the population at increased risk for developing type 2 diabetes mellitus (T2DM) and increased cardiovascular morbidity and mortality. Cardiovascular disease (CVD) affects 42.1 million women over 20 years of age in the US and is the number one cause of mortality in women.

According to the heart disease and Stroke Statistics 2007 Update, 1 in 30 female deaths could be attributed to breast cancer, whereas one in 2.6 women died from CVD. Data have shown that diabetes and hyperglycemia have poorer prognostic implications for CVD mortality in women than men, even after adjusting for age and other CVD risk factors. The aetiology of this differential impact of diabetes based on gender is unclear; however, one explanation could be the high prevalence of metabolic syndrome in female diabetic populations. An analysis of newly diagnosed patients with T2DM revealed that 82.9% met the National Cholesterol Education Program's Adult Treatment Panel III (ATP III) criteria for the metabolic syndrome at the time of T2DM diagnosis, and a significant majority of these were women rather than men (89.9% versus 78.2%; P<0. 001). Studies have associated metabolic syndrome with an increased risk for CVD and have shown that this risk is even greater among women than among men. When examining the ability of ATP-III-defined metabolic syndrome to predict cardiovascular mortality in the San Antonio Heart

Study, hazard ratios in women were 4.65 (95% CI 2.35–9.21) and in men 1.82 (95% CI 1.14–2.91). This gender difference in risk was also observed in the Atherosclerosis Risk in Communities investigation, revealing hazard ratios of 2.05 (95% CI 1.59–2.64) in women and 1.46 (95% CI 1.23–1.74) in men.

AIMS OF THE STUDY:

- To analyse the prevalence of metabolic syndrome in women with polycystic ovarian syndrome.

OBJECTIVE OF THE STUDY

- This study was performed to determine the prevalence and predictors of the metabolic syndrome in PCOS
- To study the pattern and presentation of metabolic syndrome components in women with polycystic ovarian syndrome.

REVIEW OF LITERATURE

Polycystic ovary syndrome, also called PCOS, is one of the most common endocrine system disorders among reproductive-aged women (5–10%) and the leading cause of infertility due to anovulation. PCOS affects women from in-utero life until death, leading to several health risks impairing quality of life and increasing morbidity and mortality rates. This condition includes many different phenotypes that may require additional treatments and may have various consequences, and exhibit a tremendous metabolic complexity, thus needing an urgent revision of its diagnosis.

Definition and epidemiology of PCOS:

PCOS is a complex syndrome with diagnostic criteria that have been grouped in different, somewhat controversial classifications[11,12,13]. According to the features of the syndrome considered, up to 16 phenotypes may exist with other metabolic and reproductive consequences. Some of these phenotypes will be included in the criteria of the commented classifications. The Rotterdam criteria are one of the most commonly used, although they are now over ten years old and not accepted by all[14], with calls to be updated[14].The criteria used to define oligo-anovulation(OA) are insufficient, an adequate definition of biological hyperandrogenism (HA) is yet to be established, and the characterisation of polycystic ovarian morphology (PCOM) proposed in 2003 has become obsolete in

thefaceofthelatestgenerationsofultrasoundmachines[15].Inaddition,these diagnostic criteria are not valid for early and late ages (i.e., for teenagers and aged women). High anti-Müllerian hormone(AMH) serum concentrations have emerged as a valuable marker of PCOS ,although a universally agreed cut-off has not been established[16].In any case, PCOS must always be diagnosed after all other conditions that involve HA or OA have been excluded[17].

The described prevalence of PCOS in women of reproductive age in the general population varies by geographic region, ranging from 1to19% according to population samples analysed in the USA, Western Europe, the Middle East, East Asia, and Australia[18].The varying prevalence of PCOS may be due to genetic and environmental factors. Lower socio-economic development is also associated with poorer health which can lead to hormonal alterations and/ or activate a genetic predisposition for the development of the syndrome. Lack of adequate health care provisionals results in lower rates of correct diagnosis and appropriate treatments[19].

Pathogenesis:

The pathophysiology and mechanism of PCOS appear to be the multifactorial origin and polygenic. The definition of this syndrome has been much debated. There are many extra-ovarian aspects to the pathophysiology of PCOS, yet ovarian dysfunction is central. Chronic anovulation in PCOS results from two main underlying ovarian disorders:

abnormal folliculogenesis and steroidogenesis. Although these two disorders are interlinked, it is difficult to determine the initiating disorder.

Abnormal folliculogenesis

Follicular development normally starts before birth with the daily recruitment of a cohort of those primordial follicles. Under unknown stimulus factors, these primordial follicles are transformed into primary, secondary types and then small antral follicles of 2–5mm diameter. This initial development requires low levels of follicle-stimulating hormone (FSH) and takes about 70– 80 days. Once these follicles reach that stage, they would become FSH-dependent. Without an adequate Follicular stimulating hormone stimulus, these follicles will undergo atresia by default.

At puberty and with the maturation of the hypothalamic-pituitary system, FSH rises to the levels which initiate ovulatory cycles. In the late luteal phase of normally cycling women and with the intercyclical elevation of FSH above a certain threshold, several of these small antral follicles are recruited (i.e., rescued from atresia) and undergo further growth. Once a leading follicle reaches a 9–10 mm diameter, the granulosa cells acquire luteinising hormone (LH) receptors, and further follicular development becomes LH-dependent. The rising oestrogen secretion by the leading follicle will result in a negative-feedback decline of FSH and a positive-feedback increase in LH. As a result, the dominant follicle continues to

mature due to the rising LH level, while all the other follicles undergo atresia due to the fall in FSH.

In PCOS, despite a normal stock number of primordial follicles and a normal early FSH- independent follicular development, follicular growth becomes arrested at the small antral phase, with failure of dominance and escape from the natural process of atresia. This results in an increased number of primary, secondary and small antral follicles (2–8mm in diameter). The mechanism of this disturbed folliculogenesis in PCOS remains largely unknown. Several theories have been postulated to explain the maturation arrest and the escape from atresia of the antral follicles in PCOS. Theories explaining follicular arrest include relative FSH deficiency, abnormal LH stimulus, a deficiency of certain local growth factors and abnormal ovarian steroidogenesis.

The hypothesised relative FSH deficiency may be due to an abnormally increased inhibin B secretion by the increased number of small antral follicles and/or increased ovarian and/or peripheral oestrogen production due to hyperandrogenaemia. An abnormal LH stimulus has also been postulated to explain the premature follicular arrest in PCOS. There is evidence that the granulosa cells of small antral follicles in anovulatory women with PCOS acquire LH receptors prematurely (at a follicular diameter of 4 mm), possibly due to hyperinsulinaemia. LH receptor

acquisition of the granulosa cells seems to switch the follicle from proliferation to differentiation, resulting in a suppression of granulosa cell growth and ultimately inducing an arrest of follicular development and a failure of dominance.

Abnormal steroidogenesis

Normally, the secondary follicle acquires a theca layer characterised by LH receptors and steroidogenic capacity, whereas the granulosa cells contain receptors for FSH. According to the two-cell, two-gonadotrophin model, LH stimulates the theca cells to produce androgens, the precursors for oestrogen synthesis. The androgens then diffuse to the granulosa cells, where FSH stimulates the expression of cytochrome P450 aromatase, which converts the androgens to oestrogens. The rising intraovarian oestrogen and inhibin B concentrations result in negative feedback on FSH secretion and positive feedback on LH secretion. The resulting increase in LH and the rising inhibin B level leads to increased thecal androgen production.

The granulosa cells of the dominant follicle gradually acquire LH receptors, and most of the physiological actions of FSH on granulosa cells can be exerted by LH. In the presence of increasing levels of androgen precursors, the granulosa cells of the dominant follicle, stimulated by the rising LH, continue to produce increasing levels of oestrogens despite

decreasing FSH levels. LH results in an increase in steroidogenesis, early progesterone production and luteinisation. Through a positive-feedback mechanism, progesterone, together with the high oestrogen levels, induces the midcycle LH surge, which results in ovulation.

In PCOS, excess ovarian androgen production appears to be central in the pathogenesis of PCOS. Whether hyperandrogenaemia is the cause or the result of disordered folliculogenesis remains elucidated. Although it is possible that the increased number of small antral follicles produces excess androgens, it is also possible that a genetically determined hypersecretion of androgens is responsible for disordered folliculogenesis. The excess androgen secretion by the theca cells results in increased availability of precursors for oestrogen production in the granulosa cells. In anovulatory women with PCOS, the granulosa cells show LH-induced aromatase activity in the small antral follicles (secondary to hyperinsulinaemia), resulting in enhanced oestrogen production. The elevated levels of circulating oestrogen hormones result in increased positive feedback on LH and negative feedback on FSH secretion, thus causing disordered folliculogenesis, abnormal steroidogenesis and abnormal gonadotropin secretion.

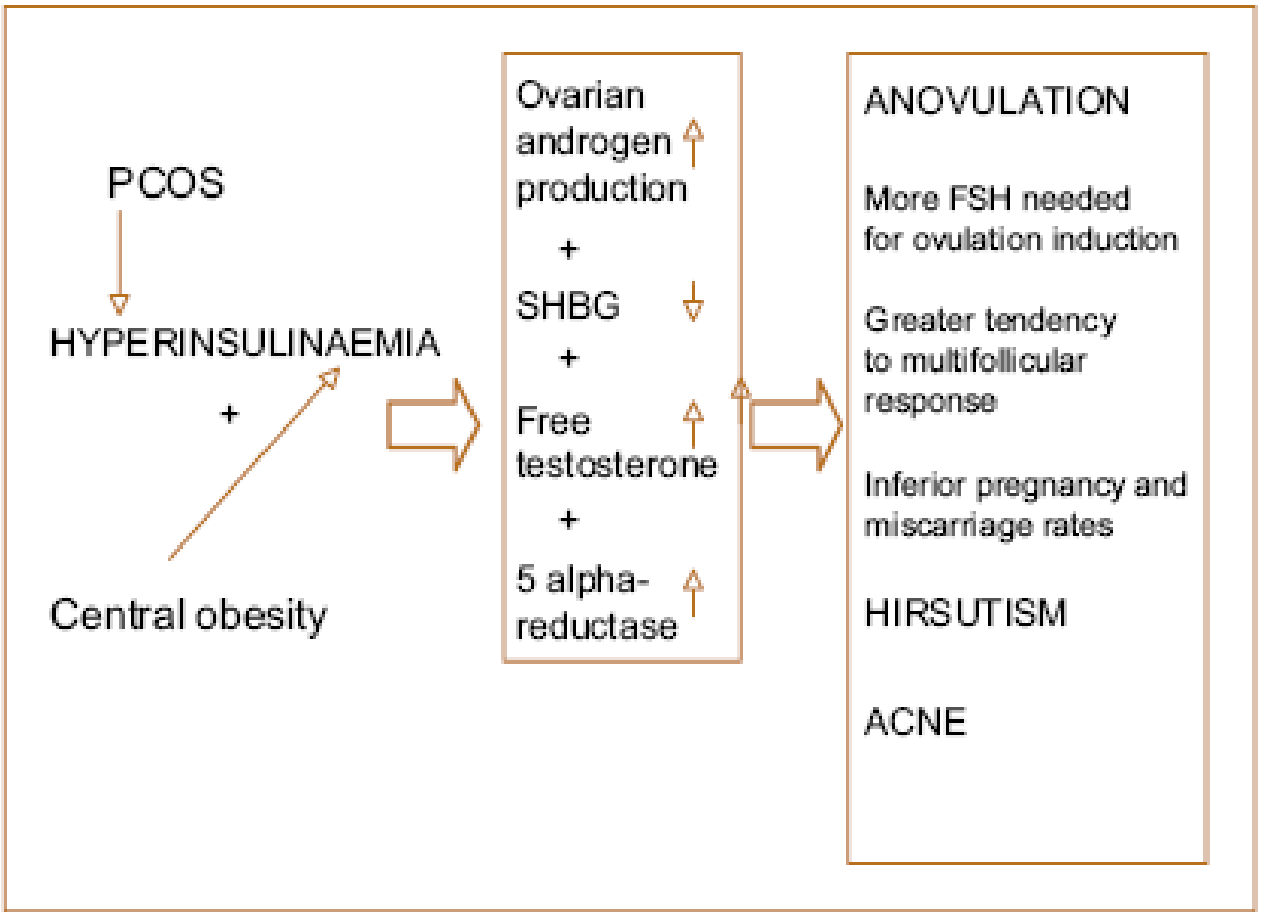

Figure 1: Pathophysiology of PCOS

Clinical features

Anovulatory symptoms Chronic anovulation is very common in women with PCOS and is often associated with menstrual irregularities, which date from the menarche. Most PCOS women present with oligo- or amenorrhoea, although other menstrual disorders such as polymenorrhoea and irregular bleeding can be seen in ~10% of women with PCOS. About 15-20% of women with PCOS have regular menstrual cycles, and some women with menstrual abnormalities may resume regular ovulatory cycles for prolonged periods.

Hyperandrogenic symptoms are common in women with PCOS and are typically mild to moderate. These include hirsutism, acne and alopecia, which have been described in ~70%, 30% and 8% of women with PCOS, respectively. Hirsutism typically starts in the decade between 15 and 25 years and progresses slowly to become noticeable one year from its onset. Virilisation (e.g., clitoromegaly, temporal baldness, deepening of voice or increase in muscle mass) is rare in PCOS and should be investigated to exclude other causes.

Metabolic symptoms Overweight/obesity (body mass index (BMI) > 25 kg/m2) affects ~ 50% of women with PCOS. It is typically characterised by upper-body obesity, defined as a ratio of waist to hip circumference exceeding 0.85. This type of distribution, which is associated with increased insulin resistance, is found even in lean women with PCOS. Acanthosis nigricans is a non-specific cutaneous marker of moderate to severe insulin resistance, which is found in some cases of PCOS and is more common among obese patients. It is characterised by patchy, velvety, hyperpigmented skin changes affecting the neck, axillae, underneath the breasts, body folds, extensor surfaces of the joints and vulva.

Definition of the metabolic syndrome:

Different criteria define the metabolic syndrome by several organisations, including the World Health Organization, the European Group for Study of Insulin Resistance, the ATP III, the American Association of Clinical Endocrinologists,[20] the International Diabetes Foundation,[21] and the recent American Heart Association (AHA) and National Heart, Lung, Blood Institute update of the ATP III criteria. The various definitions exist because of differing opinions on the thresholds that should be used for specific criteria, such as blood pressure or fasting glucose levels. In added part there is a lack of consensus on which components are fundamentally necessary and most clinically relevant for diagnosis. All of the definitions nonetheless include a measure of central obesity, glucose level, dyslipidemia, and hypertension. The focus of management is to achieve a normal level for each of the clinical and laboratory components that constitute the definition.

Because the normal thresholds for some of the components vary by gender, and within each component, there are gender-specific issues, the clinician must consider these issues to successfully prevent or manage the syndrome in women. Because the original ATP III definition has often been employed in epidemiological studies[22], we have chosen to focus this review on the metabolic syndrome defined by these ATP III criteria.

Prevalence of the metabolic syndrome in women:

The metabolic syndrome was diagnosed in 47 million US residents according to US Census data from the year 2000[23]. The age-adjusted prevalence was similar in men (24%) and women (23%); however, this gender equality was lost when comparing ethnic groups. Although there were fewer white women with metabolic syndrome than white men, there are 57% more African American women with the metabolic syndrome than African American men and 26% more Mexican American women with the metabolic syndrome than Mexican American men.18 When comparing National Health and Nutrition Examination Survey (NHANES) data obtained during 1988–1994 to data obtained during 1999–2000, the age-adjusted prevalence of the metabolic syndrome increased by 23.5% among women (P = 0.021) and 2.2% among men (P = 0.831). Therefore, regardless of ethnicity, the similarity in age-adjusted prevalence between men and women will probably be lost with time. Notably, increases in blood pressure, waist circumference, and triglyceride levels were responsible for most of the increased metabolic syndrome prevalence among women[24].

PCOS affects 6–7% of premenopausal women, with estimates varying ethnicity and environmental influences. There are currently no universally accepted criteria for the diagnosis of PCOS. Still, most definitions include

the presence of oligoovulation, hyperandrogenism, hyperandrogenemia, and the exclusion of other medical disorders that might have similar presentations[25]. PCOS has many characteristics similar to those of metabolic syndrome. The prevalence rate of metabolic syndrome in PCOS is approximately 43–47%, twice as high as the prevalence in the age-matched general population, even after adjusting for BMI[26]. Although there are nonobese women with biochemical and ultrasonographic features of PCOS, obesity is a key component of the pathology associated with PCOS[27].

The components of the metabolic syndrome most commonly present in PCOS are central obesity and low serum HDL levels; however, hypertension, increased fasting glucose levels, and impaired glucose tolerance are also commonly present[26]. PCOS has been associated with an increased prevalence of dyslipidemia, which is heritable among the sisters of women with PCOS[28]. Moreover, there is an increased prevalence of metabolic syndrome in sisters of women with PCOS who manifested the PCOS phenotype compared with the unaffected sisters (P<0. .001)[28]. Additionally, women with PCOS have a higher risk of diabetes than women of similar BMI with regular menses. Finally, data support an increased risk of BMI-independent hypertension and an increased risk of CVD in women with PCOS compared with women who have regular menstrual cycles, but these findings are controversial.

Diagnosticcriteriaforpolycysticovariansyndrome:

National Institutes of Health (NIH) 1990[11]	**Rotterdam 2003[29]**	**AE-PCOS Society 2006[13]**	**NIH 2012/International PCOS Guidelines 2018[14,15]**
▪ Hyperandrogenism ▪ Chronic Anovulation —Both criteria needed	▪ Hyperandrogenism ▪ Oligo-and/or anovulation ▪ Polycystic ovaries —2 of 3 criteria needed	• Hyperandrogenism • Ovarian dysfunction —Both criteria needed	• Hyperandrogenism Oligo-and/or anovulation ▪ Polycystic ovaries —2 of 3 criteria needed
Most commonly used criteria today	Formulated to expand on NIH diagnostic description	Formulated to provide an evidence-based description	Encouraged a name change (2012 only) and identified sub-phenotypes

Metabolic syndrome in PCOS:

Pathogenesis

Intense research and analytical studies have focused on disentangling the evolution of metabolic syndrome in PCOS. Multiple mechanisms has been proposed earlier. Complex systematic interactions inbetween the various features of metabolic syndrome mean that explaining their underlying pathophysiology is essential.

Insulin resistance

IR and its consequent hyperinsulinaemia are central to the pathogenesis of PCOS. Insulin which regulates metabolic and mitogenic pathways that function independently of each other[32]. This may explain the paradoxical insulin sensitivity aspects seen in various tissues, for example, resistance in peripheral tissues and maintained sensitivity in the ovarian cortex. Metabolic inertia to insulin had been attributed to a post-binding defect in the insulin signalling pathway caused by abnormal serine phosphorylation of the insulin receptor. Despite extensive data which are supporting the role of Insulin resistence , it remains unclear whether the relation is causal, propagative in aspect or merely co-existential.

Atherogenic dyslipidaemia

In adipocytes, Insulin resistance leads to the raised shunting of free fatty acids from fat tissue to the liver. FFAs induce hepatic synthesis of very-low-density lipoprotein (VLDL), resulting in raised triglycerides and apolipoprotein B & decreased HDL. These alterations in lipid parameter levels lead to cause of atherogenic dyslipidaemia. PCOS is related with chronic low-grade systemic inflammation, which mediates insulin resistance and stimulates atherogenesis. Besides abnormalities in genetic system , obesity and a high glycaemic diet also induce pro-inflammatory

cytokines such as tumour necrosis factor-alpha (TNFa) & interleukin-6 (IL-6)[33].

Obesity

Obesity is known to precipitate PCOS. It most likely happens from the merged effect of genetic predisposition, poor diet and a sedentary lifestyle, thus stipulating pre-existing metabolic derangements. Hyperinsulinaemia, and the raised responsiveness of the ovarian theca to insulin, causes an raise in the levels of free androgens. Hyperandrogenaemia elevates a person's predisposition for central adiposity and worsens insulin resistance & dyslipidaemia[34].

Hypertension

In obese patients, hypertension is linked to the potentiation of sympathetic outflow and the renin-angiotensin-aldosterone system, resulting from elevated insulin levels and free fatty acids. Concomitant vascular endothelial dysregulation also contributes to the development of hypertension[35]. Studying all available data on the etio-pathogenesis of PCOS, it is apparent that a paradigm shift has taken place in our acquaintance of metabolic syndrome in PCOS, from sheer ovarian dysfunction to a multisystemic, multifactorial abnormality with far more prominent metabolic consequences than initially implicated.

Consequences of Metabolic syndrome in PCOS: Pondy study

Cardiovascular disease

One meta-analysis has demonstrated that in women individual with PCOS, the risk of coronary heart disease and stroke is apparently doubled. Inspite adjusting for body mass index (BMI), there was an 55 percent increase in the risk[36]. Subjects with metabolic syndrome are 3-6 times more likely to develop CHD, with a 12% increase in mortality[6]. Where metabolic syndrome co-exists with PCOS, one will expect this risk to be importantly higher. Large-scale prospective studies that estimate long-term outcomes which are required to evaluate the magnitude of the impactness of metabolic syndrome with PCOS on cardiovascular system events.

Diabetes

Metabolic syndrome has a 5-fold times increase in risk for diabetes type II, and PCOS has been identified as a significant non-modifiable risk factor[37]. Women with PCOS with baseline normo-glycaemia have a high pronounced risk of developing impaired glucose tolerance (IGT); up to 16% of women with PCOS had been converted to IGT per yr. Women with the baseline IGT have an 2% risk of advancing to diabetes type II per year, and over 6 years, this risk may be as high as about 54%[38]. A meta-analysis on the impactness of PCOS on reproductive system outcomes has shown a 3-fold upsurge in risk for gestational diabetes[10].

Cancers

PCOS is related with an elevated likelihood of endometrial cancer (odds ratio [OR] 2.89)[40]. However, it is much difficult to establish the independent effect of PCOS because of the additive influence of hypertension, diabetes, obesity, chronic anovulation and hyper-estrogenaemia on the pathogenesis of endometrial cancer. There is no strong relation between PCOS and ovarian or breast cancer[41]. Metabolic syndrome has been related with an elevated risk of endometrial cancer (OR 1.6) and a higher incidence rate of pancreatic, post-menopausal breast and colorectal cancers (OR 1.5). Worse cancer outcomes, elevated recurrences and overall mortality have also been linked to metabolic syndrome[42].

Obstructive sleep apnoea

PCOS is related with up to a 30-fold greater risk of obstructive sleep apnoea (OSA) and a 9-fold raise in excessive daytime sleepiness. Insulin resistance has emerged as a principal predictor for OSA risk, independent of elevated testosterone levels and obesity[43]. The existence of OSA in metabolic syndrome, in addition, worsens Insulin resistance and outcomes in cardiovascular systems . Women should be screened for and educated about snoring, daytime tiredness and fatigue, and interventions when necessary.

Psychological problems

The prevalence of depression is greater in women with PCOS (OR 4.03) than the general population, with increased severity of symptoms[44]. This association is independent of BMI. These women are at greater risks to develop anxiety, eating disorders and dysfunctional relationships. Metabolic syndrome is related with depression, mainly with neuro-vegetative features such as fatigue. Although inflammation has been correlated to the development of depressive symptoms, the accurate mechanisms have yet to be elucidated[45]. Since these factors significantly affect the quality of life, it is essential to screen women for symptoms and refer them to a specialist.

Diagnostic criteria of metabolic syndrome:

Metabolic syndrome was first detected as a clinical entity with insulin resistance central to its pathogenesis. Thus, historically, the explanation of metabolic syndrome mandated the presence of Insulin resistance for its diagnosis. Subsequent explamtions included more composite criteria, for example, waist circumference as a measurement of central obesity. Multiple explanations for metabolic syndrome have been proposed over the years, reflecting a shift in the proposed pathogenetic mechanisms and clinical implications.

Table1.Diagnosticcriteriaformetabolicsyndrome

Measure	Categoricalcut-off points
1. Elevatedwaistcircumference	≥88cm ≥80cm
2. Elevatedtriglycerides	≥150mg/dl(1.7mmol/l),orhavingtreatment
3. ReducedHDL-Clevels	<50mg/dl(1.3mmol/l),orhavingtreatment
4. ElevatedBP	SystolicBP≥130 / diastolicBP≥85mmHg,ormanagementofpreviouslydiagnosedsystemic hypertension
5. Elevatedfastingglucoselevels	≥100mgs/dl(5.6mmol/l) ≥110mgs/dl(6.1mmol/l)orhavingtreatment

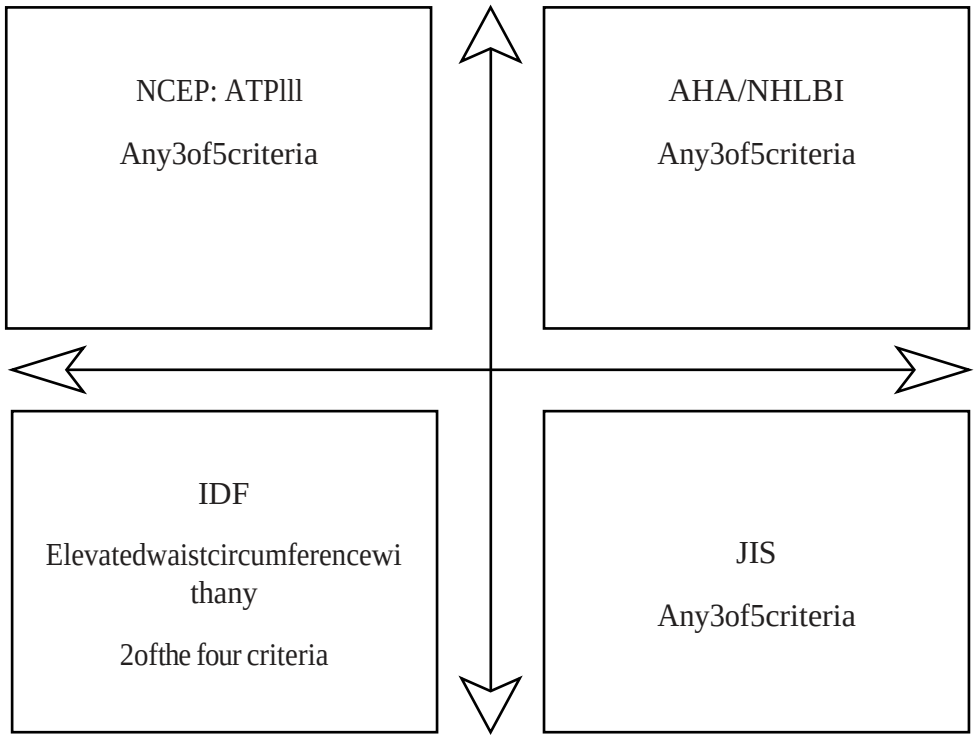

Figure1.Diagnosisofmetabolicsyndrome.

Key: NCEP: ATP III = National Cholesterol Education Programme Adult Treatment Panel III;[46] AHA/NHLBI = the American Heart Association/National Heart, Lung, and Blood Institute;[47] IDF = International Diabetes Federation;[48]JIS=Joint Interim Statement[49]

The description of metabolic syndrome proposed by the National Cholesterol Education Programme Adult Treatment Panel III (NCEP: ATP III)[46] does not build on any preconceived notion about metabolic syndrome's underlying cause(s), whether insulin resistance or obesity. In the yr 2005, the American Heart Association/National Heart, Lung, and

Blood Institute (AHA/NHLBI) statement[47] incorporated a minor modification to that of the NCEP: ATP III by revising the fasting blood glucose level cut-off to 5.6 mmol/l. In the USA, the NCEP: ATP III has recommended 88 cms as a cut-off for waist circumference in individual women. This is in contrary to the International Diabetes Federation (IDF),[48], which focuses on the impact of ethnic variations on the threshold for abdominal obesity and makes it obligatory to the description. Data analysed by the IDF has supported that waist circumference of 80 cm and higher in women across different ethnicities. However, the Joint Interim Statement (JIS),[49], highlighted the significance of ethnicity-specific waist measurements, with same emphasis placed on the patients risk-predicting factors. By unifying the diagnostic factor of metabolic syndrome, the JIS simplifies its utility as a clinical tool and which has emerged as one of the most commonly used explanations. A large cross-sectional analysis of the various diagnostic criteria in a Greek population (n = 9669). In this study, the JIS has detected a very high prevalence rate of metabolic syndrome (nearly half of the adults). Still, the AHA/NHLBI definition emerged as a better predictor of risk for cardiovascular events[50]. Consensus on same diagnostic criteria is much essential because the choice of metabolic syndrome description might affect identifying individuals at risk and the management of consequent comorbidities.

Screening for metabolic syndrome

A structured assessment for the early finding and management of metabolic syndrome, especially in individual women of reproductive age, will be of critical significance to the healthcare system. So that it could be applied in the clinical setting, this evaluation would need to majorly prioritise risks, standardise assessment methods, and establish the frequency of further testing. A comprehensive finding of the risk factors for metabolic syndrome, acclimated from evidence-based guidelines from Australia and the UK's Royal College of Obstetricians and Gynaecologists[51].

Table2.Recommendationsforscreening of metabolic syndrome risk factor in women with PCOS Cigarette smoking

Screening parameters	Frequency of assessment
Obesity (weight, BMI, waist circumference) Blood pressure	At every visit, obtain the history of recent smoking habits, if any, or cessation. At every visit
Complete lipid profile Oral glucose tolerance test(75g)	For women with a value BMI<25kg/m^2:annually For women with a value of BMI≥25kg/m^2:ateveryvisitForwo menwithanormalprofile:everytwo years For women with an abnormal profile or excess weight: annually All women: every two years Women with risk factors (age >40 years, hypertension, ethnicity, waist circumference (>80 cm), physical inactivity, smoking, BMI≥25kg/m^2,previous gestational diabetes mellitus, family history of diabetes mellitus):annually

Key:BMI=bodymas sindex

Management: pondy

With metabolic syndrome adding to the burgeoning diabetes type II and CVD epidemic, instituting early and targeted interventions is a medical necessity. Primary intervention for metabolic syndrome needs to adopting lifestyle modifications to prevent or slow progression to side effects events in higher risk individuals. In those who don't respond sufficiently to lifestyle modifications, secondary interventions include drug therapy & bariatric surgery.

Lifestyle modifications

Exercise and dietary regulation are significant components of a healthy lifestyle. Though there is no well established cure for PCOS, lifestyle modificatons have demonstrated substantial advancements in symptoms. It is recommended that individual women with PCOS aim to reduce 5–10% of their body weight in the 1st year after diagnosis for improved clinical outcomes[21,51]. Reduced body weight is associated with a reduction in metabolically active visceral fat, which leads to decreased insulin resistance and an optimised lipid profile. It may have psychological benefits such as decreased anxiety and depression. The heterogeneity of analytical designs means that the optimal duration, frequency and type of exercise is difficult to establish based on currently available data[52]. To improve certain cardio metabolic outcomes, women with PCOS are

recommended to undertake regular exercise for 150 minutes per week, including at least 90 minutes of moderate-intensity aerobic activity[51]. Several elements can be optimised in dietary goals: the quantity, quality, and spacing of meals. In obese women with PCOS, incorporating carbohydrate foods with a low glycaemic index (GI) has demonstrated significant improvement in insulin sensitivity[53]. However, low-GI foods aren't necessarily high in nutritional value. Dietary fibre is the best indigestible part of food that causes satiety, reduces cholesterol and slows the absorption of carbohydrates. Proteins may take longer to digest than carbohydrate foods. Hence they could improve the insulin profile value. Monounsaturated fatty acids improve cholesterol values and glucose levels, and insulin response. Very long-chain PUFA, including omega-3 & -6 fats, have a hypotriglyceridaemic effect and may lessen inflammation in metabolic syndrome[54]. Major omega-3 fats include eicosapentaenoic acid, docosahexaenoic acid and alpha-linolenic acid. Adequate omega-3 fat ingestion is important in PCOS; benefits include lower inflammatory markers, cholesterol and triglycerides and increased insulin sensitivity[55]. To maximise the healthy benefits of a diet, it is also very important to adopt healthy eating patterns, for example, taking small portions of calorie-appropriate meals at frequent intervals[56].

Medical management

Insulin-sensitising agents like

Metformin

Metformin corrects insulin sensitivity in women with PCOS.[37] It has also been shown to reduce fasting insulin levels, but this benefit was restricted to non obese women with PCOS (BMI <30 kg/m^2)[58]. There is no sustainable evidence for the use of metformin to merely ameliorate insulin resistance associated with PCOS, and any international organisation does not recommend its use to combat the same in normoglycaemic women

.

Women with Impaired glucose tolerance have a significant risk of conversion to diabetes mellitus type ll. Metformin can be considered for usage in women with IGT to prevent its progression to diabetes mellitus type II, although this may require further scientific validation. Metformin has been used in incremental doses which ranges from 500 to1500 mgs/day. There is no consensus on the period of treatment required. It is commonly associated with adverse gastrointestinal effects, including nausea, abdominal pain and diarrhoea. A few trials have demonstrated that the use of metformin can lead to significant weight loss, especially when combined with lifestyle modifications. Still, there is insufficient evidence to recommend its use for weight loss purposes[59].

Inositol

Inositols are compounds with insulin-mimeticproperties, particularly myoinositol(MI) and d-chiro-inositol(DCI). MI and DCI were involved in downstream regulation of signaling pathways following the activation of insulin specific receptors and are considered as mediators of insulin action[60].Various analysis have demonstrated that administration of DCI leads to decreased basal insulin levels, an improved lipid profile and reduced systolic blood pressure[61]. The clinical effectiveness of MI and DCI had been analysed in assortment and asst and alone interventions, with MI being administered at doses ranging from 1 - 4 g/day. No appreciable side effects have ever been recorded with following the administration of inositols[62]. Preliminary data suggest that supplementing with inositol could be considered for enhancing a patient's metabolic profile, but more studies are required before its use can be regularised.

Anti-obesity agents

Orlistat

Orlistat, rimonabant and sibutramine have been used to treat obesity in PCOS. Sibutramine & rimonabant agents have been withdrawn over safety concerns. Orlistat agent is an irreversible gastric lipase inhibitor that controls the breakdown of dietary fat and, thus, its absorption. In

PCOS, orlistat induces important and sustainable weight loss with same efficacy to metformin dose. Its use is also related with an improved fasting lipid profile, including considerable reductions in total cholesterol, LDL and TGs. Based on the available evidence, orlistat agent can be considered for treating overweight and obese women with PCOS for whom lifestyle changes are insufficient.[45] Patients take 60–360 mg dosage of or list at per day, divided between 2 - 3 doses. Its usage is associated with mild to moderate GI sideffects, including steatorrhoea and abdominal pain, potentially affecting treatment

compliancedomised study on the effect of orlistat on obese women with metabolic syndrome demonstrated reversal of metabolic syndrome in 43.5% of participants, with significant associated improvements in anthropometry, insulin resistance, lipid profile and blood pressure[63]. The risk of consequence of IGT to diabetes mellitus type II was also remarkably reduced (by37.3%)[64]. Extending this usable effect to cardiovascular risk, or list at agent might have arose in managing the metabolic syndrome. However, further analysis are required to assess the use of orlistat in women with PCOS and metabolic syndrome before it can be approved for therapeutic indications.

Liraglutide

Glucagon-like peptide receptor agonists(GLP-1R) are approved anti-obesity agents, including exenatide and liraglutide. The use of liraglutide

for obesity in PCOS-related metabolic syndrome remains limited. Administration of liraglutide agent is related with a significant decrease in BMI (–1.65 kg/m^2), but not in value of waist: hip ratio, which is a more sensitive indicator of visceral obesity and metabolic outcomes. No significantly positive effect have been shown on fasting insulin levels and insulin resistance [65]. The use of liraglutide agent is being limited by the need for intra venous administration and the frequent occurrence of GI adverse effects. Larger analyses were required to establish the efficacy of GLP-1R agonists agents in women with PCOS and metabolic syndrome.

Statins

In women with dyslipidaemia, statins results insignificantly and consistently improved lipid profiles. Further more, reduced levels of endothelial dysfunction and systemic inflammation suggest a decrease in cardiovascular risk factors[66]. Although statins seem to produce a favourable metabolic environment, certain risks limit their use. Long-term use can cause liver dysfunction. When prescribed with other drugs, it can lead to serious adverse effects, including teratogenicity, which is critical among women of reproductive age. Based on available outcomes, the routine clinical usage of statins could not be recommended for those women with PCOS.

Although there appear to be multiple certain options for the

pharmacological modification of metabolic derangements in women with PCOS, but no drug is currently recommended as a standard management line. The apparent benefits of medical management must be weighed against the risk of potential adverse effects of prolonged treatment.

Surgical management

Difficulty sustaining lifestyle alterations and a lack of robust evidence supporting pharmacological interventions have spurred these arch for newer modalities to manage metabolic syndrome in women with PCOS. Other management strategies have failed; bariatric surgery might be considered to those individuals with Classlll obesity (BMI ≥40 kg/m^2). Bariatric surgery might also be considered in women with Class II obesity (BMI 35–39.9 kg/m^2) and also associated chronic medical illness such as diabetes and hypertension. Women undergoing bariatric procedure have demonstrated reduced cardiometabolic risk factors, reflected by an improved lipid profile and reduced insulin resistance[67].Common bariatric surgeries include laparoscopically adjustable gastric banding, vertical banded gastroplasty and Roux-en-Y gastric bypass. These procedures are beneficial in reducing cardiovascular events, inducing remission of diabetes type II, hypertension, obstructive sleep apnoea and subsidence of metabolic syndrome[68].

A meta-analysis of women with PCOS have showed a significant reduction in the incidence of PCOS after bariatric surgery (from 45.6% to 6.8% at 12 months)[69].Bariatric surgery might also decrease the incidence rate of obesity-relatedcancers[70].Although studies involving women with PCOS were limited, the outcomes are encouraging. Further well-designed analyses are required before bariatric surgery can form part of the mainstream management of metabolic syndrome in women with PCOS.

Review of studies:

In 2004, Apridonidze T et al. [71] conducted a retrospective chart review of all women with PCOS illness seen over a 3-year duration an endocrinology clinic. Of the 161 PCOS patients reviewed, 106 met this inclusion criteria. The women were divided into 2 groups: 1) women with PCOS and the MBS (n = 46); and 2) women with PCOS who are lacking the MBS (n = 60). Prevalence of the MBS was about 43%, nearly 2-fold greater than that reported for age-matched women in the general population. Women with PCOS had consistently greater prevalence rates of the MBS than women in the public population, regardless of matched age and BMI ranges. Acanthosis nigricans is more common in women with PCOS and the MBS. Women with PCOS and the MBS had remarkably higher levels of serum free testosterone ($P < 0.002$) and lower levels of serum SHBG ($P < 0.001$) than women with PCOS without the

MBS. No differences seen in total testosterone were observed between the groups. They conclude that the MBS and its components are common in women with PCOS, placing them at increased risk for cardiovascular disease. Women with PCOS and the MBS differ from their counterparts lacking the MBS in terms of increased hyperandrogenemia, lower serum SHBG, and higher prevalence of acanthosis nigricans, all features that may reflect more severe insulin resistance.

In 2006, Ehrmann et al. [72] conducted a multicenter clinical trial. The subjects were women with PCOS who had or lacked metabolic syndrome. The main outcome measures were waist circumference, fasting glucose, high-density lipoprotein cholesterol and triglyceride concentrations, and blood pressure. Twenty-six (6.6%) subjects had diabetes; among the 368 nondiabetics, the prevalence for individual components comprising the metabolic syndrome was: waist circumference greater than 88 cm in 80%, high-density lipoprotein cholesterol less than 50 mg/dl in 66%, triglycerides greater than or equal to 150 mg/dl in 32%, blood pressure greater than or equal to 130/85 mm Hg in 21%, and fasting glucose concentrations greater than or equal to 110 mg/dl in 5%. Three or more of these individual criteria were present in 123 (33.4%) subjects overall. The prevalence of metabolic syndrome did not differ significantly between racial/ethnic groups. The prevalence of the metabolic syndrome from

lowest to the highest quartile of free testosterone concentration was 19.8, 31.3, 46.9, and 35.0%, respectively [P = 0.056 adjusted for body mass index (BMI)]. None of the 52 women with a BMI less than 27.0 kg/m2 had the metabolic syndrome; those in the top BMI quartile were 13.7 times more likely (95% confidence interval, 5.7–33.0) to have the metabolic syndrome with those in the lowest quartile. Thirty-eight per cent of those with the metabolic syndrome had impaired glucose tolerance compared with 19% without the metabolic syndrome (P < 0.001). This study concluded that metabolic syndrome and its components are common in PCOS, particularly among women with the highest insulin levels and BMI. Hyperinsulinemia is a likely common pathogenetic factor for both PCOS and metabolic syndrome.

In 2006, Marcondes et al. [73] aimed to determine the prevalence of metabolic syndrome in women with polycystic ovary syndrome and its characteristics and predictors. Seventh-three women with a body mass index of 30.4 ± 7.8 kg/m2 and 25.0 ± 6.0 yrs old, subdivided according to BMI, were studied retrospectively. There was no significant mean age difference among BMI groups (p = 0.228). Prevalence of MBS was 38.4%, with a null prevalence for normal (n = 18), 23.8% for overweight (n = 17), 62.9% for obese patients (n = 28), and 85.5% for morbidly obese women (n = 7). Women with MBS were older than women without MBS

(27.3 ± 5.3 vs 24.2 ± 4.6 vs years old; p = 0.031) and presented a higher body mass index (36.3 ± 7.7 vs 26.9 ± 5.4; p < 0.001). There was no spcific difference in the degree of hirsutism and menstrual patterns between women with and without metabolic syndrome (p = 0.593 and p = 0.119, respectively). Regarding laboratory parameters, DHEAS was lower (1,646 ± 1,007 vs. 2,594 ± 1,563; p = 0.007) and HOMA-IR were higher (9.9 ± 9.7 vs. 4.6 ± 4.7; p = 0.004) in women with metabolic syndrome (p = 0.031 and p < 0.001, respectively). The best predictors of metabolic syndrome were waist circumference > 88 cm, HDL-cholesterol < 50 mg/dL and triglycerides ≥ 150 mg/dL

In 2008, Najem et al. [74] conducted a retrospective analysis of patient records at the endocrine clinic in Benghazi was undertaken. Patient inclusion was according to Rotterdam ESHRE/ASRM criteria. Clinical features, associated diseases, family history, hormone levels, and ultrasonography results were analysed. The mean age of the 318 PCOS patients at presentation was 25.8 years (range 15-44 years), and the majority (67%) were 20-29 years old at presentation. Of all patients, 57% were obese (BMI > 30), 93% had oligo- / amenorrhea, 91% were hirsute, and 74% had ultrasound features of polycystic ovaries. Diabetes mellitus was diagnosed in 9% of all PCOS patients and hypertension in 4%. Total serum testosterone was elevated in 26% of the patients, and serum

prolactin was elevated in 31%. Thyroid disease was noted among 5.3% of the patients, and a history of diabetes or hypertension among first-degree relatives was seen in (16%) and (8%) of the patients, respectively. This study concluded that Chronic anovulation and hirsutism are the dominant features of PCOS in our patient population. More than half were obese, and the prevalence of diabetes, hypertension and thyroid disease in our patients seemed to be underestimated compared to other parts of the world.

In 2011, Moini A et al. [75] conducted a cross-sectional study in Gynecologic Clinic at Arash Hospital affiliated with Tehran University. Two hundred eighty-two women with PCOS ages between 15-40 years were included. The prevalence of MBS and its components in this population were the main outcomes. Height, weight, waist circumference, blood pressure and laboratory tests (FBS, TSH, HDL-C, serum prolactin, triglycerides and total cholesterol) were measured in this population. Results: The prevalence of MBS in PCOS women was 22.7% (64 cases). The rate of central obesity, FBS more than 110 mg/dl, triglycerides more than 150 mg/dl, high-density lipoprotein cholesterol levels (HDL-C) less than 50 mg/dl, and blood pressure ≥130/85 mmHg in PCOS women was 31% (87), 3.2% (9), 33% (93), 68.8% (194), and 10.6% (30), respectively. MBS risk was increased in older and obese women (BMI ≥30 kg/m^2).

They concluded that the present sample showed women with PCOS have a high prevalence of MBS, and its components particularly decreased HDL-C.

In 2012, Ishak et al.[76] conducted a cross-sectional study among 99 PCOS patients in Obstetrics and Gynaecology Clinics in two tertiary centres in the east coast of Peninsular Malaysia, from the period May 2008 to May 2010. Socio-demographic data, weight, waist circumference, height and blood pressure were documented. A fasting blood sample were obtained for serum glucose and lipid profile determination. Metabolic syndrome was defined following the International Diabetic Federation (IDF) 2005. The prevalence of MBS was 43.4% (N=43). Age and a family history of diabetes mellitus were significantly associated with MS. (OR=1.11, 95% CI: 1.10, 1.22) and (OR=3.07, 95% CI: 1.22-7.70), respectively. This study concluded that metabolic syndrome among PCOS patients was high. Age and a family history of diabetes strongly predicted metabolic syndrome amongst PCOS patients.

In 2012, Tabrizi et al.[77] conducted a cross-sectional study and evaluated 200 women with PCOS. PCOS was diagnosed according to Rotterdam criteria. This study defined clinical and biochemical parameters for MetS by the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) criteria. Statistical analyses were performed with

descriptive-analytical methods using SPSS software version 16. Metabolic syndrome was identified in 39.5% of PCOS women. The frequencies of individual components of metabolic syndrome among studied subjects were: high-density lipoprotein cholesterol level (HDL-C)<50 mg/ dL (99.5%), waist circumference(WC) ≥88cm (65%), triglycerides (TG) ≥150 mg/dL(98%), and blood pressure≥130/85 mmHg(34%).There were no fasting glucose concentrations≥110 mg/dL. The frequency of metabolic syndrome increased with body mass index (BMI)as follows: normal (5.4%), overweight (41.5%) and obese (85.7%) women (p<0.0001). This study further concluded that PCOS women had a high frequency of metabolic syndrome and its components, particularly decreased HDL-C and increased triglyceride levels. These data can be useful for lifestyle modification programs.

In 2014, Kim MJ et al.[78] conducted a cross-sectional observational study in Korea from May 2010 to December 2011. A total of 837 females with PCOS, aged 15–40, were recruited from Departments of Obstetrics and Gynecology at 13 hospitals. Of those, 700 subjects with either polycystic ovaries (PCO)+HA+oligomenorrhea/amenorrhea (O) or PCO+O were eligible for this study. Metabolic syndrome was found according to the modified National Cholesterol Education Program (NCEP) Adult Treatment Panel (ATP) III guidelines and the International Diabetes

Federation (IDF) criteria. Metabolic syndrome was more prevalent in the PCO+HA+O group (19.7%) than in the PCO+O (11.9%) group. There were statistically significant trends for an increased risk of Metabolic syndrome in the PCO+HA+O group compared to the PCO+O group. After adjustment for age, the odds ratio of Metabolic syndrome was 2.192 in nonobese subjects with PCO+HA+O compared to those with PCO+ O, whereas the risk of MetS was not different in obese women. Multivariate logistic regression analysis showed that high level of free androgen index and low sex hormone-binding globulin were significantly associated with Metabolic syndrome in nonobese women with PCOS, with odds ratios of 4.234 (95% CI, 1.893–9.474) and 4.612 (95% CI, 1.978–10.750), respectively. However, there were no associations between Metabolic syndrome and SHBG and FAI in obese PCOS subjects. Their results indicate that HA and its associated parameters (FAI and SHBG) are significantly associated with Metabolic syndrome in nonobese PCOS subjects, whereas this association was not observed in obese subjects.

In 2017,Liang P et al.[79] studied 299 metabolically unhealthy obese and 122 metabolically healthy obese Chinese women matched on body mass index. Metabolically healthy obese was defined as obesity with no more than one metabolic abnormality. Diagnosis of PCOS was based on the revised Rotterdam criteria. Intervention(s): Each subject underwent

physical examination, laboratory evaluation, and gynecologic ultrasound to diagnose PCOS or metabolic syndrome. The prevalence of PCOS was calculated in both groups. Insulin resistance was determined by homeostasis model assessment of insulin resistance or the insulin sensitivity index derived from Bergman's minimal model. Fat distribution was measured with a computerised tomography scan. This study noted that the prevalence of PCOS and its components did not differ between metabolically unhealthy obese and BMI-matched metabolically healthy obese groups (67.89% and 66.96%, respectively). In logistic regression analysis, Metabolic syndrome did not predict the presence of PCOS after adjusting for confounding factors. The metabolically healthy obese group had lower visceral adipose tissue, relatively higher insulin sensitivity, and better b-cell function compared with those in the metabolically unhealthy obese group. Still, there were no significant differences in sex hormones (except for free T and sex hormone-binding globulin) and ultrasound manifestations between metabolically healthy obese and metabolically unhealthy obese women. Our findings suggest that metabolic syndrome does not add additional risk for PCOS for the first time. In addition, we found that both metabolically unhealthy obese and metabolically healthy obese are associated with insulin resistance to some extent.

In 2017, Dargham et al. [80] conducted a study among 720 women with testosterone and sex hormone-binding globulin (SHBG) measurements.

PCOS was diagnosed according to the National Institute of Health (NIH) Guidelines of a raised androgen level (free androgen index >4.5 or a raised total testosterone) and menstrual irregularity after the exclusion of other conditions. All results were reported as mean values of PCOS versus control. 87 of 720 women fulfilled the NIH guidelines (12.1%) for PCOS, specifically using a free androgen index greater than 4.5 or total testosterone greater than 2.7nmol/l and menstrual irregularity. Subjects were heavier with a more metabolic profile of a greater systolic and diastolic blood pressure, higher C reactive protein levels, and insulin ($p<0.05$). This study concluded that by NIH guidelines, the prevalence of PCOS in this Qatari cohort was 12.1%, likely reflecting 20% by Rotterdam criteria, with a markedly more metabolic phenotype than Qatari controls.

In 2018, Sunita M Aghade and Jayshree S Bavikar[81] conducted a cross-sectional study that included 150 women diagnosed with PCOS between 18- and 38-years of age. Demographic variables including age, education, occupation, inhabitant area, history of infertility, and family history of diabetes mellitus and hypertension were collected. Anthropometric parameters like weight, height, body mass index (BMI), waist circumference (WC), and systolic/diastolic blood pressure (SBP/ DBP) were measured. Fasting venous blood samples are collected and analysed for biochemical parameters like glucose, total cholesterol (TC),

triglycerides (TG), and high-density lipoprotein (HDL) cholesterol. The prevalence of metabolic syndrome in women with PCOS was 38.67%. The most prevalent component was decreased HDL (84.67%), followed by increased WC (75.33%), followed by raised TG (42%). This study concluded that the analogy of PCOS with metabolic syndrome implicates that it is crucial to analyse the emerging trend of metabolic syndrome in patients with PCOS. Recognition of this high-risk group will aid in the enforcement of preventive strategies, including therapeutic lifestyle modifications and risk factor management. This will positively impact women's health and prevent or delay the outset of varying cardiometabolic complications in PCOS.

In 2019, Somayeh Haghi Karamallah et al[82]. According to the Rotterdam criteria, 62 women were diagnosed with PCOS. Metabolic syndrome was assessed according to the American National Cholesterol Panel (ATP-III criteria). The prevalence of metabolic syndrome was studied by measuring the following items: fasting glucose, triglyceride, HDL cholesterol, blood pressure, weight, height and waist circumference. We considered the patients a metabolic syndrome group with three or more cases. Based on this research, the prevalence of metabolic syndrome in Ahwaz women was estimated at 14.6%, the prevalence for individual components comprising the metabolic syndrome was: fasting glucose concentrations greater than or

equal to 110 mg/dl in 11 patients (17.7%), hypertension in 9 patients (14.5%), BMI higher or equals to 30 in 14 patients (22.6%), a waist circumference greater than or equals to 88 cm in 22 patients (35.5%), HDL less than 40 mg/dl in 10 patients (24%), triglyceride greater than or equals to 150 mg/dl in 8 patients (9.4%), IFG in 23 patients (37.1%), diabetes in 5 patients (8.07%) and dyslipidemia in 32 patients(51.62%). Their results show the metabolic syndrome and its elements frequently occur in women with PCOS, particularly those with the classic picture of the syndrome. The latter combination places them at risk for cardiovascular diseases, and screening for those disturbances is required in patients with PCOS.

MATERIALS AND METHODS

- **STUDY DESIGN:** Cross-sectional study
- **STUDY POPULATION:** Reproductive age women with PCOS attending Gynecology OPD
- **PLACE OF STUDY:** Tirunelveli Medical College and Hospital
- **PERIOD OF STUDY:** 18months
- **SAMPLE SIZE:** 120
- **INCLUSION CRITERIA:**

  Patients in the reproductive age group (18 – 45yrs) with the polycystic ovarian syndrome

  People selected according to **ROTTERDAM criteria: 2 out of 3 criteria needed**

- Hyperandrogenism - hirsutism
- Oligo-and/or anovulation – (menstrual irregularities, infertility)
- Polycystic ovaries – by ultrasound findings
- **EXCLUSION CRITERIA:**

  - Age > 45yrs
  - Hypothyroidism / Hyperthyroidism
  - Secondary causes of androgen excess

- Use of OCPill in the preceding 3months
- Chronic diseases like SLE, Diabetes mellitus, Hypertension, Cardiovascular disease

Data collection:

All the Reproductive-age women diagnosed with PCOS attending Gynaecology OPD were interviewed with a pre-tested semi-structured questionnaire, and a detailed history of the symptoms was collected. To assess the status of the metabolic syndrome, these women were measured with the following:

- Waist circumference > 88cm (measures central obesity)
- Blood pressure – (both Systolic and diastolic blood pressure)
- Fasting Lipid profile – (includes TGL and HDL)
- Fasting blood sugar – to screen for fasting hyperglycemia
- BMI – another parameter for central obesity

"National Cholesterol Education Programme Adult Treatment Panel III (NCEPATP III) guidelines defines Metabolic syndrome as having 3 or more of the following abnormalities:

- 1. waist circumference in females > 88 cm,
- 2. fasting serum glucose level at least 110mg/dl,

- 3. fasting serum triglycerides level at least 150mg/dl,
- 4. serum high-density lipoprotein cholesterol (HDL-C) < 50 mg/dl and
- 5. blood pressure at least 130/85mmHg”

The pattern and presentation of metabolic syndrome among PCOS assessed by the following parameters

BMI

Fasting lipid profile

Fasting blood sugars and blood pressure

INFORMED CONSENT:

Written informed consent will be obtained from all the study participants.

ETHICS COMMITTEE APPROVAL

was obtained from the Institutional Human Ethics Committee before starting the study.

ANALYSIS OF DATA:

All data were collected on a structural data form (sample enclosed) and analysed for descriptive statistics. Data were summarised in tables and figures. Appropriate statistical analysis will be done.

STATISTICAL METHODS:

Metabolic syndrome (study group) was considered as primary outcome variables. Age, menstrual syp, acanthosis, hirsutism, HLD, LDL, TGL, Cholesterol, fasting blood sugar, blood pressure was considered as explanatory variable.

Descriptive analysis was carried out by mean and standard deviation for quantitative variables, frequency and proportion for categorical variables. Data was also represented using appropriate diagrams like bar diagram, pie diagram.

Categorical outcome variable and explanatory variable assessed by using chi square test.

The association between categorical explanatory variables and quantitative outcome was assessed by comparing the mean values. Independent sample t-test was used to assess statistical significance.

P value < 0.05 was considered statistically significant.

RESULT

Total 120 participants included in to the final analysis.

Table 1: Descriptive analysis of age in study population (N=120)

Parameter	Mean ± SD	Median	Minimum	Maximum
Age	26.88 ± 4	26.00	18.00	38.00

Among the study population, the mean age was 26.88 ± 4 (18 to 38) **Table 1**

Table 2: Descriptive analysis of age group in the study population (N=120)

Age Group	Frequency	Percentages
18 - 25 years	48	40.00%
26 - 30 years	50	41.67%
31 - 35 years	22	18.33%

Among the study population, 40.00% of them age group were between 18 - 25 years, 41.67% of them age group were 26 - 30 years, 18.33% of them age group 31 - 35 years. **Table 2 & Figure 1**

Figure 1: Bar chart of age group in the study population (N=120)

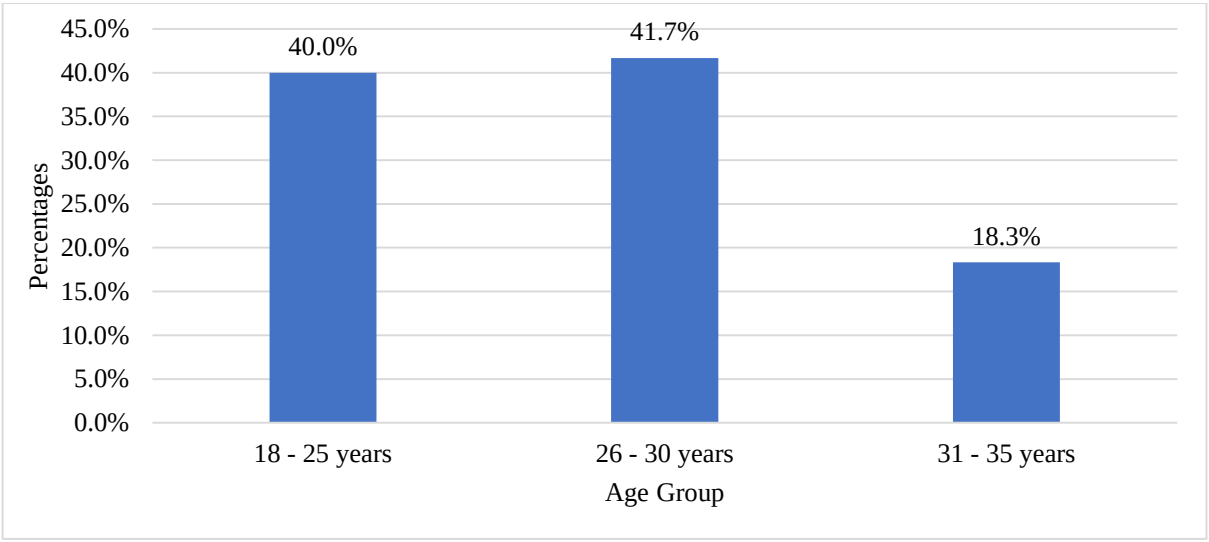

Table 3: Descriptive analysis of presentation in the study population (N=120)

Presentation	Frequency	Percentages
HIRSUTISM	2	1.67%
MEN IRR	60	50.00%
PR INF	48	40.00%
SEC INF	10	8.33%

Among the study population with presentation, 50.00% of them were MEN IRR, 40.00% of them were PR INF, 8.33% of them were SEC INF. **Table 3**

Table 4: Descriptive analysis of menstrual syp in the study population (N=120)

Menstrual Syp	Frequency	Percentages
Yes	101	84.17%
No	19	15.83%

Among the study population, 84.17% of them had menstrual syp. **Table 4 & Figure 2**

Figure 2: Pie chart of menstrual syp in the study population (N=120)

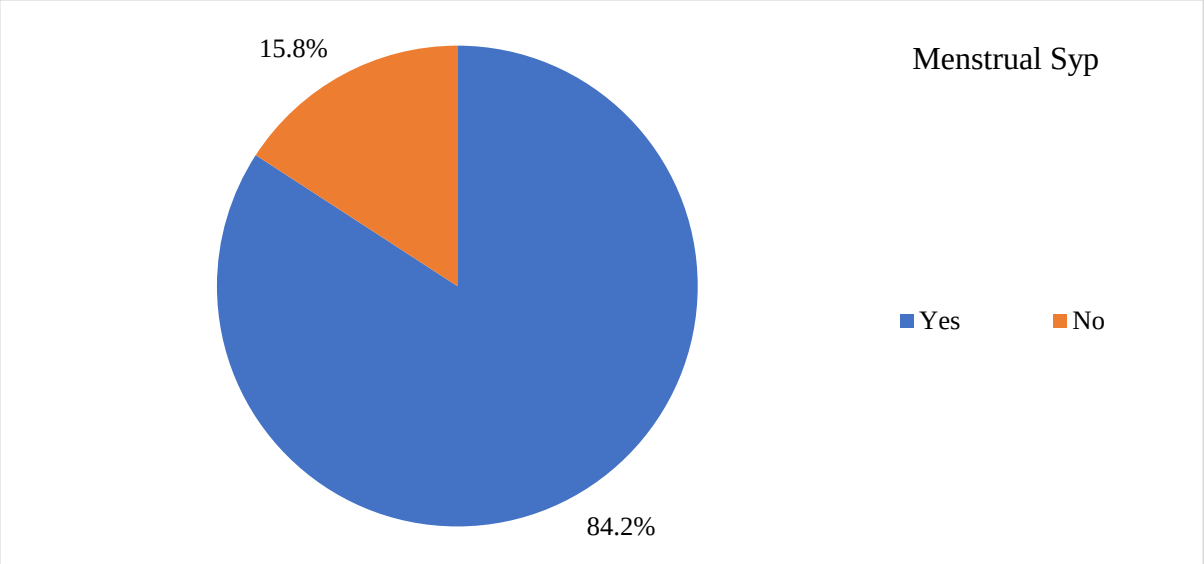

Table 5: Descriptive analysis of acanthosis in the study population (N=120)

Acanthosis	Frequency	Percentages
Yes	17	14.17%
No	103	85.83%

Among the study population, 14.17% of them had Acanthosis. **Table 5 & Figure 3**

Figure 3: Pie chart of acanthosis in the study population (N=120)

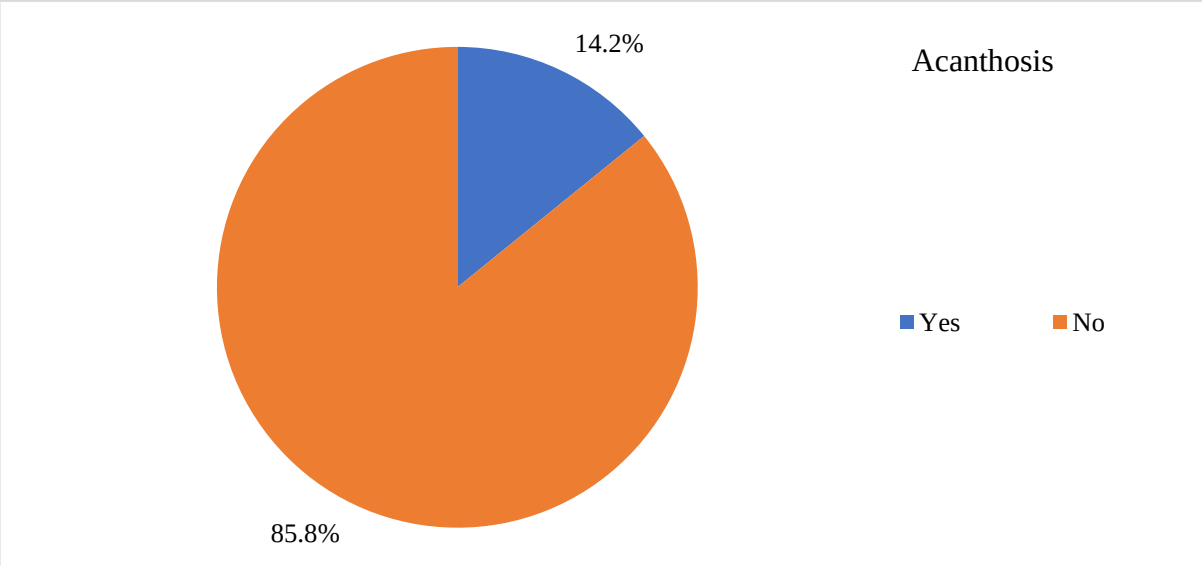

Table 6: Descriptive analysis of hirsutism in the study population (N=120)

Hirsutism	Frequency	Percentages
Yes	22	18.33%
No	98	81.67%

Among the study population, 18.33% of them had Hirsutism. **Table 6 & Figure 4**

Figure 4: Bar chart of hirsutism in the study population (N=120)

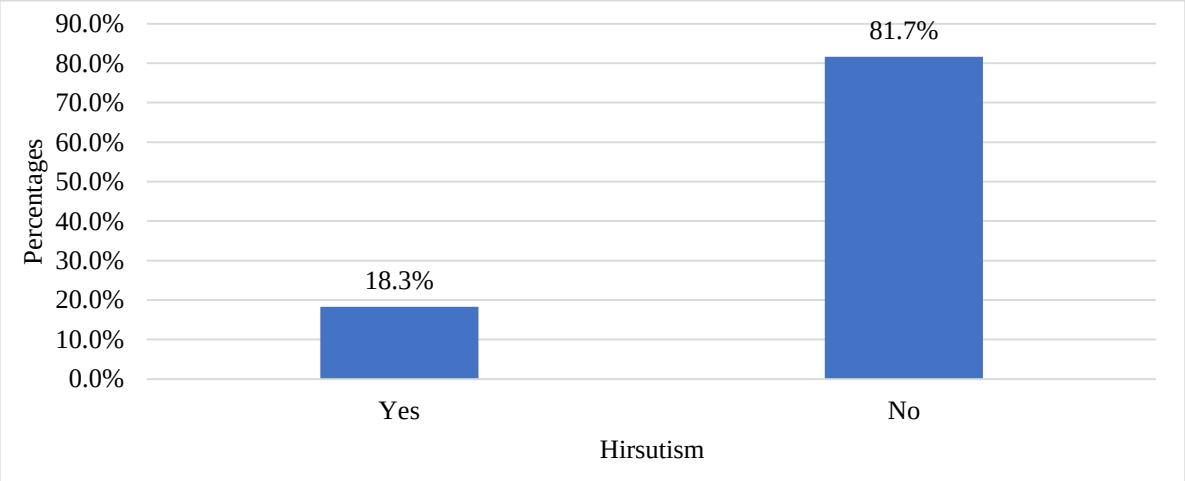

Table 7: Descriptive analysis of height, weight, bmi, waist circumference in study population (N=120)

Parameter	Mean ± SD	Median	Minimum	Maximum
Height	154.52 ± 3.47	155.00	143.00	164.00
Weight	64.41 ± 14.06	62.00	45.00	98.00
BMI	26.93 ± 5.72	25.56	20.00	40.83
Waist Circumference	87.69 ± 11.39	86.00	69.00	118.00

Among the study population, the mean height was 154.52 ± 3.47, the mean weight was 64.41 ± 14.06, the mean BMI was 26.93 ± 5.72 and the mean Waist Circumference was 87.69 ± 11.39. **Table 7**

Table 8: Descriptive analysis of s.tgl, s.hdl, s.ldl, t.chol, fbs in study population (N=120)

Parameter	Mean ± SD	Median	Minimum	Maximum
S.TGL	114.43 ± 20.45	110.00	67.00	165.00
S.HDL	51.45 ± 6.55	54.00	37.00	62.00
S.LDL	97.46 ± 16.85	90.00	70.00	163.00
Total Cholesterol	161.93 ± 19.68	156.00	104.00	229.00
FBS	93.97 ± 16.68	92.00	63.00	168.00

Among the study population, the mean S.TGL was 114.43 ± 20.45, the mean S.HDL was 51.45 ± 6.55, the mean S.LDL was 97.46 ± 16.85 and the mean Total Cholesterol was 161.93 ± 19.68, the mean FBS was 93.97 ± 16.68. **Table 8**

Table 9: Descriptive analysis of diastolic blood pressure, systolic blood pressure in study population (N=120)

Parameter	Mean ± SD	Median	Minimum	Maximum
Diastolic Blood Pressure	91.68 ± 20.6	80.00	60.00	140.00
Systolic Blood Pressure	108.3 ± 21.23	120.00	60.00	134.00

Among the study population, the mean Diastolic Blood Pressure was 91.68 ± 20.6, the mean Systolic Blood Pressure was 108.3 ± 21.23. **Table 9**

Table 10: Descriptive analysis of metabolic syndrome in the study population (N=120)

Metabolic Syndrome	Frequency	Percentages
Metabolic syndrome	45	37.50%
No metabolic syndrome	75	62.50%

Among the study population, 37.50% of them had Metabolic syndrome.

Table 10

Table 11: Comparison of age group between metabolic syndrome (N=120)

Age Gp	Metabolic Syndrome		P value
	Metabolic Syndrome	No Metabolic Syndrome	
18 - 25 Years (N=48)	12 (25%)	36 (75%)	0.002
26 - 30 Years (N=50)	18 (36%)	32 (64%)	
31 - 35 Years (N=22)	15 (68.18%)	7 (31.82%)	

Among the study population, those age group between 18 - 25 Years, 12 (25%) of them had metabolic syndrome, those age group between 26 - 30 Years, 18 (36%) of them had metabolic syndrome, those age group between 31 - 35 Years, 15 (68.18%) of them had metabolic syndrome. The difference in proportion of age group between study group was statistically significant. (p value 0.002) **Table 11 & Figure 5**

Figure 5: Cluster bar chart of comparison of age gp between metabolic Syndrome (N=120)

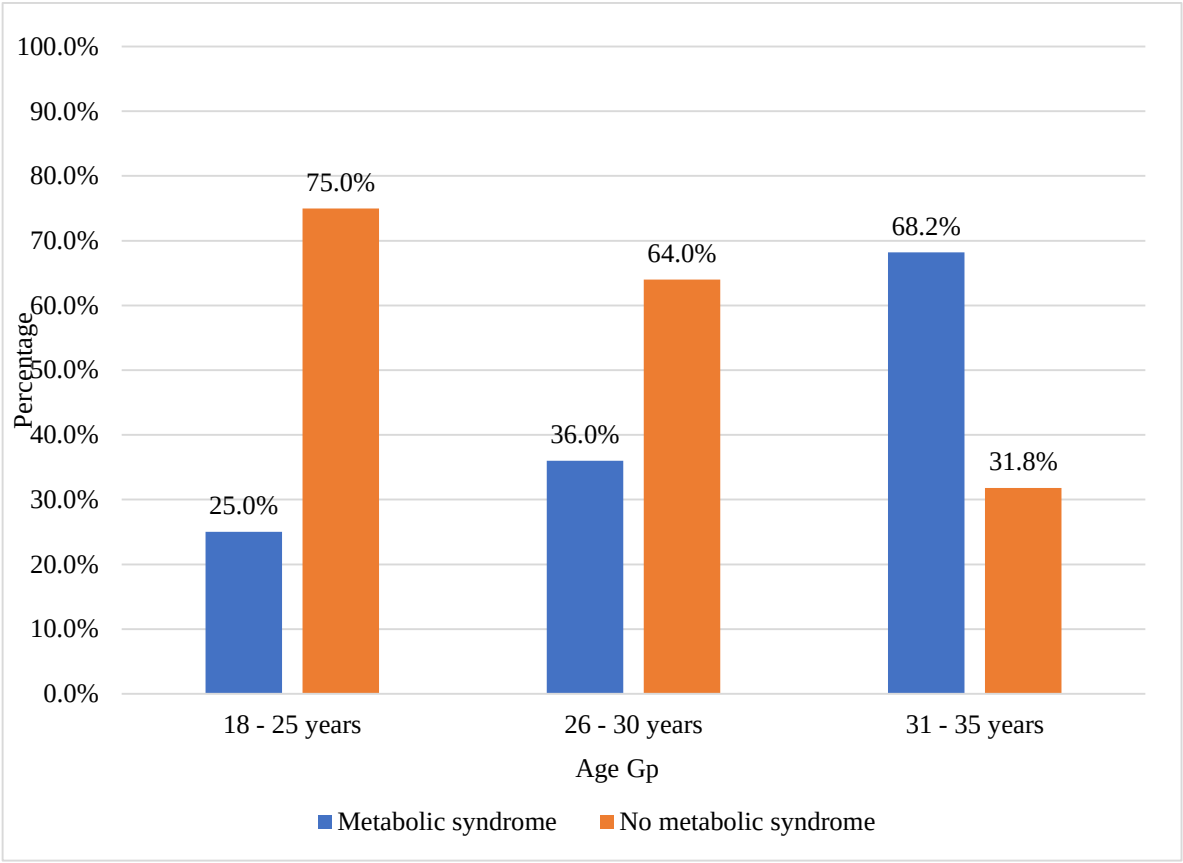

Table 12: Comparison of menstrual syp between metabolic Syndrome (N=120)

Menstrual Syp	Metabolic Syndrome		Chi square	P value
	Metabolic Syndrome	No Metabolic Syndrome		
Yes (N=101)	32 (31.68%)	69 (68.32%)	9.209	0.002
No (N=19)	13 (68.42%)	6 (31.58%)		

Among the study population, those who had menstrual syp, 32 (31.68%) of them had metabolic syndrome, those who did not had menstrual syp, 13 (68.42%)of them had metabolic syndrome. The difference in proportion of menstrual syp between study group was statistically significant. (p value 0.002) **Table 12 & Figure 6**

Figure 6: Cluster bar chart of comparison of menstrual syp between metabolic syndrome (N=120)

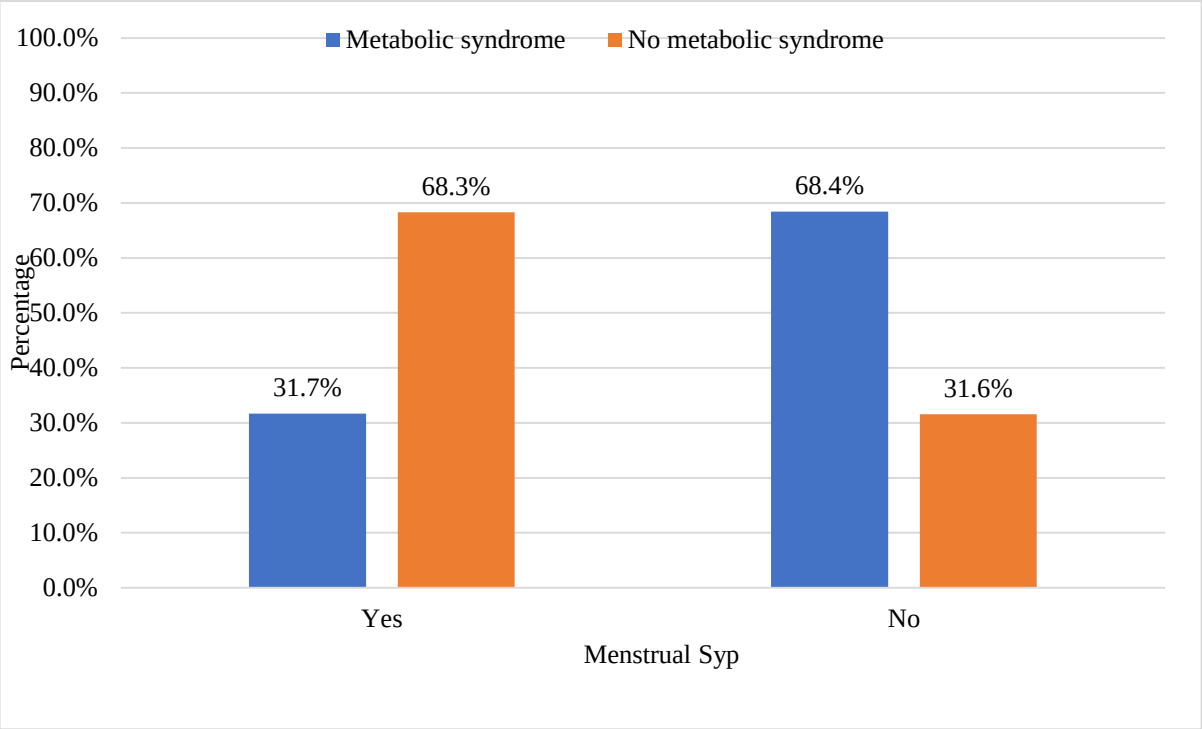

Table 13: Comparison of acanthosis between metabolic syndrome (N=120)

Acanthosis	Metabolic Syndrome		P value
	Metabolic Syndrome	No Metabolic Syndrome	
Yes (N=17)	14 (82.35%)	3 (17.65%)	<0.001
No (N=103)	31 (30.1%)	72 (69.9%)	

Among the study population, those who had acanthosis, 14 (82.35%) of them had metabolic syndrome, those who did not had acanthosis, 31 (30.1%) of them had metabolic syndrome. The difference in proportion of acanthosis between study group was statistically significant. (p value <0.001) **Table 13 & Figure 7**

Figure 7: Cluster bar chart of comparison of acanthosis between metabolic syndrome (N=120)

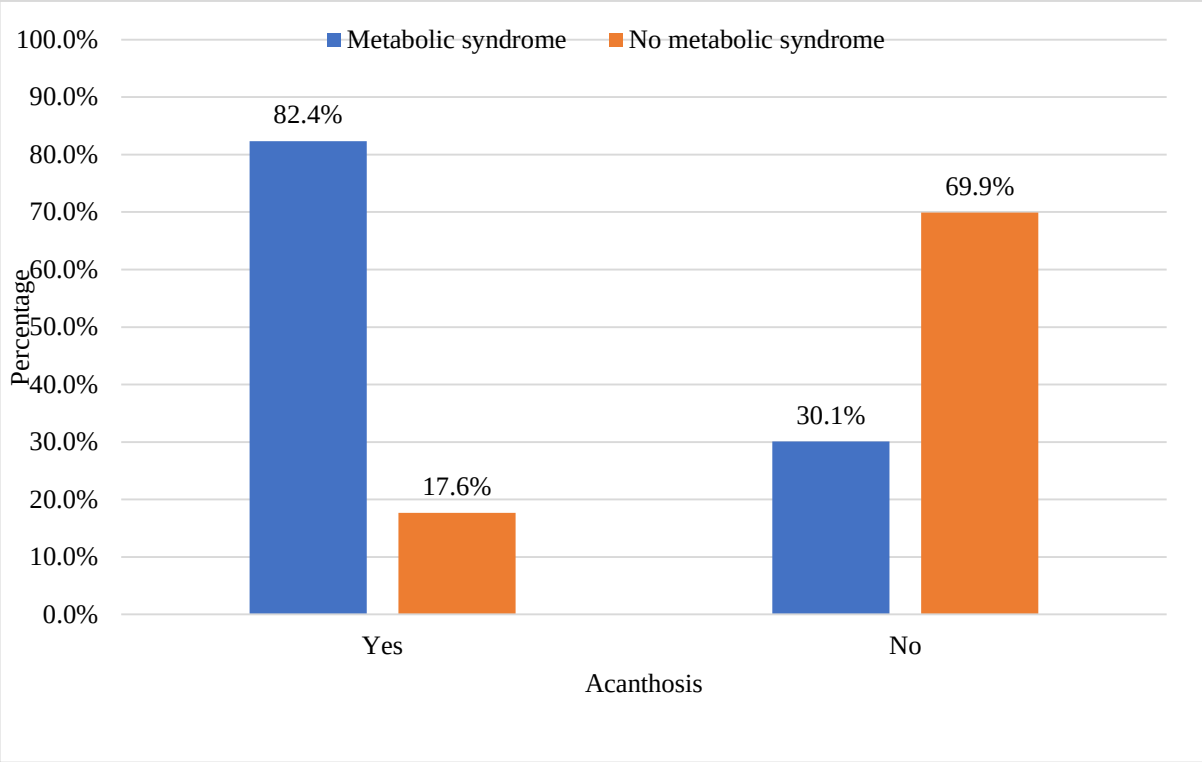

Table 14: Comparison of hirsutism between metabolic syndrome (N=120)

Hirsutism	Metabolic Syndrome		P value
	Metabolic Syndrome	No Metabolic Syndrome	
Yes (N=22)	13 (59.09%)	9 (40.91%)	0.021
No (N=98)	32 (32.65%)	66 (67.35%)	

Among the study population, those who had hirsutism, 13 (59.09%) of them had metabolic syndrome, those who did not had hirsutism, 32 (32.65%) of them had metabolic syndrome. The difference in proportion of hirsutism between study group was statistically significant. (p value <0.021) **Table 14 & Figure 8**

Figure 8: Cluster bar chart of comparison of hirsutism between metabolic syndrome (N=120)

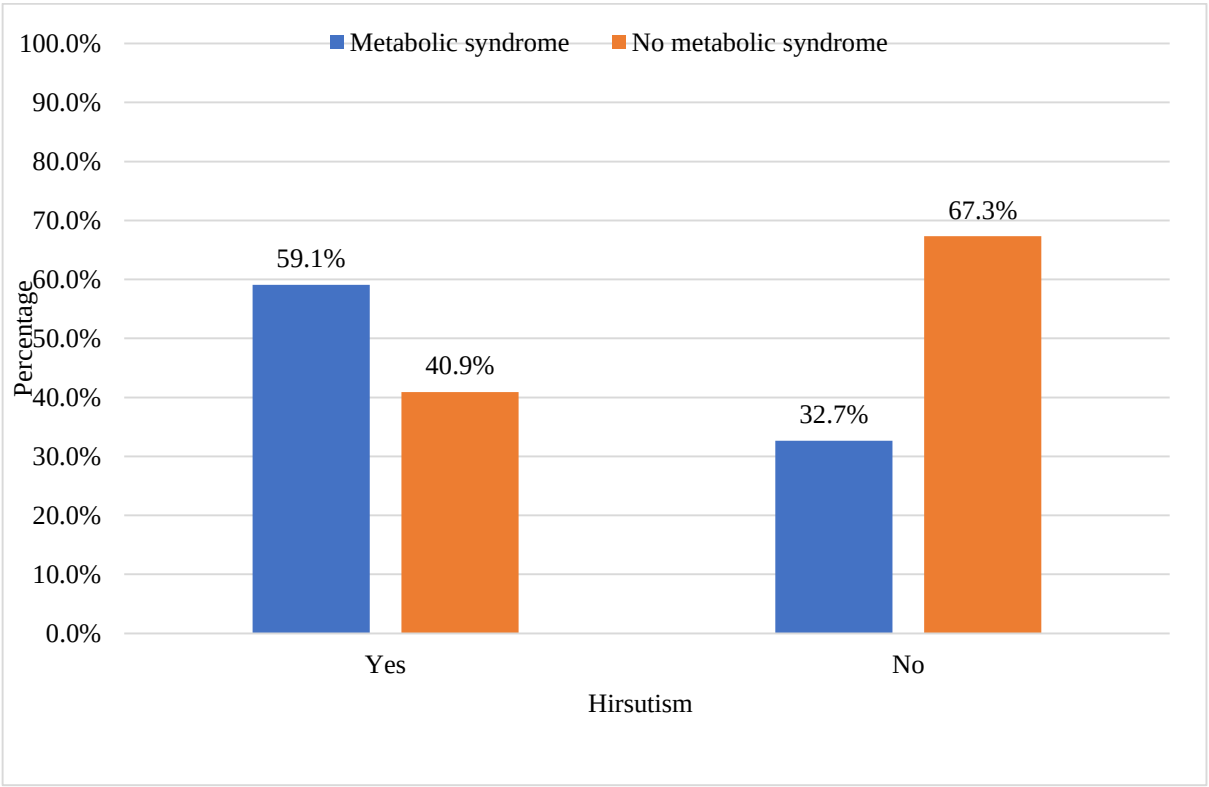

Table 15: Comparison of mean of anthropometric measurement between metabolic syndrome(N=120)

Parameter	METABOLIC SYNDROME (Mean± SD)		P value
	Metabolic syndrome (N=45)	No metabolic syndrome (N=75)	
Height	154.56 ± 3.19	154.49 ± 3.64	0.925
Weight	76.2 ± 13.46	57.33 ± 8.65	<0.001
BMI	31.96 ± 5.66	23.91 ± 2.98	<0.001
Waist circumference	97.02 ± 9.48	82.09 ± 8.4	<0.001

Among the study population, those who had metabolic syndrome, the mean height was 154.56 ± 3.19, the mean weight was 76.2 ± 13.46, the mean BMI was 31.96 ± 5.66, the mean waist circumference was 97.02 ± 9.48. The difference in mean of weight, height, weight circumference between study group was statistically significant. (p value <0.001). The difference in mean of height between study group was not statistically significant. (p value 0.925) **Table 15**

Table 16: Comparison of mean of parameter between metabolic syndrome(N=120)

Parameter	METABOLIC SYNDROME (Mean± SD)		P value
	Metabolic syndrome (N=45)	No metabolic syndrome (N=75)	
S.TGL	123.71 ± 26.12	108.87 ± 13.52	<0.001
S.HDL	45.84 ± 5.71	54.82 ± 4.35	<0.001
S.LDL	106.95 ± 20.37	91.77 ± 11.05	<0.001
Total Cholesterol	165.47 ± 27.52	159.81 ± 12.67	0.128

Among the study population, those who had metabolic syndrome, the mean S.TGL was 123.71 ± 26.12, the mean S.HDL was 45.84 ± 5.71, the mean S.LDL was 106.95 ± 20.37, the mean total cholesterol was 165.47 ± 27.52. The difference in mean of S.TGL, S.HDL, S.LDL between study group was statistically significant. (p value <0.001). The difference in mean of total cholesterol between study group was not statistically significant. (p value 0.128)**Table 16**

Table 17: Comparison of mean of blood glucose and blood pressure between metabolic syndrome(N=120)

Parameter	METABOLIC SYNDROME (Mean± SD)		P value
	Metabolic syndrome (N=45)	No metabolic syndrome (N=75)	
FBS	102.31 ± 18	88.96 ± 13.68	<0.001
Diastolic blood pressure	97.47 ± 20.79	88.21 ± 19.83	0.017
Systolic blood pressure	112.58 ± 19.77	105.73 ± 21.79	0.087

Among the study population, those who had metabolic syndrome, the mean FBS was 102.31 ± 18, the mean DBP was 97.47 ± 20.79, the mean SBP was 112.58 ± 19.77. The difference in mean of SBP between study group was not statistically significant. (p value 0.087). The difference in mean of FBS, DBP between study group was statistically significant. (p value <0.05)**Table 17**

DISCUSSION

Polycystic Ovarian Syndrome (PCOS) is a multifactorial, polygenic and multisystem endocrine disorder affecting women in reproductive age. Our study accessed the prevalence and pattern of metabolic syndrome components in women with polycystic ovarian syndrome.

Characteristics of our study participants:

The average age of the study population was 26.88 years. Among the study population, 40% were between the ages of 18 and 25 years, 41.67 percent were between the ages of 26 and 30 years, and 18.33 percent were between the ages of 31 and 35 years. In our study population with presentation, 50% were MEN IRR, 40% were PR INF, and 8.33% were SEC INF. Menstrual syp was experienced by 84.17% of the study population. Hirsutism was found in 18.33% of the participants.

The mean S.TGL was 114.43 ± 20.45, the mean S.HDL was 51.45 ± 6.55, the mean S.LDL was 97.46 ± 16.85, the mean Total Cholesterol was 161.93 ± 19.68, and the mean FBS was 93.97 ± 16.68 in the study population. The mean Diastolic Blood Pressure among our respondents was 91.68 ± 20.6, and the mean Systolic Blood Pressure was 108.3 ± 21.23.

Our Present study shows that 37.5% of women with metabolic syndrome had PCOS. This is closely related to the observations of 33.4% and 47.3% prevalence made by Ehrmann et al[83] and Dokras et al [84] respectively. Apridonidze et al [85] found a 43 percent prevalence rate in a study of 106 women with PCOS. They also demonstrated that the majority of PCOS women present clinically. Glueck et al [86] reported a 46% incidence of metabolic syndrome in a group of 138 women with confirmed PCOS. In our study 31 years and above age group has 68.2% metabolic syndrome. The age adjusted prevalence of MBS has shown that women in between 25-35 years have the highest prevalence (54%) of MBS.[87,88,89] Studies by Dey Ramprasad et al [90] also shows a high prevalence of 71.5% in the same age group.

In our study population, those who had metabolic syndrome, the mean FBS was 102.31 ± 18, the mean DBP was 97.47 ± 20.79, the mean SBP was 112.58 ± 19.77. The difference in mean of SBP between study group was not statistically significant. (p value 0.087). The difference in mean of FBS, DBP between study group was statistically significant (p value <0.05).

Indu et al [91] found that, with a significant p value of 0.04, there was an association between USG findings and PCOS. This suggests that USG can be a helpful modality in diagnosing PCOS. 31.6% had a high SBP of >

130mm of Hg while 37.3% had high DBP of > 85mm of Hg and there is a 100% significant association of high BP with PCOS. 87.8% of the cases had fasting level more than 110, which shows significant association between fasting blood sugar level with metabolic syndrome (p value = 0.001)

In our study population, those who had metabolic syndrome, the mean S.TGL was 123.71 ± 26.12, the mean S.HDL was 45.84 ± 5.71, the mean S.LDL was 106.95 ± 20.37, the mean total cholesterol was 165.47 ± 27.52. The difference in mean of S.TGL, S.HDL, S.LDL between study group was statistically significant. (p value <0.001). The difference in mean of total cholesterol between study group was not statistically significant. (p value 0.128). Indu et al [91] revealed that 84.8% of the cases had HDL<50 wherein 76.6% with HDL>50 in control, which shows significant association between HDL with metabolic syndrome (p = 0.0001). Bharatbha et al [92] have also found similar positive associations with a low HDL (<50 mg/dL) being seen in 91.7 % cases studied.

In our study, those who had metabolic syndrome, the mean height was 154.56 ± 3.19, the mean weight was 76.2 ± 13.46, the mean BMI was 31.96 ± 5.66, the mean waist circumference was 97.02 ± 9.48. The difference in mean of weight, height, weight circumference between study group was statistically significant. (p value <0.001). The difference in mean of height between study group was not statistically significant (p value 0.925). Similar study was found by Ehrmann et al [93] it was found that among their cohort of 394 women with PCOS, women in the highest quartile for BMI had a 14-fold increased chance of having the Metabolic Syndrome.

CONCLUSION

In the current study, the prevalence of metabolic syndrome was 37.5 percent, accounting for more than one-third of the women diagnosed with PCOS. This implies that it is mandatory to screen all the women with PCOS for features of metabolic syndrome. According to our findings, age above 30 years and the presence of central obesity (waist–hip ratio >0.85) were identified as risk factors for metabolic syndrome. There is a association between PCOS and high blood pressure, HDL levels, elevated FBS, TGL, and consistent USG findings. These findings can be used to develop a screening policy for metabolic syndrome, particularly in low-resource settings in developing countries.

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PROFORMA

1. NAME :
2. AGE :
3. IP NO :
4. UNIT :
5. ADDRESS :
6. OCCUPATION :
7. BOOKING STATUS :
8. REFERRED FROM :
9. PRESENTING COMPLAINTS:
    - Menstrual irregularities
    - Primary infertility
    - Secondary Infertility
    - Hirsutism
    - Weight Gain
10. EXAMINATION
    - Height
    - Weight
    - BMI

- Waist circumference
- Blood Pressure

11. LAB INVESTIGATIONS
    - Fasting Blood Sugar
    - Fasting Lipid Profile
12. Imaging
    - Ultrasound findings

நோயாளிகளுக்கு அறிவிப்பு மற்றும் ஒப்புதல் படிவம்
(மருத்துவ ஆய்வில் பங்கேற்பதற்கு)

ஆய்வு செய்யப்படும் தலைப்பு:
பங்கு பெறுவரின் பெயர்:
பங்கு பெறுவரின் வயது:

		பங்கு பெறுவர் இதனை ✓ குறிக்கவும்
1.	நான் மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்களை படித்து புரிந்து கொண்டேன். என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டுள்ளது என அறிந்து கொண்டேன்.	☐
2.	நான் இவ்வாய்வில் தன்னிச்சையாக தான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும், எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.	☐
3.	இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்து மேலும் ஆய்வு மேற்காள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன்.	☐
4.	இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக் கொள்ள மறுக்க மாட்டேன்.	☐
5.	இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன் எனக்கு கொடுக்கப்பட்டஅறிவுரைகளின் படி நடந்து கொள்வதுடன், ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ, அல்லது எதிர்பாராத, வழக்கத்திற்கு மாறான நோய்குறி தென்பட்டாலோ உடனே இதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதி அளிக்றேன்.	☐

பங்கேற்பவரின் கையொப்பம் / ..... இடம் .....
கட்டைவிரல் ரேகை
பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....
ஆய்வாளரின் கையொப்பம் /..... இடம் .....
ஆய்வாளரின் பெயர் .....
மையம் .....
கல்வியறிவு இல்லாதவற்கு (கைரேகை வைத்தவர்களுக்கு) இது அவசியம் தேவை
சாட்சியின் கையொப்பம் /..... இடம் .....
பெயர் மற்றும் விலாசம் ....

S NO	NAME	AGE	AGE GP	PRESENTATION	MENSTRUAL SYP	ACANTHOSIS	HIIRSUTISM	HT	WT	BMI	WC	S.TGL	S.HDL	S.LDL	T.CHOL	FBS	BP	BP	METABOLIC SYND
1	SATHYA	18	1	MEN IRR	YES	NO	NO	157	93	38.75	102	67	43.4	134	176	63	90	130	1
2	SUDHA	19	1	MEN IRR	YES	NO	NO	156	92	37.86	101	156	42	108	188	88	90	128	1
3	RANI	19	1	HIRSUTISM	YES	NO	YES	156	90	36.98	102	143	40.1	163	229	88	130	90	1
4	JEYA	19	1	MEN IRR	YES	NO	YES	156	92	37.86	102	153	40.1	135	217	88	130	90	1
5	VIJI	20	1	MEN IRR	YES	NO	NO	156	70	28.8	94	126	47	116	167	75	128	76	0
6	VINO	21	1	MEN IRR	YES	NO	YES	156	84	35.89	99	156	38	120	201	90	90	130	1
7	KANMANI	21	1	MEN IRR	YES	NO	NO	151	75	26.8	92	88	44	97	172	120	80	120	1
8	BANU	21	1	PR INF	NO	YES	NO	150	74	32.8	93	97	40.6	99.2	150	97	90	128	1
9	YAZHINI	21	1	MEN IRR	YES	NO	NO	157	62	25.51	79	136	58	107	177	92	128	88	0
10	VANAJA	21	1	MEN IRR	YES	NO	NO	155	64	28.44	89	157	50	102	146	107	110	70	1
11	SEETHA	22	1	PR INF	YES	NO	NO	154	56	23.62	79	135	56	86	143	76	80	116	0
12	KARTHIGA	22	1	MEN IRR	YES	NO	NO	150	45	20	79	110	54	84	156	87	60	100	0
13	VIJAYA	23	1	MEN IRR	YES	NO	NO	150	47	20.88	78	95.5	54	85	154	80	78	126	0
14	MARI	23	1	MEN IRR	YES	NO	NO	150	58	25.77	78	114	48	103	154	80	78	126	0
15	MUTHULAKSHMI	23	1	PR INF	YES	NO	NO	159	56	22.48	77	98.6	58	92	158	71	72	110	0
16	MURUGAMMAL	23	1	PR INF	YES	NO	NO	157	56	22.76	77	99	57	87	158	69	72	110	0
17	SANDHYA	23	1	MEN IRR	YES	NO	NO	150	58	25.77	82	108	48	105	154	80	90	126	1
18	VENU	23	1	PR INF	YES	NO	NO	157	56	22.76	77	99	53	87	158	69	76	110	0
19	RANJANI	23	1	PR INF	YES	NO	NO	159	56	22.48	77	98.6	55	92	158	71	70	110	0
20	SANKARI	23	1	MEN IRR	YES	NO	NO	150	47	20.88	78	93	58	85	154	80	78	126	0
21	SIVANGI	23	1	PR INF	YES	NO	NO	159	56	22.48	77	98.6	57	92	158	71	72	110	0
22	SRIDEVI	23	1	MEN IRR	YES	NO	NO	150	47	20.88	78	93	58	85	154	80	126	78	0
23	ANU	23	1	MEN IRR	YES	NO	NO	150	50	22.2	78	112	54	86	154	74	110	76	0
24	PARVATHY	23	1	MEN IRR	YES	NO	NO	150	58	25.77	80	114	48	108	154	80	126	78	0
25	SELVI	23	1	PR INF	YES	NO	NO	157	56	22.76	77	99	58	87	158	69	110	72	0
26	VENI	23	1	PR INF	YES	NO	NO	159	56	22.48	77	98.6	55	92	158	71	110	72	0
27	SHANTHI	24	1	PR INF	YES	NO	NO	157	56	22.76	79	99	58	87	158	69	72	110	0
28	SARANYA	24	1	PR INF	YES	NO	YES	155	98	40.83	98	144	44	156	216	88	85	120	1
29	SATHYA	25	1	MEN IRR	YES	NO	NO	150	45	20	79	110	54	84	156	87	60	100	0
30	MUTHULAKSHMI	25	1	MEN IRR	YES	NO	NO	157	62	25.51	84	136	54.5	104	177	92	88	128	0
31	SUBAMMAL	25	1	MEN IRR	YES	NO	NO	143	50	23.47	73	95	58	84	156	87	60	100	0
32	SUBHA	25	1	MEN IRR	YES	NO	NO	159	60	23.8	78	98.6	57	90	155	92	88	128	0
33	VAIDEGI	25	1	MEN IRR	YES	NO	NO	148	50	22.9	69	95	57	84	156	87	70	100	0
34	PONNUTHAI	25	1	MEN IRR	YES	NO	NO	156	62	25.51	78	100.4	57	89.5	176	92	88	124	0
35	VADIVU	25	1	MEN IRR	YES	NO	NO	156	62	25.51	78	102.4	56	97	177	93	70	120	0
36	LAKSHI	25	1	MEN IRR	YES	NO	NO	157	62	25.51	79	136	57	110	177	92	80	120	0
37	PRABHA	25	1	MEN IRR	YES	NO	NO	148	50	22.9	87	95	59	84	156	87	60	100	0
38	AJITHA	25	1	MEN IRR	YES	NO	NO	157	62	25.51	79	136	54.5	87	177	92	88	128	0
39	VIJAYALAKSHMI	25	1	MEN IRR	YES	NO	NO	150	45	20	79	110	51	84	156	87	60	100	0
40	BRAMALAKSHMI	25	1	MEN IRR	YES	NO	NO	157	62	25.51	79	136	52	87	177	92	80	120	0
41	RUKMINI	25	1	PR INF	YES	NO	NO	157	90	36.43	104	116	43.2	134	197	105	80	120	1
42	DEEPA	25	1	PR INF	YES	NO	NO	157	56	22.76	79	99	58	95	158	74	110	72	0
43	DIVYA	25	1	PR INF	YES	NO	YES	155	92	38.33	110	116	39.6	138	188	105	120	78	0
44	GAYATHRI	25	1	MEN IRR	YES	NO	NO	148	50	22.9	69	95	54	84	156	87	100	60	0
45	VANDHANA	25	1	MEN IRR	YES	NO	YES	157	62	25.51	79	136	58	99.5	177	92	128	88	0
46	VASUNDARA	25	1	MEN IRR	YES	NO	NO	150	45	20	79	110	55	84	156	87	100	60	0
47	TAMILARASI	25	1	MEN IRR	YES	NO	NO	150	45	20	79	110	54	84	156	87	102	60	0
48	AMUDHA	26	2	PR INF	YES	NO	NO	156	62	25.51	106	118	44	133	187	73	120	90	1
49	BHUVANA	26	2	MEN IRR	YES	NO	NO	160	64	25	88	107	55	70	146	97	70	110	0
50	KAVITHA	26	2	MEN IRR	YES	NO	NO	150	47	20.88	76	98	58	86	154	74	76	110	0
51	VENKATESHWARI	26	2	PR INF	YES	NO	NO	157	68	26.56	94	118	50	110	142	84	80	126	0
52	ANTONYAMMAL	26	2	PR INF	YES	NO	NO	157	68	26.56	90	107	43	113	144	102	80	126	1
53	JENIFFER	26	2	MEN IRR	YES	NO	NO	150	50	22.2	82	94	62	86	154	74	76	110	0
54	MAHALAKSHMI	26	2	MEN IRR	YES	NO	NO	150	50	22.2	78	96	54	86	154	74	76	110	0
55	MAHESHWARI	26	2	MEN IRR	YES	NO	NO	156	67	27.57	87	110	59	137	142	90	80	126	0
56	ESAKKIYAMMAL	26	2	PR INF	YES	NO	NO	157	68	26.56	87	118	55	90	142	84	80	126	0
57	PETCHIYAMMAL	26	2	PR INF	YES	NO	NO	157	68	26.56	87	118	57	90	142	102	80	126	0

S NO	NAME	AGE	AGE GP	PRESENTATION	MENSTRUAL SYP	ACANTHOSIS	HIIRSUTISM	HT	WT	BMI	WC	S.TGL	S.HDL	S.LDL	T.CHOL	FBS	BP	BP	METABOLIC SYND
58	SYED ALI FATHIMA	26	2	PR INF	YES	NO	NO	156	64	26.33	86	133	51	88	156	96	78	118	0
59	STELLA	26	2	MEN IRR	YES	NO	NO	155	64	28.44	110	152	48	91	146	107	74	120	1
60	AMALA	26	2	MEN IRR	YES	NO	YES	155	64	28.44	103	157	45.4	82	146	107	70	110	1
61	SUGUMARI	26	2	PR INF	YES	NO	NO	157	68	26.56	97	118	57	90	142	102	126	80	0
62	PRIYANKA	26	2	PR INF	YES	NO	NO	156	64	26.33	96	103	51	88	156	98	118	78	0
63	VIJITHA	27	2	MEN IRR	YES	NO	NO	156	70	28.8	104	120	37	116	164	89	76	128	0
64	VINOTHA	27	2	PR INF	YES	NO	NO	154	56	23.62	75	94	57	88	162	78	80	128	0
65	SUGANYA	27	2	MEN IRR	YES	NO	NO	156	63	25.92	86	126	52	97	167	75	76	128	0
66	VISHNUPRIYA	27	2	MEN IRR	YES	NO	NO	156	70	28.8	93	87	41	97	167	75	76	128	0
67	JEYALAKSHMI	27	2	MEN IRR	YES	NO	NO	155	65	27	94	126	54	97	167	75	76	128	0
68	SUJITHA	27	2	SEC INF	NO	YES	NO	151	85	37.28	101	97	41.4	99.2	150	83	90	130	1
69	SATHYA	27	2	SEC INF	NO	YES	NO	150	86	38.22	103	97	40.6	97	154	83	90	130	1
70	FATHIMA	27	2	SEC INF	NO	NO	NO	151	85	37.28	100	86	43.6	102	148	83	90	126	1
71	IRFANA	27	2	SEC INF	NO	YES	YES	151	85	37.28	103	97	42.4	112	143	145	130	90	1
72	SANTHANAMARI	27	2	SEC INF	NO	NO	NO	150	86	38.22	104	97	40.6	99.2	167	98	120	86	1
73	SUNDARI	28	2	PR INF	NO	NO	NO	164	70	26.11	90	122	56	88	158	88	86	126	0
74	MANJULA	28	2	PR INF	YES	NO	YES	157	63	25.6	91	103	58	93	144	102	80	126	0
75	PRIYA	28	2	PR INF	YES	NO	YES	158	62	24.89	86	104	58	93	144	102	80	126	0
76	BACKIYA	28	2	PR INF	YES	NO	YES	157	63	25.6	106	103	53	93	144	88	80	126	0
77	SWATHY	28	2	PR INF	YES	YES	NO	158	85	34.13	99	100.2	51	128	188	168	85	130	1
78	NIVETHA	28	2	PR INF	NO	NO	NO	164	78	29.1	115	158	57	88	158	88	86	126	1
79	SAI KEERTHI	28	2	PR INF	NO	NO	NO	164	78	29.1	94	157	44.2	88	158	88	126	86	1
80	VIGNESHWARI	28	2	PR INF	YES	NO	YES	157	63	25.6	95	103	48	93	144	102	126	80	1
81	REENA	29	2	MEN IRR	YES	NO	NO	155	50	20.8	87	113	56	89	185	115	80	120	0
82	SAMYUKTHA	29	2	MEN IRR	YES	NO	NO	153	50	21.36	70	89	58	89	178	115	80	120	0
83	CHANDRA	29	2	MEN IRR	YES	NO	NO	151	50	21.92	70	93	54.2	89	178	115	80	120	0
84	SIVA RANJANI	29	2	PR INF	YES	NO	YES	157	68	26.56	87	110	59	90	142	92	80	126	0
85	SIVABANU	29	2	MEN IRR	YES	NO	NO	155	50	20.8	78	113	59	89	185	115	80	120	0
86	HAMEETHA BANU	29	2	MEN IRR	YES	YES	NO	155	50	20.8	78	113	57	89	185	115	80	120	0
87	RANJITHA	29	2	SEC INF	YES	NO	NO	154	58	24.47	88	106	44.6	98	167	107	78	106	1
88	ESAKKIYAMMAL	29	2	MEN IRR	YES	NO	NO	153	67	28.63	92	133	43.2	99	178	115	80	120	1
89	RANI	29	2	MEN IRR	YES	YES	NO	153	50	21.36	70	89	56	89	178	115	120	80	0
90	ANITHA	29	2	MEN IRR	YES	NO	NO	155	50	20.8	78	113	57	89	185	115	120	80	0
91	ANANTHI	30	2	SEC INF	NO	YES	YES	155	50	20.8	79	92	52	74	167	108	78	120	0
92	PREMA	30	2	SEC INF	NO	NO	NO	155	50	20.8	79	110	51	88	167	108	78	120	0
93	VIJILA	30	2	SEC INF	NO	NO	NO	155	50	20.8	79	110	58.5	88	167	108	78	120	0
94	PONESAKKI	30	2	PR INF	NO	YES	NO	152	62	26.83	86	123	58	83	148	108	90	130	1
95	VIMALA	30	2	PR INF	NO	NO	NO	152	62	26.83	87	118	54	83	148	108	90	130	1
96	ESWARI	30	2	SEC INF	NO	NO	NO	155	50	20.8	79	110	59	88	167	108	120	78	0
97	PANDIYAMMAL	30	2	PR INF	NO	YES	NO	152	62	26.83	89	123	48	83	148	108	130	90	1
98	SUBBUTHAI	31	3	PR INF	YES	YES	YES	155	98	40.83	118	158	39	112	189	104	130	90	1
99	VELLAIYAMMAL	31	3	PR INF	YES	YES	YES	155	98	40.83	118	158	39	112	189	128	140	80	1
100	DEVI	32	3	MEN IRR	YES	NO	NO	154	52	21.94	78	123	56	90	146	78	78	120	0
101	PRIYA	32	3	MEN IRR	YES	NO	NO	154	55	23.2	80	123	57	90	146	78	78	120	0
102	PETCHIYAMMAL	32	3	PR INF	YES	NO	YES	156	64	26.33	96	103	51	88	156	98	118	78	0
103	VALLIYAMMAL	32	3	PR INF	YES	NO	NO	156	80	34.18	96	158	45	102	188	89	80	120	1
104	MUTHUESAKKI	32	3	MEN IRR	YES	NO	NO	154	55	23.2	80	123	57	90	146	78	120	78	0
105	SANTHA	32	3	PR INF	YES	NO	YES	156	80	34.18	96	165	45	102	188	89	120	80	1
106	GANDHIMATHI	33	3	MEN IRR	YES	YES	NO	156	62	25.5	84	117	54	87	138	107	96	134	1
107	LALITHA	33	3	PR INF	YES	YES	NO	154	56	23.62	85	109	60.5	86	143	112	90	134	1
108	BAVANI	33	3	PR INF	YES	NO	YES	154	56	23.62	81	102	52	86	143	112	80	134	1
109	SELVI	33	3	PR INF	YES	NO	NO	154	56	23.62	79	102	57	86	143	112	90	134	1
110	BARATHI	33	3	PR INF	YES	YES	NO	154	56	23.62	79	102	57.5	86	143	112	134	90	1
111	NALINI	34	3	MEN IRR	YES	NO	NO	155	68	27.2	94	99.8	44.8	87	145	109	80	120	1
112	RAMA	34	3	MEN IRR	YES	NO	NO	155	68	27.2	94	99.8	54	87	145	109	120	80	0
113	MALAR	34	3	PR INF	NO	NO	YES	157	68	26.56	87	110	59	90	142	92	80	126	0
114	NOORNISHA	34	3	PR INF	NO	YES	YES	150	82	36.4	98	142	43.2	122	104	118	126	80	1

S NO	NAME	AGE	AGE GP	PRESENTATION	MENSTRUAL SYP	ACANTHOSIS	HIIRSUTISM	HT	WT	BMI	WC	S.TGL	S.HDL	S.LDL	T.CHOL	FBS	BP	BP	METABOLIC SYND
115	MARY	35	3	HIRSUTISM	YES	NO	NO	154	75	31.64	99	156	41	102	188	96	80	128	1
116	MEKALA	38	3	PR INF	NO	YES	YES	150	82	36.4	98	142	44	122	104	118	80	126	1
117	DEVIKA	34	3	MEN IRR	YES	NO	NO	148	50	22.9	69	95	54	84	156	87	100	60	0
118	EZHIL	23	1	PR INF	YES	NO	NO	157	90	36.43	104	116	43.2	134	197	105	80	120	1
119	BOMMI	33	3	MEN IRR	YES	NO	NO	157	90	36.43	104	112	43	132	186	102	79	118	1
120	SASIKALA	35	3	MEN IRR	YES	NO	NO	157	90	36.43	104	114	44	130	190	104	81	120	1